ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

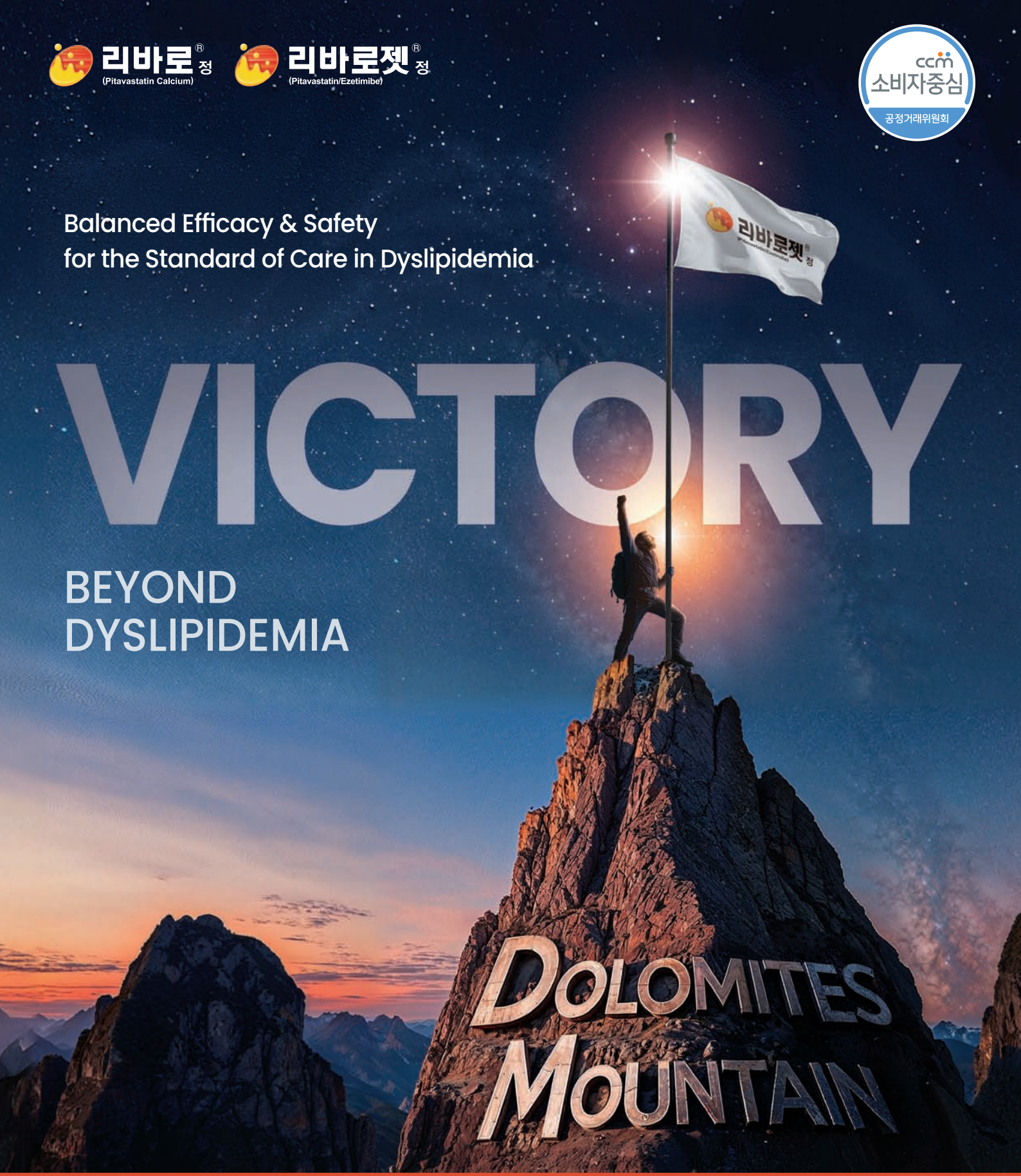
"Bridge to Excellence, Toward Cardiometabolic Health"

ABSTRACT BOOK

September
10 (Thu)-12 (Sat), 2026
Conrad Seoul, Republic of Korea

ICoLA | by KSoLA International Congress on Lipid & Atherosclerosis

KSoLA The Korean Society of Lipid and Atherosclerosis



[효능·효과] 원발성 고콜레스테롤혈증 원발성 고콜레스테롤혈증(이형접합 가족형 및 비가족형) 또는 혼합형 이상지질혈증 환자의 상승된 총 콜레스테롤(total-C), LDL-콜레스테롤(LDL-C), 아포 B 단백(Apo B), 트리글리세라이드(TG) 및 non-HDL-콜레스테롤을 감소시키고, HDL-콜레스테롤(HDL-C)을 증가시키기 위한 식이요법의 보조제로서 이 약을 투여한다. **[용법·용량]** 성인: 이 약은 식사와 관계없이 1 일 1 회 투여한다. 이 약을 투여하기 전 또는 투여 중인 환자는 반드시 표준 콜레스테롤 저하식을 지속적으로 해야 한다. 이 약의 투여량은 환자의 LDL-콜레스테롤의 기저치, 권장되는 치료목표치 및 환자의 반응에 따라 조절되어야 한다. 이 약은 초회용량으로 1 일 1 회 2/10mg 이 권장된다. LDL-콜레스테롤 수치의 저하효과가 충분하지 않은 경우 1 일 최대 4/10mg 까지 증량할 수 있다. LDL-콜레스테롤 수치, 치료 목표 및 환자의 반응에 따라 4주 또는 그 이상의 간격을 두고 용량을 적절히 조절한다. 피타바스타틴칼슘과 에제티미브를 병용으로 복용하고 있는 환자인 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있다. 간장애 환자에 대한 투여 : 간장애 환자의 경우, 피타바스타틴칼슘의 초회용량으로 1 일 1mg 이 권장되므로, 이 약(피타바스타틴칼슘/에제티미브 복합제)을 초기 치료 시 투여하는 것은 권장되지 않는다. 최대 투여량은 1 일 2/10mg 으로 한다. 중등증 간장애 환자의 경우 이 약(피타바스타틴칼슘/에제티미브 복합제) 투여가 권장되지 않는다. 중증 간장애 환자의 경우 이 약(피타바스타틴칼슘/에제티미브 복합제)을 투여하지 않는다. **[사용상주의사항]** 투여 금기 환자: 이 약의 구성성분에 과민증이 있거나, 그 병력이 있는 환자/활동성 간질환 환자 또는 원인이 밝혀지지 않은 아미노전이효소수치의 지속적 상승이 있는 환자/중증의 간장애 또는 담도폐쇄가 있는 환자 및 담즙울체 환자/사이클로스포린을 투여중인 환자/근육병증 환자/임부 또는 임신의 가능성이 있는 부인 및 수유부/소아(사용경험이 없다.)/이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption)등의 유전적인 문제가 있는 환자에게는 투여하면 안된다. 최종허가변경일 2025.02.01. 제품에 대한 자세한 정보는 식품의약품안전처 의약품안전나라(http://nedrug.mfds.go.kr)를 참고하시기 바랍니다.

Product Information
리바로®정
피타바스타틴칼슘수화물

Product Information
리바로젯®정
피타바스타틴칼슘수화물/에제티미브

JW 중외제약 JW 신약

경기도 과천시 과천대로 7길 38 (갈현동) JW과천사옥, CCM 운영팀: 1588-2675
제품에 대한 상세한 내용은 제품 설명서나 본사 홈페이지를 참조하시기 바랍니다. www.jw-pharma.co.kr

JWP380-2510001(EXP.2710)

An Excellent Solution for the Integrated Management of Dyslipidemia and Hypertension

Rosuzet Tab.
(Ezetimibe/Rosuvastatin)

- **The world's first CV outcome publication** of on the combination of Rosuvastatin and Ezetimibe[1)]
- **The first prescription drug developed by a domestic company** which took the **No.1 position in Korea's outpatient prescriptions market**[2)]
- **Eighteen clinical studies** on Korean patients with Rosuzet published in **SCIE-indexed journals** (As of Oct.2025)
- Global expansion through **R&D** and **in-house manufacturing in Hanmi Pharmaceutical.**

10 / 2.5 mg

10 / 5 mg

10 / 10 mg

10 / 20 mg

For personalized management of dyslipidemia in patients with multiple comorbidities, start with Rosuzet!

Active Form of Fenofibric Acid for Hypertriglyceridemia	Omega-3 Fatty Acid-Based Therapy for Hypertriglyceridemia	Lipid-Lowering Therapy Compatible with Statins
Fenocid Cap. (Fenofibric acid)	HANMI Omega Soft Cap. (Omega-3-acid ethyl esters 90)	Ezet Tab. (Ezetimibe)

Amoprel Tab.
(Amlodipine / Losartan / Chlorthalidone)

(1.67/16.67/4.17mg)

- **The world's first third-dose triple-combination antihypertensive medication**
- **Expected to provide stable blood pressure control** through **three complementary antihypertensive mechanisms**[3,4,5)]
- Demonstrated non-inferior blood pressure-lowering efficacy compared to Amlodipine 5 mg monotherapy[6)]
- **Demonstrated superior BP-lowering efficacy** compared to **Losartan 50mg monotherapy**[7)]
- **Low-dose combination therapy is expected to minimize dose-dependent adverse reactions**[8)]

For essential hypertension with various pathological causes, start with Amoprel!

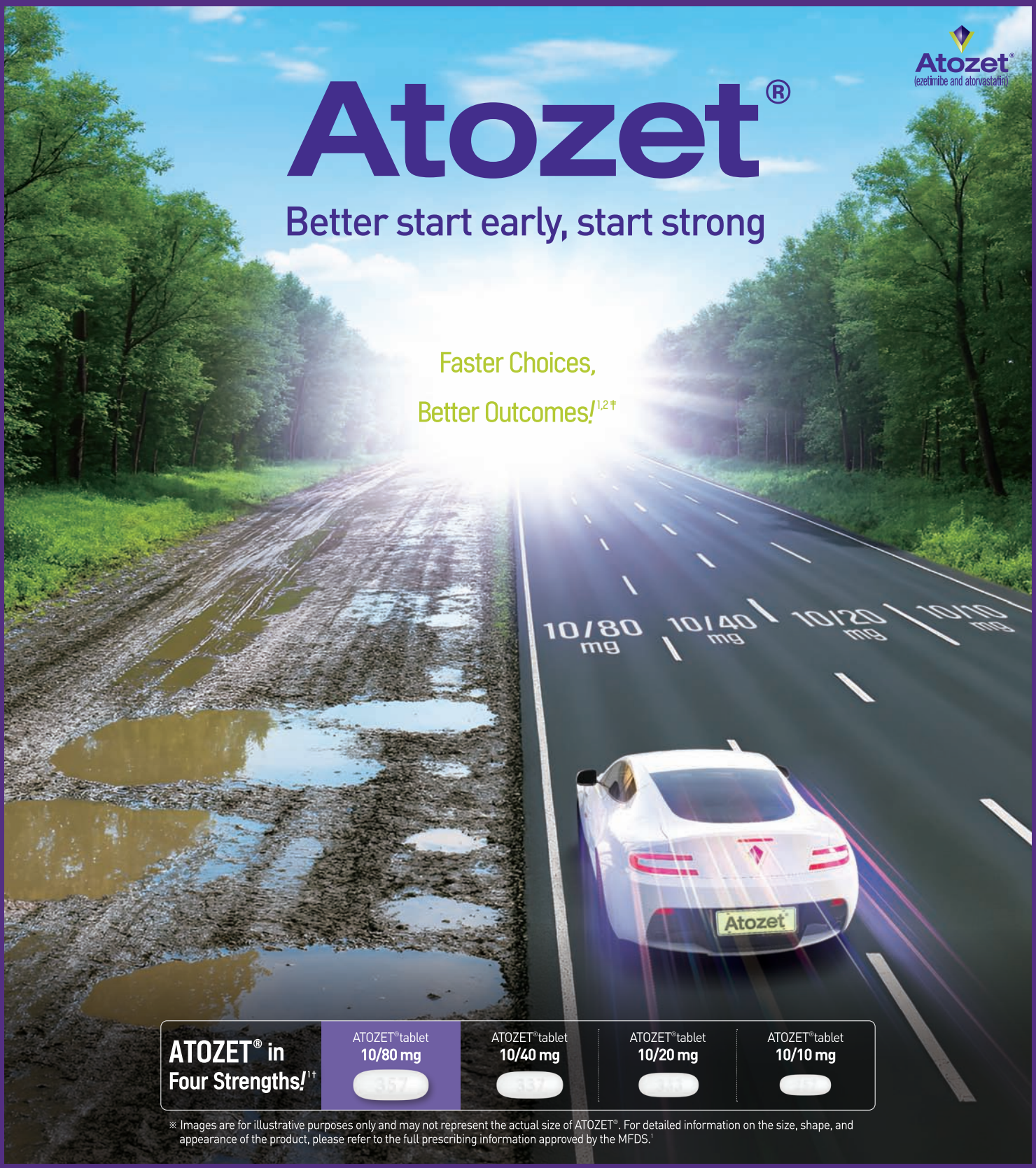
The World's First Combination of Amlodipine Camsylate + Losartan Potassium	The World's First Triple Combination of CCB/ARB/Chlorthalidone	The World's First Triple Combination of CCB/ARB/Rosuvastatin	The World's First Quadruple Combination of CCB/ARB/Rosuvastatin/Ezetimibe
Amosartan Tab. (Amlodipine camsylate/Losartan potassium)	Amosartan Plus Tab. (Amlodipine/Losartan/Chlorthalidone)	Amosartan Q Tab. (Amlodipine/Losartan/Rosuvastatin)	Amosartan XQ Tab. (Amlodipine/Losartan/Rosuvastatin/Ezetimibe)

CV, cardiovascular; **SCIE,** science citation index expanded; **R&D,** research and development; **CCB,** calcium channel blocker; **ARB,** angiotensin II receptor blocker; **BP,** blood pressure

Ref. 1) Kim BK, et a. *Lancet.* 2022 Jul;400(10349):380-390. **2)** UBIST D1 Sales data. (Based on the 2024 outpatient prescription expenditure.) **3)** McEvoy JW, et al. *Eur Heart J.* 2024 Oct;45(38):3912-4018. **4)** Waeber B, et al. Antihypertensive agents: mechanisms of drug action. Atlas of Heart Diseases. 2001, pp. 144-161. **5)** Mancia Chairperson G, et al. *J Hypertens.* 2023 Dec;41(12):1874-2071. **6)** Data on file. [Protocol No. HM-APOLLO-301]. CSR v1.0. 2024. **7)** Data on file. [Protocol No. HM-APOLLO-302]. CSR v1.0. 2024. **8)** Law MR, et al. *BMJ.* 2003 Jun;326(7404):1427.

Hanmi Hanmi Pharm.

AMPL-II-2510-02 HM-Design 2510_01



※ Images are for illustrative purposes only and may not represent the actual size of ATOZET®. For detailed information on the size, shape, and appearance of the product, please refer to the full prescribing information approved by the MFDS.[1]

[Study design[2]] This prospective, randomized, double-blind, placebo-controlled, balanced-parallel group trial was conducted to test the primary hypothesis that the coadministration of ezetimibe with atorvastatin would result in a significantly greater reduction in LDL-C than atorvastatin alone. 628 patients (aged 18 years) with baseline LDL-C 145 to 250 mg/dL and triglycerides 350 mg/dL were randomly assigned to receive 1 of the following for 12 weeks: ezetimibe (10 mg/d, n=65); atorvastatin (10, 20, 40, or 80 mg/d, n=248); ezetimibe (10 mg) plus atorvastatin (10, 20, 40, or 80 mg/d, n=255); or placebo (n=60). The primary efficacy end point was the percentage reduction in direct LDL-C from baseline to final assessment for the intent-to-treat population.

† ATOZET® is available in four strengths: 10/10 mg, 10/20 mg, 10/40 mg, and 10/80 mg.

†**[Primary endpoint result]** Mean change in direct LDL-C from baseline: Ezetimibe/Atorvastatin overall vs. Atorvastatin overall = -54.5% vs. -42.4% (P<0.01).[2]

LDL-C, low-density lipoprotein cholesterol; **MFDS,** Ministry of Food and Drug Safety.

References 1. MFDS. ATOZET® Product information. **2.** Ballantyne CM, *et al.* Effect of ezetimibe coadministered with atorvastatin in 628 patients with primary hypercholesterolemia: a prospective, randomized, double-blind trial. *Circulation.* 2003;107(19):2409-2415.

Atozet®
Selected Safety Information

[Importer / Marketing Authorization Holder / Distributor]

ORGANON 한국오가논㈜ **Organon Korea** | 4F, 50 Jong-ro 1-gil, Jongno-gu, Seoul 03142, Korea (Tel) 1577-8582 https://www.organon.com/korea

Copyright © 2026 Organon group of companies. All rights reserved.

KR-ATO-116093 03/2028

보건의료전문가용

공동판매원 GC녹십자 수입판매원 GSK

오늘 싱그릭스#

대상포진이 환자의 여정에 걸림돌이 되지 않도록[2]

오늘 선생님께서 직접, 접종 권유를 시작해주세요.

#싱그릭스 효능 효과[1]
대상포진 (HZ; herpes zoster)의 예방
- 만 50세 이상의 성인
- 만 18세 이상에서 질병 혹은 치료로 인한 면역저하 또는 면역억제로 인하여 대상포진의 위험이 높거나 높을 것으로 예상되는 사람
(예. 자가조혈모세포이식자, 고형암, 혈액암, 고형장기 이식 환자)

[References] 1. 싱그릭스 국내 제품설명서. 2. Piazza MF, et al. Vaccines (Basel). 2020 Jan 10;8(1):20.

* 실제 약의 크기는 이미지와 다를 수 있습니다.

[Integrated Safety Information[3]] 1. 다음 환자에는 투여하지 말 것. 이 백신의 구성 성분에 과민반응이 있는 자 2. 다음 환자에게는 신중히 투여할 것 1) 급성 중증 열성 질환을 앓고 있는 자(급성 중증 열성질환 또는 급성 감염이 있는 경우, 이 백신의 접종을 연기해야 한다. 감기와 같이 경미한 감염으로 이 백신의 접종을 연기할 필요는 없다.) 2) 혈소판 감소증이나 다른 혈액응고장애가 있는 환자(근육주사 시 출혈이 일어날 수 있으므로 주의하여 투여 하여야 한다.) 3. 약물이상반응 ◎ 만 50세 이상인 성인 만 50세 이상 성인 약 17,000명이 17건의 임상시험에서 최소 1회 이 백신을 투여 받았다. 이 백신의 안전성은 만 50세 이상 성인 29,305명을 대상으로 실시한 2건의 주요 3상 위약 대조 임상시험 ZOE-50 (Zoster-006) 및 ZOE-70 (Zoster-022)의 데이터를 통합하여 평가되었다. 시험대상자들은 0, 2개월 접종 일정에 따라 이 백신 (N = 14,645) 또는 위약 (N = 14,660)을 최소 1회 투여 받았다. 예상된 약물이상반응 만 70세 이상 시험대상자에 비해 만 50–69세 시험대상자에서 주사부위 통증 및 종창, 피로, 근육통, 두통, 땀림, 열 및 위장관증상의 발생률이 더 높았다. 이들 약물 이상반응 대부분은 오래 지속되지 않았다 (지속시간 중앙값 2–3일). 중증 약물이상반응으로 보고된 반응은 1–2일간 지속되었다. ◎ 만 18세 이상의 질병 혹은 치료로 인한 면역저하 또는 면역억제로 대상포진의 위험이 높거나 높을 것으로 예상되는 자 (이하 "면역저 하자 (immunocompromised)"로 지칭함) 예상된 약물이상반응 자가조혈모세포이식 환자를 대상으로 한 연구 Zoster-002에서, 이 백신 투여 후 7일 이내 만 18-49세, 만 50세 이상에서 각각 보고된 예상된 국소 약물이상반응은 통증 (87.5%, 82.7%), 발적 (29.9%, 34.6%) 그리고 종창 (20.5%, 18.0%)이었고, 예상된 전신 약물이상반응은 피로 (64.1%, 53.8%), 근육통 (57.8%, 52.4%), 두통 (43.9%, 30.1%), 위장관증상 (21.1%, 28.2%), 땀림 (31.4%, 24.6%) 그리고 열 (27.8%, 17.8%)이었다 (최소 1회 증상을 보고한 대상자의 백분율 기준). 4. 일반적 주의 1) 이 백신을 혈관내 또는 피내로 투여하지 않는다. 2) 이 백신은 수두의 예방을 적응증으로 하지 않는다. 3) 다른 백신과 마찬가지로, 백신 접종 후 아나필락시스 반응이 일어날 경우에 대비하여 적절한 의학적 치료와 감독이 항상 준비되어 있어야 한다. 4) 만 65세 이상을 대상으로 한 시판 후 관찰 연구(미국, 2017.10.01. – 2020.02.29.)에서, 이 백신 접종 후 42일 동안 길랑-바레증후군의 발생 위험 증가가 관찰되었다 (백신투여량 1,000,000 dose 당 발생 건수가 약 3건 증가). 이 백신 투여와의 인과관계를 판단하기에는 정보가 충분하지 않다. 5. 임부 및 수유부에 대한 투여 1) 임부: 임부에서 이 백신을 사용한 자료는 없다. 동물시험에서는 임신, 배태자 발달, 분만 또는 출생 후 발달과 관련하여 직접적 또는 간접적인 유해한 영향이 나타나지 않았다. 예방적 조치로서, 임신 중에는 이 백신의 사용을 피하는 것이 좋다. 2) 수유: 이 백신의 접종이 수유 중인 영아나 모체에 미치는 영향은 연구된 바 없다. 이백신이 사람의 모유로 분비되는지 여부는 알려져 있지 않다. 3) 수태능: 동물시험에서 수컷 또는 암컷의 수태 능력에서 직접적 또는 간접적인 유해한 영향이 나타나지 않았다.

자세한 사항은 (주)글락소스미스클라인 백신 담당자에게 문의하여 주십시오.
GSK 제품 사용 중 발생한 이상사례(부작용)는 080-901-4100 또는 koreasafetyreporting.co.kr으로 보고해 주시기 바랍니다. Trademarks are owned by or licensed to the GSK group of companies. ©2025 GSK group of companeies or its licensor.

수입판매원 GSK (주)글락소스미스클라인 서울특별시 용산구 한강대로 92 Tel. 080-901-4100

공동판매원 Kwangdong 광동제약 광동제약(주) 경기도 과천시 과천대로7다길52 (갈현동)

GC녹십자 GC녹십자 경기도 용인시 기흥구 이현로 30번길 107 (보정동)

싱그릭스주 [대상포진바이러스백신 (유전자재조합)]

[Product Information]
싱그릭스주
[대상포진바이러스백신 (유전자재조합)]

처방하시기 전 QR 코드 또는 식품의약품안전처 의약품통합정보시스템 (http://nedrug.mfds.go.kr)을 통해 상세제품정보를 참조하시기 바랍니다.

PM-KR-SGX-JRNA-250005 Nov 2025

LEQVIO®
(inclisiran) injection 284 mg/1.5 mL

1년에 2번*, 일상을 돌려주는

6개월의 약속

치료목표 도달 및 유지†까지 가능해진 LDL-C 치료의 새로운 패러다임[2,3]

한국인 포함 대규모 3상 임상에서 LDL-C **57.2%** 감소[4]
실제 진료 환경에서 LDL-C 치료목표 **94.7%** 달성[5,‡]

† LDL-C 70 mg/dL 기준, statin±ezetimibe 환자군 대상, 9개월 추적관찰 시.

* 1일 1회 1프리필드시린지 (인클리시란으로서 284 mg)를 피하 투여하며, 최초, 3개월 후, 이후 6개월 마다 투여합니다.[1]
† 초기 두 번의 인클리시란 투여 후, 각 6개월 간격의 투여 기간 동안 LDL-C 감소가 유지되었습니다.

References **1.** 렉비오프리필드시린지(인클리시란나트륨)) 식품의약품안전처 의약품통합정보시스템 (nedrug.mfds.go.kr) **2.** Lamb YN. Inclisiran: First Approval. Drugs. 2021 Feb;81(3):389-395. **3.** Wright RS, et al. Inclisiran administration potently and durably lowers LDL-C over an extended-term follow-up: the ORION-8 trial. Cardiovasc Res. 2024 Oct 14;120(12):1400-1410. **4.** Hua Y, et al. Efficacy and Safety of Inclisiran in Asian Patients: Results From ORION-18. JACC Asia. 2023 Nov 14;4(2):123-134. **5.** Gargiulo P, et al. Real-World Efficacy and Safety of Inclisiran: A Single-Country, Multicenter, Observational Study (CHOLINET Registry). J Am Coll Cardiol. 2025 Feb 11;85(5):536-540.

NOVARTIS 한국노바티스주식회사
서울시 영등포구 국제금융로 10 Three IFC, 49층 07326 TEL: 02)768-9000 FAX: 02)785-1939

※ Product Information 처방하시기 전 QR 코드 또는 식품의약품안전처 의약품통합정보시스템 (https://nedrug.mfds.go.kr)를 통해 상세 제품정보를 참조하시기 바랍니다.

렉비오프리필드시린지
(인클리시란나트륨)

FA-114364423 2027May31

보건의료전문가용

CRESTOR®
rosuvastatin

START STRONG! STAY SUPER![1,2]

WITH CRESTOR®

고콜레스테롤혈증 치료 시작을 함께할 강력한 슈퍼 스타틴[1,2], 크레스토®

크레스토®는 국내 이상지질혈증 가이드라인에서 1차 치료제로 권고[3]하는 스타틴으로
강력한 LDL-C 강하 효과[1]와 심혈관계 주요 적응증*을 보유한 오리지널 로수바스타틴입니다.[4]

*고콜레스테롤혈증, 죽상동맥경화증 진행 지연, 심혈관 질환 발생 위험 감소

※ 보다 자세한 사항은 크레스토®정(로수바스타틴칼슘) 제품설명서 전문을 참고하시기 바랍니다.

크레스토®정 5 mg　크레스토®정 10 mg　크레스토®정 20 mg

References. 1. Jan M. Rosuvastatin-'the super statin'. J Ayub Med Coll Abbottabad. 2001 Apr-Jun;13(2):1-2. 2. FDA Drug Safety and Availability. Relative LDL-lowering Efficacy of Statin and Statin-based Therapies. Available from: https://www.fda.gov/drugs/drug-safety-and-availability/fda-drug-safety-communication-new-restrictions-contraindications-and-dose-limitations-zocor#Relative. Accessed Mar 2026. 3. KSoLA 이상지질 진료지침 제 5판. 한국지질동맥경화학회 진료지침위원회. 2022;1-281. 4. 크레스토®정 허가사항. 식품의약품안전처 의약품안전나라 의약품통합정보시스템 [Revised on 2024-12-24].

KR-221111 | Exp. 2028-03 (Prep. 2026-03)

AstraZeneca
수입(수입자): 한국아스트라제네카
서울 강남구 영동대로 517 아셈타워 21층. 전화: (02)2188-0800 팩스: (02)2188-0852

DAEWOONG
공동판매자: (주)대웅제약
서울 강남구 봉은사로 114길 12 대웅제약빌딩. 고객센터: 080-550-8308~9

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

"Bridge to Excellence, Toward Cardiometabolic Health"

ABSTRACT BOOK

September
10 (Thu)-12 (Sat), 2026

Conrad Seoul, Republic of Korea

ICoLA by KSoLA International Congress on Lipid & Atherosclerosis

KSoLA The Korean Society of Lipid and Atherosclerosis

Welcome Message

Dear Colleagues,

On behalf of the Organizing Committee, it is our great pleasure to welcome you to the 15th International Congress on Lipid and Atherosclerosis (ICoLA 2026), held from September 10 to 12, 2026, at Conrad Seoul, Republic of Korea.

This year's theme, "Bridge to Excellence, Toward Cardiometabolic Health," embodies our shared commitment to connecting scientific discovery with clinical practice and translating knowledge into better cardiometabolic health. It also reflects the essential bridges we continue to build—across disciplines, institutions, countries, and generations—to address the increasingly complex and interconnected challenges of dyslipidemia, atherosclerosis, and cardiovascular disease.

The scientific program of ICoLA 2026 has been designed to explore the full spectrum of lipid and cardiometabolic medicine, from molecular mechanisms and emerging therapeutic targets to prevention strategies, clinical implementation, and population health. Plenary and special lectures will offer perspectives on major advances and persistent challenges in cardiovascular prevention, while the main symposia will examine a broad range of timely topics, including inflammation and residual cardiovascular risk, lipoprotein(a), familial and severe dyslipidemia, obesity, fatty liver disease, chronic kidney disease, diabetes, vascular aging, novel lipid-lowering therapies, artificial intelligence, and environmental cardiovascular risk.

A particularly meaningful highlight of this year's congress is the official launch of the 2026 KSoLA Guidelines for the Management of Dyslipidemia, providing an important opportunity to consider how evolving evidence can be translated into more effective and practical patient care. Joint symposia with leading academic societies from Asia and Europe will further broaden the discussion by bringing together regional experience and international perspectives on the challenges shared across diverse healthcare settings.

The oral and moderated poster presentation sessions also form an essential part of the congress. We are especially pleased to provide early-career investigators with opportunities to present their work, engage with established experts, and contribute fresh ideas to our scientific community. We hope these exchanges will inspire new questions, collaborations, and discoveries extending well beyond the congress itself.

Now that we are gathered together in Seoul, we encourage you to take full advantage of the many opportunities for learning, discussion, and connection throughout the next three days. Your expertise, curiosity, and active participation are what give this congress its true meaning and vitality.

We sincerely thank all invited speakers, chairpersons, panelists, presenters, participants, partner societies, sponsors, and organizing members whose dedication has made ICoLA 2026 possible.

Once again, welcome to ICoLA 2026. We hope your time in Seoul will be scientifically rewarding, professionally enriching, and truly memorable.

Sincerely,

Sung Rae Kim

President, Board of Directors, The Korean Society of Lipid and Atherosclerosis

Sang-Hyun Kim

Chairman, Board of Directors, The Korean Society of Lipid and Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

CONTENTS

Program at a Glance 04

Program in Detail
- Key Lectures 07
- Main Symposia 08
- Joint Symposia 24
- Satellite Symposia 27
- Oral Presentation 31
- Moderated Poster Presentation 35
- E-Poster 42

Author Index 377

Program at a Glance

[Day 1] September 10(Thu), 2026

(K): Korean Session
(K+E): Korean & English Session (The language of each lecture may differ within the same session.)

	Room 1 (Grand Ballroom 3, 3F)	Room 2 (Grand Ballroom 2, 3F)	Room 3 (Grand Ballroom 1, 3F)	Room 4 (Park Ballroom 3, 5F)
12:00–	Registration			
13:00–14:30	**Symposium 1** Nutritional Immunometabolism in Atherosclerosis	**Symposium 2 (K)** From Biobank to Biology: Integrative Platforms for Cardiometabolic Discovery	**Symposium 3** Inflammation in ASCVD: From LDL Lowering to Immune Modulation	**Symposium 4** *Session Time: 13:00–14:00 Publication Committee Session: JLA Award for Outstanding Article
14:30–16:00	**Oral Presentation 1**	**Symposium 5** FH/Severe Dyslipidemia: Optimizing FH Management	**IHA–KSoLA Joint Symposium** Optimizing Lipid-Lowering Strategies: Facing Challenges in Asian High-Risk Patients	**Symposium 6** Neurocardiometabolic Benefits of GLP1 RA and SGLT2 Inhibitors
16:00–16:10	*Break*			
16:10–17:40	**Symposium 7** Real-world LDL-C Management: From Guideline to Prescription	**JAS–KSoLA Joint Symposium** Comprehensive Lipid Management in East Asians: Integrating LDL-C Optimization, Residual Risk, and Genetic Factors	**Symposium 8** Metabolic Regulators of Lipid Metabolism: From Mechanisms to Cardiovascular Outcomes	**Symposium 9** KSN–KSoLA Joint Symposium Chronic Kidney Disease and Atherosclerosis
17:40–	**Welcome Reception (Lobby, Studio 4–7, 6F)**			

Program at a Glance

[Day 2] September 11(Fri), 2026

(K): Korean Session
(K+E): Korean & English Session (The language of each lecture may differ within the same session.)

	Room 1 (Grand Ballroom 3, 3F)	Room 2 (Grand Ballroom 2, 3F)	Room 3 (Grand Ballroom 1, 3F)	Room 4 (Park Ballroom 3, 5F)	Room 5 (Studio 2,3, 6F)
07:30–08:30	**Breakfast Symposium 1 (K+E)**	**Breakfast Symposium 2**	**Breakfast Symposium 3**	**Breakfast Symposium 4**	
08:30–10:00	**Oral Presentation 2**	**Symposium 10** New Frontiers in Lipid Lowering: Beyond Statins (New Drugs 1)	**Symposium 11** Lipid-lowering Therapy in Complex Clinical Settings: HIV, Cancer, and Chronic Kidney Disease	**Symposium 12** KPS-KSoLA Joint Symposium Bridging Cellular Physiology and Atherogenic Lipids	
10:00–10:10	***Break***				
10:10–10:20	**Opening Address**				
10:20–11:00	**Plenary Lecture 1** Lipoprotein(a): the next frontier in cardiovascular prevention				
11:00–11:40	**Special Lecture 1 (K)** 도자기로 보는 한국 전통 미술의 생명력 (The vitality of traditional Korean art through ceramics)				
11:45–12:45	**Luncheon Symposium 1 (K)**	**Luncheon Symposium 2**	**Luncheon Symposium 3**	**Luncheon Symposium 4 (K)**	**Luncheon Symposium 5**
12:45–13:45	**Poster Presentation Session 1 (Studio 5, 6, 7, 6F)**				
	Symposium 13 (K) 2026 KSoLA Awards for Scientific Excellence & Digital Health Media and Ethics	**Symposium 14** Sex-specific Lipid Biology and Cardiometabolic Risk across the Lifespan			
13:45–15:15	**Symposium 15** Official Launch of the 2026 KSoLA Guidelines for the Management of Dyslipidemia	**VAS-KSoLA Joint Symposium** Residual Cardiovascular Risk Beyond LDL-C: The Emerging Role of Lipoprotein(a) in Asia	**Symposium 16** Evolution of High-Risk Coronary Plaques: From Detection to Prevention	**Symposium 17** Metabolic Dysfunction, Fatty Liver, and Atherosclerosis	
15:20–16:00	**Plenary Lecture 2** Closing the gap between guidelines and reality in dyslipidemia management				
16:00–16:10	***Break***				
16:10–17:40	**Symposium 18** Intensive LDL-C Lowering in Primary Prevention	**Symposium 19** Next-generation Lipid-lowering Therapies: CETP, APOC3, and ANGPTL3 Inhibition (New Drugs 2)	**Symposium 20** Residual Cardiovascular Risk: Beyond LDL to Inflammation and Novel Targets	**Symposium 21** KSSO-KSoLA Joint Symposium Atherogenic Obesity: Beyond BMI to Lipid Dysfunction	

Program at a Glance

[Day 3] September 12(Sat), 2026

(K): Korean Session
(K+E): Korean & English Session (The language of each lecture may differ within the same session.)

	Room 1 (Grand Ballroom 3, 3F)	Room 2 (Grand Ballroom 2, 3F)	Room 3 (Grand Ballroom 1, 3F)	Room 4 (Park Ballroom 3, 5F)
07:50–08:50	**Breakfast Symposium 5**	**Breakfast Symposium 6**	**Breakfast Symposium 7**	**Breakfast Symposium 8**
08:50–10:20	**Oral Presentation 3**	**TSLA–KSoLA Joint Symposium** Contemporary Management of Residual Cardiovascular Risk in Asia	**Symposium 22** Lp(a) beyond the Known: From Metabolic Pathways to Clinical Translation	**MSA–KSoLA Joint Symposium** From Diversity to Action: Ethnicity, Genetics, and Closing the Treatment Gap in Asian Dyslipidemia
10:20–10:40	*Coffee Break*			
10:40–11:20	**Plenary Lecture 3** Beyond LDL-C lowering: strategies for maximizing residual ASCVD risk reduction			
11:20–12:00	**Plenary Lecture 4** Perspective on residual risk, inflammation, and atherosclerosis: from epidemiology to precision prevention			
12:00–13:00	**Luncheon Symposium 6**	**Luncheon Symposium 7**	**Luncheon Symposium 8**	
13:00–14:00	**Poster Presentation Session 2 (Studio 5, 6, 7, 6F)**			
	Symposium 23 KSH–KSoLA Joint Symposium Hypertension, Lipids, and Vascular Aging: Integrated Cardiovascular Risk Management	**Symposium 24** KSCVI–KSoLA Joint Symposium Beyond Stenting: Contemporary Management of Residual Coronary Risk	**Symposium 25** KDA–KSoLA Joint Symposium Integrated Cardiometabolic Risk Management in Diabetes	**Special Lecture 2** *Session Time: 13:20–13:50 Atherosclerosis and atherosclerotic cardiovascular disease in Asia: the knowledge gap for the determinants
14:00–14:10	*Break*			
14:10–15:40	**Symposium 26** Integrated Cardiovascular–kidney–metabolic Care: A Multidisciplinary Approach to CKM Syndrome	**Symposium 27** AI-driven Innovations in Atherosclerosis	**Symposium 28** Primary Prevention in Cardiovascular Disease: Beyond Traditional Risk Factors	**Symposium 29** KES–KSoLA Joint Symposium Lipid Metabolism Across Endocrine Disorders: From Mechanism to Clinical Practice
15:40–17:10	**Symposium 30** Neural and Genetic Control of Eating Behavior and Cardiometabolic Health	**EAS–KSoLA Joint Symposium** Bridging Europe and Korea in Dyslipidemia Management: Guidelines, Gaps, and Future Directions	**Symposium 31** From Cells to Systems: Integrated Control of Cardiometabolic Health	**Symposium 32** Environmental Cardiovascular Risk
17:10–	**Closing Ceremony (Room 1,2,3, 3F)**			

Program in Detail

Key Lectures p. 47

Plenary Lecture 1

Sep 11(Fri) 10:20–11:00 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Sung Rae Kim (The Catholic University of Korea, Republic of Korea)

10:20–11:00 **Lipoprotein(a): the next frontier in cardiovascular prevention**
Sotirios Tsimikas (University of California, San Diego, USA)

Plenary Lecture 2

Sep 11(Fri) 15:20–16:00 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Woo Je Lee (University of Ulsan, Republic of Korea)

15:20–16:00 **Closing the gap between guidelines and reality in dyslipidemia management**
Michal Vrablik (Charles University, Czech Republic)

Plenary Lecture 3

Sep 12(Sat) 10:40–11:20 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Sang-Hyun Kim (Seoul National University, Republic of Korea)

10:40–11:20 **Beyond LDL-C lowering: strategies for maximizing residual ASCVD risk reduction**
Nathan D. Wong (University of California, Irvine, USA)

Plenary Lecture 4

Sep 12(Sat) 11:20–12:00 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Ick Mo Chung (Ewha Womans University, Republic of Korea)

11:20–12:00 **Perspective on residual risk, inflammation, and atherosclerosis: from epidemiology to precision prevention**
Borge G. Nordestgaard (University of Copenhagen, Denmark)

Special Lecture 1 (K)

Sep 11(Fri) 11:00–11:40 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Jae Hyoung Park (Korea University, Republic of Korea)

11:00–11:40 **도자기로 보는 한국 전통 미술의 생명력 (The vitality of traditional Korean art through ceramics)**
Ae-Ryung Lee (National Museum of Korea, Republic of Korea)

Special Lecture 2

Sep 12(Sat) 13:20–13:50 | Room 4 (5F, Conrad Seoul)

CHAIRPERSON : Seong Hoon Choi (Hallym University, Republic of Korea)

13:20–13:50 **Atherosclerosis and atherosclerotic cardiovascular disease in Asia: the knowledge gap for the determinants**
Dong Zhao (Capital Medical University, China)

Main Symposia p. 73

Symposium 1

Sep 10(Thu) 13:00–14:30 | Room 1 (3F, Conrad Seoul)

Nutritional Immunometabolism in Atherosclerosis

CHAIRPERSONS : Myoung Sook Lee (Sungshin Women's University, Republic of Korea)
Sung Nim Han (Seoul National University, Republic of Korea)

13:00–13:20 **Gut microbiota remodeling in infection and inflammation**
Eun-Kyeong Jo (Chungnam National University, Republic of Korea)

13:20–13:40 **Ketogenic adaptation to intermittent fasting: implications for nutritional immunometabolism**
Kyoung-Han Kim (University of Ottawa, Canada)

13:40–14:00 **Spatiotemporal immune activation drives metabolic quiescence in brown adipose tissue**
Su Myung Jung (Sungkyunkwan University, Republic of Korea)

14:00–14:30 **Panel Discussion**
Hyunju Kang (Kyungpook National University, Republic of Korea)
Jun Namkung (Yonsei University, Republic of Korea)
Chan Joo Lee (Yonsei University, Republic of Korea)

Symposium 2 (K)

Sep 10(Thu) 13:00–14:30 | Room 2 (3F, Conrad Seoul)

From Biobank to Biology: Integrative Platforms for Cardiometabolic Discovery

CHAIRPERSONS : Chi Dae Kim (Pusan National University, Republic of Korea)
Young-Guen Kwon (Curacle Advanced Institute of Technology, Republic of Korea)

13:00–13:20 **Biobank resources for human metabolic and vascular research**
Hyun-Joung Lim (National Institute of Health, Republic of Korea)

13:20–13:40 **Leveraging biobank platforms to study cardiometabolic physiology and disease**
Heiko Bugger (Medical University of Graz, Austria)

13:40–14:00 **Integrative analysis of metabolic dysfunction**
Young Yang (Sookmyung Women's University, Republic of Korea)

14:00–14:30 **Panel Discussion**
Yongseek Park (Kyung Hee University, Republic of Korea)
Jun Hwan Cho (Chung-Ang University, Republic of Korea)
Jae-Hoon Choi (Hanyang University, Republic of Korea)

Symposium 3

Sep 10(Thu) 13:00–14:30 | Room 3 (3F, Conrad Seoul)

Inflammation in ASCVD: From LDL Lowering to Immune Modulation

CHAIRPERSONS : Young Joon Hong (Chonnam National University, Republic of Korea)
Weon Kim (Kyung Hee University, Republic of Korea)

13:00–13:18 **LDL versus inflammation: what remains after aggressive lipid lowering?**
Osung Kwon (The Catholic University of Korea, Republic of Korea)

13:18–13:36 **Emerging anti-inflammatory therapies in ASCVD: canakinumab, NLRP3 inhibitors, and beyond**
SungA Bae (Yonsei University, Republic of Korea)

13:36–13:54 **Colchicine for secondary prevention in ASCVD**
Dae Young Cheon (Hallym University, Republic of Korea)

13:54–14:12 **Immune mechanisms of residual cardiovascular risk: from oxidation-specific epitopes to therapeutic targets**
Christoph J. Binder (Medical University of Vienna, Austria)

14:12–14:30 **Panel Discussion**
Ung Kim (Yeungnam University, Republic of Korea)
Chang-Hoon Woo (Yeungnam University, Republic of Korea)
Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

Symposium 4

Sep 10(Thu) 13:00–14:00 | Room 4 (5F, Conrad Seoul)

Publication Committee Session: JLA Award for Outstanding Article

CHAIRPERSONS : In-Kyung Jeong (Kyung Hee University, Republic of Korea)
Hyun Kang (Chung-Ang University, Republic of Korea)

13:00–13:15	**Arterial tissue gene expression associated with telomere length attrition in coronary artery disease** **Rajkumar Dorajoo** (Genome Institute of Singapore, Singapore)
13:15–13:30	**Administration of sodium–glucose cotransporter–2 inhibitors following acute myocardial infarction: a systematic review, meta–analysis, and meta–regression of randomized trials** **Timotius Ivan Hariyanto** (Universitas Airlangga, Indonesia)
13:30–13:45	**Sex differences in the diagnosis and treatment of Japanese patients with familial hypercholesterolemia** **Hayato Tada** (Kanazawa University, Japan)
13:45–14:00	**Tigloylgomisin P inhibits endothelial inflammation via NFκB and Smad1/5/9 pathway regulation** **Sanguk Yun** (Inje University, Republic of Korea)

Symposium 5

Sep 10(Thu) 14:30–16:00 | Room 2 (3F, Conrad Seoul)

FH/Severe Dyslipidemia: Optimizing FH Management

CHAIRPERSONS : Sang-Hak Lee (Yonsei University, Republic of Korea)
Hayato Tada (Kanazawa University, Japan)

14:30–14:48	**Refined risk assessment in FH** **Sayaka Funabashi** (Kyorin University, Japan)
14:52–15:10	**Cause of death in FH** **Chan Joo Lee** (Yonsei University, Republic of Korea)
15:14–15:32	**Experience on new drugs for FH: including lomitapide & evinacumab** **Hayato Tada** (Kanazawa University, Japan)
15:36–15:54	**Genome editing for precision medicine** **HyongBum Kim** (Yonsei University, Republic of Korea)
	Panel **Sung Hee Choi** (Seoul National University, Republic of Korea)

Symposium 6

Sep 10(Thu) 14:30–16:00 | Room 4 (5F, Conrad Seoul)

Neurocardiometabolic Benefits of GLP1 RA and SGLT2 Inhibitors

CHAIRPERSONS : Geun Sik Hong (Inje University, Republic of Korea)
Kwang-Yeol Park (Chung-Ang University, Republic of Korea)

14:30–14:50	**GLP1 RA for stroke prevention** **Kang-Ho Choi** (Chonnam National University, Republic of Korea)
14:50–15:10	**SGLT2 inhibitor for stroke survivors** **Jeong-Min Kim** (Seoul National University, Republic of Korea)
15:10–15:30	**GLP-1 receptor agonists in Alzheimer's disease** **Sang-Won Han** (Seoul National University, Republic of Korea)
15:30–16:00	**Panel Discussion** **Bum Joon Kim** (University of Ulsan, Republic of Korea) **Hack-Lyoung Kim** (Seoul National University, Republic of Korea) **Se-eun Park** (Sungkyunkwan University, Republic of Korea)

Symposium 7

Sep 10(Thu) 16:10–17:40 | Room 1 (3F, Conrad Seoul)

Real-world LDL-C Management: From Guideline to Prescription

CHAIRPERSONS : Youngkeun Ahn (Chonnam National University, Republic of Korea)
Min Kyong Moon (Seoul National University, Republic of Korea)

16:10–16:28	**Choosing the right combination therapy: statin, ezetimibe, PCSK9 inhibitor, or beyond?** **Kyung Hoon Cho** (Chonnam National University, Republic of Korea)
16:28–16:46	**Korean Fact Sheet: Current status of LDL-C control from general population to high-risk groups** **Ye Seul Yang** (Seoul National University, Republic of Korea)
16:46–17:04	**Korean Fact Sheet: Real-world utilization of lipid-lowering agents** **Kyuho Kim** (The Catholic University of Korea, Republic of Korea)
17:04–17:22	**Why are LDL-C targets still unmet? common errors and practical solutions in the clinic** **Sang Yeub Lee** (Chung-Ang University, Republic of Korea)
17:22–17:40	**Panel Discussion** **Young Soo Lee** (Daegu Catholic University, Republic of Korea) **Jun Hwan Cho** (Chung-Ang University, Republic of Korea) **Sangmo Hong** (Hanyang University, Republic of Korea)

Symposium 8

Sep 10(Thu) 16:10–17:40 | Room 3 (3F, Conrad Seoul)

Metabolic Regulators of Lipid Metabolism: From Mechanisms to Cardiovascular Outcomes

CHAIRPERSONS : Eun Gyoung Hong (Hallym University, Republic of Korea)
Chul Sik Kim (Yonsei University, Republic of Korea)

16:10–16:30 **Sex differences in lymphatic function and atherosclerosis outcomes**
Catherine Martel (University of Montreal, Canada)

16:30–16:50 **Tirzepatide and cardiometabolic outcomes: implications of dual GLP-1/GIP agonism**
Yun Kyung Cho (University of Ulsan, Republic of Korea)

16:50–17:10 **Effects of SGLT2 inhibitors on lipid metabolism: mechanisms and clinical implications**
Jun Hwa Hong (Eulji University, Republic of Korea)

17:10–17:40 **Panel Discussion**
Shinae Kang (Yonsei University, Republic of Korea)
JiHyun Ahn (Korea Medical Institute, Republic of Korea)
Jung-Joon Cha (Korea University, Republic of Korea)

Symposium 9 – KSN-KSoLA Joint Symposium

Sep 10(Thu) 16:10–17:40 | Room 4 (5F, Conrad Seoul)

Chronic Kidney Disease and Atherosclerosis

CHAIRPERSONS : Sang-Hyun Kim (Seoul National University, Republic of Korea)
Bum Soon Choi (The Catholic University of Korea, Republic of Korea)

16:10–16:30 **Lipid abnormalities in CKD**
Hong Sang Choi (Chonnam National University, Republic of Korea)

16:30–16:50 **Why patients with CKD develop accelerated atherosclerosis**
Cheol Ho Park (Yonsei University, Republic of Korea)

16:50–17:10 **Cardiovascular prevention in CKD: an endocrinologist's perspective**
Eun Roh (Seoul National University, Republic of Korea)

17:10–17:40 **Panel Discussion**
Wonjin Kim (CHA University, Republic of Korea)
Ji Won Ryu (Seoul National University, Republic of Korea)
Eun Hui Bae (Chonnam National University, Republic of Korea)

Symposium 10

Sep 11(Fri) 08:30-10:00 | Room 2 (3F, Conrad Seoul)

New Frontiers in Lipid Lowering: Beyond Statins (New Drugs 1)

CHAIRPERSONS : Sang-Hak Lee (Yonsei University, Republic of Korea)
Sungha Park (Yonsei University, Republic of Korea)

08:30-08:50 **Bempedoic acid: LDL-C lowering beyond statins – evidence and clinical role**
Chewan Lim (Catholic Kwandong University, Republic of Korea)

08:50-09:10 **Optimizing non-statin therapy: PCSK9 inhibitors and inclisiran**
Kyung-Hee Kim (Incheon Sejong Hospital, Republic of Korea)

09:10-09:30 **Emerging Lp(a)-lowering therapies: siRNA, ASO, and beyond**
Yu-Chen Wang (Asia University, Taiwan)

09:30-10:00 **Panel Discussion**
Gyu Chul Oh (The Catholic University of Korea, Republic of Korea)
Ji-Yong Jang (National Health Insurance Service Ilsan Hospital, Republic of Korea)
Yun Kyung Cho (University of Ulsan, Republic of Korea)

Symposium 11

Sep 11(Fri) 08:30-10:00 | Room 3 (3F, Conrad Seoul)

Lipid-lowering Therapy in Complex Clinical Settings: HIV, Cancer, and Chronic Kidney Disease

CHAIRPERSONS : Junghyun Noh (Inje University, Republic of Korea)
Jung-Sun Kim (Yonsei University, Republic of Korea)

08:30-08:48 **Lipid-lowering therapy in people living with HIV (PLWH)**
So Ree Kim (Korea University, Republic of Korea)

08:48-09:06 **Lipid-lowering therapy in cancer patients and survivors**
Hiroshi Akazawa (Kanazawa Medical University, Japan)

09:06-09:24 **Lipid-lowering therapy in patients with CKD**
Eun Sil Koh (The Catholic University of Korea, Republic of Korea)

09:24-09:42 **Cardiovascular prevention in hemodialysis: is omega-3 back?**
Sang Ho Jo (Hallym University, Republic of Korea)

09:42-10:00 **Panel Discussion**
Sang Yong Kim (Chosun University, Republic of Korea)
Ji Woong Yoo (Hallym University, Republic of Korea)
Mi-Hyang Jung (The Catholic University of Korea, Republic of Korea)

Symposium 12 – KPS–KSoLA Joint Symposium

Sep 11(Fri) 08:30–10:00 | Room 4 (5F, Conrad Seoul)

Bridging Cellular Physiology and Atherogenic Lipids

CHAIRPERSONS : **Yong Sook Kim** (Chonnam National University, Republic of Korea)
Hyoung Kyu Kim (Inje University, Republic of Korea)

08:30–08:50	**Ion channels in atherosclerosis: from calcium flux to vascular dysfunction** **Sung Joon Kim** (Seoul National University, Republic of Korea)
08:50–09:10	**Energy metabolism and lipid oxidation in cardiometabolic pathophysiology** **Jin Han** (Inje University, Republic of Korea)
09:10–09:30	**Lp(a): from lipoprotein biology to atherosclerotic cardiovascular disease** **Jung–Woo Son** (Yonsei University, Republic of Korea)
09:30–10:00	**Panel Discussion** **Nari Kim** (Inje University, Republic of Korea) **Jong–Gil Park** (Korea Research Institute of Bioscience and Biotechnology, Republic of Korea) **Dong Kyu Choi** (Kyungpook National University, Republic of Korea)

Symposium 13 (K)

Sep 11(Fri) 12:45–13:40 | Room 1 (3F, Conrad Seoul)

2026 KSoLA Awards for Scientific Excellence & Digital Health Media and Ethics

CHAIRPERSONS : **Sung Rae Kim** (The Catholic University of Korea, Republic of Korea)
Sang–Hyun Kim (Seoul National University, Republic of Korea)

12:45–12:50	2026 KSoLA Award Ceremony for Scientific Excellence
12:50–12:55	2026 KSoLA Award Ceremony for Young Investigator
12:55–13:15	**The quiet signal, no longer – triglyceride at the crossroads of insulin resistance and cardiometabolic risk** **Cheol–Young Park** (Sungkyunkwan University, Republic of Korea)
13:15–13:20	Q&A
13:20–13:40	**How reliable is statin information on Korean YouTube? validating AI–assisted reliability assessment against expert raters** **Ilhak Lee** (Yonsei University, Republic of Korea)
13:40–13:45	Q&A

Symposium 14

Sep 11(Fri) 12:45–13:45 | Room 2 (3F, Conrad Seoul)

Sex-specific Lipid Biology and Cardiometabolic Risk across the Lifespan

CHAIRPERSONS : Mi-Seung Shin (Gachon University, Republic of Korea)
Jun-Hee Lee (Hallym University, Republic of Korea)

12:45–13:00	**Sex differences in lipids and lipoproteins and residual cardiovascular risk: beyond LDL cholesterol** **Soo-Jin Kim** (Kosin University, Republic of Korea)
13:00–13:15	**Early-onset dyslipidemia in young adults: screening strategies and lifetime ASCVD risk assessment** **Jaehyun Bae** (Hallym University, Republic of Korea)
13:15–13:30	**Menopause and cardiometabolic transition: optimizing lipid management in midlife women** **In-Jeong Cho** (Ewha Womans University, Republic of Korea)
13:30–13:45	**Panel Discussion** **Hack-Lyoung Kim** (Seoul National University, Republic of Korea) **Ye Seul Yang** (Seoul National University, Republic of Korea) **Seok Oh** (Chonnam National University, Republic of Korea)

Symposium 15

Sep 11(Fri) 13:45–15:15 | Room 1 (3F, Conrad Seoul)

Official Launch of the 2026 KSoLA Guidelines for the Management of Dyslipidemia

CHAIRPERSONS : Sang-Hyun Kim (Seoul National University, Republic of Korea)
Woo Je Lee (University of Ulsan, Republic of Korea)

13:45–13:50	**What's new in the 2026 Korean guidelines for the management of dyslipidemia** **Hyun-Jin Kim** (Hanyang University, Republic of Korea)
13:50–14:08	**Korean epidemiology and risk modifiers for establishing LDL-C goal strategies** **Jong Han Choi** (Konkuk University, Republic of Korea)
14:08–14:26	**Practical algorithm for intensive lipid lowering therapy in the 2026 Korean guidelines** **Jaehoon Chung** (Gachon University, Republic of Korea)
14:26–14:44	**Nutritional management and lifestyle modification tailored to the Korean dietary pattern** **Garam Jo** (Hannam University, Republic of Korea)
14:44–15:02	**Optimized lipid management for special populations** **Byung Sik Kim** (Hanyang University, Republic of Korea)
15:02–15:15	**Panel Discussion** **Hyun-Jin Kim** (Hanyang University, Republic of Korea) **Eunju Park** (Kyungnam University, Republic of Korea) **Jee Hee Yoo** (Chung-Ang University, Republic of Korea) **Hye Moon Chung** (Kyung Hee University, Republic of Korea)

Symposium 16

Sep 11(Fri) 13:45–15:15 | Room 3 (3F, Conrad Seoul)

Evolution of High-Risk Coronary Plaques: From Detection to Prevention

CHAIRPERSONS : Byung Jin Kim (Sungkyunkwan University, Republic of Korea)
Jung Rae Cho (Hallym University, Republic of Korea)

13:45–14:03 **Evolution of vulnerable plaques: from natural history to preventive intervention**
Jinho Lee (Kyung Hee University, Republic of Korea)

14:03–14:21 **High-risk plaque features on CCTA: identification and clinical implications**
Sang Yoon Lee (Sungkyunkwan University, Republic of Korea)

14:21–14:39 **Intravascular imaging of vulnerable plaques: insights from IVUS and OCT**
Ju Hyeon Kim (University of Ulsan, Republic of Korea)

14:39–14:57 **Coronary physiology and plaque vulnerability: integrative relationship and prognostic implications**
Seokhun Yang (Seoul National University, Republic of Korea)

14:57–15:15 **Panel Discussion**
Bom Lee (CHA University, Republic of Korea)
Hyun Sung Joh (Seoul National University, Republic of Korea)
Hyung Joon Joo (Korea University, Republic of Korea)

Symposium 17

Sep 11(Fri) 13:45–15:15 | Room 4 (5F, Conrad Seoul)

Metabolic Dysfunction, Fatty Liver, and Atherosclerosis

CHAIRPERSONS : Seok-Min Kang (Yonsei University, Republic of Korea)
Ki Chul Sung (Sungkyunkwan University, Republic of Korea)

13:45–14:05 **Fatty liver and residual cardiovascular risk: epidemiology and clinical implications**
Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea)

14:05–14:25 **From lipotoxicity to atherosclerosis: MASLD/MetALD subtypes and cardiovascular risk – A hepatologist's perspective**
Su Jong Yu (Seoul National University, Republic of Korea)

14:25–14:45 **Therapeutic strategies for cardiometabolic risk reduction in MASLD**
Toru Miyoshi (Okayama University, Japan)

14:45–15:15 **Panel Discussion**
Bo Kyung Koo (Seoul National University, Republic of Korea)
Kyung-Jin Kim (Ewha Womans University, Republic of Korea)
Heesun Lee (Seoul National University, Republic of Korea)

Symposium 18

Sep 11(Fri) 16:10–17:40 | Room 1 (3F, Conrad Seoul)

Intensive LDL-C Lowering in Primary Prevention

CHAIRPERSONS : Eung Gyu Kim (Inje University, Republic of Korea)
Sang-Hyun Lim (The Catholic University of Korea, Republic of Korea)

16:10–16:30 **PCSK9 inhibition in primary prevention: insights from VESALIUS-CV and clinical implications**
Doo Sun Sim (Chonnam National University, Republic of Korea)

16:30–16:50 **Low LDL-C and brain safety: risk of hemorrhagic stroke and cognitive dysfunction**
Kwang-Yeol Park (Chung-Ang University, Republic of Korea)

16:50–17:10 **Early combination therapy in primary prevention**
Hoyoun Won (Chung-Ang University, Republic of Korea)

17:10–17:40 **Panel Discussion**
Hyung-Min Kwon (Seoul National University, Republic of Korea)
Minjeong Kim (Ewha Womans University, Republic of Korea)
Garam Jo (Hannam University, Republic of Korea)

Symposium 19

Sep 11(Fri) 16:10–17:40 | Room 2 (3F, Conrad Seoul)

Next-generation Lipid-lowering Therapies: CETP, APOC3, and ANGPTL3 Inhibition (New Drugs 2)

CHAIRPERSONS : Sang Yup Lim (Korea University, Republic of Korea)
Soon Jun Hong (Korea University, Republic of Korea)

16:10–16:30 **CETP inhibition revisited: obicetrapib for LDL-C reduction and ASCVD risk management**
Mariko Harada-Shiba (Osaka Medical and Pharmaceutical University, Japan)

16:30–16:50 **APOC3 inhibition: targeting triglyceride-rich lipoproteins and residual risk**
Jin Wi (Gachon University, Republic of Korea)

16:50–17:10 **ANGPTL3 inhibition: expanding therapeutic opportunities beyond LDL-C**
Hye Moon Chung (Kyung Hee University, Republic of Korea)

17:10–17:40 **Panel Discussion**
Byung Sik Kim (Hanyang University, Republic of Korea)
Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea)
Janghoon Lee (Kyungpook National University, Republic of Korea)

Symposium 20

Sep 11(Fri) 16:10–17:40 | Room 3 (3F, Conrad Seoul)

Residual Cardiovascular Risk: Beyond LDL to Inflammation and Novel Targets

CHAIRPERSONS : Seung Woo Park (Sungkyunkwan University, Republic of Korea)
Young-Hoon Jeong (Chung-Ang University, Republic of Korea)

16:10–16:30	**Defining residual cardiovascular risk in the post-LDL era: unmet needs and mechanisms** **Hyung Joon Joo** (Korea University, Republic of Korea)
16:30–16:50	**Targeting residual risk: Lp(a), triglyceride-rich lipoproteins, and inflammation** **Sung Gyun Ahn** (Yonsei University, Republic of Korea)
16:50–17:10	**Beyond less is more: navigating residual thrombotic and inflammatory risk in AF and stable CAD** **Satoshi Yasuda** (Tohoku University, Japan)
17:10–17:40	**Panel Discussion** **Jaehyun Bae** (Hallym University, Republic of Korea) **Jung Myung Lee** (Sahmyook Medical Center, Republic of Korea) **Jong Han Choi** (Konkuk University, Republic of Korea)

Symposium 21 – KSSO-KSoLA Joint Symposium

Sep 11(Fri) 16:10–17:40 | Room 4 (5F, Conrad Seoul)

Atherogenic Obesity: Beyond BMI to Lipid Dysfunction

CHAIRPERSONS : Min Sun Kim (University of Ulsan, Republic of Korea)
In-Kyung Jeong (Kyung Hee University, Republic of Korea)

16:10–16:30	**Lipid remodeling in adipose tissue and liver: a hidden link between obesity and atherosclerosis** **Yun-Hee Lee** (Seoul National University, Republic of Korea)
16:30–16:50	**Remnant cholesterol and residual cardiovascular risk in obesity** **Young-Sang Lyu** (Chosun University, Republic of Korea)
16:50–17:10	**Dual targeting of lipid and weight: emerging therapies and clinical strategies** **Chang Hee Jung** (University of Ulsan, Republic of Korea)
17:10–17:40	**Panel Discussion** **Song-Yi Kim** (Jeju National University, Republic of Korea) **Jee Hee Yoo** (Chung-Ang University, Republic of Korea) **Heung Yong Jin** (Jeonbuk National University, Republic of Korea)

Symposium 22

Sep 12(Sat) 08:50–10:20 | Room 3 (3F, Conrad Seoul)

Lp(a) beyond the Known: From Metabolic Pathways to Clinical Translation

CHAIRPERSONS : Donghoon Choi (Yonsei University, Republic of Korea)
Keeho Song (Konkuk University, Republic of Korea)

08:50–09:10 **Oxidized phospholipids on lipoprotein(a): a key driver of atherothrombosis?**
Sotirios Tsimikas (University of California, San Diego, USA)

09:10–09:30 **Does lipoprotein(a) promote thrombosis? evidence from genetics and population studies**
Borge G. Nordestgaard (University of Copenhagen, Denmark)

09:30–09:50 **Epidemiology and cardiovascular events of Lp(a): what have we learned?**
Sang-Rok Lee (Jeonbuk National University, Republic of Korea)

09:50–10:20 **Panel Discussion**
Eu Jeong Ku (Seoul National University, Republic of Korea)
Young Sup Byun (Inje University, Republic of Korea)
Janghoon Lee (Kyungpook National University, Republic of Korea)

Symposium 23 – KSH-KSoLA Joint Symposium

Sep 12(Sat) 13:00–14:00 | Room 1 (3F, Conrad Seoul)

Hypertension, Lipids, and Vascular Aging: Integrated Cardiovascular Risk Management

CHAIRPERSONS : Kwang-il Kim (Seoul National University, Republic of Korea)
Jun Hwa Hong (Eulji University, Republic of Korea)

13:00–13:15 **Residual cardiovascular risk in hypertension despite optimal blood pressure control**
Hyue Mee Kim (Chung-Ang University, Republic of Korea)

13:15–13:30 **Lipid management in hypertensive patients**
Jaehoon Chung (Gachon University, Republic of Korea)

13:30–13:45 **Obesity, insulin resistance, and hypertension-related vascular injury**
Mihye Seo (Soonchunhyang University, Republic of Korea)

13:45–14:00 **Panel Discussion**
Eu Jeong Ku (Seoul National University, Republic of Korea)
Hyo-Suk Ahn (The Catholic University of Korea, Republic of Korea)
Han-Mo Yang (Seoul National University, Republic of Korea)

Symposium 24 – KSCVI–KSoLA Joint Symposium

Sep 12(Sat) 13:00–14:00 | Room 2 (3F, Conrad Seoul)

Beyond Stenting: Contemporary Management of Residual Coronary Risk

CHAIRPERSONS : **Joon Hyung Doh** (Inje University, Republic of Korea)
Sang Yeub Lee (Chung-Ang University, Republic of Korea)

13:00–13:15	**Impact of lipid-lowering therapy on plaque stabilization and regression** **Jong Young Lee** (Hallym University, Republic of Korea)
13:15–13:30	**Coronary microvascular dysfunction after PCI** **Hyun Sung Joh** (Seoul National University, Republic of Korea)
13:30–13:45	**Discordance between coronary artery calcium and coronary stenosis** **Sanghoon Shin** (Ewha Womans University, Republic of Korea)
13:45–14:00	**Panel Discussion** **Yong Hyun Park** (Pusan National University, Republic of Korea) **Hyun-Woong Park** (Chungnam National University, Republic of Korea) **Jong Shin Woo** (Kyung Hee University, Republic of Korea) **Soo Young Lee** (Chung-Ang University, Republic of Korea)

Symposium 25 – KDA–KSoLA Joint Symposium

Sep 12(Sat) 13:00–14:00 | Room 3 (3F, Conrad Seoul)

Integrated Cardiometabolic Risk Management in Diabetes

CHAIRPERSONS : **Sung Rae Kim** (The Catholic University of Korea, Republic of Korea)
Hyuk-Sang Kwon (The Catholic University of Korea, Republic of Korea)

13:00–13:15	**Hypertriglyceridemia and remnant cholesterol in diabetes** **Sung Hee Choi** (Seoul National University, Republic of Korea)
13:15–13:30	**Arterial stiffness and vascular aging in diabetes** **Hack-Lyoung Kim** (Seoul National University, Republic of Korea)
13:30–13:45	**Coronary artery calcium and subclinical atherosclerosis in diabetes** **Janghoon Lee** (Kyungpook National University, Republic of Korea)
13:45–14:00	**Panel Discussion** **Joon Ho Moon** (Seoul National University, Republic of Korea) **Ki-Hyun Jeon** (Seoul National University, Republic of Korea) **A Ram Hong** (Chonnam National University, Republic of Korea)

Symposium 26

Sep 12(Sat) 14:10–15:40 | Room 1 (3F, Conrad Seoul)

Integrated Cardiovascular–kidney–metabolic Care: A Multidisciplinary Approach to CKM Syndrome

CHAIRPERSONS : Yong-Jae Kim (Kim's Neurology Clinic, Republic of Korea)
Sung Kee Ryu (Ewha Womans University, Republic of Korea)

14:10–14:30	**Cardiologist perspective: implementing novel cardiometabolic therapies in CVD** **Donna Shu-Han Lin** (Shin Kong Wu Ho-Su Memorial Hospital, Taiwan)
14:30–14:50	**Nephrologist's perspective: kidney protection strategies to improve cardio-kidney outcomes** **Jong Hyun Jhee** (Yonsei University, Republic of Korea)
14:50–15:10	**Endocrinologist perspective: optimizing combination therapy in complex CKM patients** **Jae-Seung Yun** (The Catholic University of Korea, Republic of Korea)
15:10–15:40	**Panel Discussion** **Jaeseok Kim** (Yonsei University, Republic of Korea) **Min Kyung Lee** (Hanyang University, Republic of Korea) **Min-Ho Lee** (Sahmyook Medical Center, Republic of Korea)

Symposium 27

Sep 12(Sat) 14:10–15:40 | Room 2 (3F, Conrad Seoul)

AI-driven Innovations in Atherosclerosis

CHAIRPERSONS : Yongseek Park (Kyung Hee University, Republic of Korea)
Hyun Jae Kang (Seoul National University, Republic of Korea)

14:10–14:28	**AI-CVD: opportunistic testing of coronary calcium and beyond** **Nathan D. Wong** (University of California, Irvine, USA)
14:28–14:46	**A+4DCT: angio enhanced dynamic CT for non-invasive functional diagnosis of coronary artery disease** **Joon Sang Lee** (Yonsei University, Republic of Korea)
14:46–15:04	**Application of AI to coronary angiography** **Si-Hyuck Kang** (Seoul National University, Republic of Korea)
15:04–15:22	**AI-based retinomics: cardiovascular risk prediction using retinal imaging** **Sahil Thakur** (Medical Affairs, Mediwhale Inc., Singapore)
15:22–15:40	**Panel Discussion** **Kyu-Yong Ko** (Inje University, Republic of Korea) **Hee-Dong Kim** (Soonchunhyang University, Republic of Korea) **Kwan Yong Lee** (The Catholic University of Korea, Republic of Korea)

Symposium 28

Sep 12(Sat) 14:10–15:40 | Room 3 (3F, Conrad Seoul)

Primary Prevention in Cardiovascular Disease: Beyond Traditional Risk Factors

CHAIRPERSONS : **Bon-kwon Koo** (Seoul National University, Republic of Korea)
Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea)

14:10–14:30	**Lipid management in primary prevention: who, when, and how aggressive?** **Yeoree Yang** (The Catholic University of Korea, Republic of Korea)
14:30–14:50	**Every minute counts: exercise snacks for better health and life's essential 8** **SoJung Lee** (Kyung Hee University, Republic of Korea)
14:50–15:10	**Advancing cardiovascular management through vaccination** **Jae Hyoung Park** (Korea University, Republic of Korea)
15:10–15:40	**Panel Discussion** **Mihye Seo** (Soonchunhyang University, Republic of Korea) **Hyun Ju You** (Seoul National University, Republic of Korea) **Jun Hwa Hong** (Eulji University, Republic of Korea)

Symposium 29 – KES-KSoLA Joint Symposium

Sep 12(Sat) 14:10–15:40 | Room 4 (5F, Conrad Seoul)

Lipid Metabolism Across Endocrine Disorders: From Mechanism to Clinical Practice

CHAIRPERSONS : **Sung Hee Choi** (Seoul National University, Republic of Korea)
Sun Wook Cho (Seoul National University, Republic of Korea)

14:10–14:30	**Diabetic dyslipidemia: targeting residual risk beyond LDL-C lowering** **Kyung-Soo Kim** (CHA University, Republic of Korea)
14:30–14:50	**Subclinical thyroid dysfunction: when should we treat for cardiovascular and metabolic health?** **Eu Jeong Ku** (Seoul National University, Republic of Korea)
14:50–15:10	**Atherogenic dyslipidemia in women: PCOS, menopause, and cardiovascular risk** **Jeong Rang Park** (Gyeongsang National University, Republic of Korea)
15:10–15:40	**Panel Discussion** **Dae-Young Kim** (Inha University, Republic of Korea) **Seo Young Sohn** (Inje University, Republic of Korea) **You-Bin Lee** (Sungkyunkwan University, Republic of Korea)

Symposium 30

Sep 12(Sat) 15:40–17:10 | Room 1 (3F, Conrad Seoul)

Neural and Genetic Control of Eating Behavior and Cardiometabolic Health

CHAIRPERSONS : Yang Ha Kim (Ewha Womans University, Republic of Korea)
Min-Jeong Shin (Korea University, Republic of Korea)

15:40–16:00 **Dietary and social determinants of the human microbiome: implications for metabolic health**
Soyoung Kwak (Kyungpook National University, Republic of Korea)

16:00–16:20 **Understanding gene–diet interactions in cardiometabolic health: genetic risk scores and their modification by diet in Korean and UK biobank cohorts**
Sunmin Park (Hoseo University, Republic of Korea)

16:20–16:40 **Interaction effects of genetic variants and dietary intake on the development of metabolic syndrome**
Dayeon Shin (Ewha Womans University, Republic of Korea)

16:40–17:10 **Panel Discussion**
Mogan Gim (Hankuk University of Foreign Studies, Republic of Korea)
Jeong-Min Kim (Seoul National University, Republic of Korea)
Seung-Hwan Lee (The Catholic University of Korea, Republic of Korea)

Symposium 31

Sep 12(Sat) 15:40–17:10 | Room 3 (3F, Conrad Seoul)

From Cells to Systems: Integrated Control of Cardiometabolic Health

CHAIRPERSONS : Goo Taeg Oh (Ewha Womans University, Republic of Korea)
Young Mi Park (Ewha Womans University, Republic of Korea)

15:40–16:00 **Non-inflammatory function of dendritic cell in the regulation of obesity-associated heart failure**
Kae Won Cho (Soonchunhyang University, Republic of Korea)

16:00–16:20 **Hepatic lipotoxicity and fibrotic remodeling in metabolic liver disease**
KyeongJin Kim (Inha University, Republic of Korea)

16:20–16:40 **From lipidomics to systems biology: decoding metabolic networks**
Guanghou Shui (Chinese Academy of Sciences, China)

16:40–17:10 **Panel Discussion**
Yoo-Wook Kwon (Seoul National University, Republic of Korea)
Hyue Mee Kim (Chung-Ang University, Republic of Korea)
Kyung-Sun Heo (Chungnam National University, Republic of Korea)

Symposium 32

Sep 12(Sat) 15:40–17:10 | Room 4 (5F, Conrad Seoul)

Environmental Cardiovascular Risk

CHAIRPERSONS : Sun Ha Jee (Yonsei University, Republic of Korea)
Oh-Yoen Kim (Dong-A University, Republic of Korea)

15:40–16:00 **Air pollution and cardiovascular disease**
Sera Kim (Korea Environment Institute, Republic of Korea)

16:00–16:20 **Microplastics and cardiovascular health: current epidemiological evidence and challenges**
Dong-Wook Lee (Seoul National University, Republic of Korea)

16:20–16:40 **Climate change and cardiometabolic disease**
Whanhee Lee (Korea University, Republic of Korea)

16:40–17:10 **Panel Discussion**
Si-Hyuck Kang (Seoul National University, Republic of Korea)
Jong Young Lee (Hallym University, Republic of Korea)
Jun Young Hong (Yonsei University, Republic of Korea)

Joint Symposia p. 219

IHA-KSoLA Joint Symposium

Sep 10(Thu) 14:30–16:00 | Room 3 (3F, Conrad Seoul)

Optimizing Lipid-Lowering Strategies: Facing Challenges in Asian High-Risk Patients

CHAIRPERSONS : Ick Mo Chung (Ewha Womans University, Republic of Korea)
Ade Meidian Ambari (University of Indonesia, Indonesia)

14:30–14:50 **The race to the bottom: intensive LDL-C lowering in very high-risk Asian patients**
Ade Meidian Ambari (University of Indonesia, Indonesia)

14:50–15:10 **Combination therapy in high-risk patients: early vs stepwise approach**
Han Na Jung (Hallym University, Republic of Korea)

15:10–15:30 **Practical approaches to statin intolerance: from clinical trials to real-world solutions**
Jung-Joon Cha (Korea University, Republic of Korea)

15:30–16:00 **Panel Discussion**
Kyung An Kim (The Catholic University of Korea, Republic of Korea)
Hyeonju Jeong (Hanyang University, Republic of Korea)
Hun Jee Choe (Hallym University, Republic of Korea)

JAS-KSoLA Joint Symposium

Sep 10(Thu) 16:10-17:40 | Room 2 (3F, Conrad Seoul)

Comprehensive Lipid Management in East Asians: Integrating LDL-C Optimization, Residual Risk, and Genetic Factors

CHAIRPERSONS : Sung Rae Kim (The Catholic University of Korea, Republic of Korea)
Jae Hyoung Park (Korea University, Republic of Korea)

16:10-16:28	**Do East Asians need lower statin intensity? ethnic differences in LDL-C response and safety** **Eun Jeong Cho** (Chung-Ang University, Republic of Korea)
16:28-16:46	**Why TG is important in Asians?** **Daisaku Masuda** (Rinku General Medical Center, Japan)
16:46-17:04	**Genetic stratification and treatment response in Korean severe hypertriglyceridemia: a prospective multicenter registry study** **Hun Jee Choe** (Hallym University, Republic of Korea)
17:04-17:22	**Chylomicronemia: past, present and future** **Hiroaki Okazaki** (Teikyo University, Japan)
17:22-17:40	**Panel Discussion** **Osung Kwon** (The Catholic University of Korea, Republic of Korea) **Gyu Chul Oh** (The Catholic University of Korea, Republic of Korea) **Dae Young Cheon** (Hallym University, Republic of Korea)

VAS-KSoLA Joint Symposium

Sep 11(Fri) 13:45-15:15 | Room 2 (3F, Conrad Seoul)

Residual Cardiovascular Risk Beyond LDL-C: The Emerging Role of Lipoprotein(a) in Asia

CHAIRPERSONS : Donghoon Choi (Yonsei University, Republic of Korea)
Kyung Woo Park (Seoul National University, Republic of Korea)

13:45-14:05	**Lipoprotein(a) in Korean patients: real-world data, cardiovascular risk, and clinical implications** **Sang Min Park** (Eulji University, Republic of Korea)
14:05-14:25	**Lipoprotein(a) and coronary atherosclerosis in Vietnam: real-world data and emerging cardiovascular risk** **Anh Tien Hoang** (Hue University, Vietnam)
14:25-14:45	**Measuring Lipoprotein(a) in clinical practice: assay challenges, standardization, and interpretation** **Sang-Guk Lee** (Yonsei University, Republic of Korea)
14:45-15:15	**Panel Discussion** **Jaehyun Bae** (Hallym University, Republic of Korea) **Heung Yong Jin** (Jeonbuk National University, Republic of Korea) **Jung-Kyu Han** (Seoul National University, Republic of Korea)

TSLA-KSoLA Joint Symposium

Sep 12(Sat) 08:50-10:20 | Room 2 (3F, Conrad Seoul)

Contemporary Management of Residual Cardiovascular Risk in Asia

CHAIRPERSONS : Sang-Hyun Kim (Seoul National University, Republic of Korea)
Chin-Chou Huang (Taipei Veterans General Hospital, Taiwan)

08:50-09:10	**The emerging role of ApoB and triglyceride-rich lipoproteins** **Sin Yung Woo** (Konyang University, Republic of Korea)
09:10-09:30	**Beyond LDL-C: targeting inflammation and lipoprotein(a) to address residual cardiovascular risk in Asia** **Ping-Yen Liu** (National Cheng Kung University, Taiwan)
09:30-09:50	**Cardiometabolic residual risk in Asia: diabetes, metabolic dysfunction, and vascular aging** **Minkwan Kim** (Yonsei University, Republic of Korea)
09:50-10:20	**Panel Discussion** **Young-Sang Lyu** (Chosun University, Republic of Korea) **Hong Seok Lim** (Ajou University, Republic of Korea) **Hansaem Jung** (Jeong Hansaem Internal Medicine Clinic, Republic of Korea)

MSA-KSoLA Joint Symposium

Sep 12(Sat) 08:50-10:20 | Room 4 (5F, Conrad Seoul)

From Diversity to Action: Ethnicity, Genetics, and Closing the Treatment Gap in Asian Dyslipidemia

CHAIRPERSONS : Sang Ho Jo (Hallym University, Republic of Korea)
Hack-Lyoung Kim (Seoul National University, Republic of Korea)

08:50-09:10	**Dyslipidemia across ethnicities in Malaysia: who is at risk, and who is being left behind?** **Nor Ashikin Md Sari** (University of Malaya, Malaysia)
09:10-09:30	**Genetic architecture of dyslipidemia and cardiovascular risk in East Asians: from monogenic variants to polygenic risk** **Soo Heon Kwak** (Seoul National University, Republic of Korea)
09:30-09:50	**Epidemiological trends of dyslipidemia in Korea and their implications for Asian populations** **Hokyou Lee** (Yonsei University, Republic of Korea)
09:50-10:20	**Panel Discussion** **Ji Hyun Kim** (The Catholic University of Korea, Republic of Korea) **Joon Ho Moon** (Seoul National University, Republic of Korea) **Junghwan Park** (Hanyang University, Republic of Korea)

EAS-KSoLA Joint Symposium

Sep 12(Sat) 15:40-17:10 | Room 2 (3F, Conrad Seoul)

Bridging Europe and Korea in Dyslipidemia Management: Guidelines, Gaps, and Future Directions

CHAIRPERSONS : **Cheol-Young Park** (Sungkyunkwan University, Republic of Korea)
Borge G. Nordestgaard (University of Copenhagen, Denmark)

15:40-16:00 **ESC/EAS dyslipidemia guidelines: recent updates and evidence base**
Meral Kayikcioglu (Ege University, Türkiye)

16:00-16:20 **Korean perspectives on dyslipidemia guidelines: recent update and challenges**
Jaehoon Chung (Gachon University, Republic of Korea)

16:20-16:40 **Translating guidelines into practice**
Wonjin Kim (CHA University, Republic of Korea)

16:40-17:10 **Panel Discussion**
Seon Mee Kang (Soonchunhyang University, Republic of Korea)
Min Kyung Lee (Hanyang University, Republic of Korea)
Ji Eun Lee (Korea University, Republic of Korea)

Satellite Symposia p. 247

Breakfast Symposium 1 (K+E)

Sep 11(Fri) 07:30-08:00 | Room 1 (3F, Conrad Seoul)

Amgen, Hanlim Pharm

CHAIRPERSONS : **Myung-gon Kim** (Catholic Kwandong University, Republic of Korea)
Hee-Yeol Kim (Incheon Sejong Hospital, Republic of Korea)

07:30-07:45 **Evolving LDL-C management: guidelines and the role of evolocumab**
Sang Min Park (Eulji University, Republic of Korea)

07:45-08:00 **Is LDL-C goal attainment enough? pitavastatin/fenofibrate in mixed dyslipidemia**
Jae Hyun Bae (Seoul National University, Republic of Korea)

Breakfast Symposium 2

Sep 11(Fri) 07:30-08:00 | Room 2 (3F, Conrad Seoul)

Jeil Pharmaceutical

CHAIRPERSON : **Jae Hyoung Park** (Korea University, Republic of Korea)

07:30-07:50 **Bridging guidelines and practice: strategic implementation of combination therapy in lipid management**
Inki Moon (Soonchunhyang University, Republic of Korea)

Breakfast Symposium 3

Sep 11(Fri) 07:30-08:00 | Room 3 (3F, Conrad Seoul)

Yuhan

CHAIRPERSON : Jeong-Taek Woo (Kyung Hee University, Republic of Korea)

07:30-07:50 **Paving the road for optimal LDL-C management: new evidence on intensive LDL-C targeting from the Ez-PAVE trial**
Yong-Joon Lee (Yonsei University, Republic of Korea)

Breakfast Symposium 4

Sep 11(Fri) 07:30-08:00 | Room 4 (5F, Conrad Seoul)

Dong-A ST, HK inno.N

CHAIRPERSONS : Byung Ryul Cho (Kangwon National University, Republic of Korea)
Bong-Ki Lee (Kangwon National University, Republic of Korea)

07:30-07:45 **The role of DPP-4 inhibitors in Korean diabetes care: focusing on K-evidence**
Yun Kyung Cho (University of Ulsan, Republic of Korea)

07:45-08:00 **The smaller the better: rovazet**
Jeehoon Kang (Seoul National University, Republic of Korea)

Breakfast Symposium 5

Sep 12(Sat) 07:50-08:20 | Room 1 (3F, Conrad Seoul)

Viatris

CHAIRPERSON : Myung Ho Jeong (Gwangju Veterans Hospital, Republic of Korea)

07:50-08:10 **Statin therapy strategies for newly diagnosed dyslipidemia patients in clinical practice**
Michal Vrablik (Charles University, Czech Republic)

Breakfast Symposium 6

Sep 12(Sat) 07:50-08:20 | Room 2 (3F, Conrad Seoul)

Chong Kun Dang, GC Biopharma

CHAIRPERSONS : Kwang-Won Kim (Gachon University, Republic of Korea)
Kwang Kon Koh (Gachon University, Republic of Korea)

07:50-08:05 **Low-dose atorvastatin: addressing an unmet clinical need**
Sehoon Park (Seoul National University, Republic of Korea)

08:05-08:20 **The role of low-dose fenofibrate in the management of dyslipidemia**
Jaeseok Kim (Yonsei University, Republic of Korea)

Breakfast Symposium 7

Sep 12(Sat) 07:50-08:20 | Room 3 (3F, Conrad Seoul)

Daiichi-Sankyo

CHAIRPERSON : Sang-Wook Lim (CHA University, Republic of Korea)

07:50-08:10 **A new oral LDL-C lowering agent with proven CV benefit: bempedoic acid**
Chang Hee Jung (University of Ulsan, Republic of Korea)

Breakfast Symposium 8

Sep 12(Sat) 07:50-08:26 | Room 4 (5F, Conrad Seoul)

Organon, GSK, Novartis

CHAIRPERSONS : Shung Chull Chae (Kyungpook National University, Republic of Korea)
Soon Jun Hong (Korea University, Republic of Korea)

07:50-08:02 **Integrated lipid management across the CKM spectrum: from evidence to practice**
Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

08:02-08:14 **Making vaccination routine for patients with cardiovascular disease**
Timothy C. Tan (Western Sydney University, Australia)

08:14-08:26 **Why is LDL-C control still insufficient? finding the breakthrough with leqvio cases in the aLLT era**
Jung-Joon Cha (Korea University, Republic of Korea)

Luncheon Symposium 1 (K)

Sep 11(Fri) 11:45-12:15 | Room 1 (3F, Conrad Seoul)

Organon

CHAIRPERSON : Young-Bae Park (Seoul National University, Republic of Korea)

11:45-12:00 **Start early: upfront combination therapy for faster LDL-C goal attainment in the CKM era**
Soo-Jin Kim (Kosin University, Republic of Korea)

12:00-12:15 **Start strong: elevating LDL-C management in ASCVD with high-dose atorvastatin plus ezetimibe**
Jeehoon Kang (Seoul National University, Republic of Korea)

Luncheon Symposium 2

Sep 11(Fri) 11:45–12:15 | Room 2 (3F, Conrad Seoul)

Hanmi Pharmaceutical

CHAIRPERSONS : Kyong Soo Park (Konkuk University, Republic of Korea)
Myung A Kim (Jeju National University, Republic of Korea)

11:45–12:00 **Beyond the statin, ROSUZET; start early, switch now**
Gyu Chul Oh (The Catholic University of Korea, Republic of Korea)

12:00–12:15 **Be the standard ROSUZET; evidence-driven lipid management**
Wonjin Kim (CHA University, Republic of Korea)

Luncheon Symposium 3

Sep 11(Fri) 11:45–12:15 | Room 3 (3F, Conrad Seoul)

JW Pharmaceutical

CHAIRPERSON : Hyo-Soo Kim (Seoul National University, Republic of Korea)

11:45–12:00 **Integrated management of hypertension and dyslipidemia for cardiovascular disease prevention**
Minjeong Kim (Ewha Womans University, Republic of Korea)

12:00–12:15 **Management of inadequately controlled patients with diabetes mellitus**
Hae Kyung Kim (Yonsei University, Republic of Korea)

Luncheon Symposium 4 (K)

Sep 11(Fri) 11:45–12:15 | Room 4 (5F, Conrad Seoul)

Novartis

CHAIRPERSON : Moon-Kyu Lee (Eulji University, Republic of Korea)

11:45–12:00 **Protecting the vulnerable period: early and sustained LDL-C control with inclisiran**
In Tae Jin (Yonsei University, Republic of Korea)

12:00–12:15 **Proven in the real world: consistent LDL-C control with inclisiran**
Seok Oh (Chonnam National University, Republic of Korea)

Luncheon Symposium 5

Sep 11(Fri) 11:45–12:15 | Room 5 (6F, Conrad Seoul)

Daewoong

CHAIRPERSON : Hyun Ho Shin (Asan Chungmu Hospital, Republic of Korea)

11:45–12:00 **Clinical updates in dyslipidemia: positioning baroezet for optimal patient care**
Jun Sung Moon (Yeungnam University, Republic of Korea)

12:00–12:15 **Strategies for high-risk dyslipidemia patients focusing on ASCVD**
Sang Yoon Lee (Sungkyunkwan University, Republic of Korea)

Luncheon Symposium 6

Sep 12(Sat) 12:00–12:30 | Room 1 (3F, Conrad Seoul)

GSK

CHAIRPERSON : Sang Hong Baek (The Catholic University of Korea, Republic of Korea)

12:00–12:20 **Embedding shingles vaccination into everyday cardiology practice**
Timothy C. Tan (Western Sydney University, Australia)

Luncheon Symposium 7

Sep 12(Sat) 12:00–12:30 | Room 2 (3F, Conrad Seoul)

Hanmi Pharmaceutical

CHAIRPERSON : Hong Seog Seo (Korea University, Republic of Korea)

12:00–12:15 **New paradigm of initial therapy for hypertension: which patients benefit from amoprel?**
Young-Sang Lyu (Chosun University, Republic of Korea)

12:15–12:30 **Combination treatment in hypertension: why is losartan-based therapy effective?**
Hoyoun Won (Chung-Ang University, Republic of Korea)

Luncheon Symposium 8

Sep 12(Sat) 12:00–12:30 | Room 3 (3F, Conrad Seoul)

JW Pharmaceutical

CHAIRPERSON : Hak Chul Jang (Seoul National University, Republic of Korea)

12:00–12:15 **Cutting-edge care of pitavastatin with ezetimibe combination therapy**
Yeoree Yang (The Catholic University of Korea, Republic of Korea)

12:15–12:30 **Beyond LDL-C reduction comprehensive lipid control in DM patients**
Jun Hwa Hong (Eulji University, Republic of Korea)

Oral Presentation p. 303

Oral Presentation 1

Sep 10(Thu) 14:30–16:00 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Hyun Kook (Chonnam National University, Republic of Korea)
Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

OP1-1 Improved survival and in vivo cholesterol-lowering efficacy of microencapsulated Lactobacillus plantarum LAB12

Ismail M. Fareez[1,2*], Siong-Meng Lim[2], Fei Tieng Lim[2], Kalavathy Ramasamy[2]

[1]School of Biology, Faculty of Applied Sciences, Universiti Teknologi MARA 40450 Shah Alam, Selangor Darul Ehsan, Malaysia, [2]Collaborative Drug Discovery Research (CDDR) Group, Faculty of Pharmacy, Universiti Teknologi MARA (UiTM) Cawangan Selangor, Kampus Puncak Alam, 42300 Bandar Puncak Alam, Selangor Darul Ehsan, Malaysia

OP1-2 Lipid accumulation product and visceral adiposity indices as markers of hyperuricemia and gout: a meta-analysis of 575,000 patients

Khalid Sarhan*, Ibrahim Serag

Department of Internal Medicine, Mansoura University Hospitals, Mansoura, Egypt

OP1-3 Distinct vascular targets of metabolic and kidney dysfunction in adults without advanced

Hyo-In Choi*

Cardiology, Kangbuk Samsung Hospital, Republic of Korea

OP1-4 A randomized, double-blind, placebo-controlled, crossover trial to comparatively evaluate the effects of magnesium hydride (MgH2) on endothelial function: a pilot study

Yasuhiro Endo[1*], Makoto Ayaori[2], Kei Sasaki[3], Manami Teramoto[3], Junko Arakawa[3], Katsunori Ikewaki[3]

[1]Faculty of Medical Technology, Niigata University of Pharmacy and Medical and Life Sciences, Japan, [2]Department of Cardiology, Tokorozawa Heart Center, Japan, [3]Division of Anti-aging and Vascular Medicine, National Defense Medical College, Japan

OP1-5 Association of dietary sodium-to-potassium ratio with triglyceride-glucose index and metabolic syndrome risk in Korean adults: using data from the 2022-2023 Korea National Health and Nutrition Examination Survey

Jisu Lee*, Sunhye Shin

Department of Food and Nutrition, Seoul Women's University, Republic of Korea

OP1-6 A newly developed grain-based cereal product for glycemic spike management: effects on glycemic index, glycemic load, and postprandial glucose responses

Soyeon Park[1*], Dabin Lee[1], Minji Kim[1], Soyoon Cha[1], Seohyeon Lee[1], Jimin Kim[1], Jihyun Park[1,2], Seulgi Lee[3], Oh Yoen Kim[1,4,5]

[1]Department of Health Sciences, Clinical Nutrition major, Graduate School of Dong-A University, Republic of Korea, [2]Department of Cancer AI and Digital Health, Graduate School of Cancer Science and Policy, National Cancer Center, Republic of Korea, [3]Wellnable Co., Ltd, Wellnable Co., Ltd, Republic of Korea, [4]Center for Food & Bio Innovation, DAU G-LAMP Project Group, Dong-A University, Republic of Korea, [5]Department of Food Science and Nutrition, Dong-A University, Republic of Korea

OP1-7 Nutraceutical modulation of the oral-vascular axis: emerging therapeutic strategies for periodontitis-associated atherosclerosis

Viza Atiq Umar*

Clinical Clerkship Student, Universitas Islam Sultan Agung, Indonesia

Oral Presentation 2

Sep 11(Fri) 08:30-10:00 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Cheol-Ung Choi (Korea University, Republic of Korea)
Jung-Kyu Han (Seoul National University, Republic of Korea)

OP2-1 Renal lipidomic profiling reveals boerhaavia diffusa extract ameliorates saturated fatty acid lipotoxicity and mitochondrial decay independent of systemic adiposity

Lee On Ying*

The Health Technology and Informatics, The Hong Kong Polytechnic University, China

OP2-2 Extrahepatic bile acid synthesis reprograms multiorgan cholesterol flux in cardiovascular disease

Hosung Bae[1,2*]

[1]Biological Chemistry, University of California, Irvine, United States, [2]Endocrinology and Metabolism, Internal Medicine, University of Iowa, United States

OP2-3 Prdx3 deficiency triggers cGAS-STING signaling pathway induced vascular smooth muscle cell senescence and phenotypic switching in abdominal aortic aneurysm

Moajury-Jung[2*], Seong keun Sonn[1], Goo Taeg Oh[1,2]

[1]Heart-Immune-Brain Network Research Center, Department of Life Sciences, Ewha Womans University, Republic of Korea, [2]Division of Life Science, Ewha Womans Universit, Republic of Korea

OP2-4 Sphingosine-1-phosphate receptor 1 deletion in smooth muscle cells protects against atherosclerosis in apoE-deficient mice by reducing lipid droplet formation

Inbar Habaz[1,2*], Narmadaa Thyagarajan[1,2], Alexander Qian[1,2], Wei Wang[1,2], Bernardo Trigatti[1,2]

[1]Thrombosis and Atherosclerosis Research Institute, Hamilton Health Sciences, Canada, [2]Department of Biochemistry and Biomedical Sciences, McMaster University, Canada

OP2-5 Senolytic therapy attenuates atherosclerosis and metabolic dysfunction by targeting cellular senescence in western diet-fed Ldlr KO mice

Hye Rang Park[1,3*], Young Hoon Seo[1,3], Jimin Lee[1,3], Haeun Jang[1], Yongmin Lee[1], Daeun Kim[1,3], Mi-Ran Lee[4], Min ji Cho[2], Jong-Gil Park[1,3]

[1]Biotherapeutics Translational Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, [2]Genome Editing Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, [3]Department of Bioscience, KRIBB School of Bioscience, Korea University of Science and Technology (UST), Republic of Korea, [4]Department of Biomedical Laboratory Science, Jungwon University, Republic of Korea

OP2-6 The role of LGI3 in adipocytes and macrophages in atherosclerosis

Do Hee Yu*, Jin Jing, Goo Taeg Oh

Heart-Immune-Brain Network Research Center, Department of Life Sciences, Ewha Womans University, Republic of Korea

OP2-7 Cyclic induction of OSKM expression mitigates atherosclerosis through vascular anti-inflammatory remodeling independent of systemic metabolic effects

Young Hoon Seo[1,3*], Hye Rang Park[1,3], Haeun Jang[1], Yongmin Lee[1], Jimin Lee[1,3], Daeun Kim[1,3], Mi-Ran Lee[4], Min Ji Cho[2], Jong-Gil Park[1,3]

[1]Biotherapeutics Translational Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, [2]Genome Editing Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, [3]KRIBB School of Bioscience, Korea University of Science and Technology (UST), Republic of Korea, [4]Department of Biomedical Laboratory Science, Jungwon University,Republic of Korea

OP2-8 Extracellular vesicles produced by M2-polarized macrophages drive atherosclerosis regression by reprograming Ly-6Chi monocytes & enhancing efferocytosis

Robert L. Raffai*, Ngan K. Vu, Tuan Anh Phu, Martin Ng, Alex Gao, Rebecca Kim, Angela Win, Akiko Searles

Vascular and Endovascular Surgery, University of California, San Francisco, United States

Oral Presentation 3

Sep 12(Sat) 08:50-10:20 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Hyeong Kyu Park (Soonchunhyang University, Republic of Korea)
Han-Mo Yang (Seoul National University, Republic of Korea)

OP3-1 Residual risk of remnant cholesterol in patients treated to LDL-C goal: a landmark analysis of the RACING trial

Sungyeon Park[1*], Jung-Hee Lee[1], Sung-Jin Hong[2], Byeong-Keuk Kim[2], Sung Gyun Ahn[1]

[1]Cardiology, Yonsei University Wonju College of Medicine, Republic of Korea, [2]Cardiology, Yonsei University College of Medicine, Republic of Korea

OP3-2 Lower cardiovascular event risk with bempedoic acid versus ezetimibe in statin-intolerant atherosclerotic cardiovascular disease (ASCVD): a 3,540-patient TriNetX analysis

Ahmed Elaraby[1*], Ahmed Fouad[1], Ahmed Hassan[2], Ali Bakr[1]

[1]Medicine, Faculty of Medicine, Al-Azhar University, Cairo, Egypt, [2]Medicine, Faculty of Medicine, Al-Azhar University, Asyut, Egypt

OP3-3 Age-dependent association of atherogenic dyslipidemia with incident cardiovascular disease: a nationwide Korean cohort study

Ye Seul Yang[1,2*], Kyuho Kim[3], Seung-Hyun Ko[3], Kyuna Lee[4], Bongseong Kim[5], Kyungdo Han[4], Min Kyong Moon[6]

[1]Department of Medicine, Seoul National University College of Medicine, Republic of Korea, [2]Department of Internal Medicine, Seoul National University Bundang Hospital, Seoul National University College of Medicine, Republic of Korea, [3]Division of Endocrinology and Metabolism, Department of Internal Medicine, St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Republic of Korea, [4]Department of Statistics and Actuarial Science, Soongsil University, Republic of Korea, [5]Department of AI-Bio, Graduate School of AI Soongsil University, Republic of Korea, [6]Department of Internal Medicine, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul National University College of Medicine, Republic of Korea

OP3-4 Deep learning analysis of BMI, metabolic risk factors, and sex hormones in U.S. women: a NHANES study

Saheed Oladele Amusat[1,2*], Saheed[1,2]

[1]Biosciences, School of Science and Technology, Nottingham Trent University, United Kingdom, [2]Chemical Pathology Department, The Directorate of Laboratory Medicine, Beaumont Hospital, Dublin, Republic of Ireland, Ireland

OP3-5 Prevalence of high inflammatory risk in atherosclerotic cardiovascular disease with and without chronic kidney disease: findings from POSEIDON - a global cross-sectional prevalence study

Hong-Ki Jeon[1*], Ann Marie Navar[2], Ling Bai[3], Jesper Falkesgaard Højen[4], Søren Lund Kristensen[4], Jimmi Mathisen[4], Hongye Ren[4], Fabrice Ivanes[4], Carolyn S P Lam[6], Ulf Landmesser[7], Kelvin Lee[8], Giovanna Liuzzo[9], Lilia Nigro Maia[10], Adam J Nelson[12], Amritanshu Shekhar Pandey[13], Pablo E Pergola[14], Kenichi Tsujita[15], Nikolaus Marx[16]

[1]Division of Cardiology, Severance Cardiovascular Hospital, Yonsei University, Republic of Korea, [2]Departments of Internal Medicine and Population and Data Sciences, UT Southwestern Medical Center, Dallas, TX, USA, United States, [3]Department of Cardiovascular Medicine, The First Affiliated Hospital of Xi'an Jiaotong University, China, [4]Novo Nordisk A/S, Søborg, Denmark, [5]Service de Cardiologie, Centre Hospitalier Universitaire de Tours, Université de Tours, France, [6]Department of Cardiology, National Heart Centre Singapore, Singapore, [7]Department of Cardiology, Angiology and Intensive Care Medicine, Deutsches Herzzentrum der Charité (DHZC), Charité - Universitätsmedizin Berlin, Germany, [8]Diabetes, Metabolism & Inflammation, Joseph Bank Laboratories, University of Lincoln, Lincoln Heart Centre, United Lincolnshire Teaching Hospitals NHS Trust, United Kingdom, [9]Department of Cardiovascular Medicine, Fondazione Policlinico Universitario A. Gemelli IRCCS, Catholic University School of Medicine, Italy, [11]Centro Integrado de Pesquisas, Hospital de Base, Sao Jose Do Rio Preto, Brazil, [12]Monash Victorian Heart Institute, Department of Cardiology, Monash University, Royal Adelaide Hospital, University of Adelaide, Australia, [13]Cambridge Cardiac Care, Department of Medicine, Cambridge Cardiac Care, McMaster University, Canada, [14]Nephrology, Renal Associates, PA, San Antonio, TX, USA, United States, [15]Department of Cardiovascular Medicine, Graduate School of Medical Sciences, Kumamoto University, Japan, [16]Department of Internal Medicine I - Cardiology, University Hospital Aachen (RWTH Aachen), Germany

OP3-6 Real-world efficacy and tolerability of inclisiran in Malaysian patients with atherosclerotic cardiovascular disease: a single-centre experience

Kim Fong Ng[1*], Khai Ling Koh[1], Wan Tying Soh[2], Azreen Natasha Binti Azmi[2], Shiau Chin Chong[3]

[1]Cardiology, KPJ Kluang Specialist Hospital, Malaysia, [2]Pharmacy, KPJ Kluang Specialist Hospital, Malaysia, [3]Pharmacy, Hospital Sultan Ismail, Malaysia

OP3-7 A Korean multidimensional social determinants of health index identifies individuals at elevated risk of severe coronary artery calcification: evidence from the CMERC cohort

Jooeun Jeon[1,2*], Hokyou Lee[2,3], Hyoeun An[3], Hyeok-Hee Lee[4], Jee-Seon Shim[2,3], Hansol Choi[2,3], Kyoung Hwa Ha[2,3], Hae Won Chung[5], Ji-Hye Kim[5], Yoosik Youm[6], Sungha Park[7], Dae Jung Kim[8], Hyeon Chang Kim[2,3]

[1]Institute of Medical and Convergence, Ewha Womans University Mokdong Hospital, Republic of Korea, [2]Yonsei Institute of Digital Health, Yonsei University, Republic of Korea, [3]Department of Preventive Medicine, Yonsei University College of Medicine, Republic of Korea, [4]Richard A. and Susan F. Smith Center for Outcomes Research in Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA, United States, [5]Department of health promotion, Severance health check-up, Yonsei University Health System, Republic of Korea, [6]Department of Sociology, Yonsei University College of Social Sciences, Republic of Korea, [7]Division of Cardiology, Severance Cardiovascular Hospital and Cardiovascular Research Institute, Yonsei University College of Medicine, Republic of Korea, [8]Department of Endocrinology and Metabolism, Ajou University School of Medicine, Republic of Korea

OP3-8 Efficacy and safety of enlicitide decanoate, an oral macrocyclic peptide inhibitor of PCSK9, compared with bempedoic acid, ezetimibe, or bempedoic acid co-administered with ezetimibe in statin-treated adults with hypercholesterolemia: phase 3 CORALreef AddOn trial

Catapano AL[1,2], Mikhailova E[3], Navar AM[4], Banka P[5], Kordahi A[5], Corral P[6], Saxena M[7], Steg PG[8], Verma S[9], Mendizabal G[10], Zhu P[11], Ballantyne CM[12], Maria Carolina Emilia Villanueva[13*] on behalf of the authors

[1]University of Milan, Milan, Italy, [2]IRCCS Multimedica, Milan, Italy, [3]MSD (UK) Limited, London, United Kingdom, [4]University of Texas Southwestern Medical Center, Dallas, United States, [5]Merck & Co., Inc., Rahway, NJ, United States, [6]FASTA University, Mar del Plata, Argentina, [7]Barts NIHR Biomedical Research Centre, Queen Mary University of London, United Kingdom, [8]Université Paris-Cité, Assistance Publique-Hôpitaux de Paris, INSERM Unité 1148, Paris, France, [9]St. Michael's Hospital, University of Toronto, Toronto, Canada, [10]MSD Argentina, Buenos Aires, Argentina, [11]MSD China, Shanghai, China, [12]Texas Heart Institute at Baylor College of Medicine, Houston, Texas, United States, [13]MSD Philippines, Manila, Philippines

Moderated Poster Presentation p. 323

Moderated Poster Presentation 1

Sep 11(Fri) 12:45-13:45 | Studio 5 (6F, Conrad Seoul)

MODERATOR : Jun Namkung (Yonsei University, Republic of Korea)

MPP1-1 Comparative lipid profiling of celastrol-treated atherosclerotic ApoE-/- mice on a high-fat diet

Nurin Yasmin Mohd Khairudin[1,2*], Vimala R.M.T Balasubramaniam[3], Suhaila Abd Muid[4], Nurdiyana Nasrudin[5], Nasibah Azme[6,7]

[1]Laboratory Animal Care Unit (LACU), Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [2]Institute of Medical Molecular Biotechnology, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [3]Nutrition, Metabolism & Cardiovascular Research Centre, Institute for Medical Research, Malaysia, [4]Department of Biochemistry and Molecular Medicine, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [5]Department of Pediatrics, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [6]Department of Physiology, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [7]Department of Medical Education, Faculty of Medicine, Universiti Teknologi MARA, Malaysia

MPP1-2 MMP11 promotes endothelial senescence and atherosclerosis through proteolytic cleavage of FLRT2

Hyun Jung Hwang[1,2,3*], Jae-Seon Lee[1,2,3]

[1]Research Center for Controlling Intercellular Communication, College of Medicine, Inha University, Incheon, Republic of Korea, [2]Department of Molecular Medicine, College of Medicine, Inha University, Incheon, Republic of Korea, [3]Program in Biomedical Science and Engineering, Inha University, Incheon, Republic of Korea

MPP1-3 LGI3 as a novel therapeutic target in atherosclerosis: regulation of macrophage inflammation and plaque progression

Jing Jin*, Goo Taeg Oh

Life Science, Heart-Immune-Brain Network Research Center, Ewha Womans University, Republic of Korea

MPP1-4 Involvement of vimentin in oxLDL-induced microparticle release from macrophages

Soonhyeong Oh*, Wonkyoung Cho, Young Mi Park

Department of Molecular Medicine, College of Medicine, Ewha Womans University, Republic of Korea

MPP1-5 Aging rewires the vascular-wall regulatory landscape to redirect diet-induced atherosclerosis

Ju Young Kim[1,2*], Hyeonmin Gil[1], Jong Kyoung Kim[1]

[1]Department of Life Sciences, Pohang University of Science and Technology (POSTECH), Republic of Korea, [2]Biomedical Research Institute, Kyungpook National University Hospital, Republic of Korea

MPP1-6 A novel cyclodextrin derivative modulates macrophage lipid accumulation and inflammatory responses relevant to atherosclerosis

Soobin Chang[1*], Heegon Kim[2], Yujin Ko[1], ChaeYoung Kim[1], Shinhee Park[1], Sangeun Park[1], Kyuseong Park[1], Jae-hoon Choi[1]

[1]Department of Life Science, Hanyang University, Republic of Korea, [2]Renatus Co., Ltd., Republic of Korea

Moderated Poster Presentation 2

Sep 11(Fri) 12:45-13:45 | Studio 6 (6F, Conrad Seoul)

MODERATOR : Jaehoon Chung (Gachon University, Republic of Korea)

MPP2-1 Deciphering the differential impact of ART classes on vascular morphology and lipid profiles in HIV-positive patients with coronary artery disease

Nuriiat Efendieva*, Oleg Petrovich Shevchenko

Cardiology, Pirogov Russian National Research Medical University, Russian Federation

MPP2-2 Autophagy-based restoration of glucosylceramide catabolism in Gaucher disease

Minji Kim[1*], Eun Nam Choi[1], Yong Tae Kwon[2], Joo-Won Park[1]

[1]Department of Biochemistry, College of Medicine, Ewha Womans University, Republic of Korea, [2]Cellular Degradation Biology Center and Department of Biomedical Sciences, College of Medicine, Seoul National University, Republic of Korea

MPP2-3 Loss of PCSK9 enhances ANP expression to attenuate post-MI cardiac inflammation

Na Hyeon Yoon*, Goo Taeg Oh

Life Science, Ewha Womans University, Republic of Korea

MPP2-4 BMI-mediated inflammatory pathways and miR-92a-3p dysregulation: novel insights into cardiovascular risk stratification

Fitri Bin Kahar[1*], Abd-Malek Fatin-Syazwani[1], Nurul Izzati Abdullah[1], Fitri Kahar[1], Amrina Mohamad Amin[1], Sabariah Md Noor[1], Md Parvez Eusof Izzudin[2], Shariff-Ghazali Sazlina[2], Norshariza Nordin[3]

[1]Department of Pathology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia, [2]Family Medicine Clinic, Hospital Sultan Abdul Aziz Shah, Persiaran Mardi, Universiti Putra Malaysia, 43400, Serdang, Selangor, Malaysia, [3]Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia

MPP2-5 Whole-body 18F-FDG PET demonstrates coordinated splenic and arterial inflammatory activation in acute ischemic stroke

Sue Young Ha*, Jiyeon Ha, Jeong-Min Kim

Department of Neurology, Seoul National University Hospital, Seoul National University College of Medicine, Republic of Korea

MPP2-6 Revealing the hidden signals: prognostic impact of pericoronary fat attenuation index (FAI) in major adverse cardiovascular events (MACE) and coronary plaque instability - a systematic review and meta-analysis

Muhamad Fajri Addai*, Elen, Celly A. Atmadikoesoemah, Bambang Widyantoro

Cardiology and Vascular Medicine, National Cardiovascular Center Harapan Kita - Faculty of Medicine, Universitas Indonesia, Indonesia

MPP2-7 Circulating PD-L1+CD10- immature neutrophils and integrated immunometabolic signatures associated with diabetic kidney disease severity in type 2 diabetes

Junseo Kim[1,2,5*], Seong Eun Lee[1,5], Na Rae Choi[1], Jiyeon Yoon[1,2,5], Zhang Jing[1,2,5], Junyoung O. Park[4], Da Hyun Kang[3], Bon Jeong Ku[1,3], Yea Eun Kang[1,3,5]

[1]Research Center for Endocrine and Metabolic Diseases, Integrated Disease Research Institute, Chungnam National University Hospital & College of Medicine, Republic of Korea, [2]Department of Medical Science, Chungnam National University College of Medicine, Republic of Korea, [3]Department of Internal Medicine, Chungnam National University Hospital & College of Medicine, Republic of Korea, [4]Department of Chemical and Biomolecular Engineering, University of California, Los Angeles, United States, [5]System Network Inflammation Control Research Center, Chungnam National University Hospital & College of Medicine, Republic of Korea

Moderated Poster Presentation 3

Sep 11(Fri) 12:45-13:45 | Studio 7 (6F, Conrad Seoul)

MODERATOR : Mi-Hyang Jung (The Catholic University of Korea, Republic of Korea)

MPP3-1 Comparative cardiovascular risk in monogenic familial hypercholesterolemia versus polygenic hypercholesterolemia: a meta-analysis of long-term cohorts

Moaz Abouelmagd*, Amr Elrosasy, Mohammed Fathi Abduljalil

Kasr Alainy Faculty of medicine, Cairo University, Egypt

MPP3-2 Lysosomal acid lipase deficiency misdiagnosed as familial hypercholesterolaemia: a family case series with a novel homozygous LIPA variant and dramatic response to enzyme replacement therapy

Koukab AL Farsi[1*], Prof Khalid AL Rassadi[2]

[1]Clinical Biochemistry/Lipid Clinic, Department of Clinical Biochemistry, Sultan Qaboos University Hospital, Muscat, Sultanate of Oman, Oman, [2]clinical Biochemistry, College of Medicine at Sultan Qaboos University, Oman

MPP3-3 Effect of roxadustat on lipid profile in adults with chronic kidney disease: a systematic review and meta-analysis of randomized controlled trials

Ammar Yasser Ali*, Mohamed Rizk, Ahmed R. A. Moustafa, Rashad G. Mohamed, Nada Mahmoud Abdelkarim, Radwa M. Abdelsattar, Mohamed A. Wafa

Faculty of Medicine, Mansoura University, Egypt

MPP3-4 Real-world hsCRP awareness and clinical practice patterns in ASCVD, CKD, and heart failure: South Korea findings from the FLARE (Focus on Levels of Awareness and perceptions REgarding hs-CRP in identifying SI in ASCVD, CKD and HF management) study

Kyungsoo Won[1*], Jungs-Sun Kim[2]

[1]Medical Affairs, Novo Nordisk Korea, Republic of Korea, [2]Division of Cardiology, Severance Cardiovascular Hospital, Yonsei University College of Medicine, Republic of Korea

MPP3-5 Does early PCSK9 inhibition improve outcomes after acute ischemic stroke? a systematic review and meta-analysis

Amir Hegazi*, Rashad Mohamed, Hussien Osama

Faculty of Medicine, Mansoura University, Egypt

MPP3-6 Effect of alirocumab/evolocumab on lipid levels and MACEs after PCI for ACS: a meta-analysis of 3474 patients

Khalid Sarhan*

Department of Internal Medicine, Mansoura University Hospitals, Egypt

MPP3-7 Clinical impact of imaging-guided percutaneous coronary intervention in heavily calcified coronary lesions: a systematic review and meta-analysis of randomized controlled trials

Ahmed Adel[1*], Nourhan Hatem[2], Basant Ahmed Mahmoud[3], Abdalrahman Salah Shehata[6], Amr Khalid Abdallah[5], Alaa Eldeeb[8], Shafaq Jabeen[7], Ahmed Kertam[4]

[1]Faculty of Medicine, Zagazig University, Zagazig, Egypt, [2]Faculty of Medicine, Suez canal University, Ismailia, Egypt, [3]October 6 University, Faculty of Medicine, Egypt, [4]Tu Lab for Diagnostic Research, Yale School of Medicine, New Haven, CT, Tompkin's East 2, New Haven, CT 06510, United States, [5]Faculty of Medicine, Menofia University, Menofia, Egypt, [6]Faculty of Medicine, Qena University, Qena, Egypt, [7]Karachi Medical and Dental College, Pakistan, [8]Faculty of Medicine, Alexandria University, Egypt

Moderated Poster Presentation 4

Sep 12(Sat) 13:00-14:00 | Studio 5 (6F, Conrad Seoul)

MODERATOR : Su Myung Jung (Sungkyunkwan University, Republic of Korea)

MPP4-1 Unravelling the divergent pathological and transcriptional profile of lipotoxic renal tubular injury and lipoprotein glomerulopathy in APOE-deficient and humanised hAPOE4 knock-in mice

Martin Ho Yin Yeung[1,2,3*], Ching In Chung[1], Angela Zaneta Chan[2]

[1]The Department of Health Technology and Informatics, The Hong Kong Polytechnic University, Hong Kong SAR, China, [2]Department of Anatomical and Cellular Pathology, The Chinese University of Hong Kong, Hong Kong SAR, China, [3]Education Department, The English Schools Foundation, Hong Kong SAR, China

MPP4-2 Regulation of cholesterol and bile acid homeostasis by ceramide chain length

Eunji Noh[1*], Seonwoo Lee[1], Woo-Jae Park[2], Joo-Won Park[1]

[1]Department of Biochemistry, College of Medicine, Ewha Womans University, Republic of Korea, [2]Department of Biochemistry, Chung-Ang University College of Medicine, Republic of Korea

MPP4-3 Docosahexaenoic acid attenuates vascular calcification with regulation of miR-29b1

Hyeonji Lee[1*], Soo-jin Ann[2], Sang-Hak Lee[3]

[1]Division of Biomedical Engineering, Yonsei University, Republic of Korea, [2]Integrative Research Center for Cerebrovascular and Cardiovascular Diseases, Yonsei University College of Medicine, Republic of Korea, [3]Division of Cardiology, Yonsei University College of Medicine, Republic of Korea

MPP4-4 Lipid droplet sequestration protects vascular smooth muscle cells from saturated fatty acid-induced NFkB activation

Laura Alonso-Herranz[1,2*], Jacob Fog Bentzon[1,2]

[1]Department of Clinical Medicine, Denmark, [2]Steno Diabetes Center Aarhus, Denmark

MPP4-5 CIAPIN1 orchestrates vascular phenotypic switching as a redox-sensitive repressor of Tp53

Seongpyo Lee[*], Joo-Hui Han

College of Pharmacy, Chungbuk National University, Republic of Korea

MPP4-6 Endothelial CAP1 acts as a gatekeeper of vascular homeostasis via the formation of an adenylyl cyclase and caveolin-1 complex

You Ji Kim[1,2,3*], Yoo-Wook Kwon[1,3]

[1]Biomedical Research Institute, Seoul National University Hospital, Republic of Korea, [2]Program in Stem Cell Biology, Seoul National University College of Medicine, Republic of Korea, [3]Department of Medicine, Seoul National University College of Medicine, Republic of Korea

Moderated Poster Presentation 5

Sep 12(Sat) 13:00-14:00 | Studio 6 (6F, Conrad Seoul)

MODERATOR : Jeong-Min Kim (Seoul National University, Republic of Korea)

MPP5-1 Association of a body shape index and the 5-year incidence of dyslipidemia and hypertension among Filipinos in the LIFECARE cohort

Bayani Pocholo T. Maglinte[1*], Raymond Oliva[1], Lourdes Ella G. Santos[1], Deborah Ignacia D. Ona[1], Jerahmeel Aleson L. Mapili[1], Bernadette Alexis M. Mariño[1], Bryan F. Elvambuena[1], Manuel John A. Dayao[1], Rio May E. Llanes[1], Celia Catherine C. Uy[1], Olivia T. Sison[2], Nina T. Castillo-Carandang[2], Felix Eduardo R. Punzalan[1], Elmer Jasper B. Llanes[1], Wilbert Allan G. Gumatay[1], Rody G. Sy[1]

[1]Division of Cardiovascular Medicine, University of the Philippines - Philippine General Hospital, Philippines, [2]Department of Clinical Epidemiology, University of the Philippines - Manila, Philippines

MPP5-2 Burden of ischemic stroke attributable to high LDL cholesterol and high body mass index in adults aged 55+years: a comparative analysis of mortality, DALYs, and YLDs by sex across socio-demographic index levels

Zavia Putri Salsabila[1*], Nazzala Safira[2], Zulfania Rahmah[3]

[1]Medical Internship Program, Islamic Weleri Hospital, Indonesia, [2]Medical Internship Program, Banjarnegara Muhammadiyah Hospital, Indonesia, [3]Medical Internship Program, Al Aziz Islamic Hospital, Indonesia

MPP5-3 Association between triglyceride-to-HDL cholesterol ratio and coronary artery spasm: differential effect according to diabetes mellitus status

You Jin Lee[1*], Se Yeon Choi[1], Byoung Geol Choi[2], Jae Kyeong Byun[1], Abiseka Panji Baskoro[1], Aditya Reza Partama[1], Soohyung Park[1], Seung-woon Rha[1]

[1]Cardiovascular Center, Korea University Guro Hospital, Republic of Korea, [2]Department of Biomedical Laboratory Science, Honam University, Republic of Korea

MPP5-4 Decoupling atherosclerosis from vasospasm: insights from the ApoB/ApoA1 ratio

Abiseka Panji Baskoro[1,2*], Aditya Reza Pratama[1], Byoung Geol Choi[3], Se Yeon Choi[1], Jae Kyeong Byun[1], Youjin Lee[1], Won Sang Chu[1], Soohyung Park[1], Cheol Ung Choi[1], Seung-Woon Rha[1]

[1]Cardiovascular, Korea University Guro Hospital, Republic of Korea, [2]Cardiology, Universitas Sebelas Maret Hospital, Indonesia, [3]Department of Biomedical Laboratory Science, Honam University, Republic of Korea

MPP5-5 Carbohydrate-fat dietary patterns modify protein-cardiometabolic association: a cross-sectional study in Singaporean adults

Weili Hu[1*], Yueying Yao[1], Ian Mak[1], Lingyin Yu[1], Shuqi Li[1], Nicole Wong Min Yee[1], Marcus Ting[1], Chin Meng Khoo[3], Jung Eun Kim[1,2]

[1]Department of Food Science and Technology, National University of Singapore, Singapore, [2]Bezos Center for Sustainable Protein, National University of Singapore, Singapore, [3]Department of Endocrinology, National University Health System, Singapore

MPP5-6 Dietary macronutrient intake modifies the association between cognitive function and cardiovascular disease risk: a cross-sectional study in Singapore adults

Shuqi Li[1*], Yueying Yao[1], Ian En Kai Mak[1], Lingyin Yu[1], Nicole Min Yee Wong[1], Marcus Ting[1], Jung Eun Kim[1,2]

[1]Department of Food Science and Technology, National University of Singapore, Singapore, [2]Bezos Center for Sustainable Protein, National University of Singapore, Singapore

MPP5-7 Distribution of diet-attributable ischemic heart disease mortality across Indonesian provinces: an ecological analysis

Zulfania Rahmah[1*], Nazala Safira[2], Zavia Putri Salsabila[3]

[1]Medical Internship Program, Al Aziz Hospital, Indonesia, [2]Medical Internship Program, Banjarnegara Muhammadiyah Hospital, Indonesia, [3]Medical Internship Program, Weleri Islamic Hospital, Indonesia

Moderated Poster Presentation 6

Sep 12(Sat) 13:00-14:00 | Studio 7 (6F, Conrad Seoul)

MODERATOR : Jae-Han Jeon (Kyungpook National University, Republic of Korea)

MPP6-1 Association of obesity status and LDL-cholesterol levels with all-cause mortality in statin users aged 75 years and older: a nationwide population-based cohort study

Mihye Seo[1*], Kyungdo Han[2], Hyeong Kyu Park[1], Kyoil Suh[1]

[1]Endocrinology and Metabolism, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, Seoul, Republic of Korea, [2]Statistics and Actuarial Science, Soongsil University, Seoul, Republic of Korea

MPP6-2 Beyond revascularization: Wharton's jelly stem cells as a promising limb-salvage strategy in diabetic chronic limb-threatening ischemia: a systematic review and meta-analysis

Ghazy Wira Pradipta[1*], Najwa Dean Hasana[1], Ghazy Wira Pradipta[2], Affan Rayhan Ismail[3]

[1]General Practitioner, Faculty of Medicine, Trisakti University Jakarta and SADIA Private Clinic, Bandung, Indonesia, [2]Internal Medicine Department, Faculty of Medicine, Sebelas Maret University, Surakarta, Indonesia, [3]General Practitioner, Simo General Hospital, Boyolali, Indonesia

MPP6-3 Atherogenic index of plasma in metabolic syndrome: a systematic review and meta-analysis

Ahmed Almezaine[*], Abdelrahman Hatata, Ibrahim Elbably

Ministry of Health, Egypt

MPP6-4 Diagnostic accuracy of artificial intelligence for image-based unstable carotid plaque identification: a systematic review and meta-analysis

Youssef Soliman*, Nariman Ayman

Faculty of Medicine, Assiut University, Egypt

MPP6-5 Regional variation in the clinical impact of intravascular imaging for left main coronary atherosclerotic disease

Hendrianus[1*], Hoyoun Won[3], Sang Yeub Lee[2], Jun Hwan Cho[2], Jinhwan Jo[2], Soo-Young Lee[2], Kyung-Taek Park[3], Gyu-Tae Park[3], Young-Hoon Jeong[2], Sang-Wook Kim[2]

[1]Division of Cardiology, Natuna General Hospital, Indonesia, [2]Heart and Brain Hospital, Chung-Ang University Gwangmyeong Hospital, Republic of Korea, [3]Division of Cardiology, Chung-Ang University Hospital, Republic of Korea

MPP6-6 Effectiveness and safety of pravastatin in postmenopausal women with dyslipidemia in Korea: results from the MELODY study

Jun Hwa Hong[1*], Sangmo Hong[2*], Ji-Hwan Bae[3], Sung Hee Choi[4]

[1]Division of Endocrinology, Department of Internal Medicine, Daejeon Eulji Medical Center, Eulji University School of Medicine, Daejeon, Republic of Korea, [2]Department of Internal Medicine, College of Medicine, Guri Hospital, Hanyang University, Seoul, Republic of Korea, [3]Medical & Pharmacovigilance Department, Daiichi Sankyo Korea Co., Ltd., Seoul, Republic of Korea, [4]Department of Internal Medicine, Seoul National University Bundang Hospital, Seoul National University College of Medicine, Seongnam, Republic of Korea

MPP6-7 Triglyceride-to-HDL-cholesterol ratio as a marker of adiposity in young Korean adults: a single-center cross-sectional study

Sehyun Kim[1*], Ye Seul Yang[1,2], Min Kyong Moon[3]

[1]Department of Medicine, Seoul National University College of Medicine, Republic of Korea, [2]Department of Internal Medicine, Seoul National University Bundang Hospital, Seoul National University College of Medicine, Republic of Korea, [3]Department of Internal Medicine, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul National University College of Medicine, Republic of Korea

E-Poster p. 357

PE01 Synthetic HDL-based therapy for selective modulation of cholesterol efflux in atheroma macrophages

Khalfa Benadouda[1*], Georgios Kolypetris[1], Sirine Nouri[1], Menke De Leede[1], Tuomas Niemi-Aro[2], Noora Sjöstedt[1], Heidi Kidron[1], Artturi Koivuniemi[1]

[1]Faculty of Pharmacy, University of Helsinki, Finland, [2]Institute of Biotechnology, University of Helsinki, Finland

PE02 Hepatic LPLAT10 overexpression alters lipoprotein cholesterol distribution in diabetic mice

Kahori Shimizu[1*], Hideo Shindou[2,3], Hiroyuki Mizuguchi[1,4,5,6]

[1]Laboratory of Biochemistry and Molecular Biology, Graduate School of Pharmaceutical Sciences, The University of Osaka, Japan, [2]Department of Lipid Life Science, National Institute of Global Health and Medicine, Japan Institute for Health Security, Japan, [3]Department of Medical Lipid Science, Graduate School of Medicine, The University of Tokyo, Japan, [4]Global Center for Medical Engineering and Informatics, The University of Osaka, Japan, [5]Integrated Frontier Research for Medical Science Division, Institute for Open and Transdisciplinary Research Initiatives, The University of Osaka, Japan, [6]Center for Infectious Disease Education and Research, The University of Osaka, Japan

PE03 ANGPTL4 attenuates multicellular atherogenic remodeling: validation in a human iPSC-derived atheroid model

Dong Im Cho[1*], Bo Gyeong Kang[1], Hyang Hee Cho[1], Meeyoung Cho[1], Sooji Yoo[1], Jin Yoo[1], Chanyul Kim[1], Yong Sook Kim[4], Youngkeun Ahn[2,3]

[1]Cell Regeneration Research Center, Chonnam National University Hospital, Republic of Korea, [2]Cardiology, Chonnam National University Hospital, Republic of Korea, [3]Cardiology, Chonnam National University Medical School, Republic of Korea, [4]Medical Research Center, Chonnam National University Medical School, Republic of Korea

PE04 Boerhaavia diffusa ameliorates renal dyslipidaemia by enhancing fatty acid oxidation in high-fat diet-induced chronic kidney disease mouse models

Ching In Chung[1*], On Ying Lee[1], Angela Zaneta Chan[2], Martin Ho Yin Yeung[1,2]

[1]Department of Health Technology and Informatics, The Hong Kong Polytechnic University, Hong Kong, China, [2]Department of Anatomical and Cellular Pathology, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, China

PE05 Ret finger protein deficiency attenuates adipogenesis in mice with high fat diet-induced obesity

Yun-Gyeong Lee[*], Anna Jeong, Sera Shin, Duk-Hwa Kwon, Hyun Kook

Department of Pharmacology, Chonnam National University Medical School, Republic of Korea

PE06 Fermented pear extract ameliorates free fatty acid-induced hepatic steatosis in HepG2 cells

Van-Long Truong[2*], You-Jin Park[1], Woo-Sik Jeong[1]

[1]Division of Food Science and Biotechnology, Kyungpook University, Republic of Korea, [2]BK21 FOUR, Infectious Disease Healthcare, Kyungpook University, Republic of Korea

PE07 Nicotinamide promotes lipophagy and cholesterol efflux through SIRT1-LXRα deacetylation-ABCA1 axis in vascular smooth muscle cells

Poo-Reum Choi[1,2*], Seul-Gi Kim[1,2], Hyoung-Chul Choi[1,2]

[1]Department of Pharmacology, College of Medicine, Yeungnam University, Republic of Korea, [2]Yeungnam University, Senotherapy-based Metabolic Disease Control Research Center, Republic of Korea

PE08 APOA1 potentiates the protective effects of platelet-derived extracellular vesicles on lymphatic function during atherosclerosis

Élizabeth Lacroix[1,2*], Nolwenn Tessier[1], Sara Babran[1,2], Julie Guillemette[1], Béatrice Bohmer-Cardinal[1,2], Mary Sorci-Thomas[3,4,5], Catherine Martel[1,2]

[1]Montreal Heart Institute, Canada, [2]Department of Medicine, Université de Montréal, Canada, [3]Department of Medicine, Medical College of Wisconsin, United States, [4]Division of Pharmacology & Toxicology, Medical College of Wisconsin, United States, [5]Cardiovascular Center, Medical College of Wisconsin, United States

PE09 Clinical significance of PAI-1 and lipid metabolism disturbances in patients with ischemic heart disease

Kadirova Nargiza*, Nurillaeva Nargiza, Nargiza

The First Department of Internal Diseases in Family Medicine and the Basics of Preventive Medicine, Tashkent State Medical University, Uzbekistan

PE10 Post-stroke changes associated with glucocorticoid dysregulation

Huiju Jo*, Goo Taeg Oh

Department of Life Science, Ewha Womans University, Republic of Korea

PE11 Effects of a self-care model on the prevention of kidney complications in patients with type 2 diabetes: a quasi-experimental study

Mr.Natthawirot Choodum[1*], Assistant Professor Sasinaporn Lohitthai[2], Mrs.Sirinthra Futrakul[3]

[1]Community Nurse Practitioner Program, Faculty of Nursing, Songkhla Rajabhat University, Thailand, [2]Master of Nursing Science Program in Advanced Midwifery, Faculty of Nursing, Rambhai Barni Rajabhat University, Thailand, [3]Doctor of Philosophy (Educational Administration), Faculty of Nursing, Nakhon Ratchasima Rajabhat University, Thailand

PE12 Impact of high-sensitivity C-reactive protein cutoff selection on cardiovascular risk classification beyond lipid measurements in Korean adults: a KNHANES 2024 study

Rihwa Choi[1,2*], Sang Gon Lee[1,3], Sung-Eun Cho[3]

[1]Laboratory Medicine Center, GC Labs, Republic of Korea, [2]Department of Laboratory Medicine and Genetics, Sungkyunkwan University School of Medicine, Republic of Korea, [3]Endocrine Substance Analysis Center, GC Labs, Republic of Korea

PE13 The metabolic knife: how bariatric surgery transforms blood pressure and cardiometabolic risk in obese hypertensive patients (a systematic review and meta-analysis)

Withdrawn

Elen Putri Cintya[1*], Lutfi Hafiz Zunardi[2]

[1]Medicine, Universitas Brawijaya, Indonesia, [2]Cardiology, Sunan Kalijaga General Hospital Indonesia, Indonesia

PE14 Integrative post-GWAS analyses identify HEIDI-clean causal genes for hypertension in East Asian populations

Yongho Jee[1*], Keum Ji Jung[2], Wes Spiller, Tae-Jin Song[3]

[1]Advanced Biomedical Research Institute, Ewha Womans University Seoul Hospital, Republic of Korea, [2]Institute for Health Promotion, Department of Epidemiology and Health Promotion, Graduate School of Public Health, Yonsei University, Republic of Korea, [3]Department of Neurology, Seoul Hospital, Ewha Womans University College of Medicine, Republic of Korea

PE15 Trends in lipid-lowering drug prescription patterns in patients with dyslipidemia: a real-world data from 2010 to 2023

Seung-Hyun Ko[1*], Kyungdo-Han[3], Bongseong Kim[3], Kyuho Kim[1], Yeseul Yang[2], Kyuna Lee[1]

[1]Endocrinology & Metabolism, The Catholic University of Korea, Republic of Korea, [2]Department of Internal Medicine, Seoul National University College of Medicine, Republic of Korea, [3]Statistics and Actuarial Science, Soongsil University, Republic of Korea

PE16 Sex differences of lipoprotein (a) levels and cardiovascular risk: retrospective multicenter cohort study of Korean patients

Subin Lim[1*], Hyung Joon Joo[2]

[1]Cardiology, Ewha Womans University Seoul Hospital, Republic of Korea, [2]Cardiology, Korea University Anam Hospital, Republic of Korea

PE17 Provincial variation in non-alcoholic fatty liver disease burden and associated risk factors among adults aged 50–69 years in Indonesia: a population-based study

Nazala Safira[1*], Zulfania Rahmah[2], Zavia Putri Salsabila[3]

[1]Medical Internship Program, Banjarnegara Muhammadiyah Hospital, Indonesia, [2]Medical Internship Program, Al-Aziz Islamic Hospital, Indonesia, [3]Medical Internship Program, Islamic Weleri Hospital, Indonesia

PE18 Baseline atherogenic dyslipidemia and early NT-proBNP clearance in acute heart failure: a retrospective cohort study

Muhammad Ayyub Aiman bin Mohd Fauzi[1,2,3*], Mohamad Rahal Yusoff[4], Mohamad Rodi Isa[5], Noor Alicezah Mohd Kasim[1,2]

[1]Cardiovascular Advancement and Research Excellence Institute (CARE Institute), Universiti Teknologi MARA, Malaysia, [2]Department of Pathology, Universiti Teknologi MARA, Malaysia, [3]Near Peer Instructor Unit, AIMST University, Malaysia, [4]Department of Internal Medicine, Universiti Teknologi MARA, Malaysia, [5]Department of Public Health and Preventive Medicine, Universiti Teknologi MARA, Malaysia

PE19 Resolving the genetic dark matter of inherited dyslipidemia: structural variation and polygenic susceptibility identify 24.4% of previously unexplained cases

Muhammad Hamza Mubarak[1*], Muhammad Kharisma[3], Evy Sulistyoningrum[2]

[1]Department of Clinical Pathology, Universitas Islam Indonesia, Indonesia, [2]Department of Histology, Universitas Islam Indonesia, Indonesia, [3]Department of Microbiology, Universitas Islam Indonesia, Indonesia

PE20 Lipid-lowering efficacy in omani patients with familial hypercholesterolemia

Suaad Al Mukhaini[1*], Mohsin Al Lawati[2], Khalid Al Rasadi[2]

[1]Directorate of Nursing, Sultan Qaboos University Hospital, University Medical City, Oman, [2]Department of Biochemistry, College of Medicine & Health Sciences, Sultan Qaboos University, Oman

PE21 Beyond weight loss: psychiatric and eating behavior safety signals associated with GLP-1 therapies in FAERS

Ekram Hassan Hasanin[*]

Faculty of Medicine, University of Tripoli, Libyan Arab Jamahiriya

PE22 Hepatic insulin resistance index and associated metabolic profiles in Korean adults with type 2 diabetes mellitus

Paul Kim[1,2*], Minji Kang[1,2], Hyunji Sang[3,4], Sang Youl Rhee[3,4], Hyunjung Lim[1,2]

[1]Department of Medical Nutrition, Graduate School of East-West Medical Science, Kyung Hee University, Giheong-gu, Yongin, Republic of Korea, [2]Research Institute of Medical Nutrition, Kyung Hee University, Dongdaemoon-gu, Seoul, Republic of Korea, [3]Department of Endocrinology and Metabolism, Kyung Hee University Hospital, Kyung Hee University College of Medicine, Seoul, Republic of Korea, [4]Center for Digital Health, Medical Science Research Institute, Kyung Hee University Medical Center, Kyung Hee University College of Medicine, Seoul, Republic of Korea

PE23 Cumulative burden of lipid-related indices as a predictor of incident type 2 diabetes in young adults: a nationwide cohort study

Jung Heo[1*], Jae-Hyuk Lee[1], Oak-Kee Hong[2], Bongsung Kim[3], Kyungdo Han[3], Min-Kyung Lee[1]

[1]Endocrinology and Metabolism, Myongji Hospital, Hanyang University College of Medicine, Republic of Korea, [2]Endocrinology and Metabolism, The Catholic University of Korea, Republic of Korea, [3]Statistics and Actuarial Science, Soongsil University, Republic of Korea

PE24 Genetic spectrum of familial hypercholesterolaemia in multigenerational families in Oman

Mohsin Al-Lawati[1*], Khalid Al Waili[2], Fahad Zadjali[3], Mohammed Nasser Al Kindi[1], Siamak Saber[4], Koukab Al Farsi[2], Suad Al Mukhaini[5], Khalsa Al Lamki[1], Manal Al Khambashi[1], Asma Al Mubaihsi[1], Khalid Al-Rasadi[1]

[1]Department of Biochemistry, College of Medicine & Health Sciences, Sultan Qaboos University, Oman, [2]Department of Clinical Biochemistry, Sultan Qaboos University Hospital, University Medical City, Oman, [3]Oman College of Health Sciences, Ministry of Health, Oman, [4]Medical Genetic, Bion Genetic Laboratory, Oman, [5]Department of Nursing, Sultan Qaboos University Hospital, University Medical City, Oman

PE25 Higher diabetes-related distress predicts failure to achieve LDL-C targets and poor glycemic recovery in diabetes mellitus patients following myocardial infarction

Marcelino Adiska Megantara[1,2*]

[1]Internal Medicine, Sebelas Maret University, Indonesia, [2]Internal Medicine, Siloam Sentosa Hospital, Indonesia

PE26 From measurement to motivation: an integrated framework connecting 3D anthropometry, AI-driven phenotyping, and behavioral theory for precision obesity intervention

Minseo Kim[*], Jane J Lee

Food and Nutrition, Sookmyung Women's University, Republic of Korea

PE27 Analysis of miRNAs expression in abdominal aortic aneurysm and their potential as biomarkers

Kenshi Yoshimura[2*], Keiko Urushino[2], Ryotaro Nagashima[1], Tomoyuki Wada[4], Takashi Shuto[1], Takayuki Kawashima[1], Hirofumi Anai[1], Kensuke Toyama[3], Shinji Miyamoto[1]

[1]Cardiovascular Surgery, Oita University, Japan, [2]Cardiovascular Surgery, Nakatsu Municipal Hospital, Japan, [3]Cardiovascular Surgery, Kurume University, Japan, [4]Cardiocvascular Surgery, Japanese Red Cross Oita Hospital, Japan

PE28 Admission NT-proBNP level as a marker of biological frailty and cause-specific mortality after percutaneous coronary intervention

Aditya Reza Pratama[1,2*], Abiseka Panji Baskoro[1], Byoung Geol Choi[3], Se Yeon Choi[1], You Jin Lee[1], Jae Kyeong Byun[1], Wonsang Chu[1], Soohyung Park[1], Cheol Ung Choi[1], Seung Woon Rha[1]

[1]Cardiovascular, Korea University Guro Hospital, Republic of Korea, [2]Cardiology and Vascular Medicine, Ulin General Hospital, Indonesia, [3]Department of Biomedical, Honam University, Republic of Korea

PE29 High obstructive sleep apnea risk association with carotid atherosclerosis and impaired vitality in obese hypertensive patients with type 2 diabetes

Borys Shelest[1*], Yuliia Kovalova[2], Oleksiy Shelest[2], Iulia Rodionova[3]

[1]Department of Internal and Occupational Diseases, Kharkiv National Medical University, Ukraine, [2]Department of Internal Medicine No. 2, Clinical Immunology and Allergology Named After Academician L.T. Malaya, Kharkiv National Medical University, Ukraine, [3]Department of Prevention and Treatment of Emergency Conditions, The Government Institution L.T. Malaya Therapy National Institute of the National Academy of Medical Sciences of Ukraine, Ukraine

PE30 Impact of statin use on net adverse clinical and cerebral events after ESUS

Kangho Choi[1*], Ja-Hae Kim[2]

[1]Neurology, Chonnam National University Hospital, Republic of Korea, [2]Nuclear Medicine, Chonnam National University Hospital, Republic of Korea

PE31 Blood pressure variability and cerebral autoregulation failure: accelerated cognitive decline in the NACC cohort

Moaz Abouelmagd*, Amr Elrosasy, Mohammed Fathi Abduljalil

Kasr Alainy Faculty of Medicine, Cairo University, Egypt

PE32 The preventive effect of routine dental scaling on acute myocardial infarction in patients with diabetes: a long-term follow-up study

Tsai-Wei Hsu*, Chien-Chang Liao

Department of Anesthesiology, Taipei Medical University Hospital, Taiwan

PE33 Deficiency of p300/CBP-associated factor exacerbates cardiac remodeling by disrupting CAMKK2 acetylation

Anna Jeong*, Yun-Gyeong Lee, Duk-Hwa Kwon, Sera Shin, Ayeong Han, Chae Ryeong Han, Hyun Kook

Department of Pharmacology, Chonnam National University Medical School, Republic of Korea

PE34 Synergistic attenuation of adipose inflammation and hepatic steatosis by combined taurine and leucine supplementation in a diet-induced model of sarcopenic obesity

Amara Zulfiqar[1*], Jung-Eun Yim[1,2]

[1]Interdisciplinary Program in Senior Human Ecology, Changwon National University, Republic of Korea, [2]Department of Food and Nutrition, Changwon National University, Republic of Korea

PE35 Can the 4-year cholesterol screening gap be compensated? A machine learning imputation simulation for high-risk classification

ChanSeok Son[1,2*], HyongGin An[3]

[1]Department of Public Health, Korea University Graduate School, Republic of Korea, [2]Client Services, IQVIA Korea, Republic of Korea, [3]Department of Biostatistics, Korea University College of Medicine, Republic of Korea

PE36 Distinct cardiometabolic and pulmonary profiles across obesity-metabolic health phenotypes

Dabin Lee[1*], Minji Kim[1], Soyeon Park[1], Seohyeon Lee[1], Soyoon Cha[1], Jimin Kim[1], Oh Yoen Kim[1,2,3]

[1]Department of Health Sciences, Clinical Nutrition Major, Graduate School of Dong-A University, Republic of Korea, [2]Center for Food & Bio Innovation, DAU G-LAMP Project Group, Dong-A University, Republic of Korea, [3]Department of Food Science and Nutrition, Dong-A University, Republic of Korea

PE37 Peripheral microcurrent stimulation promotes neuroprotection and remyelination in a rat model of hypoxia-ischemia

Dong Rak Kwon[1*], Eun Sang Kwon[2]

[1]Department of Rehabilitation Medicine, Catholic University of Daegu School of Medicine, Daegu, Republic of Korea, [2]Department of Medicine, College of Medicine, Keimyung University, Daegu, Republic of Korea

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Key Lectures

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Plenary Lecture 1

Sep 11(Fri) 10:20–11:00 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Sung Rae Kim (The Catholic University of Korea, Republic of Korea)

10:20–11:00 **Lipoprotein(a): the next frontier in cardiovascular prevention**

Sotirios Tsimikas (University of California, San Diego, USA)

CURRICULUM VITAE

Sotirios Tsimikas

Professor of Medicine and Director of Vascular Medicine,
University of California San Diego School of Medicine, San Diego, USA

Education and Training

1988	University of Massachusetts School of Medicine, Worcester, MA, M.D., Medicine
1988-1991	University of Massachusetts Medical Center, Worcester, MA, Internship & Residency, Internal Medicine
1992-1995	University of California San Diego Medical Center, Fellowship, Cardiovascular Diseases
1994-1996	University of California San Diego, Specialized Center for Research on Arteriosclerosis and Molecular Medicine, Research Fellowship, Atherosclerosis & Molecular Medicine
1996-1997	University of California San Diego Medical Center, Fellowship, Interventional Cardiology
1980-1984	Boston College, Chestnut Hill, MA, B.S., Magna cum Laude, Chemistry

Employment and Position

2007-Present	University of California San Diego School of Medicine, Professor of Clinical Medicine
2002-Present	University of California San Diego, Division of Cardiovascular Medicine, Founding Director of Vascular Medicine
2003-2007	University of California San Diego School of Medicine, Associate Professor of Clinical Medicine
1997-2003	University of California San Diego Medical Center, Assistant Clinical Professor of Medicine

Important Publications

1. Tsimikas S, Karwatowska-Prokopczuk E, Gouni-Berthold I, Tardif J-C, Baum SJ, Steinhagen-Thiessen E, Shapiro MD, Stroes ESG, Moriarty PM, Nordestgaard BG, Guerriero J, Viney NJ, O'Dea L, Witztum JL. Antisense oligonucleotide for lipoprotein(a) reduction in patients with established cardiovascular disease. N Engl J Med. 2020;382:244-255.
2. Bhatia HS, Dweck MR, Craig N, Capoulade R, Pibarot P, Trainor PJ, Whelton SP, Rikhi R, Lidani KCF, Post WS, Tsai MY, Criqui MH, Shapiro MD, Budoff MJ, DeFilippis AP, Thanassoulis G, Tsimikas S. Oxidized phospholipids and calcific aortic valvular disease. J Am Coll Cardiol. 2024;84:2430-2441.
3. Witztum JL, Que X, Tang W, Tsimikas S. Antibody E06 and the role of oxidized phospholipids in atherogenesis and inflammation. Circ Res. 2026;138:e327359. doi: 10.1161/CIRCRESAHA.126.327359.
4. Tsimikas S, Januzzi JL Jr, et al. Balance of non-Lp(a) apoB and Lp(a) with coronary plaque phenotypes and MACE: Insights from PROSPECT II and CASABLANCA. J Am Coll Cardiol. 2026.

Lipoprotein(a): the next frontier in cardiovascular prevention

Sotirios Tsimikas

University of California, San Diego, USA

Lipoprotein(a) [Lp(a)] is a genetically determined, independent, and likely causal risk factor for atherosclerotic cardiovascular disease (ASCVD) and calcific aortic valve stenosis (CAVS). Approximately 20% of the population has elevated Lp(a), representing more than one billion individuals worldwide, yet Lp(a) remains markedly underdiagnosed in clinical practice.

The unique biology of Lp(a) extends beyond its apolipoprotein B-containing lipoprotein core. Lp(a) is the major lipoprotein carrier of pro-inflammatory oxidized phospholipids (OxPL), which may contribute to vascular and valvular inflammation and provide a mechanistic link between elevated Lp(a) and cardiovascular disease. Recent studies further demonstrate that the lipid composition of Lp(a), including its cholesterol and triglyceride content, is heterogeneous and may provide additional information regarding coronary plaque vulnerability and cardiovascular risk. In CAVS, genetic, epidemiologic, imaging, and biomarker studies implicate both Lp(a) and associated OxPL in disease initiation and progression.

The therapeutic landscape has advanced rapidly. RNA-targeted therapies directed against hepatic *LPA* expression, including antisense oligonucleotides and small interfering RNAs, can reduce circulating Lp(a) by approximately 80-100%. Pelacarsen also markedly reduces OxPL-apoB and OxPL-apo(a), providing evidence that these pro-inflammatory cargo molecules can be therapeutically modified. Several cardiovascular outcomes trials are now evaluating whether profound Lp(a) lowering translates into fewer cardiovascular events, while dedicated studies are beginning to test whether this strategy can modify CAVS progression.

The field is therefore approaching a pivotal transition from identifying Lp(a) as a causal risk factor to determining whether it is a modifiable cause of cardiovascular disease. The results of Lp(a) HORIZON and subsequent outcome trials will test the "Lp(a) hypothesis" and may establish Lp(a) measurement and targeted treatment as a new pillar of cardiovascular prevention.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Plenary Lecture 2

Sep 11(Fri) 15:20–16:00 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Woo Je Lee (University of Ulsan, Republic of Korea)

15:20–16:00 **Closing the gap between guidelines and reality in dyslipidemia management**

Michal Vrablik (Charles University, Czech Republic)

CURRICULUM VITAE

Michal Vrablik

Professor of Internal Medicine, 1st Faculty of Medicine,
Charles University, General University Hospital, Prague, Czech Republic

Education and Training

1997.06	Charles University, Prague, Czechia, M.D., General Medicine
2005.06	Charles University, Prague, Czechia, Ph.D., Physiology and Pathophysiology

Employment and Position

1997-1998	Institute of Biology, Charles University, Prague, Czechia, Research Fellow
1998-Present	3rd Department of Medicine, Charles University and General University Hospital, Prague, Czechia, Deputy Head of the Dept.
2011-Present	Czech Society for Atherosclerosis, President
2012-2016	European Atherosclerosis Society, Board Member
2019-Present	International Atherosclerosis Society, Federation for Europe, Chairman
2024	Fellow of European Federation of Internal Medicine, FEFIM
2025	Fellow of European Society of Cardiology, FESC
2026.02-Present	Charles University, Prague, Czech Republic, Vice Rector for International Affairs

Important Publications

1. Ridker, P. M.; Revkin, J.; Amarenco, P.; et al., Cardiovascular Efficacy and Safety of Bococizumab in High-Risk Patients, NEW ENGLAND JOURNAL OF MEDICINE, 2017, Volume 376, Issue16, pages: 1527-1539, DOI:10.1056/NEJMoa1701488
2. Cicero, Arrigo F. G.; Colletti, Alessandro; Bajraktari, Gani; et al., Lipid lowering nutraceuticals in clinical practice: position paper from an International Lipid Expert Panel, NUTRITION REVIEWS, 2017, Volume 75,Issue 9, page 731-767, DOI 10.1093/nutrit/nux047
3. Ray, KK., Molemans, B.,Schoonen, WM., Giovas, P., Bray, S., et al., EU-Wide Cross- Sectional Observational Study of Lipid- Modifying Therapy Use in Secondary and Primary Care: the DA VINCI study, EUROPEAN JOURNAL OF PREVENTIVE CARDIOLOGY, 2021, Volume 28, Issue 11, pages 1279-1289. DOI:10.1093/eurjpc/zwaa047
4. Banach, Maciej; Patti, Angelo Maria; Giglio, Rosaria Vincenza; et al., The Role of Nutraceuticals in Statin Intolerant Patients, JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY, 2018, Volume 72, Issue1, pages: 96-118 DOI:10.1016/j.jacc.2018.04.040
5. Fruchart, JC., Santos, RD., Aguilar-Salinas, C., Aikawa, M., Rasadi, K., et al., The selective peroxisome proliferator-activated receptor alpha modulator (SPPARM) paradigm: conceptual framework and therapeutic potential: A consensus statement from the International Atherosclerosis Society (IAS) and the Residual Risk Reduction Initiative (R3i) Foundation, CARDIOVASCULAR DIABETOLOGY, 2019, Volume 18, Article Number 71, DOI: 10.1186/s12933-019-0864-7

Closing the gap between guidelines and reality in dyslipidemia management

Michal Vrablik

1st Faculty of Medicine, Charles University, General University Hospital, Prague, Czech Republic

Atherosclerotic cardiovascular disease (ASCVD) remains a leading cause of morbidity and mortality in Europe, with substantial regional disparities and a particularly high cardiovascular burden persisting in Central and Eastern Europe (CEE). Although contemporary ESC/EAS guidelines provide clear, evidence-based recommendations for cardiovascular risk stratification, low-density lipoprotein cholesterol (LDL-C) targets, and lipid-lowering therapy (LLT), their implementation in routine clinical practice remains suboptimal.

LDL-C is a causal and modifiable determinant of ASCVD, and extensive evidence from genetic, epidemiological, and randomized intervention studies demonstrates that the magnitude and duration of exposure to LDL-C are critically related to cardiovascular risk. Accordingly, current recommendations emphasize early initiation and appropriate intensification of LLT, increasingly using combination strategies to achieve stringent risk-based LDL-C goals. Nevertheless, real-world data reveal major treatment gaps. European observational studies demonstrate particularly low LDL-C goal attainment in CEE, especially among patients in secondary prevention. The adoption of more stringent guideline-recommended targets further increases the proportion of patients classified as being above goal.

Several factors contribute to this implementation gap. These include insufficient treatment intensity, delayed therapy initiation and escalation, therapeutic inertia, suboptimal adherence and persistence, and underestimation of LDL-C as a major cardiovascular risk factor by healthcare professionals. Even among patients following acute coronary syndromes, LDL-C may receive lower priority than other established risk factors.

Bridging the gap between guidelines and clinical practice therefore requires a multifaceted approach. Earlier initiation of effective LLT, timely treatment intensification, greater use of combination therapy, simplification of treatment regimens, and strategies supporting long-term adherence are essential. Shared decision-making and improved physician awareness should complement pharmacological innovation. The key challenge in contemporary dyslipidemia management is no longer the lack of effective therapies, but ensuring that available evidence and therapeutic options are translated consistently and promptly into clinical practice.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Plenary Lecture 3

Sep 12(Sat) 10:40–11:20 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Sang-Hyun Kim (Seoul National University, Republic of Korea)

10:40–11:20 **Beyond LDL-C lowering: strategies for maximizing residual ASCVD risk reduction**

Nathan D. Wong (University of California, Irvine, USA)

CURRICULUM VITAE

Nathan D. Wong

Professor and Director, Heart Disease Prevention Program,
Mary and Steve Wen Cardiovascular Division and Co-Director,
Center for Global Cardiometabolic Health and Nutrition
Past President, American Society for Preventive Cardiology and
Pacific Lipid Association Chapter of the National Lipid Association
University of California, Irvine School of Medicine, Irvine, USA

Education and Training

1985	Yale University, MPH, Epidemiology
1987	Yale University, PhD, Epidemiology

Employment and Position

1988-Present	University of California, Irvine School of Medicine, Professor

Important Publications

1. Wong ND. Epidemiological studies of CHD and the evolution of preventive cardiology. Nat Rev Cardiol. 2014 May;11(5):276-89. doi: 10.1038/nrcardio.2014.26. Epub 2014 Mar 25. PMID: 24663092 (IF=41.7).
2. Wong ND, Zhao Y, Patel R, Patao C, Malik S, Bertoni AG, Correa A, Folsom AR, Kachroo S, Mukherjee J, Taylor H, Selvin E. Cardiovascular Risk Factor Targets and Cardiovascular Disease Event Risk in Diabetes: A Pooling Project of the Atherosclerosis Risk in Communities Study, Multi-Ethnic Study of Atherosclerosis, and Jackson Heart Study. Diabetes Care. 2016; 39(5):668-76 (IF=16.6).
3. Wong ND, Sattar N. Cardiovascular risk in diabetes mellitus: epidemiology, assessment and prevention. Nat Rev Cardiol. 2023; 20(10):685-695 (IF=41.7).
4. Wong ND, Budoff MJ, Ferdinand K, Graham IM, Michos ED, Reddy T, Shapiro MD, Toth PP. Atherosclerotic cardiovascular disease risk assessment: An American Society for Preventive Cardiology clinical practice statement. Am J Prev Cardiol. 2022 Mar 15;10:100335 (IF=5.9)
5. Wong ND, Fan W, Hu X, Ballantyne C, Hodgeveen R, Tsai MY, Browne A, Budoff MJ Lipoprotein(a) and Long-Term Cardiovascular Risk in a Multi-Ethnic Pooled Prospective Cohort. J Am Coll Cardio 2024 Apr 23;83(16):1511-1525 (IF=22.3).

Awards and Honors

2023	Master, American Society for Preventive Cardiology
2022	Lifetime Achievement Award in Clinical Research, UCI School of Medicine
2021	Joseph Stokes MD III Award (lifetime achievement in preventive cardiology), American Society for Preventive Cardiology

Beyond LDL-C lowering: strategies for maximizing residual ASCVD risk reduction

Nathan D. Wong

University of California, Irvine, USA

Many patients with prior atherosclerotic cardiovascular disease (ASCVD) are at very high risk of future events due to the presence of multiple comorbidities and/or multiple ASCVD events. While efforts to reduce ASCVD risk have focused on aggressive LDL-C control, substantial residual risk exist due to other lipid and non-lipid factors, including excess apoB containing lipoproteins, elevated lipoprotein(a), residual hypertriglyceridemia, inflammation, thrombosis, as well as inadequately controlled hypertension, diabetes, and obesity. Inadequate adherence to lifestyle behaviours, including diet and physical activity, as well as increased polygenic risk and the presence of other risk enhancing factors also contributes to residual ASCVD risk. While statins form the foundation for ASCVD risk reduction through LDL-C lowering, the use of non-statins, including PCSK9 inhibitors, which can also reduce lipoprotein(a)-associated residual risk, remains limited, even in our highest risk ASCVD patients. In addition, icosapent ethyl provides further reduction of ASCVD risk among persons with moderately elevated triglycerides, yet this remains seldom prescribed. Moreover, GLP1-RA therapy, in particular from evidence from semaglutide showing reductions in ASCVD events both among persons with diabetes as well as obesity/overweight with prior ASCVD, has become a valuable new therapy for the cardiologists' armamentarium. SGLT2i therapy also reduces CVD events, particularly through its reduction in heart failure outcomes. The efficacy of newer therapies for targeting inflammation has been less consistent in their impact on ASCVD events, with the best evidence remaining for low-dose colchicine. In conclusion, there has been a dramatic increase in recent years of newer therapeutic options to address residual risk beyond LDL-C control, with the promise of future therapies in development to reduce ASCVD outcomes, including those targeting lipoprotein(a).

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Plenary Lecture 4

Sep 12(Sat) 11:20–12:00 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Ick Mo Chung (Ewha Womans University, Republic of Korea)

11:20–12:00 **Perspective on residual risk, inflammation, and atherosclerosis: from epidemiology to precision prevention**

Borge G. Nordestgaard (University of Copenhagen, Denmark)

CURRICULUM VITAE

Borge G. Nordestgaard

Professor, Chief Physician,
Copenhagen University Hospital, University of Copenhagen, Denmark

Education and Training

1985	University of Copenhagen, Denmark, M.D., Clinical Chemistry and Cardiology
1990	University of Copenhagen, Denmark, DMSc, Medicine

Employment and Position

1999-2026	Copenhagen University Hospital, Denmark, Chief Physician
2005-2026	University of Copenhagen, Denmark, Professor

President European Atherosclerosis Society

Prof Nordestgaard studied and worked in Copenhagen, New York, and London.

He has for more than 40 years continued his interest in the pathogenesis, diagnosis and treatment of severe hypertriglyceridemia, familial hypercholesterolemia, hyperlipidemia, lipoprotein(a), remnant cholesterol, triglycerides, atherosclerosis, diabetes, and liver, renal, and cardiovascular disease, and has written extensively on these conditions.

He is chairing the Copenhagen General Population Study and is a steering committee member of the Copenhagen City Heart Study and five phase 3 cardiovascular intervention trials.

Prof. Børge G. Nordestgaard has supervised 75 Ph.D. students and 36 postdoctoral fellows, and has published 1050 original articles and 175 reviews, book chapters, consensus statements, & editorials. His H-index is 188 in Web of Science and he is listed among the top 0.1% researchers worldwide with the most highly cited papers.

Original articles include publications in NEJM, Lancet, JAMA, Nature, Science, baby Nature journals, BMJ, baby Lancet journals, Circulation, European Heart Journal, JACC and baby JAMA journals.

Perspective on residual risk, inflammation, and atherosclerosis: from epidemiology to precision prevention

Borge G. Nordestgaard

University of Copenhagen, Denmark

Despite significant advances in lipid-lowering therapeutics, residual lipid risk persists in patients with or at risk of atherosclerotic cardiovascular disease (ASCVD), even after optimizing low-density lipoprotein (LDL) cholesterol. Emerging evidence highlights the role of non-LDL cholesterol fractions such as remnant cholesterol, lipoprotein(a) (Lp(a)), apolipoprotein B (apoB), and non-high-density lipoprotein (HDL) cholesterol, as key contributors to residual ASCVD risk. Remnant cholesterol, Lp(a), and LDL cholesterol represent three independent lipoprotein species causing ASCVD, while apoB and non-HDL cholesterol integrate the other three variables. Thus, clinically interpreting elevated apoB and non-HDL cholesterol is potentially complicated since remnants, Lp(a), and LDL cause ASCVD by different mechanisms and by varying proportions in different patients. Indeed, recent research into the pathophysiology of lipid-driven atherogenesis and development of ASCVD has revealed novel mechanisms that in turn suggest new therapeutic strategies targeting non-LDL lipid components. Elevated remnant cholesterol jointly with elevated LDL cholesterol contributes to arterial wall cholesterol deposition, plaque development and ASCVD end points. Furthermore, the additional triglyceride content in remnant particles may theoretically promote intimal inflammation and possibly plaque rupture and erosion, independently contributing to atherogenesis and ASCVD. The lipid component and pro-inflammatory properties of Lp(a) could similarly contribute directly to atherosclerotic plaque development and ASCVD. In addition, the homology with plasminogen of the defining apo(a) moiety of Lp(a) has long been speculated to confer anti-fibrinolytic and pro-thrombotic properties that could produce more severe ASCVD outcomes independent of atherogenesis. This review explores the evolving understanding of residual lipid risk in ASCVD, practical guidance for clinicians today, recent advances in therapeutic interventions, and their implications for clinical practice, aiming to optimize lipid management beyond LDL cholesterol reduction today and in the future.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Special Lecture 1 (K)

Sep 11(Fri) 11:00-11:40 | Room 1,2,3 (3F, Conrad Seoul)

CHAIRPERSON : Jae Hyoung Park (Korea University, Republic of Korea)

11:00-11:40 **도자기로 보는 한국 전통 미술의 생명력 (The vitality of traditional Korean art through ceramics)**

Ae-Ryung Lee (National Museum of Korea, Republic of Korea)

CURRICULUM VITAE

Ae-Ryung Lee

Deputy Director, Chief Curator, National Museum of Korea, Republic of Korea

Education and Training

Ewha Womans University, Korea, M.D.
이화여자대학교 대학원 미술사학과 석사

Employment and Position

2024-2026	Deputy Director, Chief Curator National Museum of Korea 국립중앙박물관 학예연구실장
2023-2024	Director, Gwangju National Museum 국립광주박물관장
2021-2023	Director, Fine Arts Division, National Museum of Korea 국립중앙박물관 미술부장

Important Special Exhibition

최근 5년간 큐레이팅과 감독을 맡은 주요 특별전
2026 The Korean Table: Food, Nature, and Life 특별전 <우리들의 밥상>
2025 Art of Early Joseon 특별전 <새 나라 새 미술, 조선 전기 미술 대전>
2024 Sculpted Celadon of the Goryeo Dynasty 특별전 <고려 상형청자>
2022 A Collector's Invitation 특별전 <어느 수집가의 초대, 고 이건희 회장 기증 기념전>

도자기로 보는 한국 전통 미술의 생명력 (The vitality of traditional Korean art through ceramics)

Ae-Ryung Lee

Deputy Director and Chief Curator, National Museum of Korea

도자기는 흙과 불 그리고 사람이 삼위일체가 되어 만드는 예술이며, 인류의 오랜 역사와 함께 발전해 온 경험과 지혜, 미적 감성이 담겨있다. 세계 도자기 발달사에서 자기의 제작은 문화사적으로 중요한 의미를 가진다. 유약을 입혀 1,200를 넘는 높은 온도에서 환원번조한 자기는 기술력의 뒷받침과 높은 온도를 견뎌낼 수 있는 가마 설비의 완비 등 과학적 성숙의 토대 위에 꽃피우는 예술품이기 때문이다.

Ceramics are an art form created through the harmonious union of earth, fire, and human craftsmanship. They embody the accumulated experience, wisdom, and aesthetic sensibilities that have developed throughout the long history of humankind. The emergence of porcelain holds particular significance in the history of world ceramics. Glazed and fired in a reducing atmosphere at temperatures exceeding 1,200°C, porcelain is an art form that could flourish only upon a foundation of scientific and technological advancement, including sophisticated production techniques and kiln facilities capable of withstanding extremely high temperatures.

한국의 자기 문화는 천년이 넘는 역사를 자랑한다. 오랜 세월 꽃피워온 한국 도자기의 예술성은 오늘날 세계의 주목을 받고 있다. 특히 조선 15세기의 분청사기는 독특한 현대적 미감과 뛰어난 표현력으로 널리 사랑받고 있다.

Korea's porcelain tradition boasts a history spanning more than a thousand years. The artistry of Korean ceramics, cultivated over many centuries, is now attracting worldwide attention. In particular, the buncheong ware of the fifteenth-century Joseon dynasty is widely admired for its distinctive modern aesthetic and remarkable expressive power.

분청사기(Buncheong Ware)는 백자와 함께 조선(1392~1897)을 대표하는 도자기이다. '분청사기'는 회청색 태토에 흰색 화장토(white slip)를 입혀 장식한 조선 전기의 도자기를 가리킨다. 분청사기는 그릇 표면에 백토를 입히는 장식 기법에 따라 7가지 종류로 구분된다. 장식기법별로 분청사기의 미적 특징은 매우 다양하게 발전했다. 하지만 분청사기의 대표적인 아름다움을 하나만 고르라면, 뛰어난 생명력의 표현을 말할 수 있다. 모티브가 되는 생명체의 특징을 예리하게 관찰하여 건강한 미감으로 대범하게 창출해 냈다. 본 강연에서는 물고기 무늬가 그려진 분청사기를 통해서 한국 전통 미술의 생명력을 살펴보고자 한다.

Alongside white porcelain, buncheong ware represents one of the principal ceramic traditions of the Joseon dynasty (1392-1897). The term *buncheong ware* refers to early Joseon ceramics made by applying white slip to a grayish-blue clay body. Buncheong ware can be classified into seven categories according to the decorative techniques used to apply white slip to the vessel's surface. These techniques gave rise to a remarkably diverse range of aesthetic characteristics. If one defining quality of buncheong ware were to be singled out, however, it would be its extraordinary vitality. Through keen observation of living creatures, Korean potters boldly rendered their distinctive features with a vigorous and vibrant aesthetic. This lecture explores the vitality of traditional Korean art through buncheong ware decorated with fish motifs.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Special Lecture 2

Sep 12(Sat) 13:20–13:50 | Room 4 (5F, Conrad Seoul)

CHAIRPERSON : Seong Hoon Choi (Hallym University, Republic of Korea)

13:20–13:50 **Atherosclerosis and atherosclerotic cardiovascular disease in Asia: the knowledge gap for the determinants**

Dong Zhao (Capital Medical University, China)

CURRICULUM VITAE

Dong Zhao

Capital Medical University, China

Professor and Chief Scientist of Preventive Cardiology at Capital Medical University, Beijing Anzhen Hospital-Beijing Institute of Heart, Lung & Blood Vessel Diseases (BIHLBD). She is the former Deputy Director of BIHLBD.

Dr. Zhao is a member of the China National Expert Committee of Cardiovascular Diseases. She serves as the deputy editor of JACC Asia; Advisory Board member of Nature Reviews Cardiology, and editor for the American Journal of Preventive Cardiology, International Journal of Cardiology, Chinese Journal of Cardiology, and Chinese Journal of Internal Medicine. Furthermore, she is a member of the writing committee for various international and Chinese guidelines on preventive cardiology, dyslipidemia management, and hypertension management.

Recent Publications

1. Qi Y, Li J, Yang Z, Liu J*, Zhao D*. Cardiovascular disease in China: epidemiological evolution and implications. Eur Heart J. Published online March 25, 2026. doi:10.1093/eurheartj/ehaf1121
2. Zhao, D., Zhang, Y., Wang, J. et al. Cardiovascular disease prevention in China: challenges and opportunities in the artificial intelligence-enabled digital health era. Nat Rev Cardiol 23, 363–374 (2026). https://doi.org/10.1038/s41569-025-01222-2
3. Zhao, D, Liu, J. Lipid Management, Lifestyle Changes, and Cardiovascular Prevention in China: Evolving Insights From Surveillance. JACC. 2026 Mar, 87 (12) 1475–1477. https://doi.org/10.1016/j.jacc.2025.10.078
4. Yang N, Liu J, Ma C, Zhu, D, Smith S C, Eckel R, Morgan L, Hao Y, Liu J, Zhou Y, Han Y*, Zhao D*. A National Registry to Improve the Quality of Care for Patients With Acute Coronary Syndrome and Diabetes: Protocol for the China Diabetes Cardiovascular (CDCV) Project. Cardiology Discovery, 2025,5(3),208–214.
5. Zhao D, Rubini M. Improving English proficiency for scientific communication by non-fluent speakers. Nat Rev Cardiol. 2025;22(3):141–142.
6. Zhao D, Wang Y, Wong ND, Wang J. Impact of Aging on Cardiovascular Diseases: From Chronological Observation to Biological Insights: JACC Family Series. JACC Asia. 2024;4(5):345–358.
7. Yang N, Liu J, Liu J, Hao Y, Smith JSC, Huo Y, Fonarow GC, Ge J, Morgan L, Ma C, Han Y*, Zhao D*. In-hospital outcomes of ticagrelor versus clopidogrel in patients 75 years or older with acute coronary syndrome: findings from the Improving Care for Cardiovascular Disease in China (CCC)-Acute Coronary Syndrome Project. Age and Ageing. 2022;51(11):afac231.
8. Zhou M, Zhang J, Liu J, Smith SC Jr, Ma C, Ge J, Huo Y, Fonarow GC, Liu J, Hao Y, Gao F, Sun Y, Morgan L, Yang N, Hu G, Zeng Y, Han Y*, Zhao D*. Proton Pump Inhibitors and In-Hospital Gastrointestinal Bleeding in Patients With Acute Coronary Syndrome Receiving Dual Antiplatelet Therapy. Mayo Clin Proc. 2022;97(4):682–692.
9. Dong Zhao, Key roles of five lifestyle risk factors in the harmful transition from a healthy condition to multimorbidity in the Chinese population, European Heart Journal, Volume 42, Issue 34, 7 September 2021, Pages 3385–3387, https://doi.org/10.1093/eurheartj/ehab516

Atherosclerosis and atherosclerotic cardiovascular disease in Asia: the knowledge gap for the determinants

Dong Zhao

Capital Medical University, China

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Main Symposia

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 1

Nutritional Immunometabolism in Atherosclerosis

Sep 10(Thu) 13:00–14:30 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Myoung Sook Lee (Sungshin Women's University, Republic of Korea)
Sung Nim Han (Seoul National University, Republic of Korea)

13:00–13:20	**Gut microbiota remodeling in infection and inflammation** Eun-Kyeong Jo (Chungnam National University, Republic of Korea)
13:20–13:40	**Ketogenic adaptation to intermittent fasting: implications for nutritional immunometabolism** Kyoung-Han Kim (University of Ottawa, Canada)
13:40–14:00	**Spatiotemporal immune activation drives metabolic quiescence in brown adipose tissue** Su Myung Jung (Sungkyunkwan University, Republic of Korea)
14:00–14:30	**Panel Discussion** Hyunju Kang (Kyungpook National University, Republic of Korea) Jun Namkung (Yonsei University, Republic of Korea) Chan Joo Lee (Yonsei University, Republic of Korea)

CURRICULUM VITAE

Eun-Kyeong Jo

Distinguished Professor, Chungnam National University, Republic of Korea

Education and Training

1991.02	Chungnam National University, Korea, M.D., Medicine
1996.02	Chungnam National University, Korea, Ph.D., Medical Microbiology

Employment and Position

2008-Present	Chungnam National University, College of Medicine, Korea, Professor
2007-2024	Medical Research Center, Chungnam National University, Korea, Director

Important Publications

1. Seo W, Jung B, Roh T, Shin HJ, Song IC, Chung C, Jo EK*. Inflammasomes and pyroptosis in cancer: mechanisms and therapeutic advances. J Hematol Oncol. 2025 Nov 25;18(1):113.
2. Jeon SM, Lee YJ, Lee SH, Kim SI, Lee B, Roh T, Kim YJ, Kim HJ, Kim IS, Whang J, Kim SY, Jhun BW, Chung C, Kang DH, Yeo MK, Kim JM, Jang J, Min JJ, Komatsu M, Kim JK, Park WY, Jo EK*. ATG7 in innate immune cells is required for host defense against nontuberculous mycobacterial pulmonary infections. Nat Commun. 2025 Jul 29;16(1):6966.
3. Roh T, Ju S, Park SY, Ahn Y, Chung J, Nakano M, Ryu G, Kim YJ, Kim G, Choi H, Lee SG, Kim IS, Lee SI, Chung C, Shimizu T, Miyoshi E, Jung SS, Park C, Yamasaki S, Park SY, Jo EK*. Fucosylated haptoglobin promotes inflammation via Mincle in sepsis: an observational study. Nat Commun. 2025 Feb 4;16(1):1342.
4. Roh T, Seo W, Won M, Yang WS, Sapkota A, Park EJ, Yun SH, Jeon SM, Kim KT, Lee B, Ryu G, Lee SH, Shin JM, Shin HJ, Kim YJ, Lee Y, Chung C, Song IC, Song HK, Jo EK*. The inflammasome-activating poxvirus peptide IAMP29 promotes antimicrobial and anticancer responses. Exp Mol Med. 2024 Nov;56(11):2475-2490
5. Kim YJ, Lee SG, Park SY, Jeon SM, Kim SI, Kim KT, Roh T, Lee SH, Lee MJ, Lee J, Kim HJ, Lee SE, Kim JK, Heo JY, Kim IS, Park C, Paik S, Jo EK*. Ubiquitin regulatory X (UBX) domain-containing protein 6 is essential for autophagy induction and inflammation control in macrophages. Cell Mol Immunol. 2024 Oct 23.

Gut microbiota remodeling in infection and inflammation

Accumulating evidence suggests that the gut-lung axis represents a promising target for host-directed therapeutics against infectious diseases. However, its role in protective immunity against nontuberculous mycobacteria (NTM) and *Mycobacterium tuberculosis*, two major causes of pulmonary infections in both immunocompromised and immunocompetent individuals, remains poorly understood. In this study, we identified gut microbes and microbial metabolites that enhance pulmonary host immunity against mycobacterial infections. Metabolomic analysis revealed significantly reduced serum levels of L-arginine in patients with NTM pulmonary disease (NTM-PD). Oral administration of L-arginine markedly enhanced pulmonary antimicrobial activity by expanding IFN-γ-producing effector T cells and promoting the polarization of microbicidal (M1) macrophages in the lungs of NTM-PD model mice. Moreover, fecal microbiota transplantation from L-arginine-treated mice conferred enhanced protection against NTM infection, whereas depletion of the gut microbiota with antibiotics markedly attenuated the protective effects of L-arginine. Mechanistically, L-arginine treatment reshaped the gut microbiota, resulting in the enrichment of Bifidobacterium species. These microbial alterations triggered metabolic signaling that promoted M1 macrophage polarization while suppressing pathological inflammation. In the latter part of this presentation, I will discuss our recent findings on the role of estrogen-related receptor α (ERRα/ESRRA) in maintaining intestinal homeostasis through autophagy activation and regulation of the gut microbiota. I will highlight how ESRRA-mediated control of mitochondrial quality and intestinal microbial composition protects the host from excessive inflammation and mitochondrial dysfunction during infection. Together, these findings establish a novel gut microbial-metabolic circuit that enhances mucosal immunity while limiting pathological inflammation, providing a promising framework for host-directed therapeutic strategies against infectious diseases.

CURRICULUM VITAE

Kyoung-Han Kim

Associate Professor & Scientist,
University of Ottawa & University of Ottawa Heart Institute, Canada

Education and Training

2011.06	University of Toronto, Canada, Ph.D., Physiology
2017.06	Hospital for Sick Children, Postdoc, Developmental Genetics

Employment and Position

2017-2023	University of Ottawa, Assistant Professor
2017-Present	University of Ottawa Heart Institute, Scientist
2023-Present	University of Ottawa, Associate Professor

Important Publications

1. Aslani et al., Sex- and Ketogenesis-Dependent Effects of Intermittent Fasting on Diet-Induced Fatty Liver Disease in mice. Diabetes 2026;75:1-16 (In Press)
2. Oh et al., Transcriptional regulation of the postnatal cardiac conduction system heterogeneity. Nature Communications 2024;15(1):6550
3. Asif et al., Hmgcs2-mediated ketogenesis modulates high-fat diet-induced hepatosteatosis. Molecular Metabolism 2022;101494 (cited > 120 times)
4. Kim et al., Intermittent fasting promotes adipose thermogenesis and metabolic homeostasis via VEGF-mediated alternative activation of adipose macrophage. Cell Research 2017;27(11):1309-1326 (cited > 270 times)
5. Smemo et al., Obesity-associated variants within FTO form long-range functional connections with IRX3. Nature 2014;507(7492):371-5. (cited > 1600 times)

Awards and Honors

1. Mentor of the Year - Basic science, Faculty of Medicine, University of Ottawa (2025)
2. Early Research Award, Government of Ontario, Canada (2024)
3. National New Investigator Award with McDonald Scholarship, Heart and Stroke Foundation of Canada (2021; ranked #1)

Ketogenic adaptation to intermittent fasting: implications for nutritional immunometabolism

Intermittent fasting (IF) has emerged as a promising nutritional strategy for improving cardiometabolic health. In addition to reducing adiposity, IF can improve glucose homeostasis, hepatic lipid metabolism, and vascular risk factors while suppressing inflammation associated with obesity, fatty liver disease, and atherosclerosis. However, the metabolic adaptations that distinguish IF from simple caloric restriction remain incompletely understood.

A defining feature of fasting is the shift from glucose utilization toward fatty acid oxidation and hepatic ketogenesis. Ketone bodies, particularly β-hydroxybutyrate (BHB), serve not only as alternative energy substrates but also as signaling metabolites that regulate cellular stress responses and immune-cell activity.

Our studies investigated the contribution of hepatic ketogenesis to the benefits of an isocaloric IF regimen in diet-induced obesity and fatty liver disease. IF reduced adiposity, improved glucose homeostasis, and attenuated hepatic steatosis and inflammation, with sexually dimorphic responses. Suppression of HMGCS2, the rate-limiting enzyme for hepatic ketogenesis, diminished several protective effects of IF. Fasting-induced ketogenesis was also associated with reduced neutrophil activation and hepatic infiltration, while BHB directly suppressed inflammatory responses in neutrophil-like cells.

These findings identify hepatic ketogenesis as an active mediator of fasting adaptation and highlight a nutrient-ketone-immune axis linking metabolic flexibility to cardiometabolic protection.

CURRICULUM VITAE

Su Myung Jung

Associate Professor, Sungkyunkwan University, Republic of Korea

Education and Training

2009.02	Sungkyunkwan University, B.S., Biological Sciences
2014.02	Sungkyunkwan University, Ph.D., Molecular Cell Biology

Employment and Position

2016–2021	University of Massachusetts Chan Medical School, Postdoc
2021–Present	Sungkyunkwan University, Associate Professor

Important Publications

1. Park G*, Haley JA*, Le J*, Jung SM* et al., Quantitative analysis of metabolic fluxes in brown fat and skeletal muscle during thermogenesis. Nature Metabolism (2023) Jun. PMID: 37337122
2. Jung SM et al., In vivo Isotope Tracing Reveals the Versatility of Glucose as a Brown Adipose Tissue Substrate. Cell Reports (2021) Jul. PMID: 34320357
3. Jung SM et al., Non-canonical mTORC2 Signaling Regulates Brown Adipocyte Lipid Catabolism through SIRT6-FoxO1. Molecular Cell (2019) Aug. PMID: 31442424
4. Hsiao WY, Jung SM et al., The lipid handling capacity of subcutaneous fat is programmed by mTORC2 during development. Cell Reports (2020) Oct. PMID: 33027655
5. Lee JH *, Jung SM* et al., A20 promotes metastasis of aggressive basal-like breast cancers through multi-monoubiquitylation of Snail1. Nature Cell Biology (2017) Oct. PMID: 28892081

Awards and Honors

2018	American Diabetes Association (ADA) Postdoctoral Fellowship (#1-18-PDF-128)
2018	Keystone Symposia scholarship: "Bioenergetics and Metabolic Disease"
2014	Excellent Doctoral Thesis Award, The International Conference of the KSMCB

Research Interest

1. Adipose tissue's nutrient handling and its roles in systemic metabolic homeostasis.
2. Understanding the significance metabolic exchange between organs during disease.
3. Crosstalk between cellular signaling and metabolic pathways.

Spatiotemporal immune activation drives metabolic quiescence in brown adipose tissue

Brown adipose tissue (BAT) exhibits remarkable metabolic flexibility in response to ambient temperature. Under warm conditions, BAT enters a metabolically quiescent state, known as BAT whitening, characterized primarily by lipid accumulation and reduced mitochondrial content. However, this transition is often oversimplified as a mere consequence of reduced sympathetic tone, while the underlying cellular mechanisms remain poorly understood. By integrating spatiotemporal transcriptomics with single-cell analysis, we construct a cellular map of BAT undergoing metabolic inactivation. Time-course profiling during thermoneutral adaptation reveals dynamic shifts in immune cell populations. Notably, we identify spatially organized immune activation zones (IAZs) and immune core zones (ICZs) within whitened BAT, frequently adjacent to mature adipocytes with reduced thermogenic potential. IFN-γ-producing $CD4^+$ T cells localized to IAZs/ICZs emerge as a key regulatory population suppressing BAT thermogenesis. Functional studies and adoptive transfer experiments targeting BAT-resident IFN-γ signalling indicate a causal role for these T cells in promoting metabolic quiescence of BAT. Our findings provide fundamental insight into tissue intrinsic immune-parenchymal interactions that mediate BAT inactivation, highlighting therapeutic potential of immune modulation in metabolic disease.

Symposium 2 (K)

From Biobank to Biology: Integrative Platforms for Cardiometabolic Discovery

Sep 10(Thu) 13:00–14:30 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Chi Dae Kim (Pusan National University, Republic of Korea)
Young-Guen Kwon (Curacle Advanced Institute of Technology, Republic of Korea)

13:00–13:20	**Biobank resources for human metabolic and vascular research** Hyun-Joung Lim (National Institute of Health, Republic of Korea)
13:20–13:40	**Leveraging biobank platforms to study cardiometabolic physiology and disease** Heiko Bugger (Medical University of Graz, Austria)
13:40–14:00	**Integrative analysis of metabolic dysfunction** Young Yang (Sookmyung Women's University, Republic of Korea)
14:00–14:30	**Panel Discussion** Yongseek Park (Kyung Hee University, Republic of Korea) Jun Hwan Cho (Chung-Ang University, Republic of Korea) Jae-Hoon Choi (Hanyang University, Republic of Korea)

CURRICULUM VITAE

Hyun-Joung Lim

Director, National Institute of Health, Republic of Korea

Education and Training

2004.08	Yonsei University, Korea, Ph.D, Biomedical Science

Employment and Position

2005-2019	NIH, Korea/Division of Cardiovascular Research, Senior Researcher
2020-2023	NIH, Korea/Division of Allergy and Respiratory Diseases Research, Deputy Scientific Director
2024-Present	NIH, Fine Dust Research Task Force, Head
2024-Present	NIH, Korea/Division of Cardiovascular Research, Director

Important Publications

1. Eunmi Kim, Haeni Lee, Hyun-Joung Lim, Geun-Young Kim. Comparative expression analysis of tnni1 paralogs during zebrafish embryonic development. Gene Expression Patterns 58(2026)119413
2. Han Byul Jang, Sarang Jeong, Min-Ju Kim, Hyun-Joung Lim, Kyung Eun Lee. Temporal Dietary Patterns and Frailty in Korean Older Adults: Evening-Skewed and Morning-Evening Eating Patterns Associated with Frailty Risk. Scientific Reports (2026)
3. Yu A, Yoon D, An HB, Choi U, Heo HR, Chae DH, Park HS, Park JH, Han MH, Heo J, Cho D, Park KS, Yoo HM, Kim HS, Kang KS, Oh MK, Lim HJ*, Yu KR*. The regulatory role of ACP5 in the diesel exhaust particle-induced AHR inflammatory signaling pathway in a human bronchial epithelial cell line. Scientific Reports (2025) 15:8826. (* co-corresponding)
4. Park ES, Kim BY, Lee S, Son KH, Bang J, Hong SH, Lee JW, Uhm KO, Kwak HJ, Lim HJ. Diesel exhaust particle exposure exacerbates ciliary and epithelial barrier dysfunction in the multiciliated bronchial epithelium models. Ecotoxicology and Environmental Safety 273 (2024) 116090. (Corresponding)
5. Hyun-Joung Lim, and Hyun Jeong Kwak. Selective PPARδ Agonist GW501516 Protects Against LPS-Induced Macrophage Inflammation and Acute Liver Failure in Mice via Suppressing Inflammatory Mediators. Molecules 2024, 29, 5189.
6. Bang J, Son KH, Heo HR, Park ES, Kwak HJ, Uhm KO, Chung MH, Kim YY, Lim HJ. Exogenous 8-Hydroxydeoxyguanosine Attenuates PM2.5-Induced Inflammation in Human Bronchial Epithelial Cells by Decreasing NLRP3 Inflammasome Activation. Antioxidants 2023, 12, 1189. (Corresponding)

Research Interest

Cardiovascular diseases, Cardiomyopathy, Fine Particulate Matter & Health Effects.

Biobank resources for human metabolic and vascular research

In the era of precision medicine, high-quality human biospecimens constitute the core infrastructure for metabolic and cardiovascular disease research. As multifactorial conditions shaped by the interplay of genetic predisposition and environmental factors, metabolic and cardiovascular diseases require large-scale, population-based, long-term follow-up resources and standardized biospecimens to elucidate their etiology and to advance preventive and therapeutic technologies.

The National Institute of Health (NIH) Biobank has established and continues to expand both general-population and disease-specific resource collections, which are made available to researchers nationwide. This presentation provides an overview of the academic and policy significance of biobanks, and introduces the NIH's key biobank resources and specimen distribution procedures that are directly applicable to metabolic and vascular disease researchers—thereby aiming to contribute to broader utilization of national biobank infrastructure in the field of lipid and atherosclerosis research.

CURRICULUM VITAE

Heiko Bugger

Full Professor, Vice chair Department of Cardiology, Medical University of Graz, Austria

Education and Training

2005	University of Freiburg, Germany, M.D.
2006	University of Freiburg, Germany, Ph.D., Medicine
2014	Board certification Internal Medicine
2018	Board certification Cardiology
2021	Board certification Intensive Care

Employment and Position

2005-2009	Division of Endocrinology, Metabolism and Diabetes, University of Utah School of Medicine, UT, USA, Post-Doc
2009-2018	Division of Cardiology, Heart Center Freiburg University, Freiburg, Germany, Resident/Fellow/Consultant in Internal Medicine/Cardiology
2016	Faculty of Medicine, University of Freiburg, Assistant professor
Since 2018	Division of Cardiology, Medical University of Graz, Graz, Austria, Senior Consultant in Cardiology, Interventional Cardiology
2019	Medical University of Graz, Austria, Associate professor
2021	Medical University of Graz, Austria, Full professor
2025	Medical University of Graz, Austria, Vice chair, Division of Cardiology

Important Publications

1. Bugger H, Byrne NJ, Abel ED. Animal models of dysregulated cardiac metabolism. Circ Res. 2022; 130(12):1965-1993
2. Tsushima K*, Bugger H*, Wende AR, Soto J, Jenson GA, Tor AR, McGlauflin R, Kenny HC, Zhang Y, Souvenir R, Hu XX, Black CL, Pereira RO, Lira VA, Spitzer K, Sharp TL, Shoghi KI, Sparagna GC, Rog-Zielinska EA, Kohl P, Khalimonchuk O, Schaffer JE, Abel ED. Mitochondrial Reactive Oxygen Species in Lipotoxic Hearts Induces Post-Translational Modifications of AKAP121, DRP1 and OPA1 That Promote Mitochondrial Fission. Circ Res. 2018; 122(1):58-73. *equally contributed

Leveraging biobank platforms to study cardiometabolic physiology and disease

The recognition of the interconnection of metabolic abnormalities with onset and progression of cardiovascular (CV) disease has shaped the term cardiometabolic disease, describing a heterogenous cluster of CV diseases that are linked to and driven by common metabolic conditions such as obesity, insulin resistance and type 2 diabetes. The increasing prevalence and the option to prevent such metabolic conditions drives widespread scientific research activities aiming to unravel the underlying biology of cardiometabolic disease in search of therapeutic strategies. Multiplex technologies such as multiomics, single cell sequencing, or spatial mapping of molecules within tissues allows generation of high-dimensional datasets to explore molecular circuits and mechanisms, which is particularly useful for uncovering disease physiology in humans. Most approaches require only small amounts of tissue or body fluids that can be recovered from storage, thereby allowing their scientific processing at any convenient timepoint during a study. Thus, specialized biobanks have been established in recent years, saving millions of samples, ensuring high quality tissue conservation, and providing a well structured scientific resource which can be easily accessed by both local and collaborating researchers. In this talk, insights will be provided about the Biobank Graz, how sample collection was integrated into clinical routine at our university hospital, and how clinical research benefits from such infrastructure. Integrating biobanking into clinical cohort studies opens the door for valuable scientific analyses to decipher complex disease physiology, to advance biomarker profiling and to identify treatment targets to achieve improved diagnostics, risk prediction and precision therapy in cardiometabolic disease.

CURRICULUM VITAE

Young Yang

Professor, Sookmyung Women's University, Republic of Korea

Education and Training

1988	Seoul National University, BS, Agricultural Biology
1990	Korea Advanced Institute of Science and Technology, MS, Molecular Biology
1997	Korea Advanced Institute of Science and Technology, Ph.D., Molecular Immunology
1999	NINDS, NIH, Postdoc, Neuroscience

Employment and Position

1990-2005	Korea Research Institute of Bioscience and Biotechnology, Principal Investigator
1997-1999	NINDS, NIH, MA, USA, Postdoctoral fellow
2011-2012	MD Anderson Cancer Center. FL, USA, Visiting Associate Professor
2016-2018	BK Plus Program, Chairman
2018-2020	National Research Foundation, Director
2005-Present	Dept of Biological Sciences, Sookmyung Women's University, Professor
2020-Present	Research Institute of Women's Health, Sookmyung Women's University, Head
2022-2023	the College of Science, Sookmyung Women's University, Dean
2023-Present	the PM Ethics Committee, National Research Foundation, Member
2023-Present	the LAMP Management Committee, Ministry of Education, Member
2024-Present	National Research Foundation, Special Evaluation Group for Selection Leader Research, Member

Integrative analysis of metabolic dysfunction

Sora Han, Sehwan Mun, Young Yang

Background: The genome-transcriptome-proteome-metabolome axis is a single flow of biological information, but change at one layer does not scale linearly to the next: mRNA abundance typically explains less than half of the variance in protein abundance, the remainder being determined by translational efficiency and protein half-life. β-TrCP2 (FBXW11), the substrate-recognition subunit of an SCF-type E3 ubiquitin ligase, acts precisely on that residual term — it governs how long a protein survives, not whether its gene is transcribed. Loss of β-TrCP2 stabilises rate-limiting metabolic enzymes including PFKFB3, ATGL, CPT1A and CACT, and accelerates glycolysis, lipolysis and fatty acid oxidation simultaneously. Critically, transcriptomic profiling of β-TrCP2-deficient cells fails to recover carbohydrate or lipid metabolic processes at all, because the phenotype is generated post-translationally. This makes β-TrCP2 a system in which single-layer transcriptomics is uninformative in principle, and multi-omics integration a precondition rather than a refinement of the study design. Because MASLD arises from imbalance among hepatic lipid uptake, de novo synthesis, oxidation and export — processes whose rate-limiting enzymes are ubiquitin ligase substrates — we asked whether hepatic β-TrCP2 governs susceptibility to diet-induced steatotic liver disease.

Methods: Liver-specific β-TrCP2 knockout and wild-type mice were maintained on control diet or Western diet in a two-by-two factorial design. From the same liver tissue we generated bulk RNA-sequencing, LC-MS/MS proteomics, metabolomics and spatial transcriptomics. The primary statistical axis was the genotype × diet interaction (the "reversal axis"), which asks whether the knockout reverses diet-induced change, rather than any single pairwise contrast; this distinction separates a diet-independent baseline effect of the knockout from genuine modification of the dietary response. Directional validation was performed against independent human MASLD liver transcriptomic and plasma proteomic cohorts spanning the disease spectrum.

Results: Liver-specific β-TrCP2 knockout mice were protected from Western diet-induced hepatic steatosis. Along the reversal axis, induction of the de novo lipogenesis programme (*Fasn*, *Srebf1*) and of fibrotic markers (*Col1a1*, *Acta2*) was attenuated. The cholesterol homeostasis / SREBP axis was suppressed concordantly in both the transcriptome and the proteome, providing cross-layer reproducibility from two technically independent measurements. In contrast, components of the fatty acid oxidation axis were discordant between the two layers — a pattern compatible with regulation at the post-translational or enzyme-activity level, which is the signature expected given that CPT1A and CACT are established β-TrCP2 substrates. Lysosomal and lipophagy-associated modules were coordinately upregulated, suggesting that enhanced lipid droplet catabolism may act in parallel with reduced lipogenic input. In human cohort comparison, the knockout signature was inversely correlated with the direction of progressive steatohepatitis, indicating that the murine pathway state moves away from, rather than along, the human disease trajectory.

Conclusions: Hepatic β-TrCP2 deficiency protects against diet-induced MASLD, and integrative analysis localises the effect to reduced lipogenic drive combined with enhanced lysosomal lipid catabolism. Beyond the phenotype itself, this study demonstrates the operational value of cross-layer discordance: concordance between transcriptome and proteome raises confidence in a conclusion, whereas discordance nominates the next experiment. Here it specifies which metabolomic measurements are required to convert correlation into mechanism — acylcarnitine profiling to resolve the fatty acid oxidation discordance, sterol intermediate quantification to distinguish feedback inhibition from reduced synthetic drive, and stable-isotope flux analysis to establish that the observed changes are changes in flux rather than in pool size.

Symposium 3

Inflammation in ASCVD: From LDL Lowering to Immune Modulation

Sep 10(Thu) 13:00–14:30 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Young Joon Hong (Chonnam National University, Republic of Korea)
Weon Kim (Kyung Hee University, Republic of Korea)

13:00–13:18	**LDL versus inflammation: what remains after aggressive lipid lowering?** Osung Kwon (The Catholic University of Korea, Republic of Korea)
13:18–13:36	**Emerging anti-inflammatory therapies in ASCVD: canakinumab, NLRP3 inhibitors, and beyond** SungA Bae (Yonsei University, Republic of Korea)
13:36–13:54	**Colchicine for secondary prevention in ASCVD** Dae Young Cheon (Hallym University, Republic of Korea)
13:54–14:12	**Immune mechanisms of residual cardiovascular risk: from oxidation-specific epitopes to therapeutic targets** Christoph J. Binder (Medical University of Vienna, Austria)
14:12–14:30	**Panel Discussion** Ung Kim (Yeungnam University, Republic of Korea) Chang-Hoon Woo (Yeungnam University, Republic of Korea) Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

CURRICULUM VITAE

Osung Kwon

Associate Professor,
The Catholic University of Korea, Eunpyeong St. Mary's Hospital, Republic of Korea

Education and Training

2000-2006	College of Medicine, Hallym University, M.D., Medicine
2009-2011	College of Medicine, Ulsan University, Master, Internal Medicine
2017-2020	College of Medicine, Ulsan University, Ph.D., Internal Medicine

Employment and Position

2019-2022	Division of Cardiology, Internal Medicine, Eunpyeong St. Mary's Hospital, The Catholic University of Korea, Clinical assistant professor
2022-2026	Division of Cardiology, Internal Medicine, Eunpyeong St. Mary's Hospital, The Catholic University of Korea, Assistant professor
2025-2026	Institut Cardiovasculaire Paris Sud (ICPS), Massy, France, Visiting Physician and Clinical Research Fellow
2026-Present	Division of Cardiology, Internal Medicine, Eunpyeong St. Mary's Hospital, The Catholic University of Korea, Associate professor

Important Publications

1. Hayoung Ahn 1, Sungwoo Hur 1, Cheol Hyun Lee 2, Se Hun Kang 3, Daeung Ohn 4, SookJung Kim 4, Junghoon Lee 4, Yeon-Jik Choi 4, Jeong-Eun Yi 4, Suk Min Seo 4, Sung-Won Jang 4, Won Hwa Kim 1 5, Osung Kwon. A Transformer-Based Machine Learning Framework for Risk Stratification of Left Bundle Branch Block After Transcatheter Aortic Valve Replacement; Diagnostics (Basel). 2026 May 7;16(10):1422
2. Osung Kwon, Sang-Yeub Lee, 3 Bokyoung Kim, 4 Kyungdo Han, 5 Jihyun Ahn, 6 On behalf of the Committee of Public Relations of the Korean Society of Lipid and Atherosclerosis Dyslipidemia Fact Sheet in South Korea, 2024, J Lipid Atheroscler. 2025 Sep;14(3):298-311

LDL versus inflammation: what remains after aggressive lipid lowering?

Despite remarkable advances in lipid-lowering therapy, including high-intensity statins, ezetimibe, and PCSK9 inhibitors, a substantial proportion of patients continue to experience recurrent cardiovascular events. This persistent burden, often referred to as residual cardiovascular risk, suggests that aggressive low-density lipoprotein (LDL)-cholesterol reduction alone is not sufficient to fully prevent atherosclerotic disease progression.

Among the various contributors to residual risk, inflammation has emerged as a major therapeutic target. Evidence from recent clinical trials and observational studies has consistently demonstrated that inflammatory activity, commonly assessed by high-sensitivity C-reactive protein (hs-CRP), predicts adverse cardiovascular outcomes independently of LDL-cholesterol levels. These findings have shifted the focus of secondary prevention beyond lipid lowering toward immune modulation.

This presentation explores the evolving concept of residual inflammatory risk in patients with atherosclerotic cardiovascular disease (ASCVD). Rather than considering LDL-cholesterol and inflammation as competing mechanisms, it discusses how these pathways interact and why inflammation remains clinically relevant even after optimal lipid lowering has been achieved. Contemporary evidence regarding residual cholesterol and inflammatory risk, including recent registry data and randomized clinical trials, is reviewed to illustrate both the strengths and the limitations of current evidence.

The presentation also highlights the emerging therapeutic landscape targeting vascular inflammation, providing the clinical context for subsequent discussions on colchicine and novel immune-modulating therapies. Understanding what remains after successful LDL-cholesterol lowering may help redefine future approaches to secondary prevention and support a more comprehensive strategy addressing both lipid- and inflammation-driven residual cardiovascular risk.

CURRICULUM VITAE

SungA Bae

Assistant Professor, Division of Cardiology,
Department of Internal Medicine (MD, PhD, FACC, FESC),
Yongin Severance Hospital, Yonsei University College of Medicine, Republic of Korea

Education and Training

2016-2021	Chonnam National University School of Medicine, Korea, Ph.D., Medicine (Cardiology)
2010-2014	Chonnam National University School of Medicine, Korea, M.D., Medicine
2003-2010	Chonnam National University College of Engineering, Korea, B.S., Electronic and Computer Engineering

Employment and Position

2021-Present	Yongin Severance Hospital, Yonsei University College of Medicine, Korea, Assistant Professor, Division of Cardiology
2020	Korea University Anam Hospital, Korea, Clinical Fellow, Echocardiography (Cardiology)
2019	Chonnam National University Hospital, Korea, Clinical Instructor, Department of Cardiology
2015-2018	Chonnam National University Hospital, Korea, Resident, Department of Internal Medicine
2014	Chonnam National University Hospital, Korea, Intern

Important Publications

1. Cha JJ, Bae S (co-first author), Park DW, et al. Clinical outcomes in patients with delayed hospitalization for non-ST-segment elevation myocardial infarction. J Am Coll Cardiol. 2022;79(4):311-323.
2. Lee OH, Heo SJ, Kim MH, Park JW, Bae S (corresponding author), et al. Therapeutic hypothermia in patients with acute myocardial infarction complicated by out-of-hospital cardiac arrest. BMC Med. 2025;23(1):179.
3. Han C, Kim DW, Kim S, You SC, Park JY, Bae S, Yoon D. Evaluation of GPT-4 for 10-year cardiovascular risk prediction: insights from the UK Biobank and KoGES data. iScience. 2024;27(2):109022.
4. Bae S, Cha JJ, Lim S, et al. Early invasive strategy based on the time of symptom onset of non-ST-segment elevation myocardial infarction. JACC Cardiovasc Interv. 2023;16(1):64-75.

Emerging anti-inflammatory therapies in ASCVD: canakinumab, NLRP3 inhibitors, and beyond

In 2017 the CANTOS trial gave the first proof that inflammation itself can be targeted in coronary disease. Over 10,000 post-MI patients with hs-CRP above 2 mg/L received canakinumab, an antibody against IL-1β, or placebo, and cardiovascular events fell by 15% with no change in LDL cholesterol. Low-dose methotrexate (CIRT) did nothing, so the pathway mattered, not anti-inflammatory therapy in general. Canakinumab never reached the clinic, but every agent in this talk descends from it.

One step upstream sits the NLRP3 inflammasome, the intracellular sensor that responds to cholesterol crystals and switches on IL-1β and IL-18. Colchicine reaches it indirectly and is the subject of the next lecture. Direct oral inhibitors are new. VTX3232 lowered hs-CRP by 78% at 12 weeks in a 175-patient phase 2 study in late 2025, BGE-102 lowered it by 86% within two weeks in phase 1, and NT-0796, DFV890 and dapansutrile are in phase 1 or 2. None has outcome data, and I will discuss what an outcome trial for this class would need to look like.

One step downstream sits IL-6, until this summer the cleanest-looking target of all. On July 31, 2026, ZEUS, a trial of the IL-6 antibody ziltivekimab in 6,376 patients with ASCVD, chronic kidney disease and hs-CRP of 2 mg/L or more, reported no reduction in cardiovascular death, MI or stroke (HR 0.99) despite the expected fall in IL-6 and hs-CRP. Serious infections rose. Four weeks later at ESC 2026, another IL-6 antibody, pacibekitug, lowered hs-CRP by up to 89% in TRANQUILITY, and clazakizumab is heading for phase 3 in dialysis patients. Lowering the biomarker is no longer the question.

I will close with how to read ZEUS, what ARTEMIS (after acute MI) and HERMES (in HFpEF) can still tell us in 2027, and where this leaves the clinic.

CURRICULUM VITAE

Dae Young Cheon

Assistant Professor, Hallym University Dongtan Sacred Heart Hospital, Republic of Korea

Education and Training

2006-2012	Hallym University, M.D., Internal Medicine
2018-2023	Korea National Open University, M.S., Bioinformatics & Statistics

Employment and Position

2020-2021	Division of Cardiology, Seoul National University Hospital, Clinical Fellow
2023-Present	Division of Cardiology, Hallym University Dongtan Sacred Heart Hospital, Assistant Professor

Important Publications

1. Combined Pre-Hypertension, Pre-Diabetes, and Pre-Hyperlipidemia and Long-Term Cardiovascular Risk in Young Adults: A Nationwide Cohort Study. European Journal of Preventive Cardiology. 2026. Co-corresponding author.
2. Blood Pressure Status and Risk of Cardiovascular Disease in Older Adults Aged 75+ Without Prior Cardiovascular Events: A Nationwide Cohort Study. European Journal of Preventive Cardiology. 2026. Co-corresponding author.
3. Dementia Risk Across Blood Pressure Categories: A South Korean Nationwide Study. European Heart Journal. 2025. Co-author.
4. Cardiovascular Risk Across Blood Pressure Categories Defined by the 2024 ESC and 2023 ESH Hypertension Guidelines: Insights From a Korean Nationwide Cohort Study. European Journal of Preventive Cardiology. 2025. Co-first author.
5. Association Between Metabolic Syndrome and Young-Onset Dementia: A Nationwide Population-Based Study. Neurology. 2025. Co-corresponding author.

Colchicine for secondary prevention in ASCVD

Atherosclerotic cardiovascular disease (ASCVD) is increasingly recognized as a chronic inflammatory disorder as well as a lipid-driven disease. Despite intensive low-density lipoprotein cholesterol (LDL-C) lowering and contemporary secondary prevention strategies, substantial cardiovascular risk persists. Residual inflammatory risk therefore represents a potentially modifiable pathway beyond conventional lipid management.

Low-dose colchicine has emerged as a practical anti-inflammatory strategy for secondary prevention of ASCVD. By disrupting microtubule assembly and attenuating neutrophil activation and inflammasome-mediated inflammatory signaling, colchicine targets pathways implicated in atherosclerotic plaque progression and destabilization. Landmark randomized trials, including COLCOT in patients with recent myocardial infarction and LoDoCo2 in patients with chronic coronary disease, demonstrated reductions in recurrent cardiovascular events with low-dose colchicine. More recent trials, however, have yielded heterogeneous results, emphasizing that the magnitude of benefit may depend on clinical setting, timing of treatment, patient selection, and background preventive therapy. Nevertheless, contemporary meta-analyses incorporating approximately 30,000 patients continue to demonstrate an overall reduction in major cardiovascular events, with the most consistent evidence observed in patients with established coronary artery disease.

These findings establish inflammation as a clinically actionable component of residual cardiovascular risk but also argue against indiscriminate anti-inflammatory therapy for all patients with ASCVD. The clinical role of colchicine should be considered within comprehensive secondary prevention after optimization of LDL-C lowering, antithrombotic therapy, blood pressure, metabolic risk factors, and lifestyle interventions. Appropriate patient selection, potential drug interactions, renal and hepatic function, and gastrointestinal tolerability should also be considered.

Low-dose colchicine provides proof of concept that targeting inflammation can reduce recurrent atherosclerotic events independently of further LDL-C lowering. Future strategies should move toward identifying patients with meaningful residual inflammatory risk and determining who derives the greatest net clinical benefit from targeted anti-inflammatory therapy. Colchicine therefore represents an important transition in ASCVD management—from treating atherosclerosis predominantly as a lipid-storage disorder toward simultaneously addressing its lipid and inflammatory components.

CURRICULUM VITAE

Christoph J. Binder

Professor of Atherosclerosis, Medical University of Vienna, Austria

Education and Training

1997.03	University of Vienna, Austria, M.D., Medicine
2002.09	University of California San Diego, USA, Ph.D., Molecular Pathology
2005-2013	Medical University of Vienna, Austria, Residency in Laboratory Medicine, Clinical Pathology
2013.09	Medical University of Vienna, Austria, Certified specialist, Medical and Chemical Laboratory Diagnostics

Employment and Position

2016	"BBA Molecular and Cell Biology of Lipids" Journal, Guest Editor, Special Review Series "Lipid modification and lipid peroxidation products in innate immunity and inflammation"
2016-2019	European Atherosclerosis Society, Member of the Executive Committee
Since 2017	Familial hypercholesterolemia (FH) registry of the Austrian Atherosclerosis Society, Principal Investigator
2018-2019	"Atherosclerosis" Journal, Associate Editor
Since 2018	"Thrombosis and Haemostasis" Journal, Section Editor
Since 2019	"Atherosclerosis" Journal, Co-Editor
2019-2022	Austrian Atherosclerosis Society, President
Since 2019	Medical University of Vienna, Member of the Senate
Since 2020	Medical University of Vienna, Coordinator of the PhD program "Vascular Biology"
Since 2020	Leducq Transatlantic Network of Excellence "B cells in Cardiovascular Disease", Coordinator
2021-2024	European Atherosclerosis Society, Vice-President
Since 2024	Austrian Science Fund (FWF), Vice President for Biology and Medicine

Immune mechanisms of residual cardiovascular risk: from oxidation-specific epitopes to therapeutic targets

Despite effective lipid-lowering therapies, patients with atherosclerotic cardiovascular disease (ASCVD) remain at substantial risk of recurrent cardiovascular events. Persistent vascular inflammation is a key determinant of this residual risk and involves complex interactions between innate and adaptive immune responses. Oxidation-specific epitopes (OSEs), which are generated by lipid peroxidation and exposed on oxidized LDL, dying cells and extracellular vesicles, represent major drivers of inflammation in atherosclerosis. OSEs constitute a prominent class of danger-associated molecular patterns recognized by multiple arms of humoral and cellular immunity. These include natural IgM antibodies, complement factors, soluble pattern-recognition proteins and macrophage scavenger receptors. Under physiological conditions, OSE-specific immune responses maintain tissue homeostasis by neutralizing the pro-inflammatory activities of OSEs and promoting the clearance of oxidized lipoproteins, dying cells and cellular debris. However, when these protective mechanisms are insufficient or become dysregulated, OSEs activate inflammatory and thrombotic pathways, thereby sustaining chronic arterial inflammation and promoting atherosclerotic plaque progression and its clinical complications. Natural IgM antibodies are particularly important components of this protective response. By binding OSEs, they can inhibit inflammatory signalling, facilitate the disposal of damaged structures and limit thrombosis and other downstream consequences of oxidative stress. Low levels of OSE-specific natural IgM are associated with an increased risk of cardiovascular events, whereas higher levels may provide an enhanced protective capacity against OSE-mediated inflammation. These observations identify OSEs as attractive therapeutic targets in ASCVD. Immunization strategies that induce or enhance protective OSE-specific immune responses offer a particularly promising approach. This presentation will summarize evidence linking natural IgM antibodies inversely with cardiovascular events, the mechanisms involvd and discuss new experimental approaches demonstrating that vaccination against OSE-related antigens can attenuate atherosclerosis in mice.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 4

Publication Committee Session: JLA Award for Outstanding Article

Sep 10(Thu) 13:00–14:00 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : In-Kyung Jeong (Kyung Hee University, Republic of Korea)
Hyun Kang (Chung-Ang University, Republic of Korea)

13:00–13:15	**Arterial tissue gene expression associated with telomere length attrition in coronary artery disease** Rajkumar Dorajoo (Genome Institute of Singapore, Singapore)
13:15–13:30	**Administration of sodium–glucose cotransporter–2 inhibitors following acute myocardial infarction: a systematic review, meta–analysis, and meta–regression of randomized trials** Timotius Ivan Hariyanto (Universitas Airlangga, Indonesia)
13:30–13:45	**Sex differences in the diagnosis and treatment of Japanese patients with familial hypercholesterolemia** Hayato Tada (Kanazawa University, Japan)
13:45–14:00	**Tigloylgomisin P inhibits endothelial inflammation via NFκB and Smad1/5/9 pathway regulation** Sanguk Yun (Inje University, Republic of Korea)

CURRICULUM VITAE

Rajkumar Dorajoo

Associate Professor, Lab of Metabolic Disease and Ageing Genomics,
Genome Institute of Singapore, Agency for Science, Technology and Research (A*STAR),
Singapore, Department of Paediatrics, Yong Loo Lin School of Medicine,
National University of Singapore, Singapore

Education and Training

2013.10	Imperial College London, Ph.D., Genomic Medicine

Employment and Position

2013-2018	Human Genomics, Genome Institute of Singapore, Post-doctoral Fellow
2018-2022	Human Genomics, Genome Institute of Singapore, Research Scientist
2023-Present	Department of Paediatrics, Yong Loo Lin School of Medicine, National University of Singapore, Adjunct Associate Professor
2022-Present	Metabolic Disease and Ageing Genomics, Genome Institute of Singapore, Principal Scient

Important Publications

1. Gurung RL, Wu H, Zheng H, Liu S, Le Ivan Tan J, Liu JJ, Tay JH, Sim KS, Liu JJ, Dorajoo R, Lim SC. Distinct epigenetic ageing patterns are associated with heterogeneity in kidney function decline in type 2 diabetes. Geroscience. 2026 Jul 13. doi: 10.1007/s11357-026-02396-4. PMID: 42440189.
2. Chang X, Shih CC, Chen J, Lee AS, Tan P, Wang L, Liu J, Li J, Yuan JM, Khor CC, Koh WP, Dorajoo R. Predictive capabilities of polygenic scores in an East-Asian population-based cohort: the Singapore Chinese health study. Commun Biol. 2025 Aug 15;8(1):1228. doi: 10.1038/s42003-025-08675-8. PMID: 40817126.

Arterial tissue gene expression associated with telomere length attrition in coronary artery disease

Shortened telomere length (TL) in circulating blood cells has been associated with coronary artery disease (CAD), but whether telomere attrition reflects similar biological processes within disease-relevant arterial tissues remains unclear. We investigated compartment-specific molecular signatures associated with TL in matched blood granulocytes and arterial wall tissues from patients with CAD.

We studied 155 Chinese men undergoing coronary artery bypass graft surgery. TL was measured in matched blood granulocytes and arterial wall tissues using quantitative polymerase chain reaction. Arterial wall RNA sequencing was performed in a subset of 34 patients. Participants were stratified into short- and long-TL groups according to the median TL within each tissue compartment, and differential gene expression and gene-set enrichment analyses were performed.

TL in arterial wall tissues was not significantly correlated with TL in matched granulocytes (R=0.107, P=0.19), indicating independent regulation across tissue compartments. In arterial wall tissues, TL attrition was associated with 10 significant differentially expressed genes (FDR<0.045), including upregulation of *ADRA2A*, which encodes an adrenergic receptor involved in coronary vasoconstriction. Gene-set enrichment analysis also demonstrated enrichment of the vasoconstriction pathway in arterial tissues with shorter TL (FDR=0.012). In contrast, these transcriptional signatures were not observed in granulocytes. Instead, shorter granulocyte TL was associated with negative enrichment of ATP synthesis and oxidative phosphorylation pathways, suggesting impaired mitochondrial bioenergetics.

These findings demonstrate that telomere attrition in arterial wall tissues and circulating granulocytes is associated with distinct molecular pathways in CAD. Arterial wall TL shortening is linked to vasoconstriction, whereas granulocyte TL shortening is associated with mitochondrial energy dysfunction. The lack of correlation between arterial wall and blood TL suggests that circulating telomere length does not fully reflect telomere biology within disease-relevant vascular tissue. These findings provide additional insight into the tissue-specific mechanisms of telomere attrition underlying atherosclerotic coronary artery disease.

CURRICULUM VITAE

Timotius Ivan Hariyanto

Medical Doctor (M.D.), Faculty of Medicine, Universitas Airlangga, Indonesia

Education and Training

2020.03	Faculty of Medicine, Universitas Pelita Harapan, M.D., Medicine

Employment and Position

2020-2021	Rumah Sakit Umum Daerah Tidar, Magelang, Internship
2021-2022	Siloam Hospitals Yogyakarta, Resident Medical Officer (RMO)
2022-2026	Apotek Triana Muntilan, General Practitioner
2023-2026	RSUD Bukit Menoreh Salaman, ER Doctor
2026-Now	Faculty of Medicine, Universitas Airlangga, Resident

Important Publications

1. Administration of Sodium-Glucose Cotransporter-2 Inhibitors Following Acute Myocardial Infarction: A Systematic Review, Meta-Analysis, and Meta-Regression of Randomized Trials. J Lipid Atheroscler. 2025 Sep;14(3):326-338. https://doi.org/10.12997/jla.2025.14.3.326
2. Comparative Efficacy and Safety of Radiofrequency Ablation and Microwave Ablation in the Treatment of Benign Thyroid Nodules: A Systematic Review and Meta-Analysis. Korean J Radiol. 2024 Mar;25(3):301-313. https://doi.org/10.3348/kjr.2023.1004
3. Efficacy and safety of tirzepatide as novel treatment for type 2 diabetes: A systematic review and meta-analysis of randomized clinical trials. Diabetes Metab Syndr. 2022 Nov;16(11):102640. https://doi.org/10.1016/j.dsx.2022.102640
4. Pre-admission glucagon-like peptide-1 receptor agonist (GLP-1RA) and mortality from coronavirus disease 2019 (Covid-19): A systematic review, meta-analysis, and meta-regression. Diabetes Res Clin Pract. 2021 Sep;179:109031. https://doi.org/10.1016/j.diabres.2021.109031
5. Obstructive sleep apnea (OSA) and outcomes from coronavirus disease 2019 (COVID-19) pneumonia: a systematic review and meta-analysis. Sleep Med. 2021 Apr 1;82:47-53. https://doi.org/10.1016/j.sleep.2021.03.029

Administration of sodium-glucose cotransporter-2 inhibitors following acute myocardial infarction: a systematic review, meta-analysis, and meta-regression of randomized trials

Objective: Individuals with acute myocardial infarction (AMI) face a considerable mortality risk. Recent evidence suggests that sodium-glucose cotransporter-2 inhibitors (SGLT-2i) may provide cardioprotective benefits, particularly in cases of heart failure (HF). However, the evidence regarding SGLT-2i use in AMI patients remains contradictory and ambiguous. This study investigates the efficacy and safety of SGLT-2i administration following AMI.

Methods: We systematically searched Scopus, Cochrane Library, MEDLINE, and ClinicalTrials.gov for potential articles using specific keywords, with the search extending until December 15th, 2024. All published randomized controlled trials (RCTs) assessing SGLT-2i use following AMI were included. Outcomes were expressed as risk ratios (RRs) and standardized mean differences.

Results: A total of 7 RCTs were included. Our pooled analysis demonstrated that SGLT-2i administration post-AMI was associated with a reduced risk of HF hospitalization (RR, 0.72; 95% confidence interval [CI], 0.60, 0.86; p=0.0004, I2=0%) and an improved left ventricular ejection fraction (LVEF) (SMD, 0.29; 95% CI, 0.03, 0.55; p=0.03; I2=60%) compared with placebo. However, there were no significant differences in all-cause mortality (p=0.94), cardiovascular (CV)-related mortality (p=0.77), stroke/transient ischemic attack (p=0.17), or major adverse CV events (p=0.16). The incidences of serious adverse events, diabetic ketoacidosis, and urinary tract infections were comparable between the 2 groups.

Conclusion: The administration of SGLT-2i following AMI may reduce the risk of HF hospitalization and improve LVEF, without affecting mortality outcomes. Overall, SGLT-2i proved to be a comparatively safe treatment option.

CURRICULUM VITAE

Hayato Tada

Assistant Professor, Kanazawa University, Japan

Education and Training

2003.03	Kanazawa University, Japan, M.D, Medicine
2011.11	Kanazawa University, Japan, Ph.D, Cardiovascular Medicine

Employment and Position

2003-2004	Kanazawa University Hospital, Resident
2004-2005	Kouseiren Takaoka Hospital, Resident
2005-2006	Fukui Cardiovascular Center, Clinical Fellow
2006-2012	Kanazawa University Hospital, Medical Staff
2012-2014	Massachusetts General Hospital, Center for Human Genetic Research, Research Fellow
2014-	Kanazawa University Hospital, Assistant Professor

Important Publications

1. Tada H, Kawashiri MA, Ikewaki K, Terao Y, Noguchi T, Nakanishi C, Tsuchida M, Takata M, Miwa K, Konno T, Hayashi K, Nohara A, Inazu A, Kobayashi J, Mabuchi H, Yamagishi M. Altered metabolism of low-density lipoprotein and very-low-density lipoprotein remnant in autosomal recessive hypercholesterolemia: results from stable isotope kinetic study in vivo. Circ Cardiovasc Genet. 2012 Feb 1;5(1):35-41
2. Tada H, Won HH, Melander O, Yang J, Peloso GM, Kathiresan S. Multiple associated variants increase the heritability explained for plasma lipids and coronary artery disease. Circ Cardiovasc Genet. 2014 Oct;7(5):583-7
3. Tada H, Shiffman D, Smith JG, Sjögren M, Lubitz SA, Ellinor PT, Louie JZ, Catanese JJ, Engström G, Devlin JJ, Kathiresan S, Melander O. Twelve-single nucleotide polymorphism genetic risk score identifies individuals at increased risk for future atrial fibrillation and stroke. Stroke. 2014 Oct;45(10):2856-2862
4. Tada H, Melander O, Louie JZ, Catanese JJ, Rowland CM, Devlin JJ, Kathiresan S, Shiffman D. Risk prediction by genetic risk scores for coronary heart disease is independent of self-reported family history. Eur Heart J. 2016 Feb 7;37(6):561-7

Sex differences in the diagnosis and treatment of Japanese patients with familial hypercholesterolemia

Objective: Significant sex differences have been observed in the diagnosis and treatment of familial hypercholesterolemia (FH). This study aimed to clarify sex differences among Japanese FH patients.

Methods: We assessed Japanese patients who had been clinically diagnosed with heterozygous FH (HeFH) at our hospital from 1990 to 2022 (n=1,273). We compared variables that were associated with the diagnosis and treatment of HeFH and the attainment rate of low-density lipoprotein (LDL)-cholesterol treatment target between male and female patients. We used the Cox proportional hazards model to assess factors associated with major adverse cardiac events (MACEs) between male and female sexes.

Results: Female patients diagnosed with HeFH were significantly older than male patients (51 vs. 46 years, p<0.001). The median follow-up period was 10.9 years. Compared with female patients, male patients had significantly lower median LDL-cholesterol levels at baseline (236 vs. 244 mg/dL, p<0.001) and follow-up (110 vs. 116 mg/dL, p<0.001). Female patients with HeFH were less likely to achieve the LDL-cholesterol treatment target than male patients (22% vs. 34%, p<0.001). During the follow-up period, 142 MACEs were observed. Male sex was significantly associated with MACEs (hazard ratio, 1.64; 95% confidence interval, 1.14-2.14; p<0.001). Factors associated with MACEs did not significantly differ between male and female.

Conclusion: Female patients with HeFH were diagnosed 5 years later than male patients. Moreover, they were less likely to achieve the guideline-recommended LDL-cholesterol level targets. This sex bias represents a significant barrier to clinical care.

CURRICULUM VITAE

Sanguk Yun

Assistant Professor, Inje University, Republic of Korea

Education and Training

2008.02	POSTECH, Korea, Ph.D., Biochemistry
1999.02	Yonsei University, Korea, BS, Biochemistry

Employment and Position

2020-Present	Inje University, Korea, Assistant Professor
2016-2020	Yale University, USA, Research Scientist

Important Publications

1. Minjeong Shin, Junhyeon Ku, Jenita Immanuel, Nayeong Kwon and Sanguk Yun (2025) Tigloylgomisin P Inhibits Endothelial Inflammation by Regulating the NF-κB and Smad1/5/9 Pathways. J Lipid Atheroscler. 15(1):173-182.
2. Yoon Ji Choi, Jimin An, Ji Hye Kim, Sa Bin Lee, Bo Seok Lee, Chae Young Eom, Hyohi Lee, Nayeong Kwon, Il Shin Kim, Kyoung-Su Park, Sooah Park, Jung-Woog Shin, Sanguk Yun (2024) Mexenone protects mice from LPS-induced sepsis by EC barrier stabilization. PLos One 19(5):e0302628.
3. Sanguk Yun, Rui Hu, Melanie E. Schwaemmle, Alexander N. Scherer, Zhenwu Zhuang, Anthony J. Koleske, David C. Pallas, and Martin A. Schwartz (2019) Integrin α5β1 regulates PP2A complex assembly through PDE4D in atherosclerosis. Journal of Clinical Investigation 130. pii: 127692.
4. Sanguk Yun, Madhusudhan Budatha, James E. Dahlman, Brian G. Coon, Ryan T. Cameron, Robert Langer, Daniel G. Anderson, George Baillie and Martin A. Schwartz (2016) Interaction between integrin a5 and PDE4D regulates endothelial inflammatory signaling. Nature Cell Biology 18, 1043-1053.

Research Interest

- Vascular inflammation and atherosclerosis
- Fluid shear stress-dependent mechanotransduction
- ECM/integrin-dependent endothelial signaling and phenotype

Tigloylgomisin P inhibits endothelial inflammation via NFκB and Smad1/5/9 pathway regulation

Vascular inflammation underlies many chronic human diseases. Inflammatory stimuli such as Interleukin-1β (IL-1β) or disturbed blood flow induce endothelial activation, promoting leukocyte recruitment and transmigration through inflammatory signaling. We aimed to identify novel compounds that can block vascular inflammation, which would be useful for treatment of vascular inflammatory diseases such as atherosclerosis. Natural compound library was screened to identify drug candidates that can block IL-1β-induced endothelial inflammation. Anti-inflammatory effects of tigloylgomisin P, one of hit compounds, were tested in BAECs (Bovine aortic endothelial cells) stimulated with IL-1β or oscillatory (disturbed) flow. Endothelial inflammation was examined by measuring NFκB phosphorylation and nuclear translocation, monocyte adhesion to endothelial monolayers and Smad1/5/9 phosphorylation in vitro. Vascular inflammation was also examined in the aortas of ApoE knockout mice administered with tigloylgomisin P using immunohistochemistry. Tigloylgomisin P suppressed IL-1β-induced NFκB activation and monocyte adhesion. Furthermore, it blocked oscillatory shear stress-induced endothelial inflammation mediated by NFκB activation and Smad1/5/9 phosphorylation. Administration of tigloylgomisin P reduced inflammatory marker proteins in atheroprone inner curvatures of aortic arches in mice. Our findings suggest that tigloylgomisin P may serve as a therapeutic agent for vascular inflammatory diseases such as atherosclerosis.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 5

FH/Severe Dyslipidemia: Optimizing FH Management

Sep 10(Thu) 14:30–16:00 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : **Sang-Hak Lee** (Yonsei University, Republic of Korea)
Hayato Tada (Kanazawa University, Japan)

14:30–14:48	**Refined risk assessment in FH** **Sayaka Funabashi** (Kyorin University, Japan)
14:52–15:10	**Cause of death in FH** **Chan Joo Lee** (Yonsei University, Republic of Korea)
15:14–15:32	**Experience on new drugs for FH: including lomitapide & evinacumab** **Hayato Tada** (Kanazawa University, Japan)
15:36–15:54	**Genome editing for precision medicine** **HyongBum Kim** (Yonsei University, Republic of Korea)
	Panel **Sung Hee Choi** (Seoul National University, Republic of Korea)

CURRICULUM VITAE

Sayaka Funabashi

Assistant Professor, Kyorin University, Japan

Education and Training

2011.03	Kyorin University, Japan, M.D., Medicine
2023.03	Kyorin University, Japan, Ph.D., Cardiovascular Medicine

Employment and Position

2011-2013	Kyorin University Hospital, Japan, Internship
2013-2016	Kyorin University Hospital, Japan, Resident
2016-2019	National Cerebral and Cardiovascular Center, Japan, Resident
2019-2021	National Cerebral and Cardiovascular Center, Japan, Fellow
2021-2022	Kyorin University Hospital, Japan, Clinical Fellow
2022-	Kyorin University, Japan, Assistant Professor

Important Publications

1. Funabashi S, Kataoka Y, Nicholls SJ, Kitahara S, Makino H, Matsubara M, Matsuo M, Omura-Ohata Y, Koezuka R, Tochiya M, Tamanaha T, Tomita T, Honda-Kohmo K, Noguchi M, Murai K, Sawada K, Iwai T, Matama H, Honda S, Fujino M, Nakao K, Yoneda S, Takagi K, Otsuka F, Asaumi Y, Hosoda K, Yasuda S, Noguchi T. Circulating apolipoprotein B levels in statin-treated type 2 diabetic patients with coronary artery disease: Implications for coronary atheroma progression and instability. J Clin Lipidol. 2025:S1933-2874(25)00279-X.
2. Funabashi S, Kataoka Y, Hori M, Ogura M, Doi T, Morita Y, Kiyoshige E, Nishimura K, Noguchi T, Harada-Shiba M. Asymptomatic Intracranial Artery Stenosis/Occlusion in Heterozygous Familial Hypercholesterolemia: Its Frequency and Implications for Cerebrovascular and Cardiovascular Events. J Am Heart Assoc. 2024;13:e033972.
3. Funabashi S, Kataoka Y, Hori M, Ogura M, Doi T, Noguchi T, Harada-Shiba M. Characterization of Polyvascular Disease in Heterozygous Familial Hypercholesterolemia: Its Association with Circulating Lipoprotein(a) Levels. J Am Heart Assoc. 2022:e025232.
4. Funabashi S, Kataoka Y, Hori M, Ogura M, Nakaoku Y, Nishimura K, Doi T, Nishikawa R, Tsuda K, Noguchi T, Harada-Shiba M. Substantially Elevated Atherosclerotic Risks in Japanese Severe Familial Hypercholesterolemia Defined by the International Atherosclerosis Society. JACC Asia. 2021;1:245-255.

Refined risk assessment in FH

I am a cardiologist in Japan with a clinical and research interest in familial hypercholesterolemia (FH), lipoprotein(a) [Lp(a)], and atherosclerotic cardiovascular disease (ASCVD). My research has focused particularly on cardiovascular risk stratification in patients with FH.

FH is characterized by lifelong exposure to elevated LDL cholesterol and a substantially increased risk of ASCVD. However, cardiovascular risk varies considerably among patients with FH, highlighting the importance of individualized risk assessment.

In this presentation, I will discuss three approaches to refining risk stratification in FH: genetic, clinical, and imaging-based assessment. Genetic factors, including monogenic variants and polygenic background, contribute to heterogeneity in cardiovascular risk. Clinical risk assessment remains essential, particularly when genetic testing is not readily available. I will also present our studies of Severe FH in Japanese patients and the FRISK-Lp(a) score, a simple clinical score developed to predict first ASCVD events. Finally, imaging modalities, including coronary artery calcium scoring, coronary CT angiography, and carotid plaque assessment, can directly evaluate subclinical atherosclerotic burden.

Integrating genetic, clinical, and imaging information may enable more individualized cardiovascular risk assessment and preventive strategies for patients with FH.

CURRICULUM VITAE

Chan Joo Lee

Professor, Division of Cardiology, Severance Hospital,
Yonsei University College of Medicine, Republic of Korea

Education and Training

2010.3-2015.2	Graduate School, Yonsei University, Seoul, Republic of Korea, Ph.D., Cellular and Molecular Biology
2007.9-2010.2	Graduate School, Yonsei University, Seoul, Republic of Korea, M.S., Internal Medicine
1999.3-2005.2	Yonsei University College of Medicine, Seoul, Republic of Korea, M.D., Medicine

Employment and Position

2026.2-Present	Division of Cardiology, Department of Internal Medicine, Severance Hospital, Yonsei University College of Medicine, Professor
2025.3-2026.2	Division of Cardiology, Department of Internal Medicine, Severance Hospital, Yonsei University College of Medicine, Clinical Professor
2020.3-2025.2	Division of Cardiology, Department of Internal Medicine, Severance Hospital, Yonsei University College of Medicine, Clinical Associate Professor
2019.3-2020.2	Division of Cardiology, Department of Internal Medicine, Severance Hospital, Yonsei University College of Medicine, Clinical Assistant Professor
2017.3-2019.2	Department of Health Promotion, Severance Hospital, Yonsei University College of Medicine, Clinical Assistant Professor
2016.3-2017.2	Division of Cardiology, Department of Internal Medicine, Severance Hospital, Yonsei University College of Medicine, Clinical Research Assistant Professor

Cause of death in FH

Familial hypercholesterolemia (FH) causes lifelong exposure to very high LDL-cholesterol and is uniformly labeled a high-risk condition. Yet cardiovascular risk within FH is not homogeneous, and general-population risk equations, such as PREVENT and SCORE2, poorly capture the cumulative burden that defines these patients. Cohorts including SAFEHEART have shown that observed atherosclerotic events vary widely among individuals with the same diagnosis, and a "resilient" subset remains event-free. Despite this, a fundamental question is rarely addressed: what do patients with severe hypercholesterolemia actually die of, and does the cause of death differ within this population?

This lecture presents evidence from a Korean nationwide cohort of 3,753,191 statin-naive adults followed for a median of 10.3 years, in which cause-specific mortality was examined across LDL-cholesterol categories, including a very severe category (≥225 mg/dL) enriched for an FH phenotype. Although cancer remained the leading cause of death at every LDL-cholesterol level, higher LDL-cholesterol was associated with a selective, disproportionate increase in cardiovascular mortality (+114% in the very severe category versus +13.5% for cancer), reshaping the distribution of causes of death. This excess was specific to cardiovascular causes and driven predominantly by ischemic heart disease.

Crucially, the cardiovascular mortality risk was markedly heterogeneous. Through prespecified subgroup analyses, the association was substantially stronger in men than women and steepest in young adults, in whom ischemic heart disease mortality showed the highest relative risk. These patterns suggest that sex, age, and the timing of exposure shape not only whether but how patients with severe hypercholesterolemia die.

The talk will integrate these findings with current concepts of FH risk stratification, arguing that "severe FH" is not a single risk. Recognizing cause-specific and subgroup-specific heterogeneity may refine how we prioritize early, individualized lipid-lowering management in the highest-risk phenotypes, particularly young men.

CURRICULUM VITAE

Hayato Tada

Assistant Professor, Kanazawa University, Japan

Education and Training

2003.03	Kanazawa University, Japan, M.D, Medicine
2011.11	Kanazawa University, Japan, Ph.D, Cardiovascular Medicine

Employment and Position

2003-2004	Kanazawa University Hospital, Resident
2004-2005	Kouseiren Takaoka Hospital, Resident
2005-2006	Fukui Cardiovascular Center, Clinical Fellow
2006-2012	Kanazawa University Hospital, Medical Staff
2012-2014	Massachusetts General Hospital, Center for Human Genetic Research, Research Fellow
2014-	Kanazawa University Hospital, Assistant Professor

Important Publications

1. Tada H, Kawashiri MA, Ikewaki K, Terao Y, Noguchi T, Nakanishi C, Tsuchida M, Takata M, Miwa K, Konno T, Hayashi K, Nohara A, Inazu A, Kobayashi J, Mabuchi H, Yamagishi M. Altered metabolism of low-density lipoprotein and very-low-density lipoprotein remnant in autosomal recessive hypercholesterolemia: results from stable isotope kinetic study in vivo. Circ Cardiovasc Genet. 2012 Feb 1;5(1):35-41
2. Tada H, Won HH, Melander O, Yang J, Peloso GM, Kathiresan S. Multiple associated variants increase the heritability explained for plasma lipids and coronary artery disease. Circ Cardiovasc Genet. 2014 Oct;7(5):583-7
3. Tada H, Shiffman D, Smith JG, Sjögren M, Lubitz SA, Ellinor PT, Louie JZ, Catanese JJ, Engström G, Devlin JJ, Kathiresan S, Melander O. Twelve-single nucleotide polymorphism genetic risk score identifies individuals at increased risk for future atrial fibrillation and stroke. Stroke. 2014 Oct;45(10):2856-2862
4. Tada H, Melander O, Louie JZ, Catanese JJ, Rowland CM, Devlin JJ, Kathiresan S, Shiffman D. Risk prediction by genetic risk scores for coronary heart disease is independent of self-reported family history. Eur Heart J. 2016 Feb 7;37(6):561-7
5. Tada H, Kawashiri MA, Nohara A, Inazu A, Mabuchi H, Yamagishi M. Impact of clinical signs and genetic diagnosis of familial hypercholesterolaemia on the prevalence of coronary artery disease in patients with severe hypercholesterolaemia. Eur Heart J. 2017 May 21;38(20):1573-1579

Awards and Honors

1. Japanese Circulation Society Young Investigator's Award (1st Prize), 2017
2. Japanese College of Cardiology Young Investigator's Award (2nd Prize), 2017
3. 15th Japan Atherosclerosis Society Goto Yuichiro Award, 2020

Experience on new drugs for FH: including lomitapide & evinacumab

Homozygous FH has long been a difficult-to-treat, refractory condition, with poor prognosis due to complications like very early-onset heart attacks. However, with the development of statins, ezetimibe, PCSK9 inhibitors, and the advent of LDL apheresis, LDL-C levels have improved, and the outlook has been getting better. In this context, more recently, new LDL-C lowering drugs that work independently of LDL receptors, like lomitapide and evinacumab, have become available, leading to a potential paradigm shift in treatment for this condition. Using a comprehensive treatment approach, including these drugs, early on now allows us to almost completely control the complications of this disease. In this talk, I will share the history of diagnosing and treating homozygous FH in Japan, the current status of genetic testing, and usage data for lomitapide and evinacumab. By properly understanding these drugs' characteristics, not only can we treat the condition more effectively, but early diagnosis becomes more valuable, and we hope it will promote broader screening as well.

CURRICULUM VITAE

HyongBum Kim

Professor, Yonsei University, Republic of Korea

Education and Training

2001.02	Yonsei University, Korea, M.D., General Medicine
2006.02	Yonsei University, Korea, Ph.D., Nano-science and technology

Employment and Position

2007-2010	Emory/Tufts University, USA, Postdoctoral Fellow
2010-2011	CHA University, Korea, Assistant Professor
2011-2015	Hanyang University, Korea, Assistant Professor, Associate Professor
2015-Present	Yonsei University, Korea, Assistant Professor, Associate Professor, Professor

Important Publications

1. Oh HC, Chang Y, Park J, Cheong Y, Lee KS, Han H, Kim HH+ (+Corresponding author). High-resolution functional mapping of androgen receptor variants. Nat. Biomed. Eng. 2026. doi: 10.1038/s41551-026-01647-1.Online ahead of print.
2. Jung Y, Yu G, Oh H-C, Lee J-H, Jeon E, Bae J, Sim T, Kim HH+ (+Corresponding author). Comprehensive resistance profiling of chronic myeloid leukaemia associated ABL1 variants against five tyrosine kinase inhibitors using prime editing. Nat. Biomed. Eng. 2025. doi: 10.1038/s41551-025-01531-4. Online ahead of print.
3. Yeo JH, Lee S, Kim S, Min JG, Gopalappa R, Oh HC, Kim HK, Nam EJ, Kim HH+. (+Corresponding author). High-throughput evaluation of in vitro CRISPR activities enables optimized large-scale multiplex enrichment of rare variants. Nat. Biomed. Eng. 2025. Online ahead of print.
4. Kim HK, Kim HH+. (+Corresponding author). Evaluation and prediction of guide RNA activities in genome-editing tools. Nat. Rev. Bioeng. 2025. https://doi.org/10.1038/s44222-025-00352-z
5. Lee KS, Min J-G, Cheong Y, Oh HC, Jung SY, Park JI, Song M, Seo JH, Cho SR, Kim HH+.(+Corresponding author). Functional assessment of all ATM SNVs using prime editing and deep learning. Cell. 2025; 188(18):5081-5099.

Genome editing for precision medicine

Precision medicine aims to tailor prevention and treatment to each patient's genome, but its progress is limited by variants of uncertain significance (VUS). Nearly half of the variants reported by clinical genetic testing are classified as VUS, leaving clinicians unable to tell whether a given mutation raises disease risk or alters drug response. When a patient carries a single missense variant in a cancer-predisposition gene such as ATM, it is often impossible to say whether her cancer risk is truly elevated.

In this lecture, I will describe how genome editing can resolve this uncertainty at scale. Using prime editing, we install every possible single-nucleotide variant of a target gene within its native genomic context and measure the functional consequence of each, an approach we implemented as PrimeScan and PEER-seq (prime editing and endogenous region sequencing). Coupling these high-throughput measurements with deep-learning models, DeepPrime and DeepATM, allows functional scores to be extended to variants that have not yet been tested experimentally.

I will present two clinical applications. First, in EGFR-mutant non-small-cell lung cancer, we profiled the drug resistance of EGFR protein variants across the tyrosine kinase inhibitor-interacting domain against afatinib and osimertinib, with and without the T790M background, expanding coverage of variant-drug pairs from roughly 1% to about 95% and enabling prediction of tyrosine kinase inhibitor response. Second, we experimentally scored 23,092 single-nucleotide variants of ATM and used DeepATM to predict the remainder; among more than 420,000 UK Biobank participants without a prior cancer history, carriers of functionally damaging variants showed a significantly higher cancer risk.

Together, these results show that prime editing combined with deep learning can convert variants of uncertain significance into clinically actionable information, supporting drug selection and genetic risk stratification, and they illustrate a broadly generalizable route toward genome-editing-enabled precision medicine.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 6

Neurocardiometabolic Benefits of GLP1 RA and SGLT2 Inhibitors

Sep 10(Thu) 14:30-16:00 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Geun Sik Hong (Inje University, Republic of Korea)
Kwang-Yeol Park (Chung-Ang University, Republic of Korea)

14:30-14:50	**GLP1 RA for stroke prevention** Kang-Ho Choi (Chonnam National University, Republic of Korea)
14:50-15:10	**SGLT2 inhibitor for stroke survivors** Jeong-Min Kim (Seoul National University, Republic of Korea)
15:10-15:30	**GLP-1 receptor agonists in Alzheimer's disease** Sang-Won Han (Seoul National University, Republic of Korea)
15:30-16:00	**Panel Discussion** Bum Joon Kim (University of Ulsan, Republic of Korea) Hack-Lyoung Kim (Seoul National University, Republic of Korea) Se-eun Park (Sungkyunkwan University, Republic of Korea)

CURRICULUM VITAE

Kang-Ho Choi

Professor, Chonnam National University Hospital, Republic of Korea

Education and Training

2005.02	Chonnam National University, Korea, M.D., Neurology
2011.02	Chonnam National University, Korea, Ph.D., Neurology

Employment and Position

2010-2011	Chonnam National University Hospital, Korea, Clinical Fellowship
2013-2013	Bristol University, United Kingdom, Research Fellowship
2011-2016	Chonnam National University Hospital, Korea, Assistant Professor
2016-2021	Chonnam National University Hospital, Korea, Associate Professor
2021-2026	Chonnam National University Hospital and Medical School, Korea, Associate Professor
2026-Present	Chonnam National University Hospital and Medical School, Korea, Professor

Important Publications

1. Nagareddy R, Sundaram A, Kim JH, Kim JH, Vasvani S, Kweon S, Park IK, Jeong YY, Choi KH (Corresponding). ROS-responsive nanocarriers for the delivery of atorvastatin in ischemic MCAO rats promoting neuroprotection. Mater Today Bio (IF 11.0). 2026 May 28;38:103296.
2. Kim JH, Kim JH, Ahmad HA, Kim H, Nagareddy R, Choi KH (Corresponding), Yoon J, Jeong YY. Effects of atorvastatin-loaded PEGylated liposomes delivered by magnetic stimulation for stroke treatment. Brain Stimul (IF 9.7). 2025 Sep-Oct;18(5):1620-1631.
3. Jung J, Kim JH, Seo JW, Lee HR, Kim HS, Park MS, Choi KH (Corresponding). Clopidogrel May Be Superior to Aspirin as Maintenance Antiplatelet Monotherapy in Patients with Non-Cardioembolic Ischemic Stroke. J Stroke (IF 11.0). 2025;27(3):422-425.
4. Kim JH, Park HJ, Cho SG, Park KS, Choi KH (Corresponding). 68Ga-FAPI PET in Visualizing Acute Ischemic Stroke at Risk of Hemorrhagic Transformation. Clin Nucl Med (IF 9.6). 2025 Jan 1;50(1):56-57.
5. Nagareddy R, Kim JH, Kim JH, Thomas RG, Choi KH (Corresponding), Jeong YY. Reactive Oxygen Species-Responsive Chitosan-Bilirubin Nanoparticles Loaded with Statin for Treatment of Cerebral Ischemia. Biomater Res (IF 9.8). 2024 Oct 24;28:0097.

GLP1 RA for stroke prevention

Ischemic stroke remains a leading cause of global morbidity and mortality, heavily driven by metabolic risk factors including Type 2 Diabetes Mellitus (T2DM) and obesity. Recently, Glucagon-Like Peptide-1 receptor agonists (GLP-1 RAs) have rapidly evolved from traditional glycemic and weight management therapies into powerful agents for cerebrovascular protection. This lecture examines the emerging clinical paradigm of utilizing GLP-1 RAs specifically for stroke prevention.

The presentation will delve into the multifaceted atheroprotective mechanisms of GLP-1 RAs that directly benefit the cerebrovascular system. We will explore their vital role in significant blood pressure reduction, improvement of endothelial dysfunction, attenuation of systemic and neuro-inflammation, and the stabilization of atherosclerotic plaques, particularly within the carotid arteries. Emerging preclinical and translational data also suggest direct neuroprotective properties, indicating that GLP-1 RAs can cross the blood-brain barrier to potentially limit infarct volume, decrease oxidative stress, and support neural cell survival following an acute ischemic event.

Furthermore, this session will synthesize compelling evidence from landmark cardiovascular outcome trials (CVOTs). These key trials have demonstrated that GLP-1 RAs confer a substantial relative risk reduction in stroke incidence among high-risk patient populations, largely independent of their glucose-lowering effects. Consequently, these robust findings are prompting a critical paradigm shift in both primary and secondary stroke prevention strategies. By bridging underlying pathophysiology with clinical data, this lecture aims to equip clinicians with a comprehensive understanding of how to strategically implement GLP-1 RAs to mitigate cerebrovascular risk and optimize long-term neurological outcomes.

CURRICULUM VITAE

Jeong-Min Kim

Professor, Seoul National University Hospital, Republic of Korea

Education and Training

2003.02	Seoul National University, M.D., Medicine
2013.02	Seoul National University, Ph.D., Medicine

Employment and Position

2003-2008	Seoul National University Hospital, Resident
2013-2020	Chung-Ang University, Associate Professor
2020-Present	Seoul National University Hospital, Professor

Important Publications

1. Yang W, Hong SA, Kim JM, Jeong HB, Nam TK, Choi HH, Kim SM, Park KY, Kim HR. The immunologic phenotype of thrombi is associated with future vascular events after cerebral infarction. J Neurointerv Surg. 2023; jnis-2023-020155.
2. Kim JM, Moon J, Yu JS, Park DK, Jung KH. Therapeutic Target MicroRNA Identification Based on Circular RNA Expression Signature After Intracerebral Hemorrhage. Mol Neurobiol. 2024;61:908-918.
3. Yang W, Kim JM, Chung M, Ha J, Kang DW, Lee EJ, Jeong HY, Jung KH, Sung H, Paeng JC, Lee SH. Yang W, Kim JM, Chung M, Ha J, Kang DW, Lee EJ, Jeong HY, Jung KH, Sung H, Paeng JC, Lee SH. Sodium-Glucose Cotransporter 2 Inhibitor Improves Neurological Outcomes in Diabetic Patients With Acute Ischemic Stroke. J Stroke. 2024 May;26(2):342-346. doi: 10.5853/jos.2023.04056.
4. Kim JM, Jeong HB, Moon Y, Hong S, Nam TK, Choi HH, Jung KH, Park KY, Lee R, Seok JW, Kim HR. Role of MicroRNAs in Thrombus and Future Vascular Events Among Patients With Stroke. J Stroke. 2025 Sep;27(3):413-417. doi: 10.5853/jos.2025.00150. Epub 2025 Sep 17.
5. Bae J, Lee EJ, Kang DW, Yang W, Jeong HY, Choi KS, Lee SH, Jung KH, Kim JM. Distinction between acute and chronic carotid disease based on computed tomography angiography in acute ischemic stroke patients with carotid artery occlusion. BMC Neurol. 2026 Feb 27;26(1):136. doi: 10.1186/s12883-026-04768-x.

Awards and Honors

1. Best Editor Award, Journal of Lipid and Atherosclerosis, 2026

Research Interest

Atherosclerosis, Stroke, Dementia, Biomarker

SGLT2 inhibitor for stroke survivors

Stroke survivors remain at high risk for recurrent vascular events despite optimal secondary prevention strategy, and there is no specific treatment modality which can prevent neurological degeneration following stroke. Sodium glucose cotransporter 2 inhibitors, originally developed as glucose lowering agents, have demonstrated robust tissue protective effects in the heart and kidneys, reducing hospitalization for heart failure and slowing the progression of chronic kidney disease. Recent translational and clinical evidence also suggests these benefits may extent to the neurovascular system.

This lecture will synthesize the latest evidence on SGLT2 inhibitor among stroke patients, contextualizing their role within the paradigm of cardiovascular kidney metabolic protection.

CURRICULUM VITAE

Sang-Won Han

Clinical Assistant Professor, Seoul Metropolitan Government-Seoul National University Boramae Medical Center, Republic of Korea

Education and Training

2016.02	CHA University, Korea, M.D., Medicine
2026.02	Seoul National University, Korea, Ph.D., Translational Medicine

Employment and Position

2017.3-2021.2	Seoul National University Bundang Hospital, Korea, Resident
2021.3-2022.2	Seoul National University Bundang Hospital, Korea, Fellow
2022.3-2023.2	Chung-Ang University Gwangmyeong Hospital, Korea, Clinical Assistant Professor
2023.3-2025.2	Chuncheon Sacred Heart Hospital, Korea, Clinical Assistant Professor
2025.3-2026.8	Soonchunhyang University Seoul Hospital, Korea, Assistant Professor
2026.9-	Seoul Metropolitan Government-Seoul National University Boramae Medical Center, Korea, Clinical Assistant Professor

GLP-1 receptor agonists in Alzheimer's disease

Recent advances in Alzheimer's disease (AD) research have highlighted metabolic dysfunction, particularly brain insulin resistance, as an important contributor to disease pathogenesis beyond the classical amyloid-β and tau hypotheses. Among emerging therapeutic strategies, glucagon-like peptide-1 receptor agonists (GLP-1RAs), originally developed for type 2 diabetes and obesity, have attracted considerable attention because of their metabolic and neuroprotective properties.

This lecture will provide a comprehensive overview of the biological mechanisms underlying GLP-1RA therapy in AD and critically review the current evidence from preclinical and clinical studies. The presentation will first discuss how impaired insulin signaling contributes to tau hyperphosphorylation, neuroinflammation, and synaptic dysfunction, and how GLP-1 receptor activation restores insulin-related signaling through the PI3K-Akt pathway while suppressing GSK-3β activity and inflammatory responses. Key pharmacological issues, including blood-brain barrier penetration and central nervous system target engagement, will also be addressed.

The lecture will then summarize representative preclinical studies demonstrating that GLP-1RAs improve cognition and reduce AD-related pathology in animal models. Subsequently, clinical evidence from the ELAD Phase 2b trial and the large Phase 3 EVOKE and EVOKE+ studies of oral semaglutide will be reviewed. Although these trials demonstrated favorable effects on several cerebrospinal fluid biomarkers associated with tau pathology, neuroinflammation, and neurodegeneration, they failed to produce significant clinical benefits, highlighting the translational challenges between experimental models and human disease.

Finally, potential explanations for these discrepancies, including limited brain drug delivery, disease heterogeneity, treatment timing, and differences between peripheral metabolic regulation and central neuroprotection, will be discussed. The lecture will conclude by introducing future therapeutic directions, including dual GLP-1/GIP receptor agonists, alternative drug delivery strategies, metabolically targeted patient selection, and next-generation combination therapies, emphasizing both the opportunities and current limitations of GLP-1-based treatments for AD.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 7

Real-world LDL-C Management: From Guideline to Prescription

Sep 10(Thu) 16:10-17:40 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Youngkeun Ahn (Chonnam National University, Republic of Korea)
Min Kyong Moon (Seoul National University, Republic of Korea)

16:10-16:28	**Choosing the right combination therapy: statin, ezetimibe, PCSK9 inhibitor, or beyond?** Kyung Hoon Cho (Chonnam National University, Republic of Korea)
16:28-16:46	**Korean Fact Sheet: Current status of LDL-C control from general population to high-risk groups** Ye Seul Yang (Seoul National University, Republic of Korea)
16:46-17:04	**Korean Fact Sheet: Real-world utilization of lipid-lowering agents** Kyuho Kim (The Catholic University of Korea, Republic of Korea)
17:04-17:22	**Why are LDL-C targets still unmet? common errors and practical solutions in the clinic** Sang Yeub Lee (Chung-Ang University, Republic of Korea)
17:22-17:40	**Panel Discussion** Young Soo Lee (Daegu Catholic University, Republic of Korea) Jun Hwan Cho (Chung-Ang University, Republic of Korea) Sangmo Hong (Hanyang University, Republic of Korea)

CURRICULUM VITAE

Kyung Hoon Cho

Associate Professor, Chonnam National University Medical School, Republic of Korea

Education and Training

2006.02	Chonnam National University, Korea, M.D, Medicine
2020.02	Chonnam National University, Korea, Ph.D., Internal Medicine

Employment and Position

2007-2011	Chonnam National University Hospital, Residency in Internal Medicine
2011-2014	Ministry of Defence, Captain
2014-2015	Chonnam National University Hospital, Fellowship training in Cardiology
2015-2018	Gangjin Medical Center, Staff
2024-2025	Monash University Victorian Heart Institute, Australia, Research Fellow
2023-Present	Chonnam National University Medical School, Associate Professor

Important Publications

1. 2026 Honda S, Cho KH, Lee SY et al. Prognostic Value of BARC-Defined Bleeding in East Asian Acute Myocardial Infarction Patients: Evidence From Multicentre Registries in Korea and Japan. Thrombosis and Haemostasis. 2026 Jan 6. doi: 10.1055/a-2773-5644.
2. 2026 Cho KH, Cho YR, Lee SR, et al. Physician Disagreement With Guidelines as a Critical Barrier to Achieving LDL-C Targets in Very High-Risk Patients: An Implementation Science Study. J Korean Med Sci. 2026 Mar 2;41(8):e74.
3. 2025 Cho KH, Ji YH, Joo S, et al. Novel Artificial Intelligence Model Using Electrocardiogram for Detecting Acute Myocardial Infarction Needing Revascularization. Eur Heart J Digit Health. 2025 May 13;6(4):608-618.
4. 2022. Cho KH, Kim MC, Choo EH et al. Impact of Low Baseline Low-Density Lipoprotein Cholesterol on Long-Term Postdischarge Cardiovascular Outcomes in Patients With Acute Myocardial Infarction. J Am Heart Assoc. 2022;11:e025958.
5. 2021. Cho KH. Han X, Ahn JH et al. Long-Term Outcomes of Patients With Late Presentation of ST-Segment Elevation Myocardial Infarction. J Am Coll Cardiol 2021;77:1859-70.

Choosing the right combination therapy: statin, ezetimibe, PCSK9 inhibitor, or beyond?

Low-density lipoprotein cholesterol (LDL-C) is the central causal and modifiable driver of atherosclerotic cardiovascular disease (ASCVD), and contemporary guidelines recommend progressively lower LDL-C goals for patients at high and very high risk. Statins remain the foundation of lipid-lowering therapy, but statin monotherapy—even at high intensity—fails to achieve guideline-recommended goals in half or more of very-high-risk patients in real-world practice. A growing body of randomized evidence, including the IMPROVE-IT, FOURIER, ODYSSEY OUTCOMES, and CLEAR Outcomes trials, demonstrates that adding non-statin agents with proven cardiovascular benefit —ezetimibe, proprotein convertase subtilisin/kexin type 9 (PCSK9) monoclonal antibodies, and bempedoic acid—produces incremental event reduction proportional to the additional absolute LDL-C lowering achieved. The Korean RACING trial and recent meta-analyses further suggest that upfront moderate-intensity statin plus ezetimibe combination is at least as effective as, and better tolerated than, high-intensity statin monotherapy. Reflecting this evidence, the 2025 European Society of Cardiology/European Atherosclerosis Society focused update and the 2026 American College of Cardiology/American Heart Association multisociety guideline endorse earlier and more intensive combination strategies, particularly during hospitalization for acute coronary syndrome. This presentation summarizes the pharmacology and outcome evidence for currently available LDL-C-lowering agents, appraises the evidence supporting early combination therapy, and proposes a practical, goal-directed framework for selecting the right combination —statin, ezetimibe, PCSK9 inhibitor, bempedoic acid, or inclisiran—for the individual patient, with particular attention to Korean and East Asian perspectives.

CURRICULUM VITAE

Ye Seul Yang

Associate Professor, Seoul National University, Republic of Korea

Education and Training

2012.02	Hallym University, Korea, M.D., Medicine
2024.08	Seoul National University, Korea, Ph.D., Medicine

Employment and Position

2020-2022	The Catholic University of Korea, Korea, Clinical Assistant Professor
2022-	Seoul National University, Korea, Assistant Professor

Important Publications

1. Yang YS, Ku EJ, Ko SH, Rhee EJ, Ihm SH, Choi SH, Lee WY; Committee of Clinical Practice Guideline; Korean Society of Cardio-cerebrovascular Disease Prevention. Physical activity for prevention of cardiovascular disease: consensus statement of Korean Society of Cardio-cerebrovascular Disease Prevention. Korean J Intern Med. 2025 Sep;40(5):696-709.
2. Yang YS, Han K, Cheon DY, Lee M. Abdominal obesity and the risk of young-onset dementia in women: a nationwide cohort study. Alzheimers Res Ther. 2025 Apr 21;17(1):86.
3. Yang YS, Kim NH, Baek JH, Ko SH, Son JW, Lee SH, Rhee SY, Kim SK, Sohn TS, Jun JE, Jeong IK, Kim CH, Song K, Rhee EJ, Noh J, Hur KY; Committee of Clinical Practice Guidelines, Korean Diabetes Association. Real-World Treatment Patterns according to Clinical Practice Guidelines in Patients with Type 2 Diabetes Mellitus and Established Cardiovascular Disease in Korea: Multicenter, Retrospective, Observational Study. Diabetes Metab J. 2024 Mar;48(2):279-289.
4. Yang YS, Kim HL, Kim SH, Moon MK. Lipid Management in Korean People with Type 2 Diabetes Mellitus: Korean Diabetes Association and Korean Society of Lipid and Atherosclerosis Consensus Statement. J Lipid Atheroscler. 2023 Jan;12(1):12-22.
5. Yang YS, Min KW, Park SO, Kim KS, Yu JM, Hong EG, Cho SR, Won KC, Kim YH, Oh S, Choi SH, Koh G, Huh W, Kim SY, Park KS. Efficacy and safety of monotherapy with enavogliflozin in Korean patients with type 2 diabetes mellitus: Results of a 12-week, multicentre, randomized, double-blind, placebo-controlled, phase 2 trial. Diabetes Obes Metab. 2023 Aug;25(8):2096-2104.

Korean Fact Sheet: Current status of LDL-C control from general population to high-risk groups

The Korean Society of Lipid and Atherosclerosis (KSoLA) periodically publishes the Dyslipidemia Fact Sheet using nationally representative data from the Korea National Health and Nutrition Examination Survey (KNHANES) to describe the current status of lipid-related disorders in Korean adults. This talk introduces key findings from the newly released Dyslipidemia Fact Sheet in Korea 2026, based on 2010-2024 KNHANES data.

The prevalence of hypercholesterolemia increased from 11.9% in 2010 to 30.3% in 2024, and remained consistently higher in women than in men (32.8% vs. 27.9%). Over the same period, awareness, treatment, and control of hypercholesterolemia improved substantially, from 45.5%, 35.8%, and 28.6% in 2010-2012 to 70.8%, 64.8%, and 58.9% in 2022-2024, respectively.

Dyslipidemia affected 47.8% of men and 45.4% of women, with a marked increase among women after menopause, reaching 67.1% at 60-69 years and 70.4% at 70 years and older. Hyper-LDL-cholesterolemia increased from 11.0% in 2010 to 26.7% in 2024, while hypo-HDL-cholesterolemia declined substantially over the same period.

This presentation will illustrate the evolving burden of LDL-cholesterol and dyslipidemia from the general population to high-risk subgroups in Korea, providing an updated evidence base for LDL-C management strategies across the risk spectrum.

CURRICULUM VITAE

Kyuho Kim

Assistant Professor, The Catholic University of Korea, Republic of Korea

Education and Training

2010.02	Yeungnam University, Korea, M.D., Medicine
2015.02	KAIST, Korea, Ph.D., Medical Science and Engineering

Employment and Position

2017-2021	Seoul National University Bundang Hospital, Korea, Resident/Fellow
2024-Present	The Catholic University of Korea, Korea, Assistant Professor

Important Publications

1. Kim K, Lee YJ, Yun JS, Ahn YB, Ko SH. Effects of the FXR agonist GW4064 on metabolic disorders in db/db mice. Lab Anim Res 2026;42:5.
2. Kim K, Kim B, Lee K, Ahn YB, Ko SH, Choi SH, Han K, Yun JS. Older Adults with Diabetes in Korea: Latest Clinical and Epidemiologic Trends. Diabetes Metab J 2025;49:183-93.
3. Kim K, Ko SH, Yun JS, Lee KW, Kim ES, Jeong IK, Kim JH, Kim SY, Won KC, Kim M, Cha BS, Kim S, Choi SH, Rhee EJ, Kim SG, Kim BH, Park KS, Ju YC, Heo TW, Ahn YB. Efficacy and safety of pioglitazone versus dapagliflozin as an add-on to metformin and alogliptin combination therapy: the EPIDOTE study. Sci Rep 2025;16:1226.
4. Kim K, Choi SH. A New Modality in Dyslipidemia Treatment: Antisense Oligonucleotide Therapy. J Lipid Atheroscler 2022;11:250-61.
5. Kim K, Ginsberg HN, Choi SH. New, Novel Lipid-Lowering Agents for Reducing Cardiovascular Risk: Beyond Statins. Diabetes Metab J 2022;46:517-32.

Korean Fact Sheet: Real-world utilization of lipid-lowering agents

Using the National Health Insurance Service-National Sample Cohort, we prepared the Korean Fact Sheet, including a comprehensive analysis of the real-world utilization patterns of lipid-lowering agents.

In 2023, the prescription rates for lipid-lowering agents were 94.4% for statins, 35.9% for ezetimibe, 10.3% for fenofibrate, and 10.3% for omega-3 fatty acids, with ezetimibe use showing a steady upward trend over time. The prescription rate of dual lipid-lowering therapy has increased over time, whereas that of lipid-lowering monotherapy has decreased. Among dual lipid-lowering therapy regimens, statin plus ezetimibe was the most frequently prescribed (78.1%), followed by statin plus omega-3 fatty acids (10.7%) and statin plus fenofibrate (10.0%). Among statin-treated patients with dyslipidemia, 92.1% received intermediate-intensity statins, 6.2% received high-intensity statins, and 1.7% received low-intensity statins. Among patients with dyslipidemia and cardiovascular disease (CVD), the prescription rates for lipid-lowering agents were 99.1% for statins, 50.7% for ezetimibe, 8.7% for omega-3 fatty acids, 6.1% for fenofibrate, and 0.5% for PCSK9 inhibitors. Among statin-treated patients with dyslipidemia and CVD, 50.1% received intermediate-intensity statins, 49.3% received high-intensity statins, and 0.6% received low-intensity statins. Among patients receiving statin plus ezetimibe therapy for dyslipidemia, 89.4% received intermediate-intensity statins, 8.9% received high-intensity statins, and 1.7% received low-intensity statins. The use of intermediate-intensity statins showed an increasing trend over time. The adherence rates for lipid-lowering agents were 95.3% for statins, 28.6% for ezetimibe, 7.3% for fenofibrate, and 7.3% for omega-3 fatty acids.

We hope that this fact sheet will serve as a valuable resource for understanding real-world prescribing patterns and contribute to improved patient care and further research.

CURRICULUM VITAE

Sang Yeub Lee

Associate Professor, Division of Cardiology,
Chung-Ang University Gwangmyeong Hospital, Republic of Korea

Education and Training

Chung-Ang University College of Medicine, Korea, M.D., Medicine
Chung-Ang University Graduate School, Korea, M.S., Medicine
Chung-Ang University Graduate School, Korea, Ph.D., Medicine

Employment and Position

1999-2000	Chung-Ang University Hospital, Korea, Intern
2000-2004	Chung-Ang University Hospital, Korea, Resident, Internal Medicine
2007-2009	Samsung Medical Center, Korea, Clinical Fellow, Cardiology
2009-2022	Chungbuk National University Hospital, Korea, Kigeum (Clinical) Professor, Cardiology
2015-2016	MedStar Washington Hospital Center, USA, Research Fellow
2022-Present	Chung-Ang University Gwangmyeong Hospital, Korea, Associate Professor, Division of Cardiology

Why are LDL-C targets still unmet? common errors and practical solutions in the clinic

Despite progressively lower LDL-cholesterol (LDL-C) targets in international guidelines, real-world attainment in Korea remains far below goal. Nationwide cohorts show that fewer than half of high-risk patients reach an LDL-C <70 mg/dL, and attainment is often worst where risk is highest. Our own survey of 407 Korean Society of Myocardial Infarction (KSMI) members found that while 94% were aware of the tightened <55 mg/dL target for very-high-risk patients, only 46% actually used it in practice for acute myocardial infarction (AMI) patients, citing concerns about efficacy, safety, and cost-effectiveness specific to Koreans.

This talk asks whether those concerns are justified. Drawing on the KAMIR-NIH registry, our new analysis shows that absolute follow-up LDL-C follows a U-shaped relationship with 5-year MACE, with the lowest risk observed at 55-69 mg/dL rather than below 55 mg/dL, whereas percentage reduction from baseline shows a continuous, monotonic benefit. This raised the question of whether a fixed <55 mg/dL target is even the right number to chase - a question now answered by two Korean randomized trials. Ez-PAVE, the first trial to randomize the LDL-C target itself in Korean ASCVD patients, showed a 33% relative risk reduction with intensive (<55 mg/dL) versus conventional (<70 mg/dL) targeting. RACING showed that moderate-intensity statin plus ezetimibe achieves target attainment non-inferior to high-intensity statin monotherapy, with better tolerability and lower discontinuation.

Together, these Korean-specific data close the evidence gap identified in our survey and support actively titrating toward <55 mg/dL with combination therapy, while tracking both absolute LDL-C and percentage reduction from baseline rather than either alone. I will also review the expanding non-statin toolbox, including bempedoic acid and PCSK9-targeted therapies, for patients who cannot reach target with statins and ezetimibe alone, and close with practical, clinic-ready strategies for narrowing Korea's persistent LDL-C treatment gap.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 8

Metabolic Regulators of Lipid Metabolism: From Mechanisms to Cardiovascular Outcomes

Sep 10(Thu) 16:10–17:40 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Eun Gyoung Hong (Hallym University, Republic of Korea)
Chul Sik Kim (Yonsei University, Republic of Korea)

16:10–16:30	**Sex differences in lymphatic function and atherosclerosis outcomes** Catherine Martel (University of Montreal, Canada)
16:30–16:50	**Tirzepatide and cardiometabolic outcomes: implications of dual GLP-1/GIP agonism** Yun Kyung Cho (University of Ulsan, Republic of Korea)
16:50–17:10	**Effects of SGLT2 inhibitors on lipid metabolism: mechanisms and clinical implications** Jun Hwa Hong (Eulji University, Republic of Korea)
17:10–17:40	**Panel Discussion** Shinae Kang (Yonsei University, Republic of Korea) JiHyun Ahn (Korea Medical Institute, Republic of Korea) Jung-Joon Cha (Korea University, Republic of Korea)

CURRICULUM VITAE

Catherine Martel

Full Professor, Department of Medicine,
Université de Montréal / Montreal Heart Institute Research Centre, Canada

Education and Training

2000–2002	Université de Montréal, B.Sc., Biomedical Sciences
2003–2004	Université de Montréal, M.Sc., Biomedical Sciences
2005–2009	Université de Montréal, Ph.D., Biomedical Sciences
2009–2011	Mount Sinai School of Medicine, New York, Postdoctoral Fellowship, Immunology and Lipid Metabolism
2012–2013	Washington University School of Medicine, St. Louis, Postdoctoral Fellowship, Pathology, Immunology and Atherosclerosis

Employment and Position

2014–Present	Montreal Heart Institute Research Centre, Researcher
2014–2019	Université de Montréal, Assistant Professor
2019–2025	Université de Montréal, Associate Professor
2022–Present	Temple University, Adjunct Associate Professor
2026–Present	Université de Montréal, Full Professor

Important Publications

1. Mesples M, Lacroix E, Tessier N, Farhat N, Milasan A, Babran S, Fernandez C, Latendresse T, Benoit J, Long V, Guillemette J, El Khakani S, Alikashani A, Leblanc CA, Higgins ME, Durocher Granger V, Fiset C, Martel C. Estrogens Differentially Modulate Lymphatic Function and Atherosclerosis in Menopause-Modeled Female Mice and Age-Matched Males. Front Cardiovasc Med. 2026:13:1699372.
2. Vachon L, Jean G, Milasan A, Babran S, Lacroix E, Guadarrama Bello D, Villeneuve L, Rak J, Nanci A, Mihalache-Avram T, Tardif JC, Ruiz M, Boilard E, Tessier N, Martel C. Platelet Extracellular Vesicles Preserve Lymphatic Endothelial Cell Integrity and Enhance Lymphatic Vessel Function. Communications Biology. 2024:7:975.

Sex differences in lymphatic function and atherosclerosis outcomes

Atherosclerosis is the leading cause of cardiovascular disease (CVD) and is driven by lipid accumulation and chronic arterial inflammation. The lymphatic system plays an essential role in cholesterol transport and immune cell clearance, and growing evidence suggests that lymphatic dysfunction contributes to cardiovascular disease progression. Previous studies have shown that impaired lymphatic function precedes atherosclerosis development in hypercholesterolemic mice and that enhancing lymphatic function can limit disease progression.

Hormonal transitions such as menopause are major determinants of cardiovascular risk and are often accompanied by metabolic disturbances associated with metabolic syndrome, including obesity, dyslipidemia, insulin resistance, and chronic inflammation. However, the impact of hormone therapy on lymphatic function and cardiovascular health remains poorly understood.

To investigate this question, we examined the effects of estrogen therapy in a mouse model of menopause predisposed to atherosclerosis. Estrogen produced marked sex-specific effects on lymphatic function and disease progression. While estrogen improved lymphatic transport and reduced atherosclerosis in males, it impaired lymphatic vessel contractility in ovariectomized female mice. Importantly, early stimulation of the lymphatic network preserved lymphatic function and limited atherosclerotic lesion development, highlighting the lymphatic vasculature as a potential therapeutic target during hormonal transitions.

These findings provide the rationale for an ongoing translational study evaluating lymphatic function in postmenopausal women receiving hormone therapy. Using near-infrared fluorescence imaging, this work aims to determine whether the lymphatic alterations observed in experimental models are also present in humans and whether they may contribute to cardiovascular vulnerability following menopause.

Together, these studies identify the lymphatic system as a potential mechanistic link between hormonal changes, metabolic syndrome, and cardiovascular disease. They support the development of novel lymphatic-targeted strategies to preserve vascular health and reduce cardiovascular risk in women undergoing menopause and hormone therapy.

CURRICULUM VITAE

Yun Kyung Cho

Assistant Professor,
Asan Medical Center, Ulsan University College of Medicine, Republic of Korea

Education and Training

2006-2012	University of Ulsan, College of Medicine, M.D, Internal Medicine
2015-2017	University of Ulsan, College of Medicine, M.S, Internal Medicine
2018-2020	Kangwon National University, School of Medicine, M.D, Internal Medicine

Employment and Position

2017-2018	Asan Medical Center, Clinical fellow
2018-2019	Kangwon National University Hospital, Clinical fellow
2019-2020	Asan Medical Center, Research fellow
2020-2022	Hallym University Sacred Heart Hospital, Clinical Assistant Professor
2022-2023	Asan Medical Center, Clinical Assistant Professor
2024-	Asan Medical Center, Assistant Professor

Important Publications

1. Defining Obesity Under the Lancet Commission Criteria: Metabolic Syndrome-Anchored Body Fat Percentage Thresholds in Korean Adults. Jung HN, Cho YK, Kim MJ, Moon JY, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH. Diabetes Obes Metab. 2026 Jun;28(6):5309-5317.
2. Beyond Weight Loss: Skeletal Considerations in Obesity Treatment. Kim MJ, Cho YK. Endocrinol Metab (Seoul). 2026 Apr;41(2):210-221.
3. Engineered nutrient-stimulated hormonal multi-agonists for precision targeting of obesity and metabolic disorders. Cho YK, Jung CH. Clin Mol Hepatol. 2026 Apr;32(2):464-486.

Tirzepatide and cardiometabolic outcomes: implications of dual GLP-1/GIP agonism

Selective GLP-1 receptor agonists established a reproducible class effect on major adverse cardiovascular events, and extended benefit to kidney and heart failure endpoints. Substantial residual glycemic, weight and cardiovascular risk nevertheless persisted, and insulin resistance improved largely as a by-product of weight loss. Dual GIP/GLP-1 receptor agonism was designed to enter this therapeutic space.

Across the SURPASS programme, tirzepatide lowered HbA1c by 1.9-2.6% and body weight by 6-13 kg, exceeding placebo, semaglutide 1.0 mg, insulin degludec and insulin glargine, with comparable effect sizes in East Asian participants. In the head-to-head SURMOUNT-5 trial it was superior to semaglutide at its full obesity dose (-20.2% versus -13.7%). Weight loss was driven predominantly by fat mass and accompanied by a weight-independent component of insulin sensitisation and a triglyceride- and remnant-directed lipid effect with only modest change in LDL cholesterol.

Cardiovascular outcome data have now arrived. SURPASS-CVOT (n=13,299) demonstrated non-inferiority to dulaglutide for three-point MACE (hazard ratio 0.92; 95.3% CI 0.83-1.01) without superiority; MACE-4, all-cause death and prespecified kidney outcomes in very-high-risk chronic kidney disease nonetheless favoured tirzepatide. In SUMMIT, tirzepatide reduced cardiovascular death or worsening heart failure by 38% in obesity-related HFpEF, with reductions in hsCRP, estimated blood volume, left ventricular mass and paracardiac adipose tissue.

This lecture will argue that these findings are mechanistically coherent rather than discordant. Four-year atherothrombotic risk is dominated by ApoB-containing lipoproteins and blood pressure, neither of which differed from dulaglutide, whereas the pathways engaged by GIP receptor agonism — adipose lipid buffering, ectopic lipid, circulatory volume and inflammation — map onto heart failure and kidney disease, precisely where the outcome evidence is strongest. Placebo-controlled proof in obesity awaits SURMOUNT-MMO.

CURRICULUM VITAE

Jun Hwa Hong

Associate Professor, M.D., Ph.D.,
Department of Endocrinology, Daejeon Eulji Medical Center, Eulji University, Republic of Korea

Introduction

Dr. Jun Hwa Hong is currently an Associate Professor in the Division of Endocrinology at Daejeon Eulji Medical Center, Eulji University. He received his M.D. and Ph.D. degrees from Eulji University College of Medicine and completed his internship and residency in internal medicine at Eulji University Hospital.

As an endocrinologist, Dr. Hong has developed a strong clinical and academic focus on obesity, diabetes, and dyslipidemia, with particular expertise in the design and conduct of clinical trials in metabolic diseases.

His research is centered on leading and participating in multicenter randomized clinical trials evaluating novel therapeutic strategies in type 2 diabetes and obesity, including SGLT2 inhibitors, incretin-based therapies, and emerging glucose-lowering agents. He has contributed to pivotal studies investigating add-on therapies to metformin, combination treatment strategies, and real-world effectiveness of anti-obesity pharmacotherapies.

Dr. Hong's work emphasizes evidence generation for optimal treatment strategies and translational application of clinical trial findings into real-world practice. His recent publications include high-quality clinical research articles in journals such as Trends in Endocrinology & Metabolism and Clinical Therapeutics, as well as investigator-initiated and multicenter trial reports in the field of cardiometabolic medicine.

He is an active member of several professional societies, including the Korean Diabetes Association, the Korean Endocrine Society, the Korean Society for the Study of Obesity, and the Korean Society of Lipidology and Atherosclerosis.

Education and Training

2004.02	Eulji University, Korea, M.D., Internal Medicine
2015.02	Eulji University, Korea, Ph.D., Internal Medicine

Effects of SGLT2 inhibitors on lipid metabolism: mechanisms and clinical implications

Sodium-glucose cotransporter 2 (SGLT2) inhibitors have transformed the management of type 2 diabetes mellitus by demonstrating robust cardiovascular and renal protection that extends beyond glucose lowering. Although their beneficial effects on heart failure and chronic kidney disease are well established, growing evidence suggests that SGLT2 inhibitors also exert important regulatory effects on lipid metabolism, contributing to their broad cardiometabolic benefits.

SGLT2 inhibition induces a metabolic shift characterized by enhanced glycosuria, negative energy balance, and increased lipid utilization. These adaptations promote fatty acid oxidation, reduce ectopic lipid accumulation, improve hepatic steatosis, and modulate key metabolic pathways involving AMP-activated protein kinase (AMPK), peroxisome proliferator-activated receptors (PPARs), fibroblast growth factor 21 (FGF21), adipokines, and ketone body metabolism. Experimental studies further suggest favorable effects on adipose tissue function, inflammation, mitochondrial efficiency, and lipoprotein remodeling, providing mechanistic insights into the reduction of cardiovascular risk observed in large outcome trials.

Clinically, SGLT2 inhibitors produce modest but consistent changes in circulating lipoproteins, including small increases in LDL cholesterol and HDL cholesterol together with reductions in triglycerides and improvements in atherogenic dyslipidemia. Emerging evidence indicates that these quantitative lipid changes may not fully reflect qualitative improvements in lipoprotein composition and function, including reductions in small dense LDL particles, remnant lipoproteins, and lipotoxicity. Furthermore, the beneficial effects of SGLT2 inhibitors on metabolic dysfunction-associated steatotic liver disease (MASLD), visceral adiposity, insulin resistance, and chronic inflammation highlight their role as metabolic modulators rather than conventional lipid-lowering agents.

This lecture will review the molecular mechanisms by which SGLT2 inhibitors regulate lipid metabolism, summarize current experimental and clinical evidence, and discuss their implications for cardiovascular risk reduction. Particular emphasis will be placed on integrating mechanistic insights with clinical trial data to better understand the role of SGLT2 inhibitors within contemporary cardiometabolic risk management and future precision medicine strategies.

Keywords: SGLT2 inhibitors, Lipid metabolism, Cardiovascular disease, Atherogenic dyslipidemia, Fatty acid oxidation, MASLD

Symposium 9 – KSN-KSoLA Joint Symposium

Chronic Kidney Disease and Atherosclerosis

Sep 10(Thu) 16:10-17:40 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Sang-Hyun Kim (Seoul National University, Republic of Korea)
Bum Soon Choi (The Catholic University of Korea, Republic of Korea)

16:10-16:30 **Lipid abnormalities in CKD**
Hong Sang Choi (Chonnam National University, Republic of Korea)

16:30-16:50 **Why patients with CKD develop accelerated atherosclerosis**
Cheol Ho Park (Yonsei University, Republic of Korea)

16:50-17:10 **Cardiovascular prevention in CKD: an endocrinologist's perspective**
Eun Roh (Seoul National University, Republic of Korea)

17:10-17:40 **Panel Discussion**
Wonjin Kim (CHA University, Republic of Korea)
Ji Won Ryu (Seoul National University, Republic of Korea)
Eun Hui Bae (Chonnam National University, Republic of Korea)

CURRICULUM VITAE

Hong Sang Choi

Associate Professor, Chonnam National University Medical School, Republic of Korea

Education and Training

2007.02	Chonnam National University, Korea, B.S., Medicine
2011.02	Chonnam National University, Korea, M.S, Internal Medicine
2020.02	Chonnam National University, Korea, Ph.D., Nephrology

Employment and Position

2015	Chonnam National University Hospital, Clinical Fellow
2016-2021	Chonnam National University Hospital, Clinical Assistant Professor
2022-	Chonnam National University Medical school, Associate Professor
2024.8-2026.7	Toronto general hospital research institute, University Health Network, Toronto, ON, Canada, Postdoctoral Researcher

Important Publications

1. Choi HS, Heo J, Won S, Suh SH, Kim CS, Bae EH, Ahn J, Kim SW. Association of Sodium-Glucose Cotransporter-2 Inhibitors With Incident Gout Risk: A Population-Based Comparative Cohort Study With Genetic Evidence From Mendelian Randomisation. Diabetes Obes Metab. 2026 Jun 21. doi: 10.1111/dom.71027. Epub ahead of print. PMID: 42324202.
2. Choi HS, Sundaram A, Vasukutty A, Mathew AP, Sathiyamoorthy P, Kim IJ, Suh SH, Kim CS, Ma SK, Kim SW, Park IK, Bae EH. Multiscale molecular modeling-directed ROS-responsive nanotherapy for dual-axis regulation of fibrotic and inflammatory signaling in alport nephropathy. J Nanobiotechnology. 2026 May 12. doi: 10.1186/s12951-026-04451-w. Epub ahead of print. PMID: 42121137.
3. Choi HS, Oh S, Ji M, Choi B, Suh SH, Kim CS, Ma SK, Kim SW, Paik MJ, Bae EH. Systematic metabolomic study on the plasma and urine of a mouse model of Alport syndrome. Sci Rep. 2025 Oct 2;15(1):34452. doi: 10.1038/s41598-025-17620-y. PMID: 41039011; PMCID: PMC12491436.

Lipid abnormalities in CKD

Dyslipidemia in chronic kidney disease (CKD) is a qualitatively distinct phenotype rather than ordinary dyslipidemia occurring in a patient with kidney disease. As kidney function declines, triglyceride-rich lipoproteins and their remnants accumulate and HDL cholesterol falls, while total and LDL cholesterol often remain within the reference range or even decrease. The defect is one of lipoprotein remodeling and clearance rather than cholesterol overproduction: reduced lipoprotein lipase and hepatic lipase activity — driven by elevated apolipoprotein C-III, downregulation of endothelial GPIHBP1, insulin resistance, secondary hyperparathyroidism, and uremic toxins — delays remnant catabolism, while hepatic LDL receptor downregulation impairs LDL clearance. Reduced apolipoprotein A-I synthesis, impaired LCAT activity, and increased CETP activity yield HDL particles that are fewer and functionally deficient in cholesterol efflux. The net phenotype — remnant-rich, shifted toward small dense LDL, HDL-dysfunctional, and with progressively rising lipoprotein(a) — is highly atherogenic yet largely invisible to conventional LDL-C measurement. Nephrotic-range proteinuria, dialysis modality, and post-transplant immunosuppression each modify this pattern further.

The association between LDL-C and myocardial infarction attenuates as eGFR falls and inverts on dialysis, and the relative risk reduction per unit of LDL-C lowering declines in parallel. Recent cohort data show that non-HDL cholesterol and remnant cholesterol predict atherosclerotic cardiovascular risk in CKD more consistently than LDL-C. The discrepancy between KDIGO's target-free approach and target-driven guidelines is thus best understood as a disagreement about which lipid measure to trust — and this lecture argues for a particle- and remnant-centric reassessment of lipids in CKD.

CURRICULUM VITAE

Cheol Ho Park

Contract Employment Doctor/Professor, Yonsei University, Republic of Korea

Education and Training

2013.02	Yonsei University, Korea, M.D., Medicine
2022.02	Yonsei University, Korea, Ph.D., Microbiology & Immunology

Employment and Position

2014-2018	Severance Hospital, Korea, Residency
2018-2022	Department Microbiology & Immunology, Yonsei University College of Medicine, Korea, Physician-Scientist
2022-2024	Division of Nephrology, Department Internal Medicine, Yonsei University College of Medicine, Korea, Clinical Instructor
2024-	Division of Nephrology, Department Internal Medicine, Yonsei University College of Medicine, Korea, Contract Employment, Doctor/Professor

Important Publications

1. Park CH, Kim Y, Kim C et al. Clinical Evaluation of a Disposable Test Strip for Potassium Level Measurement in Patients Treated with Maintenance Hemodialysis. Clin J Am Soc Nephrol. 2026;21:425-436.
2. Park CH, Kim HW, Park JT et al. BP and Kidney Disease Progression in Advanced CKD: Findings from the Chronic Renal Insufficiency Cohort and KoreaN Cohort Study for Outcome in Patients with CKD Studies. Clin J Am Soc Nephrol. 2025;20:1179-1189.
3. Park CH, Ko YE, Heo GY et al. Medication Burden and Adverse Cardiovascular Events and Death in Patients Treated with Maintenance Hemodialysis: A Nationwide Cohort Study. Clin J Am Soc Nephrol. 2025;20:72-80.
4. Park CH, Kim HW, Joo YS et al. Findings from the KNOW-CKD Study indicate that higher systolic blood pressure time in target range is associated with a lower risk of chronic kidney disease progression. Kidney Int. 2024;105:835-843.

Why patients with CKD develop accelerated atherosclerosis

Cardiovascular disease remains the leading cause of premature mortality in patients with chronic kidney disease (CKD), with many individuals dying from cardiovascular events before reaching end-stage renal disease (ESRD). First described by Lindner et al. in 1974, the phenomenon of "accelerated atherosclerosis" in the uremic population presents distinct clinical and morphological features compared to the general population. While traditional risk factors play a role, they fail to fully account for this elevated burden, highlighting the critical contribution of uremia-specific non-traditional factors.

The rapid progression of atherosclerosis in CKD is driven by a complex interplay of systemic inflammation, uremic toxicity, and altered lipid biology

Uremic Inflammation & Immune Dysregulation

Accumulated uremic toxins and chronic kidney injury trigger systemic inflammation, inducing profound dysregulation in both innate and adaptive immunity that promotes a pro-inflammatory vascular environment.

Lipoprotein Modification

The uremic milieu induces post-translational modifications of lipoproteins, such as low-density lipoprotein (LDL) carbamylation, which accelerates endothelial dysfunction and foam cell formation.

Dysfunctional HDL

High-density lipoprotein (HDL) loses its baseline atheroprotective and anti-inflammatory properties, transforming into an "acute-phase" dysfunctional particle.

Plaque-Level Activation

Within the vascular wall, the accumulation of cholesterol crystals acts as an endogenous trigger, activating local inflammatory pathways and perpetuating a maladaptive, unresolved chronic tissue response.

Accelerated atherosclerosis in CKD is not merely a consequence of traditional risk amplification, but a distinct pathological state driven by uremia-induced immune-inflammatory pathways, protein carbamylation, and dysfunctional HDL lipid biology. Understanding these non-traditional pathways is vital for developing targeted therapeutic strategies to mitigate the profound cardiovascular burden in the CKD population.

CURRICULUM VITAE

Eun Roh

Associate Professor, Seoul National University Boramae Medical Center, Republic of Korea

Education and Training

2008	Seoul National University College of Medicine, M.D., Medicine
2018	Ulsan University College of Medicine, Ph.D., Translational Medicine

Employment and Position

2023-2025	Hallym University College of Medicine, Associate Professor
2025-	Seoul National University Boramae Medical Center, Associate Professor

Important Publications

1. The weight-adjusted waist index predicts sarcopenia in community-dwelling older adults in a nationwide multicenter prospective study. Sci Rep. 2026 Feb 4;16(1):7284.
2. Impact of type 2 diabetes on the risk of cardiovascular disease and cardiovascular mortality in patients with non-alcoholic fatty liver disease: a nationwide population-based study. BMJ Public Health. 2025 Jun 18;3(1):e002020.
3. Impact of Remnant Cholesterol on the Risk for End-Stage Renal Disease in Type 2 Diabetes Mellitus: A Nationwide Population-Based Cohort Study. Diabetes Metab J. 2025 Sep;49(5):1106-1115.
4. High-Density Lipoprotein Cholesterol Levels and the Risk of Acromegaly: A Nationwide Cohort Study. Clin Endocrinol (Oxf). 2025 Aug;103(2):225-233.
5. Plasma Leukocyte Cell-Derived Chemotaxin-2 as a Risk Factor of Sarcopenia: Korean Frailty and Aging Cohort Study. Nutrients. 2025 Apr 14;17(8):1342.

Cardiovascular prevention in CKD: an endocrinologist's perspective

Chronic kidney disease (CKD) is a major cardiovascular risk condition, with cardiovascular morbidity and mortality increasing progressively as estimated glomerular filtration rate declines and albuminuria increases. Importantly, excess cardiovascular risk is evident from the early stages of CKD, well before progression to kidney failure. Cardiovascular prevention should therefore begin early and incorporate kidney function and albuminuria into comprehensive risk assessment.

Effective prevention in CKD requires an integrated cardiovascular-kidney-metabolic approach that addresses both conventional cardiovascular risk factors and kidney-specific mechanisms. Lifestyle modification, optimal blood pressure control, renin-angiotensin system blockade, and lipid-lowering therapy remain fundamental components of management. However, contemporary cardiorenal therapies provide substantial cardiovascular and kidney protection beyond conventional risk factor modification. Sodium-glucose cotransporter 2 inhibitors reduce heart failure events and kidney disease progression across a broad range of kidney function, with benefits that are largely independent of glucose lowering. Glucagon-like peptide-1 receptor agonists reduce major adverse cardiovascular events and provide additional kidney protection, particularly in patients with type 2 diabetes, obesity, and CKD. Finerenone, a nonsteroidal mineralocorticoid receptor antagonist, further reduces cardiovascular and kidney events in patients with type 2 diabetes and albuminuric CKD.

These therapeutic strategies should be considered complementary and implemented according to individual cardiovascular and kidney risk profiles rather than used as competing alternatives. From an endocrinologist's perspective, CKD represents an important opportunity for early cardiovascular intervention through integrated management of diabetes, obesity, hypertension, dyslipidemia, and kidney disease. Systematic assessment of estimated glomerular filtration rate and albuminuria, together with timely initiation of evidence-based therapies, may reduce residual cardiovascular risk and improve long-term cardiovascular and kidney outcomes across the continuum of CKD.

Symposium 10

New Frontiers in Lipid Lowering: Beyond Statins (New Drugs 1)

Sep 11(Fri) 08:30–10:00 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Sang-Hak Lee (Yonsei University, Republic of Korea)
Sungha Park (Yonsei University, Republic of Korea)

08:30–08:50	**Bempedoic acid: LDL-C lowering beyond statins – evidence and clinical role** Chewan Lim (Catholic Kwandong University, Republic of Korea)
08:50–09:10	**Optimizing non-statin therapy: PCSK9 inhibitors and inclisiran** Kyung-Hee Kim (Incheon Sejong Hospital, Republic of Korea)
09:10–09:30	**Emerging Lp(a)-lowering therapies: siRNA, ASO, and beyond** Yu-Chen Wang (Asia University, Taiwan)
09:30–10:00	**Panel Discussion** Gyu Chul Oh (The Catholic University of Korea, Republic of Korea) Ji-Yong Jang (National Health Insurance Service Ilsan Hospital, Republic of Korea) Yun Kyung Cho (University of Ulsan, Republic of Korea)

CURRICULUM VITAE

Chewan Lim

Assistant Professor, International St. Mary's Hospital,
Catholic Kwandong University, Republic of Korea

Education and Training

2005.03-2011.02	Catholic Kwandong University, Korea, M.D., Medicine
2011.03-2012.02	Myongji Hospital, Korea, Internship
2012.03-2016.02	Myongji Hospital, Korea, Residency, Internal Medicine
2015.03-2018.02	Catholic Kwandong University, Korea, M.S., Medicine
2019.03-2020.02	Severance Hospital, Yonsei University, Korea, Clinical Fellowship, Cardiology

Employment and Position

2020-2021	Severance Hospital, Yonsei University, Korea, Clinical Research Assistant Professor
2021-2024	Myongji Hospital, Korea, Clinical Assistant Professor
2024-Present	International St. Mary's Hospital, Catholic Kwandong University, Korea, Assistant Professor

Important Publications

1. Continuous Renal Replacement Therapy and Extracorporeal Membrane Oxygenation in Patients with Cardiogenic Shock: Results from the RESCUE Registry. J Clin Med. 2025.
2. Association of Timing of Revascularization on Clinical Outcomes of Percutaneous Coronary Intervention Relative to Surgery in Non-ST-Elevation Acute Coronary Syndrome Patients With Multivessel Disease. J Cardiovasc Interv. 2022.
3. Association between Body Mass Index and Clinical Outcomes of Peripheral Artery Disease after Endovascular Therapy: Data from K-VIS ELLA Registry. Korean Circ J. 2021.

Bempedoic acid: LDL-C lowering beyond statins - evidence and clinical role

Statins remain the cornerstone of atherosclerotic cardiovascular disease (ASCVD) prevention, yet a large proportion of high- and very-high-risk patients never reach guideline-recommended LDL-cholesterol (LDL-C) goals because of an inadequate response, statin-associated muscle symptoms, or genuine intolerance. Bempedoic acid, a first-in-class inhibitor of ATP-citrate lyase (ACLY), was developed for precisely this treatment gap. It acts upstream of HMG-CoA reductase but is functionally liver-selective: the prodrug requires activation by very-long-chain acyl-CoA synthetase-1, an enzyme absent from skeletal muscle, so hepatic cholesterol synthesis is suppressed without the muscle toxicity that limits statin therapy. Concomitant AMP-activated protein kinase activation may add anti-inflammatory and metabolic effects.

Across the phase 3 CLEAR programme, bempedoic acid 180 mg once daily lowered LDL-C by approximately 17-18% when added to maximally tolerated statins, by 21-28% in statin-intolerant patients, and by 38% as a fixed-dose combination with ezetimibe, with parallel reductions in high-sensitivity C-reactive protein (hsCRP). The CLEAR-J trial confirmed comparable efficacy in an East Asian population. CLEAR Outcomes, which randomised 13,970 statin-intolerant patients, showed a 13% relative reduction in four-component major adverse cardiovascular events over a median of 40.6 months (hazard ratio 0.87, 95% CI 0.79-0.96), a benefit per unit of LDL-C lowering consistent with Cholesterol Treatment Trialists' estimates for statins. Prespecified and post hoc analyses demonstrated a 30% risk reduction in primary prevention and consistent benefit in patients with diabetes or obesity, with no worsening of HbA1c or excess new-onset diabetes. Safety signals to monitor include hyperuricaemia and gout, small OAT2-mediated rises in creatinine, cholelithiasis, and tendon rupture, whereas muscle-related events were balanced against placebo.

The 2025 ESC/EAS focused update now positions bempedoic acid among non-statin agents with proven cardiovascular benefit. This lecture will review the mechanism, the efficacy and outcome data, and the safety profile, and will propose where bempedoic acid — alone or combined with ezetimibe — best fits in Korean clinical practice as an oral option for statin-intolerant patients and for those with residual LDL-C above goal.

CURRICULUM VITAE

Kyung-Hee Kim

Director, Heart Transplantation Center; Director, Education and Research;
Director, Special Cardiac Imaging; Board of Directors (Heart), ISHLT;
Associate Director, ISHLT Registry
Division of Cardiology, Department of Internal Medicine, Cardiovascular Center,
Incheon Sejong Hospital, Incheon, Republic of Korea

Education and Training

2004-2005	Seoul National University Hospital, Seoul, Korea, Internship, Internal Medicine
2005-2009	Seoul National University Hospital, Seoul, Korea, Residency, Internal Medicine
2009-2011	Seoul National University Hospital, Seoul, Korea, Research Doctor, Cardiovascular Research
2011-2013	Seoul National University Hospital, Seoul, Korea, Clinical Fellowship, Cardiology
2019-2020	Mayo Clinic, Rochester, MN, USA, Visiting & Senior Research Doctor, Cardiovascular Medicine

Employment and Position

2026-Present	International Society for Heart and Lung Transplantation (ISHLT), Board of Directors (Heart)
2025-Present	International Society for Heart and Lung Transplantation (ISHLT), Associate Director, ISHLT Registry
2020-Present	Incheon Sejong Hospital, Incheon, Korea, Director, Heart Transplantation Center
2017-Present	Incheon Sejong Hospital, Incheon, Korea, Director, Education and Research; Director, Special Cardiac Imaging
2013-Present	Sejong General Hospital & Sejong Heart Institute, Korea, Cardiologist, Division of Cardiology

Important Publications

1. Kim KH, Hsich E, Cherikh WS, et al. The International Thoracic Organ Transplant Registry of the International Society for Heart and Lung Transplantation: 2026 Adult Heart Transplantation: A contemporary analysis of trends and outcomes by recipient sex. J Heart Lung Transplant. 2026. doi:10.1016/j.healun.2026.05.025.
2. Lee SE, Joo JH, Hwang HS, et al. Spatial transcriptional landscape of human heart failure. Eur Heart J. 2025;46(31):3098-3114. doi:10.1093/eurheartj/ehaf272.

Optimizing non-statin therapy: PCSK9 inhibitors and inclisiran

Despite intensive statin therapy, a substantial proportion of patients at high or very high atherosclerotic cardiovascular disease (ASCVD) risk do not achieve guideline-recommended low-density lipoprotein cholesterol (LDL-C) goals. Contemporary lipid management therefore increasingly requires early, mechanism-based combination therapy rather than delayed stepwise escalation. PCSK9-targeted therapies are central to this strategy.

Monoclonal antibodies against PCSK9, including evolocumab and alirocumab, typically lower LDL-C by approximately 50-60% on top of statin therapy. Their cardiovascular benefit is established by FOURIER and ODYSSEY OUTCOMES, and the recent VESALIUS-CV trial further extended evidence for evolocumab to high-risk patients with atherosclerosis or diabetes but without previous myocardial infarction or stroke, demonstrating a significant reduction in first major cardiovascular events. These data support earlier consideration of PCSK9 inhibition when the absolute LDL-C reduction required to reach target is large.

Inclisiran uses small interfering RNA to suppress hepatic PCSK9 synthesis and provides sustained LDL-C lowering of approximately 50% with dosing at baseline, 3 months, and every 6 months thereafter. ORION trials established durable efficacy and an acceptable safety profile, while recent implementation studies have shown high rates of early and sustained LDL-C goal achievement. Its infrequent, healthcare-professional-administered dosing may be particularly attractive when long-term adherence to self-administered therapy is a concern. However, definitive cardiovascular outcome evidence for inclisiran is still awaited from ongoing outcomes trials, including ORION-4 and VICTORION-2 PREVENT.

This lecture will review how to choose between PCSK9 monoclonal antibodies and inclisiran according to ASCVD risk, required LDL-C reduction, urgency of treatment, adherence, safety, access, and strength of outcomes evidence. Practical treatment algorithms will be discussed in the context of the 2025 ESC/EAS focused update, emphasizing timely intensification and sustained achievement of LDL-C targets. Special attention will be given to very-low LDL-C safety, patients with familial hypercholesterolemia, recurrent events, statin intolerance, polypharmacy, and persistent therapeutic inertia in routine practice.

CURRICULUM VITAE

Yu-Chen Wang

Chief of Internal Medicine and Cardiology; Associate Professor, Asia University,
Asia University Hospital, Taichung, Taiwan

Education and Training

1994.10-2001.06	National Yang Ming Chiao Tung University, Taipei, Taiwan, M.D., Medicine
2003.07-2006.06	National Taiwan University Hospital, Taipei, Taiwan, Residency, Internal Medicine
2006.07-2008.06	National Taiwan University Hospital, Taipei, Taiwan, Fellowship, Cardiology
2010.09-2016.07	China Medical University, Taichung, Taiwan, Ph.D., Clinical Medical Science
2011.07-2012.06	Texas Heart Institute, Houston, TX, USA, Research Fellowship, Cardiology

Employment and Position

2016.08-Present	Asia University Hospital, Taichung, Taiwan, Chief, Division of Cardiology
2021.07-Present	Asia University Hospital, Taichung, Taiwan, Chief, Department of Internal Medicine
2022.08-Present	Asia University, Taichung, Taiwan, Associate Professor
2024-Present	Taiwan Society of Lipids and Atherosclerosis, Director
2024-Present	Taiwan Myocardial Infarction Society, Secretary General

Important Publications

1. Cheng CY, et al. 2026 Consensus and review of Lipoprotein(a) from Taiwan Society of Lipid and Atherosclerosis. J Formos Med Assoc. 2026;S0929-6646(26)00243-3.
2. He JM, Wang YC, Chang KC. Serial Coronary Artery Calcium Progression and Risk of Major Adverse Cardiovascular Events in an Asian Cohort. J Clin Med. 2026;15(12):4652.
3. Wang YC, et al. Clinical Effectiveness and Safety of Reduced-Dose Prasugrel in Asian Patients: The PROMISE-TW Registry. J Clin Med. 2025;14(16):5791.

Emerging Lp(a)-lowering therapies: siRNA, ASO, and beyond

Lipoprotein(a) [Lp(a)] is a genetically determined, lifelong causal risk factor for atherosclerotic cardiovascular disease (ASCVD) and calcific aortic valve stenosis. About one in five people have elevated concentrations, but Lp(a) is minimally affected by lifestyle. Statins do not lower Lp(a) and may modestly increase it; PCSK9 inhibitors provide modest reductions, while lipoprotein apheresis is effective but invasive and resource-intensive. This unmet need has accelerated development of therapies that directly target apolipoprotein(a) production or Lp(a) particle assembly.

This lecture reviews the evolving Lp(a)-lowering pipeline. Pelacarsen, a hepatocyte-directed antisense oligonucleotide, lowered Lp(a) by up to approximately 80% and is being evaluated in the phase 3 Lp(a) HORIZON trial. Small interfering RNA agents offer profound, durable suppression with infrequent dosing. Olpasiran lowered Lp(a) by more than 95% at higher doses in OCEAN(a)-DOSE and is under evaluation in OCEAN(a)-Outcomes. Lepodisiran achieved an approximately 94% time-averaged reduction after a 400-mg dose in ALPACA and is being studied in ACCLAIM-Lp(a). Zerlasiran has also shown sustained reductions with extended-interval dosing. Beyond injectable RNA therapeutics, muvalaplin, an investigational oral small molecule that inhibits apo(a)-apoB assembly, has produced substantial Lp(a) lowering and entered cardiovascular outcomes evaluation.

Although marked Lp(a) lowering is now biologically achievable, the pivotal question is whether it reduces cardiovascular events without unforeseen long-term harm. Ongoing phase 3 outcome trials will clarify the magnitude of clinical benefit, appropriate treatment thresholds, and the populations most likely to benefit. The lecture will address whom to test, how to interpret Lp(a) values, how emerging therapies may be integrated with established lipid-lowering treatment, and practical challenges involving cost, access, dosing, and long-term safety. Positive outcome data could establish Lp(a) as a major modifiable target in residual cardiovascular risk.

Symposium 11

Lipid-lowering Therapy in Complex Clinical Settings: HIV, Cancer, and Chronic Kidney Disease

Sep 11(Fri) 08:30-10:00 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Junghyun Noh (Inje University, Republic of Korea)
Jung-Sun Kim (Yonsei University, Republic of Korea)

08:30-08:48	**Lipid-lowering therapy in people living with HIV (PLWH)** So Ree Kim (Korea University, Republic of Korea)
08:48-09:06	**Lipid-lowering therapy in cancer patients and survivors** Hiroshi Akazawa (Kanazawa Medical University, Japan)
09:06-09:24	**Lipid-lowering therapy in patients with CKD** Eun Sil Koh (The Catholic University of Korea, Republic of Korea)
09:24-09:42	**Cardiovascular prevention in hemodialysis: is omega-3 back?** Sang Ho Jo (Hallym University, Republic of Korea)
09:42-10:00	**Panel Discussion** Sang Yong Kim (Chosun University, Republic of Korea) Ji Woong Yoo (Hallym University, Republic of Korea) Mi-Hyang Jung (The Catholic University of Korea, Republic of Korea)

CURRICULUM VITAE

So Ree Kim

Assistant Professor, Korea University Anam Hospital, Republic of Korea

Education and Training

2012.02	Wonkwang University, Medicine
2018.02	Sungkyunkwan University, Korea, M.D., Internal Medicine
2023.08	Korea University, Korea, Ph.D., Internal Medicine

Employment and Position

2014-2018	Samsung Medical Center, Korea/Internal Medicine, Residency
2018-2020	Samsung Medical Center, Korea/Cardiology, Fellowship
2020-2025	Korea University Anam Hospital, Korea/Cardiology, Clinical Assistant Professor
2025-	Korea University Anam Hospital, Korea/Cardiology, Assistant Professor

Important Publications

1. So Ree Kim, D-H Cho, M-N Kim, J-J Cha, BR Kim, JH Yang, and S-M Park. Relationship of cardiopulmonary exercise capacity to coronary microvascular function and body composition in patients with suspected heart failure with preserved ejection fraction. Cardiac Failure Review 2026; 12:e20. [IF 12.2]
2. So Ree Kim, Kim MN, Kim HD, Cho DH, Kim HL, Kim YH, Na JO, Yoon HJ, Shin MS, Kim MA, Hong KS, Shim WJ, Park SM. Association Between Ratio of Cholesterols and Coronary Artery Stenosis According to Low-Density Lipoprotein-Cholesterol Levels and Statin Use. Korean Circ J. 2026 Mar 16. [IF 3.1]
3. So Ree Kim, Cho D-H, Kim J-H, Park S-M, Kim M-N. Oxidative Stress Biomarkers Predict Myocardial Dysfunction in a Chemotherapy-Induced Rat Model. Diagnostics. 2025; 15(6):705. [IF 3.0]
4. So Ree Kim, Kim MN, Cho DH, Kim HD, Bae SA, Kim HL, Kim MA, Hong KS, Shim WJ, Park SM. Sex differences of sequential changes in coronary blood flow and microvascular function in patients with suspected angina. Clin Res Cardiol. 2024 Dec;113(12):1638-1649. [IF 3.8]

Lipid-lowering therapy in people living with HIV (PLWH)

People living with HIV (PLWH) face a substantially elevated burden of atherosclerotic cardiovascular disease (ASCVD), with a pooled relative risk of 2.16 (95% CI, 1.68-2.77) compared with the general population. This excess risk arises from the interplay of chronic HIV-driven immune activation, adverse metabolic effects of certain antiretroviral therapy (ART) regimens, and a higher prevalence of traditional risk factors. Conventional ASCVD risk calculators, including the Pooled Cohort Equations, SCORE2, and PREVENT, were derived from predominantly HIV-negative cohorts and consistently underestimate risk in PLWH, particularly in high-prevalence, resource-limited settings lacking locally validated models.

This presentation reviews the evidence base guiding lipid-lowering therapy in PLWH, centered on the REPRIEVE trial (N Engl J Med, 2023), the largest primary-prevention cardiovascular trial conducted in this population. REPRIEVE randomized 7,769 PLWH with low-to-moderate traditional ASCVD risk to pitavastatin 4 mg daily or placebo, demonstrating a 35% relative reduction in major adverse cardiovascular events (hazard ratio, 0.65; 95% CI, 0.48-0.90) over a median follow-up period, with a 5-year number needed to treat of 106. These findings, together with evidence for statins' pleiotropic anti-inflammatory effects beyond LDL-cholesterol lowering, have reshaped international recommendations. The 2025 ESC/EAS guidelines now recommend statin therapy for all PLWH aged 40 years or older regardless of calculated risk score or LDL-C level, while the US Department of Health and Human Services maintains a risk-threshold-based approach.

Practical prescribing considerations, particularly drug-drug interactions between statins and ART (favoring pitavastatin or pravastatin with boosted protease inhibitors), and a stepwise clinical algorithm for lipid management in PLWH are presented, providing clinicians with an evidence-based framework for cardiovascular risk reduction in this high-risk population.

CURRICULUM VITAE

Hiroshi Akazawa

Professor, Kanazawa Medical University, Japan

Education and Training

1992.03	The University of Tokyo, Japan, M.D., Internal Medicine
2009.03	Chiba University Graduate School of Medicine, Japan, Ph.D., Medicine

Employment and Position

1992-1993	The University of Tokyo Hospital, Japan, Resident
1993-1995	Jichi Medical School Hospital, Japan, Clinical Fellow
1995-1996	The University of Tokyo School of Medicine, Japan, Clinical and Research Fellow
1996-2000	The Cancer Institute, Japanese Foundation for Cancer Research, Japan, Research Fellow
2000	Graduate School of Medicine, The University of Tokyo, Japan, Clinical and Research Fellow
2000-2004	Chiba University Graduate School of Medicine, Japan, Research Fellow
2004-2009	Chiba University Graduate School of Medicine, Japan, Research Associate
2009-2010	Chiba University Graduate School of Medicine, Japan, Assistant Professor
2010-2012	Osaka University Graduate School of Medicine, Japan, Assistant Professor
2012-2013	Osaka University Graduate School of Medicine, Japan, Associate Professor
2013-2014	Graduate School of Medicine, The University of Tokyo, Japan, Associate Professor
2014-2026	The University of Tokyo Hospital, Japan, Lecturer
2026-	Kanazawa Medical University, Professor

Important Publications

1. Ueda T, Shindo A, Kadowaki H, Ishida J, Takeda N, Akazawa H#. Acute Myocardial Infarction in Cancer Patients Treated With Immune Checkpoint Inhibitors: A Nationwide Cohort Study. JACC Adv. in press.
2. Yagi H, Akazawa H#, Liu Q, Ito SM, Umei M, Kadowaki H, Matsuoka R, Shindo A, Okamura S, Ueda T, Saga-Kamo A, Ueda K, Takeda N, Takada T, Komuro I. Inverse agonist activity of angiotensin II receptor blocker is crucial for prevention of progressive aortic dilatation in Marfan syndrome. Arterioscler Thromb Vasc Biol. 46(1):132-144,2026.

Lipid-lowering therapy in cancer patients and survivors

Advances in cancer therapies have markedly improved survival, shifting attention toward long-term non-cancer comorbidities. Cardiovascular disease (CVD) is a major cause of morbidity and mortality in cancer survivors, making atherogenic risk management crucial. However, lipid management in this population remains challenging. Cancer treatment-induced dyslipidemia is increasingly recognized. Key anticancer agents—including endocrine therapies (e.g., aromatase inhibitors, androgen deprivation therapy) and targeted therapies (e.g., tyrosine kinase inhibitors)—significantly alter lipid profiles, elevating atherogenic risk. Furthermore, pediatric and adolescent/young adult (AYA) cancer survivors experience a disproportionately high prevalence of dyslipidemia and metabolic syndrome, driven by previous radiation, chemotherapy, and premature vascular aging. While statins and other lipid-lowering agents effectively mitigate cardiovascular risks and may offer pleiotropic benefits, intervention must be tailored rather than uniform. Patients with curable disease or long-term survival benefit from guideline-directed lipid-lowering therapy, whereas those in palliative settings require careful deprescribing. In addition, concomitant use of statins and specific anticancer agents (e.g., via CYP3A4) necessitates careful selection, such as switching to non-interacting statin, to avoid adverse events. Therefore, lipid management in cancer patients and survivors requires an individualized, multidisciplinary approach. Balancing long-term cardiovascular prevention against immediate oncologic status and potential drug-drug interactions is key to optimizing outcomes for cancer patients and survivors

CURRICULUM VITAE

Eun Sil Koh

Associate Professor, Division of Nephrology, Department of Internal Medicine,
The Catholic University of Korea College of Medicine, Seoul, Republic of Korea

Education and Training

2005.02	The Catholic University of Korea, M.D., Medicine
2016.08	The Catholic University of Korea, Ph.D., Internal Medicine

Employment and Position

2012-2015	The Catholic University of Korea Yeouido St. Mary's Hospital, Clinical Fellowship
2016-	The Catholic University of Korea Yeouido St. Mary's Hospital, Staff

Important Publications

1. Kim JW, Nam SA, Koh ES, Kim HW, Kim S, Woo JJ, et al. The Impairment of Endothelial Autophagy Accelerates Renal Senescence by Ferroptosis and NLRP3 Inflammasome Signaling Pathways with the Disruption of Endothelial Barrier. Antioxidants (Basel). 2024;13(8).
2. Koh ES, Kim GH, Chung S. Intrarenal Mechanisms of Sodium-Glucose Cotransporter-2 Inhibitors on Tubuloglomerular Feedback and Natriuresis. Endocrinol Metab (Seoul). 2023;38(4):359-72.
3. Lee H, Moon SJ, Kim SW, Min JW, Park HS, Yoon HE, Koh ES, and Chung BH. Prediction of intradialytic hypotension using pre-dialysis features - a deep learning-based artificial intelligence model. Nephrol Dial Transplant. 2023;38(10):2310-20
4. Koh ES, Han K, Nam YS, Wittbrodt ET, Fenici P, Kosiborod MN, et al. Renal outcomes and all-cause death associated with sodium-glucose co-transporter-2 inhibitors versus other glucose-lowering drugs (CVD-REAL 3 Korea). Diabetes Obes Metab. 2021;23(2):455-66.
5. Koh ES, Kim M, Kim MK, Han K, Shin SJ, Kwon HS, et al. Intra-individual variability in high density lipoprotein cholesterol and risk of end-stage renal disease: A nationwide population-based study. Atherosclerosis. 2019;286:135-41.

Lipid-lowering therapy in patients with CKD

Dyslipidemia is a common complication of chronic kidney disease (CKD), characterized by hypertriglyceridemia, low HDL cholesterol levels, and structural alterations in LDL cholesterol. Despite the increased cardiovascular risk in patients with CKD, major lipid management guidelines present conservative recommendations due to altered cardiovascular dynamics and a lack of robust evidence. Large-scale randomized controlled trials (RCT) such as SHARP have demonstrated that statin-based therapy effectively reduces major atherosclerotic events in non-dialysis CKD patients. However, these cardiovascular benefits are markedly diminished in dialysis-dependent end-stage kidney disease patients, among whom non-atherosclerotic events predominate. Furthermore, statins fail to delay the progression of CKD. Non-statin agents, including fibrates, omega-3 fatty acids, and PCSK9 inhibitors, provide alternative strategies to address residual cardiovascular risk and hypertriglyceridemia, although additional dedicated RCTs are required. In conclusion, lipid-lowering therapy in CKD patients requires an individualized, risk-stratified approach that considers both cardiovascular benefits and renal outcomes.

CURRICULUM VITAE

Sang Ho Jo

Div. of Cardiology, Dept. of Internal Medicine, College of Medicine, Hallym University,
Hallym University Sacred Heart Hospital, Anyang-si, Gyeonggi-do, Republic of Korea

Education

1991-1992	Premedical Course, College of Liberal Arts & Science, Seoul National University
1993-1997	Seoul National University College of Medicine (M.D.)
2002-2004	Graduate School, Seoul National University College of Medicine

Professional Activities

1998-1999	Internship, Seoul National University Hospital, Seoul, Korea
2000-2004	Residency in Internal Medicine, Seoul National University Hospital, Seoul, Korea
2004-2006	Fellowship in Cardiology, Seoul National University Hospital, Seoul, Korea
2006-2006	Full time Lecturer, Hallym University Sacred Heart Hospital, Anyang-si, Gyeonggi-do, Korea
2006-Present	Professor, Hallym University Sacred Heart Hospital, Anyang-si, Gyeonggi-do, Korea

Fields of Interest

1. Interventional cardiology
2. Myocardial salvage
3. Vascular biology
4. Antioxidant
5. Pathogenesis of atherosclerosis and coronary artery disease
6. Diabetes and vascular biology

Cardiovascular prevention in hemodialysis: is omega-3 back?

Background: Cardiovascular disease remains the leading cause of death in patients receiving maintenance hemodialysis, accounting for nearly half of all mortality in this population, yet pharmacologic strategies with proven cardiovascular benefit remain remarkably limited. Statins, despite their efficacy in the general population, have failed to reduce cardiovascular events in dialysis patients in dedicated trials such as 4D and AURORA, leaving a persistent therapeutic gap.

Omega-3 polyunsaturated fatty acids (n-3 PUFA) have a long and inconsistent history as a cardiovascular preventive strategy. Early trials including DART and GISSI-Prevenzione, and the Japanese JELIS trial using purified EPA, suggested benefit, while subsequent large contemporary trials such as ASCEND, VITAL, and OMEMI showed neutral results with lower-dose mixed EPA/DHA formulations. REDUCE-IT, using high-dose purified EPA (icosapent ethyl), demonstrated a substantial cardiovascular risk reduction, whereas STRENGTH, using a comparable dose of mixed EPA/DHA, was stopped for futility. This divergence has been attributed to formulation, dose, and the choice of comparator oil, leaving the role of omega-3 fatty acids in cardiovascular prevention unresolved and the topic largely set aside in recent years.

Recent Evidence: The PISCES Trial: This landscape has been reframed by PISCES (Protection Against Incidences of Serious Cardiovascular Events Study), a multicenter, double-blind, randomized, placebo-controlled trial conducted at 26 sites in Canada and Australia. The trial enrolled 1228 adults receiving maintenance hemodialysis and randomly assigned them to daily supplementation with 4 g of n-3 PUFA (1.6 g EPA, 0.8 g DHA) or corn-oil placebo, with a mean follow-up of approximately 3.5 years.

The primary composite endpoint, comprising sudden and nonsudden cardiac death, fatal and nonfatal myocardial infarction, fatal and nonfatal stroke, and peripheral vascular disease leading to amputation, occurred at a rate of 0.31 per 1000 patient-days in the fish-oil group versus 0.61 per 1000 patient-days in the placebo group (HR 0.57, 95% CI, 0.47-0.70, P<0.001), corresponding to a 43% relative risk reduction. Benefit was consistent across individual components: cardiac death (HR 0.55, 95% CI, 0.40-0.75), myocardial infarction (HR 0.56, 95% CI, 0.40-0.80), stroke (HR 0.37, 95% CI, 0.18-0.76), and amputation (HR 0.57, 95% CI, 0.38-0.86). A composite of first cardiovascular event or death from any cause was also reduced (HR 0.73, 95% CI, 0.61-0.87). The benefit was directionally consistent in patients both with and without prior cardiovascular disease, and adherence and adverse-event rates did not differ meaningfully between groups.

Discussion: The magnitude of benefit observed in PISCES is considerably larger than that reported in general-population trials, and this discrepancy is best understood through the distinct risk substrate of the hemodialysis population rather than as a contradiction of prior neutral results. Patients receiving maintenance hemodialysis exhibit markedly low circulating EPA and DHA levels, among the lowest reported in the medical literature, related to reduced dietary intake, increased catabolism, chronic inflammation, and the dialysis procedure itself. This baseline deficiency, combined with a cardiovascular event profile dominated by sudden cardiac death and arrhythmic events rather than atherosclerotic plaque rupture alone, plausibly creates a population in which the anti-arrhythmic, anti-thrombotic, and anti-inflammatory properties of n-3 PUFA can exert a disproportionately large effect. This mechanistic framework is consistent with the earlier, smaller FISH trial from the same investigator group, and lends biological coherence to the present result rather than positioning it as an isolated positive finding.

These results do not resolve the formulation debate raised by REDUCE-IT and STRENGTH, since PISCES used a mixed EPA/DHA preparation similar to that used in the neutral STRENGTH trial, yet produced a strongly positive result in a population with a fundamentally different risk profile and baseline omega-3 status. This suggests that patient selection, specifically the presence of omega-3 deficiency and a high burden of arrhythmic cardiovascular risk, may be as important as formulation in determining efficacy.

Clinical Implications: For Korean hemodialysis patients, in whom fish consumption and baseline omega-3 status differ from the Canadian and Australian PISCES cohort, confirmatory data will be valuable before this strategy is generalized into routine practice. Nonetheless, given the favorable safety profile observed and the persistent absence of effective cardiovascular preventive options in this population, daily fish-oil supplementation merits serious consideration as a pragmatic, low-cost addition to cardiovascular risk reduction in maintenance hemodialysis.

Conclusion: The PISCES trial provides robust evidence that daily omega-3 fatty acid supplementation reduces serious cardiovascular events in patients receiving maintenance hemodialysis, reviving the role of omega-3 fatty acids in cardiovascular prevention within a population-specific context defined by omega-3 deficiency and a high arrhythmic risk burden.

References: 1. Lok CE, Farkouh M, Hemmelgarn BR, et al. Fish-oil supplementation and cardiovascular events in patients receiving hemodialysis. N Engl J Med. 2026;394(2):128-137.

2. Bhatt DL, Steg PG, Miller M, et al. Cardiovascular risk reduction with icosapent ethyl for hypertriglyceridemia. N Engl J Med. 2019;380:11-22.

3. Nicholls SJ, Lincoff AM, Garcia M, et al. Effect of high-dose omega-3 fatty acids vs corn oil on major adverse cardiovascular events in patients at high cardiovascular risk: the STRENGTH randomized clinical trial. JAMA. 2020;324:2268-2280.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 12 – KPS–KSoLA Joint Symposium

Bridging Cellular Physiology and Atherogenic Lipids

Sep 11(Fri) 08:30–10:00 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Yong Sook Kim (Chonnam National University, Republic of Korea)
Hyoung Kyu Kim (Inje University, Republic of Korea)

08:30–08:50	**Ion channels in atherosclerosis: from calcium flux to vascular dysfunction** Sung Joon Kim (Seoul National University, Republic of Korea)
08:50–09:10	**Energy metabolism and lipid oxidation in cardiometabolic pathophysiology** Jin Han (Inje University, Republic of Korea)
09:10–09:30	**Lp(a): from lipoprotein biology to atherosclerotic cardiovascular disease** Jung-Woo Son (Yonsei University, Republic of Korea)
09:30–10:00	**Panel Discussion** Nari Kim (Inje University, Republic of Korea) Jong-Gil Park (Korea Research Institute of Bioscience and Biotechnology, Republic of Korea) Dong Kyu Choi (Kyungpook National University, Republic of Korea)

CURRICULUM VITAE

Sung Joon Kim

Chair Professor of Department of Physiology (Undergraduate)
Department of Biomedical Sciences (Graduate)
Laboratory of Cardiovascular and Ion Channel Physiology
Seoul National University College of Medicine, Seoul, Republic of Korea

Academic and Professional Backgrounds

1985.03-1991.02	Seoul National University College of Medicine (SNUCM), M.D.
1991.03-1997.02	Seoul National University, M.S. and Ph.D. (Physiology)
1991.04-1995.01	SNUCM, Education Assistant
1995.05-1998.07	SNUCM, Medical Research Institute, Research Fellow
1998.08-2000.07	University of Freiburg(프라이부르그), Germany, Humboldt Research Fellow
2000.09-2004.08	Sungkyunkwan University School of Medicine, Assistant Professor
2004.09-2006.09	SNUCM, Assistant Professor
2006.10-2011.08	SNUCM, Associate Professor
2011.09-Present	SNUCM, Professor
2012.01-2013.12	SNUCM, Associate Dean for Research Affairs (연구부학장)
2014.03-2016.02	SNUCM, Core Research Facility Center, Chief Director
2015.01-2019.03	SNUCM, Hypoxic/Ischemic Disease Institute, Director
2015.04-2016.01	Seoul National University Senate (서울대학교 평의원)
2016.01-2017.11	SNUCM, Associate Dean for Students Affairs (학생부학장)
2019.01-2020.12	Present: SNU Graduate School, Department of Biomedical Sciences, Chief professor (대학원 의과학과장)
2018.07-2022.05	SNUCM, Department of Physiology, Chair professor (생리학교실 주임교수)
2018.11-2022.05	Seoul National University Senate (서울대학교 평의원)
2022.06-2024.05	Program Manager (PM) for the Medical Research Field in Korea National Research Foundation (NRF) (한국연구재단 의약학단장)
2021.11-2025.12	Director of Foreign Affairs, Elected President of The Korean Physiological Sciences (KPS, 대한생리학회 국제이사, 차기회장)
2026.01-Present	President of The Korean Physiological Sciences (KPS, 대한생리학회 회장)

Ion channels in atherosclerosis: from calcium flux to vascular dysfunction

Calcium (Ca^{2+}) signaling, which evolved from an ancient geochemical threat into a master physiological regulator, serves as a double-edged sword in vascular health. While oscillating physiological increase in cytosolic Ca^{2+} ($[Ca^{2+}]_c$) maintain endothelial homeostasis, hemodynamic stress converts this tightly controlled system into a pathological sustained Ca^{2+} plateau, driving atherosclerosis and active vascular calcification. Under hemodynamic disturbances such as disturbed flow and high hydrostatic pressure, apical mechanosensitive channels—specifically Piezo1 and TRPV4—trigger continuous Ca^{2+} influx in endothelial cells. This induces the endothelial-mesenchymal transition (EndMT), mitochondrial ROS generation, and NLRP3 inflammasome-mediated pyroptosis. In response to inflammatory signals, recruited neutrophils rely on store-operated Ca^{2+} entry (SOCE) for firm adhesion under shear stress, a process electrically augmented by the KV1.3 potassium channel activation. Within the sub-endothelial space, matrix stiffening hyperactivates macrophage Piezo1, promoting YAP/TAZ nuclear translocation and driving pro-inflammatory M1 polarization, while TRPV4 overactivation promotes foam cell formation and ferroptosis. Concurrently, vascular smooth muscle cells (VSMCs) undergo an excitable-to-nonexcitable phenotypic switch, down-regulating voltage-gated CaV1.2 channels while up-regulating receptor-operated TRP and STIM/Orai channels. This bioenergetic rewiring forces VSMCs into a synthetic phenotype through a metabolic shift toward aerobic glycolysis, ultimately accelerating osteochondrogenic differentiation and the active exocytosis of calcifying vesicles. Consequently, a self-perpetuating vicious cycle between matrix stiffening and calcium dysregulation is established. Ultimately, modulating these ion channel networks—not through complete blockade, but via functional correction—presents a novel therapeutic paradigm to restore vascular mechanical homeostasis beyond traditional lipid-lowering strategies

CURRICULUM VITAE

Jin Han

Professor, Cardiovascular and Metabolic Disease Center, Inje University, Republic of Korea

Education and Training

1991	M.D., (Medicine) College of Medicine, Inje University (IU), Busan, Korea
1993	M.Sc., (Physiology) Seoul National University (SNU) Graduate School, Seoul, Korea
2000	Ph.D., (Physiology) SNU Graduate School, Seoul, Korea

Employment and Position

2019-2026	Director, INNOPOLIS Gimhae, Ministry of Science and ICT, Korea
2006-Present	Chair, Department of Physiology, College of Medicine, IU
2006-Present	Director, CMDC, IU
2010-Present	Professor, Department of Physiology, College of Medicine, IU
2023-Present	Director, Cardiovascular and Metabolic Disease Core Center, Ministry of Education, Korea
2024-Present	Chair, Advisory Committee for Inter-ministerial National Integrated Bio-Bigdata and Omics, Korea
2024-Present	Director, Biomedical Center, Glocal University 30, Ministry of Education, Korea
2025-Present	Director, Basic Science Research Capacity Enhancement Project (Infrastructure Advancement), Ministry of Education, Korea
2025-Present	Director, Medical Research Center, Ministry of Science and ICT, Korea

Important Publications

1. Cereblon contributes to cardiac dysfunction by degrading Cav1.2α. Eur Heart J, 2022
2. Tetrahydrobiopterin in energy metabolism and metabolic diseases. Pharmacol Res, 2020
3. C1q/TNF-α-Related Protein 1 (CTRP1) maintains blood pressure under dehydration conditions. Circ Res, 2018
4. Current and upcoming mitochondrial targets for cancer therapy. Seminars in Cancer Biol, 2017
5. FOXM1-induced PRX3 regulates stemness and survival of colon cancer cells via maintenance of mitochondrial function. Gastroenterology, 2015.

Energy metabolism and lipid oxidation in cardiometabolic pathophysiology

Current diagnostic tools often fail to detect diabetic cardiomyopathy (DCM) before irreversible structural damage, such as cardiac fibrosis, occurs. This "diagnostic void" highlights the urgent need to understand the early stages of cardiometabolic pathophysiology, characterized by lipotoxicity and mitochondrial dysfunction.

This presentation explores the critical intersection of early bioenergetic failure and fibrogenesis, highlighting a novel master regulator: Cereblon (CRBN). Conventionally known as an E3 ubiquitin ligase component, our multi-omics and clinical findings reveal CRBN as a primary identifier of early metabolic stress. In diabetic patients, CRBN is significantly upregulated, correlating strictly with subclinical inflammatory markers (e.g., IL-6, TNF-α rather than traditional myocardial necrosis, acting as an "Early Bird" signal.

Mechanistically, CRBN directly binds to and suppresses the α subunit of AMP-activated protein kinase (AMPK). In early-stage diabetic models (db/db), this disruption creates a severe bioenergetic bottleneck, reducing mitochondrial oxidative capacity and altering TCA cycle metabolites long before echocardiographic functional decline. The ensuing surge in oxidative stress and pro-inflammatory signaling acts as a potent catalyst, upregulating fibrotic markers (TGF-β, Col1a1) and driving the transdifferentiation of cardiac fibroblasts.

Crucially, mitigating this CRBN-mediated metabolic suppression offers a paradigm-shifting therapeutic approach. Our in vivo data demonstrate that targeted CRBN deletion drastically reduces cardiac fibrosis and preserves function in both doxorubicin-induced and ischemia-driven cardiomyopathy models. Furthermore, pharmacological degradation of CRBN via novel PROTACs (TD-165), or structured aerobic exercise—which significantly lowers serum CRBN in humans—robustly restores metabolic flexibility and mitochondrial integrity. Targeting the CRBN-AMPK axis serves as a vital "bridge to excellence" for preventing cardiometabolic fibrosis and preserving cardiac health.

CURRICULUM VITAE

Jung-Woo Son

Division of Cardiology, Wonju Severance Christian Hospital, Republic of Korea

Education

2004	M.D., Yonsei University Wonju College of Medicine
2026	Combined M.S.-Ph.D. Program in Medicine, Yonsei University

Professional Experience

2016-Present	Assistant Professor, Division of Cardiology, Wonju Severance Christian Hospital
2014-2015	Clinical Assistant Professor, Division of Cardiology, Hallym University Chuncheon Sacred Heart Hospital
2012-2013	Clinical Fellow, Division of Cardiology, Severance Hospital
2004-2008	Resident, Department of Internal Medicine, Severance Hospital

Areas of Interest

Echocardiography, Heart Failure, Dyslipidemia, Hypertension

Publications

1. Dobutamine Stress Echocardiography for Left Ventricular Reverse Remodeling in Idiopathic Dilated Cardiomyopathy. Int J Heart Fail. 2025 Jul 7;7(3):152-159.
2. Clinical and Echocardiographic Predictors for the Presence of Late Gadolinium Enhancement on Cardiac Magnetic Resonance Imaging in Patients with Carbon Monoxide Poisoning. Diagnostics (Basel). 2023 Dec 27;14(1):60.
3. Epidemiologic Profile of Patients With Valvular Heart Disease in Korea: A Nationwide Hospital-Based Registry Study. J Cardiovasc Imaging. 2023 Jan;31(1):51-61.
4. Temporal trends in heart failure over 11 years in the aging Korean population: A retrospective study using the national health insurance database. PLoS One. 2022 Dec 28;17(12):e0279541.

Lp(a): from lipoprotein biology to atherosclerotic cardiovascular disease

Symposium 13 (K)

2026 KSoLA Awards for Scientific Excellence & Digital Health Media and Ethics

Sep 11(Fri) 12:45–13:40 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Sung Rae Kim (The Catholic University of Korea, Republic of Korea)
Sang-Hyun Kim (Seoul National University, Republic of Korea)

12:45–12:50	2026 KSoLA Award Ceremony for Scientific Excellence
12:50–12:55	2026 KSoLA Award Ceremony for Young Investigator
12:55–13:15	**The quiet signal, no longer – triglyceride at the crossroads of insulin resistance and cardiometabolic risk** Cheol-Young Park (Sungkyunkwan University, Republic of Korea)
13:15–13:20	Q&A
13:20–13:40	**How reliable is statin information on Korean YouTube? validating AI-assisted reliability assessment against expert raters** Ilhak Lee (Yonsei University, Republic of Korea)
13:40–13:45	Q&A

CURRICULUM VITAE

Cheol-Young Park

Professor, Endocrinology & Metabolism, Kangbuk Samsung Hospital,
Sungkyunkwan University, Republic of Korea

Education and Training

1995.02	Kyunghee University, Korea, M.D., Internal Medicine
2001.02	Kyunghee University, Korea, Ph.D., Endocrinology

Employment and Position

2006-Present	Sungkyunkwan University, Korea, Professor
2012-2013	York University, Canada, Visiting Scholar

Important Publications

1. Hypertriglyceridemia in adults with diabetes mellitus in Korea: Sex, age, and lifestyle determinants. J Clin Lipidol 2026 Jul;20(7):1377-1386.
2. Cardiometabolic benefits of fenofibrate in heart failure related to obesity and diabetes. Cardiovasc Diabetol 2024 Sep 16;23(1):343.
3. Fenofibrate and risk of end-stage renal disease: A nationwide cohort study. Diabetes Obes Metab 2024 Oct;26(10):4583-4590.
4. Fenofibrate's impact on cardiovascular risk in patients with diabetes: a nationwide propensity-score matched cohort study. Cardiovasc Diabetol 2024 Jul 18;23(1):263.
5. Fenofibrate add-on to statin treatment is associated with low all-cause death and cardiovascular disease in the general population with high triglyceride levels. Metabolism 2022 Dec:137:155327.

Awards and Honors

2024	대한내분비학회 남곡학술상 수상
2024	보건복지부 장관상
2026	대한 비만학회 문석학술상

The quiet signal, no longer - triglyceride at the crossroads of insulin resistance and cardiometabolic risk

Two decades of LDL-centered therapy have transformed cardiovascular prevention, yet events continue to occur in patients who have reached their LDL-C targets. Triglyceride (TG) — long dismissed as a weak or confounded marker — is re-emerging as the most measurable and modifiable component of this residual risk.

This lecture revisits TG through three axes, drawn entirely from studies in which I served as first or corresponding author.

First, TG as residual risk. Using KNHANES and the Korean National Health Insurance Service, we established the national record of hypertriglyceridemia: 29.6% of Korean adults at ≥150 mg/dL and 16.1% at ≥200 mg/dL, peaking at 47.7% among men in their forties. Critically, 34.4% of patients on lipid-lowering therapy and 32.7% of ASCVD patients with LDL-C <70 mg/dL still have TG ≥150 mg/dL, and nearly half of Korean adults with diabetes are affected. Against this background, fenofibrate added to statin was associated with lower all-cause death (HR 0.79), myocardial infarction (HR 0.76) and stroke (HR 0.83), with consistent benefit in diabetes, a 24% lower risk of end-stage renal disease among 413,715 matched pairs, favorable effects in obesity- and diabetes-related heart failure, and — most recently — a reduced incidence of hepatocellular carcinoma.

Second, TG as metabolic signal. The triglyceride-glucose (TyG) index, computed from two routine laboratory values, identified thresholds for all-cause (8.23) and cardiovascular (8.14) mortality, and predicted nonalcoholic fatty liver disease and incident dementia, including Alzheimer's and vascular subtypes.

Third, TG as a future platform.Progress now depends on shifting from TG concentration to remnant cholesterol biology, on precision stratification of patients with diabetes, high TG and elevated TyG, and on translating Korea's nation-scale cohorts into global standards.

TG is no longer a quiet signal; it is where insulin resistance meets cardiometabolic risk.

CURRICULUM VITAE

Ilhak Lee

Professor, Department of Medical Humanities and Social Sciences,
Yonsei University College of Medicine, Seoul, Republic of Korea

Education and Training

1992-1999	Yonsei University, M.D., Medicine
2003-2010	Yonsei University, Ph.D., Bioethics

Employment and Position

2012-Present	Yonsei University College of Medicine, Korea, Professor, Dept of Medical Humanities and Social Sciences
2026-Present	Korean Bioethics Association, President
2026-Present	Korean Society for Medical Ethics, Vice-president

Important Publications

1. The communication I had with him back then is still stuck in my mind. Bereaved families of cancer patients' experiences for end-of-life communication. Supportive care in cancer (2023)
2. Characteristics of Life-Sustaining Treatment Decisions: National Data Analysis in South Korea. Asian Bioethics Review (2024)
3. Organ Donation and Transplantation Coordinators’ Experience and needs for ethics education. Nursing Ethics (2025)
4. Amendment Proposals for the Act on Decisions on Life-Sustaining Treatment to Improve End-of-Life Care. JKSME (2024)

How reliable is statin information on Korean YouTube? validating AI-assisted reliability assessment against expert raters

Patients increasingly turn to YouTube for information about statins and cholesterol, yet the reliability of this content remains uncertain. Systematic reviews consistently find health information on the platform to be of moderate-to-poor quality, and misleading videos can attract disproportionate attention. Of particular clinical concern are narratives that position omega-3 supplements as a substitute for statins, or that encourage discontinuation of therapy—claims that may translate into real behavioral risk.

This talk presents the design and rationale of an ongoing cross-sectional study evaluating Korean-language YouTube content on statins. Videos are sampled through the official API using a predefined search matrix, transcribed, and rated with a shared codebook that combines modified DISCERN, the Global Quality Score, and JAMA benchmark criteria. Two features distinguish the study. First, we examine how omega-3 is framed within statin content—as adjunct, as alternative, or as general information. Second, and as the methodological core, each video is rated independently by cardiology and endocrinology experts and by a large language model applying the identical codebook; we then quantify human-AI concordance, overall and by item type (objective versus judgment items).

The study will characterize the reliability of Korean statin-related content and its association with uploader type and popularity, while testing whether AI-assisted coding can reliably support—rather than replace—expert reliability assessment, and identifying where it cannot. A pre-registered protocol, an explicit coding principle that evaluates evidence quality and transparency rather than pro-statin stance, and a modality-sensitivity sub-study address key validity concerns.

By clarifying both the reliability landscape and the appropriate role of artificial intelligence in monitoring health misinformation, the study aims to give clinicians and lipid societies concrete, evidence-based cues for counseling patients who bring YouTube claims into the clinic.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 14

Sex-specific Lipid Biology and Cardiometabolic Risk across the Lifespan

Sep 11(Fri) 12:45-13:45 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Mi-Seung Shin (Gachon University, Republic of Korea)
Jun-Hee Lee (Hallym University, Republic of Korea)

12:45-13:00	**Sex differences in lipids and lipoproteins and residual cardiovascular risk: beyond LDL cholesterol** Soo-Jin Kim (Kosin University, Republic of Korea)
13:00-13:15	**Early-onset dyslipidemia in young adults: screening strategies and lifetime ASCVD risk assessment** Jaehyun Bae (Hallym University, Republic of Korea)
13:15-13:30	**Menopause and cardiometabolic transition: optimizing lipid management in midlife women** In-Jeong Cho (Ewha Womans University, Republic of Korea)
13:30-13:45	**Panel Discussion** Hack-Lyoung Kim (Seoul National University, Republic of Korea) Ye Seul Yang (Seoul National University, Republic of Korea) Seok Oh (Chonnam National University, Republic of Korea)

CURRICULUM VITAE

Soo-Jin Kim

Kosin University, Republic of Korea

Education and Professional Experience

- Graduated from Dong-A University College of Medicine (2009)
- Completed internship at Dong-A University Medical Center
- Completed residency in Internal Medicine at Dong-A University Medical Center
- Clinical Fellow in Cardiology, Dong-A University Medical Center
- Attending Physician, Division of Cardiology, Ulsan Hospital
- Assistant Professor, Division of Cardiology, Department of Internal Medicine, Kosin University Gospel Hospital (September 2021-Present)

- Board-Certified Internist / Board-Certified Cardiologist / PhD in Medicine

Professional Memberships

Member, Korean Society of Cardiology
Member, Korean Association of Internal Medicine
Member, Korean Society of Interventional Cardiology
Member, Korean Society of Hypertension
Member, Korean Society of Cardiometabolic Syndrome

Sex differences in lipids and lipoproteins and residual cardiovascular risk: beyond LDL cholesterol

Atherosclerotic cardiovascular disease (ASCVD) mortality is rising fastest among middle-aged women, and delayed diagnosis and undertreatment do not fully account for this burden. A complementary explanation lies in the biology of atherogenic lipoproteins, which differs between the sexes in ways that conventional lipid panels obscure.

Women display a favorable lipid phenotype only between roughly the second and fifth decades; before puberty and again from midlife onward, their profile becomes comparably or more atherogenic than that of men. In a contemporary prospective cohort of over 430 000 adults, LDL cholesterol, apolipoprotein B and lipoprotein(a) showed modestly stronger associations with incident cardiovascular disease in men, whereas triglycerides were stronger in women and apolipoprotein A1 less protective — differences that are directionally consistent but small relative to the two-fold higher absolute event rate in men. These data nonetheless suggest that residual risk in women is not fully captured by LDL cholesterol concentration alone.

First, particle quality: directly measured small dense LDL cholesterol remains an independent risk enhancer after full adjustment, whereas calculated estimates do not; LDL subclasses and apolipoprotein B rise abruptly around the final menstrual period and track with subclinical atherosclerosis. Second, lipoprotein(a): concentrations rise after menopause and fall with oral hormone therapy, yet menopausal status does not materially modify the lipoprotein(a)-ASCVD association, while venous thromboembolic risk appears conditional on oestrogen exposure; individual trajectories across the transition are heterogeneous, supporting reassessment rather than a single lifetime measurement in selected women. Third, inflammation: in long-term follow-up of initially healthy women, high-sensitivity C-reactive protein predicted events more strongly than LDL cholesterol, with the three markers contributing independently.

LDL-lowering confers equivalent proportional benefit in both sexes, yet women receive less intensive therapy and reach targets less often. Recognising sex-specific residual risk — and closing the treatment gap — is an actionable target.

Keywords: Sex differences, Residual cardiovascular risk, Lipoprotein(a), Small dense LDL, Menopause, Inflammation

CURRICULUM VITAE

Jaehyun Bae

Associate Professor, Hallym University, Republic of Korea

Education and Training

2010.02	Yonsei University College of Medicine, Korea, M.D., Medicine
2022.02	Graduate School, Yonsei University College of Medicine, Ph.D., Internal Medicine

Employment and Position

2020-2024	Catholic Kwandong University College of Medicine, Korea, Assistant Professor
2024-2025	Hallym University Kangnam Sacred Heart Hospital, Korea, Clinical Assistant Professor
2025-Present	Hallym University College of Medicine, Korea, Associate Professor

Important Publications

1. Jaehyun Bae, Minyoung Lee*, Yong-Ho Lee, Sang-Guk Lee, Byung-Wan Lee. The Impact of Ketogenic Capacity on Lipid Profile in Individuals with Prediabetes or Newly Diagnosed Type 2 Diabetes. Int J Mol Sci. 2025 Sep 3;26(17):8566. (*co-author)
2. Jaehyun Bae, Young-Eun Kim*, Keum Ji Jung, Sun Ha Jee, Byung-Wan Lee. Association between serum beta-hydroxybutyrate levels and risk of type 2 diabetes mellitus in patients with impaired fasting glucose. Nutr Diabetes. 2025 Apr 16;15(1):16. (*co-author)
3. Jaehyun Bae, Min Heui Yu*, Minyoung Lee, Bong-Soo Cha, Byung-Wan Lee. Effectiveness and Safety of Oral Quadruple Combination Therapy in Patients with Type 2 Diabetes: A Systematic Review and Meta-Analysis. Endocrinol Metab (Seoul). 2025 Apr;40(2):258-267. (*co-author)
4. Jaehyun Bae, Sejeong Lee*, Seung Up Kim, Minyoung Lee, Yong-ho Lee, Eun Seok Kang, Bong-Soo Cha, Byung-Wan Lee. Intact ketogenesis predicted reduced risk of moderate-severe metabolic-associated fatty liver disease assessed by liver transient elastography in newly diagnosed type 2 diabetes. Front Endocrinol (Lausanne). 2024 Jan 8:14:1306134. (*co-author)

Early-onset dyslipidemia in young adults: screening strategies and lifetime ASCVD risk assessment

Atherosclerosis is a lifelong process driven by cumulative exposure to atherogenic lipoproteins. Although dyslipidemia has traditionally been regarded as a health concern primarily affecting middle-aged and older adults, early-onset dyslipidemia in young adults is increasingly recognized as a silent yet important threat to long-term cardiovascular health. Recent epidemiological data indicate that the prevalence of dyslipidemia among young adults has remained persistently high or continues to increase globally. In Korea, dyslipidemia affects nearly 40% of men and 16% of women in their 30s.

Despite this substantial disease burden, young adults exhibit the lowest rates of dyslipidemia awareness and treatment, highlighting a considerable gap in preventive care. Although multiple factors likely contribute to this gap, one important explanation is the reliance on conventional short-term cardiovascular risk assessment. Ten-year risk models, such as those based on the Pooled Cohort Equations, are strongly influenced by age. Consequently, young adults—particularly those without major comorbidities or other conventional cardiovascular risk factors—are generally perceived as being at low short-term risk and are therefore less likely to be considered candidates for active preventive intervention. However, prolonged exposure to elevated low density lipoprotein (LDL) cholesterol, often conceptualized as cumulative LDL-C exposure or "LDL-years," increases lifetime cardiovascular risk. Early intervention for dyslipidemia in young adults may enable primary prevention to begin at a healthier stage, before substantial atherosclerotic burden has accumulated. Therefore, the early detection and appropriate management of early-onset dyslipidemia are essential for reducing long-term cardiovascular risk.

This presentation highlights current epidemiological trends in dyslipidemia among young adults, examines the limitations of conventional screening and risk assessment approaches, and outlines practical strategies for early detection, lifestyle modification, and risk-stratified management to reduce the lifetime burden of cardiovascular disease.

CURRICULUM VITAE

In-Jeong Cho

Cardiology Division, Ewha Womans University Seoul Hospital,
Ewha Womans University College of Medicine, Seoul, Republic of Korea

Academic Education

2002	M.D., Ewha Womans University College of Medicine, Seoul, Republic of Korea
2009	Master's Degree, Ewha Womans University Graduate School, Seoul, Republic of Korea
2018	Ph.D., Ewha Womans University Graduate School, Seoul, Republic of Korea

Postgraduate Training

2002-2003	Internship, Ewha Medical Center, Ewha Womans University College of Medicine, Seoul, Republic of Korea
2004-2008	Residency in Internal Medicine, Ewha Medical Center, Ewha Womans University College of Medicine, Seoul, Republic of Korea
2008-2009	Clinical Fellow in Cardiology, Ewha Medical Center, Ewha Womans University College of Medicine, Seoul, Republic of Korea

Licensure and Certification

2002	Diplomate, Licensure in Medicine (Korea)
2008	Diplomate, Korean Board of Internal Medicine
2011	Diplomate, Korean Cardiovascular Subspecialty Board
2011	Certification Board of Echocardiography (Korea)
2016	Certification Board of Cardiovascular Computed Tomography (USA)

Appointments and Professional Activities

2009-2011	Clinical Research Assistant Professor in Cardiology, Severance Cardiovascular Hospital, Seoul, Republic of Korea
2013-2019	Clinical Assistant Professor in Cardiology, Severance Cardiovascular Hospital, Seoul, Republic of Korea
2019-	Associate Professor in Cardiology, Ewha Womans University Seoul Hospital, Seoul, Republic of Korea
2024-	Director, Advanced Biomedical Research Institute, Ewha Womans University Seoul Hospital, Seoul, Republic of Korea

Menopause and cardiometabolic transition: optimizing lipid management in midlife women

Estrogen decline during the menopausal transition accelerates adverse lipid changes—rising LDL-C, impaired HDL function, and elevated Lp(a)—driving increased cardiovascular risk. This talk reviews mechanisms of midlife dyslipidemia, high-risk phenotypes (early/surgical menopause), hormone therapy's cardiovascular implications, and sex-specific guideline recommendations for statin initiation and intensity, offering a practical risk-based approach to lipid management in midlife women.

Symposium 15

Official Launch of the 2026 KSoLA Guidelines for the Management of Dyslipidemia

Sep 11(Fri) 13:45–15:15 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Sang-Hyun Kim (Seoul National University, Republic of Korea)
Woo Je Lee (University of Ulsan, Republic of Korea)

13:45–13:50	**What's new in the 2026 Korean guidelines for the management of dyslipidemia** Hyun-Jin Kim (Hanyang University, Republic of Korea)
13:50–14:08	**Korean epidemiology and risk modifiers for establishing LDL-C goal strategies** Jong Han Choi (Konkuk University, Republic of Korea)
14:08–14:26	**Practical algorithm for intensive lipid lowering therapy in the 2026 Korean guidelines** Jaehoon Chung (Gachon University, Republic of Korea)
14:26–14:44	**Nutritional management and lifestyle modification tailored to the Korean dietary pattern** Garam Jo (Hannam University, Republic of Korea)
14:44–15:02	**Optimized lipid management for special populations** Byung Sik Kim (Hanyang University, Republic of Korea)
15:02–15:15	**Panel Discussion** Hyun-Jin Kim (Hanyang University, Republic of Korea) Eunju Park (Kyungnam University, Republic of Korea) Jee Hee Yoo (Chung-Ang University, Republic of Korea) Hye Moon Chung (Kyung Hee University, Republic of Korea)

CURRICULUM VITAE

Jong Han Choi

Assistant Professor, Division of Endocrinology and Metabolism, Department of Internal Medicine, Konkuk University Medical Center, Konkuk University School of Medicine, Republic of Korea

Education and Training

2004.03-2010.02	Chonnam National University College of Medicine, M.D., Medicine
2014.03-2019.08	University of Ulsan College of Medicine, M.S., Medicine
2021.03-2024.02	Konkuk University School of Medicine, Ph.D., Medicine

Employment and Position

2010.03-2011.02	Asan Medical Center, Intern
2011.03-2015.02	Department of Internal Medicine, Asan Medical Center, Resident
2018.05-2020.02	Division of Endocrinology and Metabolism, Asan Medical Center, Clinical Fellow
2020.03-Present	Konkuk University Medical Center, Assistant Professor

Important Publications

1. Kim SK, Kim DL, Song K, Kang S, Lee BW, Choi JH. Contemporary Type 2 Diabetes Guidelines: Converging Evidence, Diverging Strategies, and the Position of the Korean Diabetes Association Framework. Endocrinol Metab. 2026.
2. Choi JH, Song SJ, Kim SK, Cho JW, Bae JH, Moon S, et al. Health Effects of Sugar-Sweetened and Artificially Sweetened Beverages: Umbrella Review and Evidence-Based Consensus Statement of the Korean Diabetes Association and the Korean Nutrition Society. Diabetes Metab J. 2026.
3. Choi JH, Kang S, Kim SK, Kim WJ, Kim JM, Bae JH, et al. Bridging Evidence and Practice: A Consensus Statement from the Korean Diabetes Association on Diabetes Screening, Pharmacological Treatment and Severe Diabetes. Diabetes Metab J. 2025.
4. Choi JH, Koo BK, Yang YS, Min SH, Park JS, Rhee SY, et al. Initial Pharmacological Strategies in People with Early Type 2 Diabetes Mellitus: A Systematic Review and Network Meta-Analysis. Diabetes Metab J. 2025.

Korean epidemiology and risk modifiers for establishing LDL-C goal strategies

Optimal LDL cholesterol (LDL-C) targets should reflect both the baseline cardiovascular risk of the population and the presence of clinical factors that modify individual risk. The 2026 Korean Society of Lipid and Atherosclerosis (KSoLA) Guidelines maintain a risk-based LDL-C target framework while introducing a more individualized approach through the incorporation of risk modifiers.

This lecture reviews the epidemiological evidence underlying LDL-C goal setting in Korea and explains how contemporary evidence has shaped the updated guideline recommendations. Although cardiovascular disease remains the leading cause of mortality in Korea, substantial heterogeneity exists according to age, diabetes status, and the burden of accompanying cardiovascular risk factors. The Korean guideline therefore continues to stratify treatment goals according to established risk categories while emphasizing more intensive LDL-C lowering in patients with higher baseline risk.

In addition to traditional major cardiovascular risk factors, the updated guideline highlights the role of risk modifiers—including obesity, chronic inflammatory diseases, elevated lipoprotein(a), apolipoprotein B, high-sensitivity C-reactive protein, physical inactivity, psychosocial stress, sleep apnea, and female-specific reproductive factors—to support individualized clinical decision-making. These modifiers are intended to complement, rather than replace, conventional risk assessment and may justify more intensive lipid-lowering strategies, particularly in primary prevention.

The lecture also compares the Korean approach with recent international guidelines, including the 2026 ACC/AHA and the 2025 ESC/EAS recommendations, discussing similarities and differences in risk assessment, treatment targets, and implementation strategies. Finally, the need for a Korean-specific quantitative cardiovascular risk prediction model will be highlighted as a future direction.

CURRICULUM VITAE

Jaehoon Chung

Professor, Gachon University, Republic of Korea

Education and Training

2010.02	Yeung-nam University, Korea, M.D, Medicine
2026.02	Dongguk University, Korea, Master of Internal Medicine

Employment and Position

2017-2024	National Medical Center, Clinical Doctor
2024-2026.06	Dongguk University Hospital, Assistant Professor
2026.07-Present	Gil Medical Center, Associate Professor

Important Publications

1. Chung, J., Kim, H. L., Lim, W. H., Seo, J. B., Kim, S. H., Zo, J. H., & Kim, M. A. (2019). Association between invasively measured aortic pulse pressure and orthostatic hypotension in patients undergoing invasive coronary angiography. Journal of hypertension, 37(10), 1966-1973.
2. Chung, J., Kim, H. L., Lee, J. P., Lim, W. H., Seo, J. B., Kim, S. H., Zo, J. H., & Kim, M. A. (2020). Association of the Serum Osteoprotegerin Level With Target Organ Damage in Patients at High Risk of Coronary Artery Disease. Circulation journal : official journal of the Japanese Circulation Society, 85(1), 69-76.
3. Chung, J., Han, J. K., Yang, H. M., Park, K. W., Kang, H. J., Koo, B. K., Jeong, M. H., Kim, H. S., & investigators for Korea Acute Myocardial Infarction Registry (KAMIR) (2021). Long-term efficacy of vasodilating β-blocker in patients with acute myocardial infarction: nationwide multicenter prospective registry. The Korean journal of internal medicine, 36(Suppl 1), S62-S71.
4. Chung, J., Min, K. W., Son, B. K., Kim, D. H., & Kim, H. L. (2021). Association between histological severity of Helicobacter pylori infection and cardiovascular risk scores in the Korean population. Atherosclerosis, 333, 124-130.
5. Chung, J., Kim, H. L., Lim, W. H., Seo, J. B., Zo, J. H., Kim, M. A., & Kim, S. H. (2023). New onset diabetes mellitus and cardiovascular outcomes according to statin intensity in patients after drug-eluting stent implantation in Asian patients. Scientific reports, 13(1), 16061. https://doi.org/10.1038/s41598-023-42277-w

Awards and Honors

Research Interest
Atherosclerosis
Arterial stiffness
Lipid
Hypertension
Metabolic syndrome
Coronary artery disease

Practical algorithm for intensive lipid lowering therapy in the 2026 Korean guidelines

The 2026 Korean dyslipidemia guidelines emphasize risk-based LDL-C targets and treatment intensity tailored to the required degree of LDL-C reduction. Statins remain the foundation of therapy, with early addition of non-statin therapy when substantial LDL-C reduction is required. A goal-directed strategy with early combination therapy and timely reassessment may improve rapid achievement of LDL-C targets.

CURRICULUM VITAE

Garam Jo

Assistant Professor, Department of Food and Nutrition, Hannam University, Republic of Korea

Education and Training

2021.02	Korea University, Korea, Ph.D., Public Health Science
2016.08	Korea University, Korea, B.S., Food and Nutrition

Employment and Position

2023-2026	Institute for BioMaterials, Korea University, Korea, Research Professor
2025	Harvard T.H. Chan School of Public Health, USA, Visiting Scientist
2022-2023	BK21 Four Institute of Precision Public Health, Korea University, Korea, Research Professor
2021-2022	Division of Cardiovascular Disease Research, Department of Chronic Disease Convergence Research, Korea National Institute of Health, Korea, Research Fellow

Important Publications

1. Jo G*, Park D*, Kim S, Jun HJ, Magkos F, Merino J, Sun Q, Kim R, Subramanian SV, Franks W.P, Shin MJ. Obesity Transitions by Age, Period, and Cohort: A Population-Based Application of the Lancet Commission Framework in the United States and Korea. Diabetes Metab J. 2026 Apr 10 * Equal contribution.
2. Jeong D*, Jo G*, Kim R, Subramanian SV, Shin MJ, Oh H. Nationwide trends in overall and abdominal obesity among Korean adults, 2007-2022: An age-period-cohort analysis. Int J Epidemiol. 2026;55(3):dyag068 * Equal contribution.
3. Jun HJ, Kim S, Jo G. Age-Period-Cohort Analysis of dietary sodium, potassium, and sodium-to-potassium ratio in Korea. Epidemiol Health. 2025 Nov 4:e2025062.
4. Lee J*, Jo G*, Park D, Jun HJ, Bae JH, Shin MJ. The Association between Advanced Liver Fibrosis and Mortality Is Modified by Dietary Quality among Korean Adults: Results from the Korea National Health and Nutrition Examination Survey with Mortality Data. Nutrients. 2023;15(6):1501,* Equal contribution.
5. Jo G, Park D, Lee J, Kim R, Subramanian SV, Oh H, Shin MJ. Trends in Diet Quality and Cardiometabolic Risk Factors Among Korean Adults, 2007-2018. JAMA Netw Open. 2022; 5(6):e2218297.

Research Interest

Nutrition Epidemiology; Molecular Epidemiology; Cardiometabolic health; Multi-omics; Epigenome

Nutritional management and lifestyle modification tailored to the Korean dietary pattern

Lifestyle intervention is a fundamental component of dyslipidemia care and is recommended for all patients, regardless of pharmacologic treatment. The 2026 Koren Society of Lipid and Atherosclerosis Guidelines integrate dietary modification, physical activity, smoking cessation, and alcohol restriction.

The updated nutritional recommendations focus on energy balance, weight management, and improved dietary fat and carbohydrate quality. Rather than emphasizing nutrient-specific targets alone, the guidelines promote sustainable dietary patterns such as the Mediterranean, DASH, and plant-based diet, centered on whole grains, vegetables, fruits, legumes, nuts, and fish. These dietary principles should be adapted to the Korean dietary context to enhance their feasibility and long-term adherence. Particular emphasis should be placed on reducing refined grains, processed meat, sugar-sweetened beverages, and ultra-processed foods.

Regular aerobic and resistance exercise, reduced sedentary time, and complete smoking cessation are also recommended. Alcohol intake should be minimized, and abstinence is strongly advised in patients with hypertriglyceridemia. Taken together, these recommendations support lifestyle management tailored to individual lipid profiles and Korean dietary practices.

CURRICULUM VITAE

Byung Sik Kim

Division of Cardiology, Hanyang University Guri Hospital, Republic of Korea

Education

2003-2009	M.D., College of Medicine, Hanyang University
2021-2022	M.S. in Medicine, Hanyang University
2023-2024	Ph.D. in Medicine, Hanyang University

Professional Experience

Intern and Resident, Department of Internal Medicine, Hanyang University Hospital
Military Doctor, Republic of Korea Army
Clinical Fellow, Division of Cardiology, Hanyang University Hospital
Chief, Division of Cardiology, Gwangmyeong Sungae Hospital and Seoul Sungae Hospital
Clinical Assistant Professor and Assistant Professor, Division of Cardiology, Hanyang University Guri Hospital
Present: Associate Professor, Division of Cardiology, Hanyang University Guri Hospital

Optimized lipid management for special populations

Lipid management should be individualized according to cardiovascular risk, disease-specific evidence, life stage, comorbidities, and treatment safety. The sixth edition of the Korean Guidelines for the Management of Dyslipidemia provides updated recommendations for patients with coronary artery disease, ischemic stroke, chronic kidney disease, and diabetes, including revised LDL cholesterol targets and appropriate use of combination lipid-lowering therapy.

The guidelines also address populations in whom conventional risk assessment or treatment strategies require modification, including patients with chronic inflammatory disease, HIV infection, cancer, familial hypercholesterolemia, and children, older adults, and pregnant women. Key considerations include disease-specific cardiovascular risk, drug-drug interactions, renal function, frailty, reproductive safety, and long-term cumulative exposure to atherogenic lipoproteins.

This session will summarize the major updates and provide practical guidance for individualized lipid management in these special populations.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 16

Evolution of High-Risk Coronary Plaques: From Detection to Prevention

Sep 11(Fri) 13:45-15:15 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Byung Jin Kim (Sungkyunkwan University, Republic of Korea)
Jung Rae Cho (Hallym University, Republic of Korea)

13:45-14:03	**Evolution of vulnerable plaques: from natural history to preventive intervention** Jinho Lee (Kyung Hee University, Republic of Korea)
14:03-14:21	**High-risk plaque features on CCTA: identification and clinical implications** Sang Yoon Lee (Sungkyunkwan University, Republic of Korea)
14:21-14:39	**Intravascular imaging of vulnerable plaques: insights from IVUS and OCT** Ju Hyeon Kim (University of Ulsan, Republic of Korea)
14:39-14:57	**Coronary physiology and plaque vulnerability: integrative relationship and prognostic implications** Seokhun Yang (Seoul National University, Republic of Korea)
14:57-15:15	**Panel Discussion** Bom Lee (CHA University, Republic of Korea) Hyun Sung Joh (Seoul National University, Republic of Korea) Hyung Joon Joo (Korea University, Republic of Korea)

CURRICULUM VITAE

Jinho Lee

Assistant Professor, Kyung Hee University, Republic of Korea

Education and Training

2017.02	Kyung Hee University, Korea, M.D
2024.02	Ulsan University, Korea, Ph.D., Internal Medicine
2018.02	Asan Medical Center, M.D, Intern
2021.02	Asan Medical Center, M.D, Internal Medicine
2024.02	Asan Medical Center, M.D, Cardiology

Employment and Position

2024.03-	Kyung Hee University Hospital, Korea, Assistant Professor

Evolution of vulnerable plaques: from natural history to preventive intervention

The majority of acute myocardial infarctions arise not from severely stenotic, flow-limiting lesions but from non-obstructive plaques that rupture, erode, or fracture silently — a paradox that has driven two decades of effort to define, image, and ultimately treat the "vulnerable plaque." This talk traces that evolution from conceptual origins to contemporary preventive strategy.

The story begins with Naghavi and Fuster's reframing of a lesion-centric problem as a patient-centric one: vulnerability reflects the composite biology of the arterial wall and the whole vascular tree, not a single culprit segment. Pathologically, Virmani's classification anchors this narrative, tracing progression from the fatty streak through pathological intimal thickening to the thin-cap fibroatheroma, and distinguishing the three mechanisms of acute coronary syndrome — plaque rupture, plaque erosion, and calcified nodule — each with distinct underlying biology and, as EROSION study demonstrated, distinct management implications: erosion-related events with preserved flow and non-critical residual stenosis were safely managed with antithrombotic therapy alone, without stenting.

Advances in intracoronary and non-invasive imaging — virtual-histology IVUS, near-infrared spectroscopy (NIRS)-IVUS, optical coherence tomography, and CT-based plaque characterization — enabled a series of natural history studies (PROSPECT, the Lipid-Rich Plaque study, CLIMA, and PROSPECT II/PROSPECT ABSORB) that identified thin-cap fibroatheroma, large plaque burden, and lipid-rich necrotic cores as markers of non-culprit lesion risk. Yet these same studies exposed a persistent limitation: the positive predictive value of any single imaging signature remains under 10%, and roughly half of lesion-related events occur in plaques that would not have been flagged.

This gap has reframed prevention around two complementary strategies: intensified systemic therapy — statins, PCSK9 inhibitors, and anti-inflammatory agents, whose evidence remains mixed, as the positive COLCOT and LoDoCo2 colchicine trials contrast with the neutral CLEAR-SYNERGY result — and, more recently, mechanism- and imaging-guided local intervention, exemplified by the PREVENT trial, which demonstrated that preventive percutaneous coronary intervention of non-flow-limiting, imaging-defined high-risk plaque reduced major adverse cardiac events. Taken together, these data argue for a mechanism-guided, rather than morphology-guided, approach to the vulnerable plaque — one that integrates whole-patient atherosclerotic risk with lesion-specific biology.

CURRICULUM VITAE

Sang Yoon Lee

Sungkyunkwan University, Republic of Korea

Education

Mar. 2007-Feb. 2011	School of Electrical Engineering, Seoul National University
Mar. 2011-Feb. 2015	Master of Medicine (M.M.), Sungkyunkwan University School of Medicine
Mar. 2023-Sep. 2025	Completed PhD Coursework in Internal Medicine, Sungkyunkwan University School of Medicine

Board Certification

Physician License (Feb 25, 2015)
Board Certified in Internal Medicine (Mar 2, 2020)

Professional Experience

Mar. 2015-Feb. 2016	Intern, Samsung Medical Center
Mar. 2016-Feb. 2020	Resident, Department of Internal Medicine, Samsung Medical Center
Mar. 2020-Apr. 2023	Medical Officer, Republic of Korea Air Force
May 2023-Feb. 2025	Fellow, Division of Cardiology, Samsung Medical Center
Mar. 2025-Present	Clinical Assistant Professor, Division of Cardiology, Samsung Medical Center, Sungkyunkwan University School of Medicine

Professional Memberships

Korean Society of Cardiology
Korean Association of Internal Medicine
Korean Society of Interventional Cardiology

High-risk plaque features on CCTA: identification and clinical implications

In patients with coronary artery disease, the risk associated with atherosclerotic plaques can be assessed according to their morphological characteristics. Low-risk plaques, characterized by a thick fibrous cap and a small necrotic lipid core, generally exhibit gradual progression and are more commonly associated with stable clinical presentations, such as chronic coronary syndrome. In contrast, high-risk plaques, characterized by a thin fibrous cap and a large necrotic lipid core, are prone to rupture, potentially resulting in acute thrombosis, abrupt coronary occlusion, and acute coronary syndrome. Accordingly, considerable efforts have been made to identify and treat high-risk plaques.

Traditional approaches to cardiovascular risk assessment include clinical risk scores and coronary artery calcium scoring. More recently, intravascular imaging modalities, such as intravascular ultrasound and optical coherence tomography, have enabled detailed characterization of individual plaques and evaluation of their potential to cause future coronary events. However, clinical risk scores do not directly assess coronary plaques, and coronary artery calcium scoring estimates risk using calcification as an indirect surrogate of the overall atherosclerotic burden. Although intravascular imaging provides detailed information on plaque morphology, its requirement for invasive coronary angiography limits its widespread application in routine clinical practice.

Coronary computed tomography angiography (CCTA) addresses several of these limitations by enabling noninvasive visualization and characterization of coronary atherosclerotic plaques. Several high-risk plaque features have been identified on CCTA, among which low-attenuation plaque is particularly important. A subsequent analysis of the SCOT-HEART trial demonstrated that low-attenuation plaque burden provided incremental prognostic information beyond conventional noninvasive risk measures. As the clinical use of CCTA continues to increase, recognizing and appropriately interpreting these high-risk plaque characteristics has become increasingly important. This presentation will review the key high-risk plaque features identifiable on CCTA and discuss their clinical implications and potential management strategies.

CURRICULUM VITAE

Ju Hyeon Kim

Clinical Associate Professor, Cardiology, Asan Medical Center, Seoul, Republic of Korea

Education and Training

2005.03–2011.02	Korea University College of Medicine, Korea, M.D., Medicine
2012.03–2016.02	Asan Medical Center, Korea, Residency, Internal Medicine
2014.03–2016.02	University of Ulsan College of Medicine, Korea, M.S., Medical Science
2019.05–2021.10	Asan Medical Center, Korea, Fellowship, Cardiology
2022.03–2024.08	Korea University College of Medicine, Korea, Ph.D., Medical Science

Employment and Position

2021.11–2025.02	Korea University Medical Center, Korea, Clinical Assistant Professor, Cardiology
2025.03–2026.02	Asan Medical Center, Korea, Clinical Assistant Professor, Cardiology
2026.03–Present	Asan Medical Center, Korea, Clinical Associate Professor, Cardiology

Important Publications

1. Kim JH, Kang DY, Ahn JM, et al. Optimal minimal stent area after crossover stenting in patients with unprotected left main coronary artery disease. EuroIntervention. 2025;21:e1069-e1080.
2. Kim JH, Hong SJ, Lim S, et al. Optical coherence tomography versus intravascular ultrasound-guided PCI in patients with and without acute coronary syndrome: a prespecified subanalysis of the OCTIVUS trial. EuroIntervention. 2025;21:581-583.
3. Kim JH, Kang DY, Ahn JM, et al. Optimal Minimal Stent Area and Impact of Stent Underexpansion in Left Main Up-Front 2-Stent Strategy. Circ Cardiovasc Interv. 2024;17:e013006.
4. Kim JH, Hong SJ, Cha JJ, et al. Impact of proton pump inhibitor use on clinical outcomes in East Asian patients receiving clopidogrel following drug-eluting stent implantation. BMC Med. 2024;22:335.
5. Kim JH, Lee SJ, Cha JJ, et al. Prognostic Impact of CYP2C19 Genotypes on Long-Term Clinical Outcomes in Older Patients After Percutaneous Coronary Intervention. J Am Heart Assoc. 2024;13:e032248.

Intravascular imaging of vulnerable plaques: insights from IVUS and OCT

Coronary angiography, and to a large extent computed tomography, describes the lumen; intravascular imaging describes the substrate that fails. This lecture reviews what intravascular ultrasound (IVUS), optical coherence tomography (OCT) and near-infrared spectroscopy (NIRS) actually measure, what those measurements predict, and what they do not.

Two questions are routinely conflated. Explaining why a culprit lesion failed is a retrospective task at which intravascular imaging excels: OCT distinguishes plaque rupture, erosion and eruptive calcified nodule in vivo. Predicting which untreated lesion will cause the next event is a different task, judged by predictive values and absolute event rates rather than by hazard ratios alone.

The three modalities measure largely orthogonal axes of risk. IVUS, with 5-8 mm of penetration, resolves the external elastic membrane and therefore plaque burden and remodelling; a plaque burden ≥ 70% remains the most reproducible lesion-level predictor. OCT, at 10-20 µm axial resolution, measures fibrous cap thickness, lipid arc and surface pathology, but loses the vessel wall behind a lipid pool. NIRS quantifies lipid chemistry. Risk concentrates where these axes converge: in PROSPECT II, lesions with both a plaque burden ≥70% and a maxLCBI4mm ≥ 324.7 had a 4-year lesion-level event rate of 7.0% versus 0.2%. A negative fractional flow reserve excludes ischaemia but not adverse plaque biology, as COMBINE OCT-FFR and PECTUS-obs have shown.

Even the strongest phenotype remains probabilistic. In CLIMA, lesions with all four high-risk OCT features carried a hazard ratio of 7.54, yet the 1-year event rate was 19.4%, while 97% of patients without that phenotype remained event-free. Plaque phenotype is also dynamic and modifiable — pharmacologically through lipid lowering, and mechanically, as PROSPECT ABSORB showed. Whole-pullback artificial-intelligence analysis and structural-molecular imaging are the logical next steps.

No single imaging feature identifies an inevitable culprit: IVUS, OCT and NIRS measure complementary dimensions of risk — burden, cap integrity, surface pathology and lipid biology — and morphology defines a substrate whose fate is decided by the patient and the local haemodynamic environment.

CURRICULUM VITAE

Seokhun Yang

Clinical Assistant Professor, Seoul National University Hospital, Seoul, Republic of Korea

Education and Training

2009-2015	Seoul National University College of Medicine, Korea, M.D., Medicine
2018-2020	Seoul National University College of Medicine, Korea, M.S., Medicine
2023-2025	Seoul National University College of Medicine, Korea, Ph.D., Medicine

Employment and Position

2015-2016	Seoul National University Hospital, Korea, Internship
2016-2020	Seoul National University Hospital, Korea, Residency in Internal Medicine
2020-2021	UN Command Security Battalion of Joint Security Area, Korea, Medical Officer
2021-2023	Presidential Security Service Medical Department, Korea, Medical Officer
2023-2025	Seoul National University Hospital, Korea, Clinical Fellow
2025-Present	Seoul National University Hospital, Korea, Clinical Assistant Professor

Important Publications

1. Yang S, Jung JW, Park SH, et al. Anatomical versus Physiological Lesion Characteristics in Prediction of Acute Coronary Syndrome. JACC Cardiovasc Interv. 2025;18(23):2833-2845.
2. Yang S, Hu X, Zhang J, et al. Long-term Outcomes After Fractional Flow Reserve versus Intravascular Ultrasound Guided PCI: FLAVOUR Trial Extended Follow-up. J Am Coll Cardiol. 2025;86(8):593-606.
3. Hu X, Zhang J, Yang S, et al. Angiography-derived fractional flow reserve versus intravascular ultrasound to guide percutaneous coronary intervention in patients with coronary artery disease (FLAVOUR II): a multicentre, randomised, non-inferiority trial. Lancet. 2025;405(10488):1491-1504.

Coronary physiology and plaque vulnerability: integrative relationship and prognostic implications

oronary plaque characteristics and coronary physiology are both established prognostic markers in coronary artery disease, and the two have long been regarded as separate domains with different implications for treatment. During the natural course of atherosclerosis, however, they evolve together. Plaque growth and luminal narrowing limit epicardial flow and produce myocardial ischemia on a per vessel basis, while repeated cycles of plaque rupture and healing drive acute coronary events on a per lesion basis.

Assessed in isolation, each has clear limits. Vulnerable plaque features carry a low positive predictive value for hard events, and the extent of atherosclerosis appears to matter as much as plaque quality. Physiological indexes guide revascularization reliably, yet clinical events still occur in lesions deferred on the basis of a dichotomous cutoff, and neither approach alone predicts the risk of an individual patient with sufficient accuracy.

The two aspects are linked pathophysiologically. Local hemodynamic conditions, including wall shear stress and the mechanical stress acting on the plaque, participate in plaque formation, progression, and rupture. Because pressure derived indexes reflect the pressure loss across a lesion, they can imply plaque quantity and composition and not only about the degree of luminal narrowing. Coronary microvascular dysfunction shows a similar association with a more vulnerable plaque phenotype, although the causal direction remains unclear.

These interactions explain why the two measures behave in a complementary rather than a redundant manner. Plaque assessment adds prognostic information in lesions that are not ischemic, physiological assessment adds prognostic information in lesions that appear vulnerable, and their combination identifies risk that neither captures alone.

This lecture reviews the pathophysiological basis of this interplay and discusses how integrative assessment of coronary anatomy, plaque quantity and quality, and physiology at both lesion and vessel level may refine personalized risk profiling and treatment selection, with reference to the ongoing trials addressing this question.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 17

Metabolic Dysfunction, Fatty Liver, and Atherosclerosis

Sep 11(Fri) 13:45–15:15 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Seok-Min Kang (Yonsei University, Republic of Korea)
Ki Chul Sung (Sungkyunkwan University, Republic of Korea)

13:45–14:05	**Fatty liver and residual cardiovascular risk: epidemiology and clinical implications** Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea)
14:05–14:25	**From lipotoxicity to atherosclerosis: MASLD/MetALD subtypes and cardiovascular risk – A hepatologist's perspective** Su Jong Yu (Seoul National University, Republic of Korea)
14:25–14:45	**Therapeutic strategies for cardiometabolic risk reduction in MASLD** Toru Miyoshi (Okayama University, Japan)
14:45–15:15	**Panel Discussion** Bo Kyung Koo (Seoul National University, Republic of Korea) Kyung-Jin Kim (Ewha Womans University, Republic of Korea) Heesun Lee (Seoul National University, Republic of Korea)

CURRICULUM VITAE

Eun-Jung Rhee

Professor, Department of Endocrinology and Metabolism, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Republic of Korea

Education and Training

1991-1997	Ewha Womans University, Seoul, Korea, M.D., Medicine
2002-2009	The Catholic University of Korea, Seoul, Korea, M.S., Ph.D., Medicine
1997-1998	Ewha Womans University Hospital, Seoul, Korea, Internship
1999-2003	Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Korea, Residency, Internal Medicine
2003-2005	Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Korea, Fellowship, Endocrinology and Metabolism

Employment and Position

2005-2007	Department of Endocrinology and Metabolism, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Korea, Instructor
2007-Present	Department of Endocrinology and Metabolism, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Korea, Professor
2010-2011	Cardiovascular Division, Brigham and Women's Hospital, Harvard University, Boston, USA, Visiting Professor
2013-Present	Endocrinology and Metabolism (EnM), Deputy Editor
2021-2022, 2025-Present	Journal of Obesity & Metabolic Syndrome (JOMES), Editor-in-Chief

Fatty liver and residual cardiovascular risk: epidemiology and clinical implications

Despite remarkable advances in low-density lipoprotein cholesterol lowering and blood pressure control, a substantial burden of atherosclerotic cardiovascular disease persists — the so-called residual cardiovascular risk. Metabolic dysfunction-associated steatotic liver disease (MASLD), now affecting more than 30% of adults worldwide and rising steeply in Korea alongside obesity and type 2 diabetes, is increasingly recognized as both a marker and a contributor to this residual risk. Notably, cardiovascular disease, not liver disease, is the leading cause of death in patients with MASLD. Large cohort studies and meta-analyses have shown that MASLD is associated with incident cardiovascular events independently of traditional risk factors, with a clear risk gradient according to the severity of hepatic fibrosis. Korean nationwide and health-screening cohort data, including our studies linking fatty liver to subclinical coronary atherosclerosis and to hepatorenal vulnerability signaled by glomerular hyperfiltration, further support these observations.

The recently proposed cardiovascular-kidney-metabolic (CKM) syndrome framework conceptualizes the multidirectional interplay among metabolic risk factors, chronic kidney disease, and cardiovascular disease. The liver, although not explicitly captured in current CKM staging, sits at the center of this crosstalk: MASLD shares its pathophysiologic soil with CKM syndrome — insulin resistance, visceral adiposity, atherogenic dyslipidemia, and systemic inflammation — and amplifies risk across the cardiovascular-kidney axis. Viewing MASLD through the CKM lens offers a unifying framework for identifying individuals whose risk remains hidden from conventional risk calculators.

In this lecture, I will review the epidemiologic evidence linking MASLD with residual cardiovascular risk, position the liver within the CKM syndrome construct, and discuss what these findings mean for cardiovascular risk stratification in everyday practice — setting the stage for the mechanistic and therapeutic discussions that follow in this session.

CURRICULUM VITAE

Su Jong Yu

Professor, Department of Internal Medicine, Seoul National University College of Medicine, Seoul National University Hospital, Republic of Korea

Education and Training

1997.03–2001.02	Seoul National University College of Medicine, Korea, M.D., Medicine
2004.03–2006.02	Graduate School, Seoul National University, Korea, M.S., Internal Medicine
2010.03–2012.02	Graduate School, Seoul National University, Korea, Ph.D., Medical Science

Employment and Position

2011.03–Present	Department of Internal Medicine & Liver Research Institute, Seoul National University College of Medicine, Korea, Professor
2026.07–Present	Department of Internal Medicine, Seoul National University Hospital, Korea, Chief of Medical Affairs
2016.08–2018.01	National Cancer Institute, National Institutes of Health, Bethesda, MD, USA, Visiting Scientist
2026–Present	Journal of Liver Cancer, Editor-in-Chief
2021.12–Present	Gut and Liver, Associate Editor
2026.07–Present	Korean Liver Cancer Association (KLCA), Director of Publications
2022.07–2025.06	Korean Liver Cancer Association (KLCA), Director of Academic Affairs
2023.12–2025.11	Korean Association for the Study of the Liver (KASL), Director of Planning
2019.12–2021.12	Korean Association for the Study of the Liver (KASL), Vice Secretary General & Director of the Liaison Committee
2026–Present	APASL 2028 Seoul (Asian Pacific Association for the Study of the Liver), Chair, Scientific Programme Committee

From lipotoxicity to atherosclerosis: MASLD/MetALD subtypes and cardiovascular risk – A hepatologist's perspective

Cardiovascular disease is a leading cause of morbidity and mortality in metabolic dysfunction-associated steatotic liver disease (MASLD), yet the liver remains largely absent from conventional cardiovascular risk assessment. This lecture will trace the path from the lipotoxic hepatocyte to the arterial wall and define where hepatology can add clinically useful information beyond traditional risk factors.

Hepatic steatosis develops when fatty-acid influx and de novo lipogenesis exceed the liver's capacity for oxidation and export. The pathogenic signal, however, is not triglyceride storage itself, but lipotoxic species – including saturated fatty acids, ceramides, lysophosphatidylcholine, and free cholesterol – that promote organelle stress and inflammatory signaling. In parallel, hepatic overproduction of triglyceride-rich lipoproteins drives remnant cholesterol, small dense LDL, and excess apoB-containing particles, often despite apparently acceptable LDL cholesterol. Atherogenic dyslipidemia, systemic inflammation, endothelial dysfunction, and a prothrombotic milieu thereby connect steatotic liver disease (SLD) to atherosclerosis.

The new SLD nomenclature adds clinical granularity. Korean population data show a graded increase in incident cardiovascular disease across individuals without SLD, MASLD, and MASLD with increased alcohol intake (MetALD), supporting an additive effect of alcohol and metabolic dysfunction. Risk is further modified by diabetes, aggregate cardiometabolic burden, and fibrosis severity. Conversely, PNPLA3 I148M and TM6SF2 E167K can increase hepatic fat and fibrosis without a parallel increase in atherosclerotic risk, partly through reduced export of apoB-containing lipoproteins. Hepatic fat alone is therefore an insufficient cardiovascular risk marker; its metabolic context and export phenotype matter.

A practical hepatology assessment should integrate cardiometabolic burden, quantified alcohol exposure, fibrosis risk, and atherogenic particle burden using non-HDL cholesterol or apoB rather than LDL cholesterol alone. Recognizing MASLD and MetALD as overlapping but clinically distinct phenotypes may reveal residual cardiovascular risk that is otherwise missed and strengthen the interface between hepatology, endocrinology, and cardiology.

CURRICULUM VITAE

Toru Miyoshi

Associate Professor, Department of Cardiovascular Medicine, Okayama University, Japan

Education and Training

1996.03	Okayama University, Japan, M.D.
2006.12	Okayama University, Japan, Ph.D., Medicine

Employment and Position

1996-1998	Hiroshima City Hospital, Japan, Resident
1009-2001	Dept. of Cardiology, Fukuyama City Hospital, Japan, Staff
2001-2004	Dept. of Cardiovascular Medicine, Okayama University, Japan, Staff
2004-2006	University of Virginia, USA, Research Associate
2006-2014	Dept. of Cardiovascular Medicine, Okayama University, Japan, Assistant Professor
2014-2025	Dept. of Cardiovascular Medicine, Okayama University, Japan, Lecturer
2025-Present	Dept. of Cardiovascular Medicine, Okayama University, Japan, Associate Professor

Important Publications

1. Nakashima M, Miyoshi T, Ueki Y, Nishihara T, Miki T, Hara S, Ichikawa K, Osawa K, Yuasa S. Cardio-ankle vascular index as a screening tool for coronary artery disease in patients with metabolic dysfunction-associated steatotic liver disease. J Cardiol. 2026;87(5):429-433.
2. Miyoshi T, Shirai K, Horinaka S, Higaki J, Yamamura S, Saiki A, Takahashi M, Okura T, Kotani K, Kubozono T, Yoshioka R, Kihara H, Hasegawa K, Satoh-Asahara N, Yamaguchi O, Ito H. Association Between Cardio-Ankle Vascular Index and Heart Failure Outcomes: Insights From a Prospective Multicenter Cohort. JACC Adv. 2025;4(12 Pt 2):102187.
3. Nishihara T, Miyoshi T, Nakashima M, Miki T, Toda H, Yoshida M, Ichikawa K, Osawa K, Yuasa S. Prognostic value of metabolic dysfunction-associated steatotic liver disease over coronary computed tomography angiography findings: comparison with no-alcoholic fatty liver disease. Cardiovasc Diabetol. 2024;23(1):167.

Therapeutic strategies for cardiometabolic risk reduction in MASLD

Metabolic dysfunction-associated steatotic liver disease (MASLD) is closely associated with obesity, type 2 diabetes, dyslipidaemia, hypertension, and chronic kidney disease, and is increasingly recognised as a systemic cardiometabolic disorder rather than an isolated liver disease. Cardiovascular disease remains a major cause of morbidity and mortality in patients with MASLD, highlighting the need for therapeutic strategies that extend beyond liver-directed management.

A key challenge in clinical practice is identifying patients with MASLD who are at particularly high cardiovascular risk. Conventional risk factors alone may not fully capture the heterogeneous cardiovascular risk associated with MASLD. Assessment of subclinical vascular and coronary disease may therefore provide additional information for risk stratification. The cardio-ankle vascular index (CAVI), a marker of arterial stiffness, can identify vascular dysfunction, while coronary computed tomography angiography (CCTA) enables direct assessment of coronary atherosclerosis. Our findings suggest that integrating vascular function and coronary imaging with conventional cardiometabolic risk assessment may help identify high-risk individuals with MASLD who could benefit from more intensive preventive strategies.

Effective cardiovascular risk reduction requires comprehensive management of cardiometabolic risk factors. Lifestyle modification and weight reduction remain fundamental, while contemporary therapies, including incretin-based therapies and sodium-glucose cotransporter 2 inhibitors, provide opportunities to address metabolic dysfunction while reducing cardiovascular risk. Optimal lipid-lowering and antihypertensive therapies also remain essential.

This presentation will discuss a cardiovascular-centred approach to MASLD, integrating CAVI and coronary CT for early detection of subclinical cardiovascular disease with comprehensive risk-factor management and evidence-based cardioprotective therapies. Such a strategy may enable more personalised prevention and reduce the substantial residual cardiovascular risk associated with MASLD.

Symposium 18

Intensive LDL-C Lowering in Primary Prevention

Sep 11(Fri) 16:10-17:40 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Eung Gyu Kim (Inje University, Republic of Korea)
Sang-Hyun Lim (The Catholic University of Korea, Republic of Korea)

16:10-16:30	**PCSK9 inhibition in primary prevention: insights from VESALIUS-CV and clinical implications** Doo Sun Sim (Chonnam National University, Republic of Korea)
16:30-16:50	**Low LDL-C and brain safety: risk of hemorrhagic stroke and cognitive dysfunction** Kwang-Yeol Park (Chung-Ang University, Republic of Korea)
16:50-17:10	**Early combination therapy in primary prevention** Hoyoun Won (Chung-Ang University, Republic of Korea)
17:10-17:40	**Panel Discussion** Hyung-Min Kwon (Seoul National University, Republic of Korea) Minjeong Kim (Ewha Womans University, Republic of Korea) Garam Jo (Hannam University, Republic of Korea)

CURRICULUM VITAE

Doo Sun Sim

Department of Cardiology, Chonnam National University Hospital, Republic of Korea

Education

1999	M.D., Chonnam National University Medical School
2011	Ph.D. in Medicine, Graduate School of Chonnam National University

Professional Experience

Mar. 1999-Feb. 2000	Intern, Chonnam National University Hospital
Mar. 2000-Feb. 2004	Resident, Department of Internal Medicine, Chonnam National University Hospital
Mar. 2004	Board Certification in Internal Medicine

Awards and Honors

2009	Medical Research Academic Award, Internal Medicine Category

Publications

1. 2012. Sim DS, Jeong MH, Cha KR, Park SH, Park JO, Shin YM, Shin H, Hong YJ, Ahn Y, Schwartz RS, Kang JC. A reliable porcine coronary model of chronic total occlusion using copper wire stents and a bioabsorbable levo-polylactic acid polymer. J Cardiol. 2012;60:443-447.
2. 2012. Sim DS, Jeong MH, Ahn Y, Kim YJ, Chae SC, Hong TJ, Seong IW, Chae JK, Kim CJ, Cho MC, Rha SW, Bae JH, Seung KB, Park SJ; Korea Acute Myocardial Infarction Registry (KAMIR) Investigators. Benefit of percutaneous coronary intervention in early latecomers with acute ST-segment elevation myocardial infarction. Am J Cardiol. 2012 Jul 21. [Epub ahead of print]

PCSK9 inhibition in primary prevention: insights from VESALIUS-CV and clinical implications

CURRICULUM VITAE

Kwang-Yeol Park

Full Professor, Chung-Ang University, Republic of Korea

Education and Training

1997.02	Seoul National University, Korea, M.D., Medicine
2010.02	SungKyunKwan University, Korea, Ph.D., Neuroscience

Employment and Position

2008-Present	Chung-Ang University, Korea, Professor

Important Publications

1. Hong JH, Park SH, Park KY, Kim EY. Increased Risk of Ischemic Events in Patients With Stroke Treated With Clopidogrel and a P-CAB or PPI. Stroke. 2026 May;57(5):1164-1172.
2. Kim JM, Lee R, Kim Y, Jeong HB, Lee ES, Kim HR, Park KY, Seok JW. Impact of metabolic activity of vertebra and amygdala on stroke recurrence: A Prospective cohort study. Circ Cardiovasc Imaging 2023 Jan 16(1).
3. Kim JM, Lee ES, Park KY, Seok JW, Kwon OH. Comparison of [18F]-FDG and [18F]-NaF Positron Emission Tomography on Culprit Carotid Atherosclerosis. JACC: Cardiovascular imaging. 2019 2:269-370.
4. Park KY, Ay I, Avery R, Caceres JA, Siket MS, Pontes-Neto OM, Zheng H, Rost NS, Furie KL, Sorensen AG, Koroshetz WJ, Ay H. New Biomarker for acute ischemic stroke: plasma glycogen phosphorylase isoenzyme BB. JNNP. 2018 Apr:89(4):404-409.
5. Kim J-M, Park KY, Shin D-W, Park M-S, Kwon O-S. Relation of serum homocysteine levels to cerebral artery calcification and atherosclerosis. Atherosclerosis 2016;254:200-204.

Low LDL-C and brain safety: risk of hemorrhagic stroke and cognitive dysfunction

Observational studies consistently associate low LDL-C with increased hemorrhagic stroke risk, and postmarketing surveillance has raised concern for statin-associated cognitive impairment. Whether these associations reflect causal neurotoxicity of aggressive LDL-C lowering, a statin drug-class effect independent of LDL-C, or confounding bias, remains clinically important.

Observational/Epidemiologic studies (Women's Health Study, a large Chinese prospective cohort, Asia Pacific Cohort Studies Collaboration) show a graded inverse relationship between LDL-C and hemorrhagic stroke, with adjusted hazard ratios up to 2.69 for LDL-C <50 mg/dL. Randomized data are discordant by drug class: statin-based intensive LDL-C lowering was associated with a 46% relative increase in hemorrhagic stroke in a meta-analysis of secondary-prevention trials, whereas PCSK9 inhibitor trials showed no excess risk despite very low achieved LDL-C. Notably, SPARCL found no relationship between achieved LDL-C and hemorrhagic stroke among statin-treated patients, with risk concentrated in those with prior hemorrhagic stroke (HR 1.68, rising to 5.65 for recurrent hemorrhage) — arguing for a statin-specific rather than LDL-level mechanism. A comparative meta-analysis of 36 statin RCTs versus 5 PCSK9 inhibitor RCTs corroborated this: statins showed a dose-dependent hemorrhagic stroke signal (RR 1.15 overall, 1.53 with high-potency statins) while PCSK9 inhibitors showed none. Drug-target Mendelian randomization studies proxying HMGCR inhibition (statin target) found increased intracerebral hemorrhage risk independent of lipid-lowering effect, with tissue-specific analyses suggesting brain (versus blood) HMGCR expression may drive this risk. Findings across MR studies were not fully consistent, and a larger tabular meta-analysis (23 RCTs, 19 observational studies) found no significant statin-ICH association overall, with this signal appearing largely confined to secondary prevention in prior cerebrovascular disease.

Regarding cognition, population cohort data on endogenous LDL-C and dementia/Alzheimer risk remain inconsistent. Large systematic review/meta-analyses [25 RCTs (46,836 patients for statins) and 22 RCTs (117,781 patients across statins, ezetimibe, and PCSK9 inhibitors)] found no association between LDL-lowering therapy and cognitive impairment regardless of LDL-C level achieved. Also, large cohorts found no acceleration of cognitive decline with statin use, and a separate meta-analysis found statins associated with reduced dementia/Alzheimer risk. Mendelian randomization showed dissociation by drug classes: genetically proxied HMGCR inhibition was associated with modest adverse cognitive/cortical effects, while PCSK9 inhibition was neutral.

In conclusion, observational data consistently show an inverse relationship between LDL-C and hemorrhagic stroke risk; however, randomized trial and Mendelian randomization data do not show that pharmacologically lowering LDL-C to very low levels — including with PCSK9 inhibitors — meaningfully increases hemorrhagic stroke or impairs cognition.

However, statins show a dose-dependent hemorrhagic stroke signal — most consistent in secondary prevention with prior cerebrovascular/hemorrhagic disease. Also, HMGCR inhibition (statins) shows a small but inconsistent genetic signal for cognitive effects not corroborated by trial-level testing. Safety data in patients with prior hemorrhagic stroke remain a key evidence gap warranting continued clinical caution.

CURRICULUM VITAE

Hoyoun Won

Associate Professor, Chung-Ang University Hospital, Republic of Korea

Education and Training

2007	Yonsei University, Korea, M.D.
2014	Yonsei University, Korea, Ph.D., Cardiology

Employment and Position

-2026	Chung-Ang University Hospital, Staff

Early combination therapy in primary prevention

Symposium 19

Next-generation Lipid-lowering Therapies: CETP, APOC3, and ANGPTL3 Inhibition (New Drugs 2)

Sep 11(Fri) 16:10-17:40 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Sang Yup Lim (Korea University, Republic of Korea)
Soon Jun Hong (Korea University, Republic of Korea)

16:10-16:30	**CETP inhibition revisited: obicetrapib for LDL-C reduction and ASCVD risk management** Mariko Harada-Shiba (Osaka Medical and Pharmaceutical University, Japan)
16:30-16:50	**APOC3 inhibition: targeting triglyceride-rich lipoproteins and residual risk** Jin Wi (Gachon University, Republic of Korea)
16:50-17:10	**ANGPTL3 inhibition: expanding therapeutic opportunities beyond LDL-C** Hye Moon Chung (Kyung Hee University, Republic of Korea)
17:10-17:40	**Panel Discussion** Byung Sik Kim (Hanyang University, Republic of Korea) Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea) Janghoon Lee (Kyungpook National University, Republic of Korea)

CURRICULUM VITAE

Mariko Harada-Shiba

Professor, Osaka Medical and Pharmaceutical University, Japan

Education and Training

1984.03	Shiga University of Medical Science, M.D., Internal Medicine
1988.03	Graduate School of Medicine, Shiga University of Medical Science, Ph.D., Medicine

Employment and Position

1988-1995	National Cardiovascular Center Research Institute, Postdoctoral Fellow
1996-1998	Case Western Reserve University, Postdoctoral Fellow
1998-2011	National Cerebral and Cardiovascular Center Research Institute, Laboratory Chief
2011-2020	National Cerebral and Cardiovascular Center Research Institute, Director
2020-2021	National Cerebral and Cardiovascular Center Research Institute, Senior Fellow
2022-Present	Osaka Medical and Pharmaceutical University, Professor

Important Publications

1. Nicholls SJ, Nelson AJ, Ditmarsch M, Kastelein JJP, Ballantyne CM, Ray KK, Navar AM, Nissen SE, Harada-Shiba M, Curcio DL, Neild A, Kling D, Hsieh A, Butters J, Ference BA, Laufs U, Banach M, Mehran R, Catapano AL, Huo Y, Szarek M, Balinskaite V, Davidson MH and Investigators B. Safety and Efficacy of Obicetrapib in Patients at High Cardiovascular Risk. N Engl J Med. 2025;393:51-61.
2. Harada-Shiba M, Arai H, Ohmura H, Okazaki H, Sugiyama D, Tada H, Dobashi K, Matsuki K, Minamino T, Yamashita S and Yokote K. Guidelines for the Diagnosis and Treatment of Adult Familial Hypercholesterolemia 2022. J Atheroscler Thromb. 2023;30:558-586.

CETP inhibition revisited: obicetrapib for LDL-C reduction and ASCVD risk management

Cholesteryl ester transfer protein (CETP) plays a central role in lipoprotein metabolism by mediating the exchange of cholesteryl esters from high-density lipoproteins (HDL) to apolipoprotein B-containing lipoproteins in exchange for triglycerides. Because CETP inhibition increases HDL cholesterol (HDL-C) while lowering low-density lipoprotein cholesterol (LDL-C), it has long been regarded as an attractive therapeutic strategy for preventing atherosclerotic cardiovascular disease (ASCVD).

Over the past two decades, four CETP inhibitors —torcetrapib, dalcetrapib, evacetrapib, and anacetrapib—were evaluated in large clinical development programs. However, none achieved widespread clinical use. Torcetrapib increased mortality because of off-target adverse effects, while dalcetrapib and evacetrapib failed to demonstrate meaningful cardiovascular benefit. Although anacetrapib significantly reduced cardiovascular events, concerns regarding its prolonged tissue accumulation and commercial considerations prevented regulatory approval. These experiences led many to question the future of CETP inhibition.

Obicetrapib, a highly selective and potent next-generation CETP inhibitor, has renewed interest in this therapeutic class. Unlike its predecessors, obicetrapib produces substantial LDL-C reductions of approximately 30-35% as monotherapy on top of maximally tolerated lipid-lowering therapy, with even greater reductions when combined with ezetimibe. It also consistently lowers apolipoprotein B, non-HDL cholesterol, and lipoprotein(a) [Lp(a)], an independent causal risk factor for ASCVD, while demonstrating a favorable safety and tolerability profile in Phase 3 clinical trials.

The ongoing global PREVAIL cardiovascular outcomes trial is designed to determine whether these favorable effects on atherogenic lipoproteins translate into reductions in major adverse cardiovascular events. If successful, obicetrapib may represent the long-awaited revival of CETP inhibition and become an important oral adjunctive therapy for patients with ASCVD or familial hypercholesterolemia who remain above LDL-C targets despite contemporary lipid-lowering treatment. This lecture will review the history of CETP inhibition, summarize the latest evidence for obicetrapib, and discuss its potential role in future lipid management strategies.

CURRICULUM VITAE

Jin Wi

Professor, Division of Cardiology, Gachon University Gil Medical Center, Republic of Korea

Education and Training

2005.02	Yonsei University, Korea, M.D., Medicine
2017.08	Yonsei University, Korea, Ph.D., Internal Medicine

Employment and Position

2006-2010	Yonsei University Severance Hospital, Korea, Resident, Internal Medicine
2010-2013	Yonsei University Severance Hospital, Korea, Clinical Fellow, Cardiology
2013-2019	Yonsei University Severance Hospital, Korea, Assistant Professor, Cardiology
2019-	Gachon University Gil Medical Center, Korea, Professor, Cardiology

Important Publications

1. Population Pharmacokinetics of Ticagrelor during Veno-Arterial ECMO in Acute Coronary Syndrome: Model-Informed Dosing Simulations. Clin Pharmacol Ther 2026; In Press (IF 6.300)
2. Effect of Extracorporeal Membrane Oxygenation Flow Rate on Midazolam Clearance: A Population Pharmacokinetic Study. Anesthesiology 2025;144:485-488 (IF 9.400)
3. Population pharmacokinetics and dosing optimization of piperacillin/tazobactam in critically ill patients on extracorporeal membrane oxygenation and the influence of concomitant renal replacement therapy. Microbiology Spectrum 2021;9:e0063321 (IF 9.043)
4. Dose Optimization of Cefpirome Based on Population Pharmacokinetics and Target Attainment during Extracorporeal Membrane Oxygenation. Antimicrob Agents Chemother 2020;64:e00249-20 (IF 5.938)
5. Population pharmacokinetics of intravenous sufentanil in critically ill patients supported with extracorporeal membrane oxygenation therapy. Critical Care 2019;23:248 (IF 19.346)

Awards and Honors

EXCELLENT ABSTRACT AWARD 2024, Japanese Society of Intensive Care Medicine
BEST REVIEWER AWARD 2023, Korean Society of Hypertension
ACADEMIC PRIZE 2022, Korean Society on Thrombosis and Hemostasis

Research Interest

Critical Care Cardiology, Advanced Heart Failure, ECMO, Sudden Cardiac Arrest, RNA Therapeutics

APOC3 inhibition: targeting triglyceride-rich lipoproteins and residual risk

Hypertriglyceridemia is a common clinical problem as it occurs in approximately 10 to 25 % of the population. Elevated triglyceride levels are associated with several pathologic conditions, including insulin resistance, the metabolic syndrome, diabetes, cardiovascular disease, and hereditary disorders, such as the familial chylomicronemia syndrome, familial combined hyperlipidemia, and familial hypertriglyceridemia. Strong evidence links triglycerides to atherosclerotic cardiovascular risk, and when at higher levels, hypertriglyceridemia is associated with acute pancreatitis.

Apolipoprotein C-III (APOC3) is a key regulator of lipoprotein metabolism and plays a pivotal role in regulating plasma triglyceride levels. It is a component of very low density lipoproteins (VLDL) and high density lipoproteins (HDL), which increases plasma TG levels by many mechanisms and raises atherogenic cardiovascular risk. Recently, a new category of drugs was developed targeting the inhibition of APOC3. By blocking or degrading APOC3 mRNA, these drugs reduce APOC3 and TG levels as a result.

In this lecture, I would like to deal with the mechanisms of action of these emerging new molecules and review their future possible clinical applications in detail.

CURRICULUM VITAE

Hye Moon Chung

Division of Cardiology, Department of Internal Medicine,
Kyung Hee University College of Medicine, Republic of Korea

Education

2008	M.D., Yonsei University College of Medicine
2013	M.S. in Internal Medicine, Graduate School, Yonsei University
2018	Ph.D. in Internal Medicine, Graduate School, Yonsei University

Professional Experience

2009-2012	Resident, Department of Internal Medicine, Gangnam Severance Hospital
2013-2014	Clinical Fellow, Division of Cardiology, Yonsei University Health System
2016-2019	Clinical Assistant Professor, Division of Cardiology, Kyung Hee University Medical Center
2020-Present	Assistant Professor, Division of Cardiology, Department of Internal Medicine, Kyung Hee University College of Medicine

Areas of Interest]

Heart Failure, Cardiac Imaging

Publications

1. Age-Dependent Role of Genetics and Renal Function for Atrial Fibrillation Development in Hypertrophic Cardiomyopathy. Korean Circ J. 2025 Nov;55(11):1001-1013.
2. Determinants and effects of microvascular obstruction on serial change in left ventricular diastolic function after reperfused acute myocardial infarction. Front Cardiovasc Med. 2024 Apr 26;11:1338940.
3. Multimodality Imaging in Patients with Hypertrophic Cardiomyopathy and Atrial Fibrillation. Diagnostics (Basel). 2023 Sep 25;13(19):3049.
4. Arterial Stiffness is Associated with False-Positive ST-Segment Depression in Supine Bicycle Exercise Stress Echocardiography. Rev Cardiovasc Med. 2023 Feb 6;24(2):47.
5. Emerging Indicators of Left Atrial Function Evaluation Considering the Unique Characteristics of Hypertrophic Cardiomyopathy. J Cardiovasc Imaging. 2023 Jan;31(1):49-50.

ANGPTL3 inhibition: expanding therapeutic opportunities beyond LDL-C

Symposium 20

Residual Cardiovascular Risk: Beyond LDL to Inflammation and Novel Targets

Sep 11(Fri) 16:10–17:40 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Seung Woo Park (Sungkyunkwan University, Republic of Korea)
Young-Hoon Jeong (Chung-Ang University, Republic of Korea)

16:10–16:30	**Defining residual cardiovascular risk in the post-LDL era: unmet needs and mechanisms** Hyung Joon Joo (Korea University, Republic of Korea)
16:30–16:50	**Targeting residual risk: Lp(a), triglyceride-rich lipoproteins, and inflammation** Sung Gyun Ahn (Yonsei University, Republic of Korea)
16:50–17:10	**Beyond less is more: navigating residual thrombotic and inflammatory risk in AF and stable CAD** Satoshi Yasuda (Tohoku University, Japan)
17:10–17:40	**Panel Discussion** Jaehyun Bae (Hallym University, Republic of Korea) Jung Myung Lee (Sahmyook Medical Center, Republic of Korea) Jong Han Choi (Konkuk University, Republic of Korea)

CURRICULUM VITAE

Hyung Joon Joo

Professor, Division of Cardiology, Department of Internal Medicine,
Korea University College of Medicine, Korea University Anam Hospital, Republic of Korea

Education and Training

1997-2003	Korea University College of Medicine, Korea, M.D., Medicine
2005-2007	Korea University College of Medicine, Korea, M.S., Medicine
2008-2011	KAIST Graduate School of Medical Science and Engineering, Korea, Ph.D., Medical Science
2003-2008	Korea University Anam and Guro Hospital, Korea, Internship & Residency, Internal Medicine

Employment and Position

2011-2013	KAIST Natural Science Research Institute, Korea, Postdoctoral Researcher
2013-2015	Korea University Anam Hospital, Division of Cardiology, Clinical Fellow
2015-2017	Korea University Anam Hospital, Division of Cardiology, Clinical Assistant Professor
2017-2018	Korea University Anam Hospital, Division of Cardiology, Assistant Professor
2018-2023	Korea University Anam Hospital, Division of Cardiology, Associate Professor
2023-Present	Korea University Anam Hospital, Division of Cardiology, Professor
2019-Present	Korea University Anam Hospital, Information Management Office, Director
2021-Present	Korea University College of Medicine, Department of Medical Informatics, Adjunct Professor

Defining residual cardiovascular risk in the post-LDL era: unmet needs and mechanisms

Despite unprecedented success in LDL-C lowering, a substantial burden of incident and recurrent atherosclerotic cardiovascular disease (ASCVD) persists even when guideline-recommended LDL-C goals are achieved. This lecture will reframe "residual risk" as a multidimensional construct rather than a failure of LDL-C lowering alone. Beyond residual apoB-containing particle burden and triglyceride-rich remnants, lipoprotein(a) [Lp(a)] has emerged as a genetically determined, causal risk enhancer whose LDL-like cholesterol cargo, apolipoprotein(a), oxidized phospholipids, inflammatory signaling, prothrombotic potential, and valvular calcification pathways may explain events that occur in the post-LDL era.

The talk will focus on three unmet needs. First, how should Lp(a) be interpreted when fixed thresholds do not uniformly translate into risk? Recent Korean real-world data suggest that elevated Lp(a) is clinically heterogeneous: phenotypic clustering identifies high-Lp(a) patients with markedly different event rates despite similar concentrations, emphasizing the importance of age, sex, cardiometabolic burden, renal function, inflammation, and treatment context. Second, should Lp(a) be regarded as a stable lifetime exposure or as a dynamic marker? Although Lp(a) is largely inherited and generally stable, serial measurements reveal meaningful intra-individual variability in a subset of patients, especially around the intermediate "grey zone." Emerging data from multicenter cohorts suggest that Lp(a) variability may be associated with adverse cardiovascular profiles, risk reclassification, and cardiovascular outcomes, while adding only modest incremental discrimination beyond conventional models. Third, how should future Lp(a)-lowering therapies be integrated? Antisense and siRNA approaches now achieve profound Lp(a) reductions, but outcome evidence, optimal target populations, and monitoring strategies remain unresolved.

By integrating mechanistic biology, trial evidence, and real-world data, this lecture will propose a practical framework for residual cardiovascular risk: measure once, contextualize continuously, and treat residual risk as a phenotype shaped by Lp(a), inflammation, metabolic instability, and patient-specific vulnerability.

Keywords: Lipoprotein(a), Residual cardiovascular risk, LDL-C, Atherosclerosis, Variability, Real-world evidence, Inflammation, Risk stratification

CURRICULUM VITAE

Sung Gyun Ahn

Professor of Internal Medicine, Division of Cardiology, Wonju Severance Christian Hospital, Yonsei University Wonju College of Medicine, Republic of Korea

Education and Training

1996	Yonsei University Wonju College of Medicine, Korea, M.D., Medicine
1996-1997	Ajou University Medical Center, Korea, Internship, Medicine
1997-2001	Ajou University Medical Center, Korea, Residency, Internal Medicine
2004-2006	Ajou University Medical Center, Korea, Fellowship, Cardiology
2007	Ajou University Graduate School, Korea, M.S., Medicine
2014	Ajou University Graduate School, Korea, Ph.D., Medicine
2014-2016	Emory University Hospital, USA, Visiting Scholar, Cardiovascular Imaging and Biomechanics

Employment and Position

2001-2004	The 4th Peru-Corea Hospital, Huánuco, Peru, Medical Doctor (Korea Overseas Volunteer)
2006-2010	Pohang Sunlin Hospital, Korea, Attending Physician; Chief of Cardiac Catheterization Laboratory
2010-2014	Yonsei University Wonju College of Medicine, Korea, Assistant Professor of Internal Medicine
2014-2019	Yonsei University Wonju College of Medicine, Korea, Associate Professor of Internal Medicine
2017-2022	Wonju Severance Christian Hospital, Korea, Chief of Cardiovascular Center
2019-Present	Yonsei University Wonju College of Medicine, Korea, Professor of Internal Medicine
2022-Present	Journal of Cardiovascular Intervention, Associate Editor

Important Publications

1. Jeon HK, Kim JS, Ahn SG, et al. Antithrombotic therapy in atrial fibrillation patients with prior complex percutaneous coronary intervention: a secondary analysis of the randomized ADAPT AF-DES trial. Circ Cardiovasc Interv 2026 (Epub Jul 17).

Targeting residual risk: Lp(a), triglyceride-rich lipoproteins, and inflammation

Statins and non-statin therapy have driven LDL cholesterol to historic lows, yet major adverse cardiovascular events continue to accrue. Three measurable axes account for much of this residual risk: lipoprotein(a), triglyceride-rich lipoprotein (TRL) remnants, and vascular inflammation.

Lp(a) is genetically fixed, established by age five, and causal for atherosclerotic disease and calcific aortic valve stenosis, affecting 20-30% of the global population. Its pathogenicity is threefold: greater atherogenicity per particle, carriage of oxidized phospholipids, and apo(a)-mediated inhibition of fibrinolysis. The 2026 ACC/AHA and 2025 ESC/EAS guidelines endorse universal once-in-a-lifetime measurement using isoform-insensitive, WHO-traceable assays reported in nmol/L; KSoLA sets 30 mg/dL and 50 mg/dL as the borderline and high thresholds. No approved Lp(a)-lowering agent exists, but pelacarsen, olpasiran, zerlasiran, lepodisiran, and oral muvalaplin lower Lp(a) by 80-95%. Lp(a)HORIZON and OCEAN(a)-Outcomes will show whether that reduction translates into fewer events.

TRL remnants enter the intima and are internalized by macrophages without prior oxidation, activating NF-κB signaling. Benefit tracks apoB rather than triglyceride reduction, as REDUCE-IT, STRENGTH, and PROMINENT together demonstrate. APOC3-directed olezarsen and plozasiran cut triglycerides by 49-81% and acute pancreatitis events by roughly 80%.

Inflammation completes the triad. Among 15,494 statin-treated patients undergoing percutaneous coronary intervention, hsCRP ≥2 mg/L with LDL-C <70 mg/dL predicted MACE (5.1% vs 2.6%), whereas isolated high LDL-C did not. KNHANES 2024 data show hsCRP reclassifies 4.4-14.8% of lipid-normal Korean adults as at risk, although East Asian distributions run lower than Western ones, arguing for population-specific cutoffs.

This lecture appraises the evidence for each axis and proposes a practical measurement sequence—apoB, Lp(a) once, remnant cholesterol, hsCRP—for residual-risk stratification in Korean practice.

CURRICULUM VITAE

Satoshi Yasuda

Professor and Chairman, Tohoku University Graduate School of Medicine, Japan

Education and Training

1987	Tohoku University School of Medicine, Japan, M.D., Medicine
2000	Tohoku University School of Medicine, Japan, Ph.D., Cardiovascular Medicine

Employment and Position

2006-2007	Tohoku University Hospital, Japan, Assistant Professor and Chief of Coronary Care Unit
2007-2008	Tohoku University School of Medicine, Japan, Associate Professor in Innovative Cardiovascular Medicine
2008-2011	Tohoku University School of Medicine, Japan, Associate Professor in Cardiovascular Medicine
2011-2016, Jan	National Cerebral and Cardiovascular Center, Japan, Chairman
2016. Feb-2020. Jun	National Cerebral and Cardiovascular Center, Japan, Deputy Director General
2020. Aug-Present	Tohoku University Graduate School of Medicine, Japan, Professor and Chairman

Important Publications

1. Yasuda S, et al. Antithrombotic Therapy for Atrial Fibrillation with Stable Coronary Disease. N Engl J Med. 2019 Sep 19;381(12):1103-1113.
2. Nakashima T, Noguchi T, Tahara Y, Nishimura K, Yasuda S, Onozuka D, Iwami T, Yonemoto N, Nagao K, Nonogi H, Ikeda I, Sato N, Tsutsui H. Public-access defibrillation leads to better neurological outcome even in patients without pre-hospital return of spontaneous circulation: a population-based cohort study. Lancet. 2020 Dec 21;394(10216):2255-2262.
3. Takahashi H, Yang G, Yoneshiro T, Abe Y, Ito R, Yang C, Nakazono J, Okamoto-Katsuyama M, Uchida A, Arai M, Jin H, Choi H, Tumenjargal M, Xie S, Zhang J, Sagae H, Zhao Y, Yamaguchi R, Nomura Y, Shimizu Y, Yamada K, Yasuda S, Kimura H, Tanaka T, Wada Y, Kodama T, Aburatani H, Zhu MS, Inagaki T, Osborne TF, Kawamura T, Ishihama Y, Matsumura Y, Sakai J. MYPT1-PP1β phosphatase negatively regulates both chromatin landscape and co-activator recruitment for beige adipogenesis. Nat Commun. 2022 Sep 29;13(1):5715.

Beyond less is more: navigating residual thrombotic and inflammatory risk in AF and stable CAD

Long-term antithrombotic management in patients with atrial fibrillation (AF) and stable coronary artery disease (CAD) has undergone a paradigm shift from combination therapy toward treatment simplification. The AFIRE trial first demonstrated that rivaroxaban monotherapy improved net clinical outcomes compared with combination therapy, establishing the concept that, in appropriately selected patients, "Less Is More." More recently, EPIC-CAD and ADAPT-AF-DES have extended this paradigm, supporting de-escalation across different direct oral anticoagulants and clinical settings.

Despite these advances, substantial residual cardiovascular risk persists after coronary stabilization. Emerging evidence suggests that residual risk is determined not only by thrombosis but also by chronic low-grade inflammation, which may influence both ischemic and bleeding outcomes. Inflammatory biomarkers, particularly C-reactive protein, may therefore help identify patients who derive the greatest benefit from antithrombotic de-escalation, providing a biological basis for treatment heterogeneity.

The next challenge is to move beyond a uniform "Less Is More" strategy toward a precision approach that integrates both residual thrombotic and inflammatory risk. Such a framework may refine long-term treatment selection and further improve outcomes in patients with AF and stable CAD.

Symposium 21 – KSSO-KSoLA Joint Symposium

Atherogenic Obesity: Beyond BMI to Lipid Dysfunction

Sep 11(Fri) 16:10-17:40 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Min Sun Kim (University of Ulsan, Republic of Korea)
In-Kyung Jeong (Kyung Hee University, Republic of Korea)

16:10-16:30	**Lipid remodeling in adipose tissue and liver: a hidden link between obesity and atherosclerosis** Yun-Hee Lee (Seoul National University, Republic of Korea)
16:30-16:50	**Remnant cholesterol and residual cardiovascular risk in obesity** Young-Sang Lyu (Chosun University, Republic of Korea)
16:50-17:10	**Dual targeting of lipid and weight: emerging therapies and clinical strategies** Chang Hee Jung (University of Ulsan, Republic of Korea)
17:10-17:40	**Panel Discussion** Song-Yi Kim (Jeju National University, Republic of Korea) Jee Hee Yoo (Chung-Ang University, Republic of Korea) Heung Yong Jin (Jeonbuk National University, Republic of Korea)

CURRICULUM VITAE

Yun-Hee Lee

Professor, Seoul National University, Republic of Korea

Education and Training

2001.02	Seoul National University, Korea, B.S., Pharmacy
2003.02	Seoul National University, Korea, M.S., Pharmacy/Toxicology
2012.05	Wayne State University, USA, Ph.D, Pathobiology

Employment and Position

2015-2018	Yonsei University, Korea, Assistant Professor
2018-2026 (Present.)	Seoul National University, Korea, Associate Professor, Professor

Important Publications

1. Cho, Y.K., Lee, J., Jeong, Y.L. et al. TMEM120A maintains adipose tissue lipid homeostasis through ER CoA channeling. Nature Communications 17, 1113 (2026).
2. Kim, S., Choi, C., Son, Y., Lee, J., Joo, S., & Lee, YH*. BNIP3-mediated mitophagy in macrophages regulates obesity-induced adipose tissue metaflammation. Autophagy, 21(9), 2009-2027. (2025).
3. Choi C, Jeong YL, Park KM, Kim M, Kim S, Jo H, Lee S, Kim H, Choi G, Choi YH, Seong JK, Namgoong S, Chung Y, Jung YS, Granneman JG, Hyun YM, Kim JK, Lee YH*. TM4SF19-mediated control of lysosomal activity in macrophages contributes to obesity-induced inflammation and metabolic dysfunction. Nature Communications. 2024 30;15, 2779.
4. Cho YK, Yoon YC, Im H, Son Y, Kim M, Saha A, Choi C, Lee J, Lee S, Kim JH, Kang YP, Jung YS, Ha HK, Seong JK*, Granneman JG*, Kwon SW*, Lee YH*. Adipocyte lysoplasmalogenase TMEM86A regulates plasmalogen homeostasis and protein kinase A-dependent energy metabolism. Nature Communications 13, 4084 (2022).
5. Cho YK, Son Y, Saha A, Kim D, Choi C, Kim M, Park J-H, Im H, Han J, Kim K, Jung Y-S, Yun J, Bae EJ, Seong JK, Lee M-O, Lee S, Granneman JG, Lee Y-H*: STK3/STK4 signalling in adipocytes regulates mitophagy and energy expenditure. Nature Metabolism (2021)

Lipid remodeling in adipose tissue and liver: a hidden link between obesity and atherosclerosis

Obesity-associated atherosclerosis is driven not only by increased lipid burden, but also by maladaptive lipid remodeling across metabolically connected tissues, particularly adipose tissue and liver. Adipose dysfunction alters lipid storage, lipolysis, and inflammatory signaling, thereby increasing the delivery of fatty acids and other lipid intermediates to the liver. In turn, hepatic lipid remodeling influences circulating lipoprotein composition and promotes an atherogenic systemic lipid environment. However, the molecular mechanisms that coordinate intracellular organelle function, adipose-liver communication, and lipoprotein quality remain incompletely understood.

In this presentation, we will discuss emerging roles of transmembrane proteins in the regulation of lipid homeostasis and cardiometabolic disease. Our studies identify TM4SF19 as a regulator of lysosomal lipid handling and cellular lipid homeostasis. Dysregulation of TM4SF19 is associated with impaired lysosomal control, altered lipid accumulation, and exacerbation of atherosclerosis-related phenotypes, suggesting that lysosomal dysfunction may represent an important link between obesity-associated metabolic stress and vascular disease. We will also present evidence that TMEM120A contributes to lipid metabolic adaptation in adipose tissue and liver, potentially influencing lipid storage and tissue-specific remodeling under metabolic stress. Finally, we will highlight TMEM86A as a candidate regulator of hepatic lipid and lipoprotein metabolism.

Together, these findings support a model in which transmembrane protein-mediated lipid remodeling in adipose tissue and liver governs organelle function, inter-organ lipid flux, and lipoprotein quality. Defining these pathways may reveal new therapeutic targets for obesity-associated dyslipidemia and atherosclerotic cardiovascular disease.

CURRICULUM VITAE

Young-Sang Lyu

Associate Professor, Chosun University Hospital, Republic of Korea

Education and Training

2005-2011	Chosun University College of Medicine, M.D.
2017-2019	Chosun University, M.S., Internal Medicine
2019-2021	Chosun University, Ph.D., Internal Medicine

Employment and Position

2021-	Chosun University Hospital, Assistant Professor

Important Publications

1. Lyu et al. Safety and Effectiveness of Naltrexone-Bupropion in Korean Adults with Obesity: Post-Marketing Surveillance Study. Drug Des Devel Ther. 2024 Nov 19;18:5255-5268.
2. Lyu et al. The Effect of Periodontitis on Body Size Phenotypes in Adults without Diagnosed Chronic Diseases: The Korean National Health and Nutrition Examination Survey 2013-2015. Int J Environ Res Public Health. 2024 Sep 4;21(9):1180.
3. Lyu et al. Efficacy and safety of enavogliflozin vs. dapagliflozin as add-on therapy in patients with type 2 diabetes mellitus based on renal function: a pooled analysis of two randomized controlled trials. Cardiovasc Diabetol. 2024 Feb 15;23(1):71.
4. Lyu et al. Comparison of SGLT2 inhibitors with DPP-4 inhibitors combined with metformin in patients with acute myocardial infarction and diabetes mellitus. Cardiovasc Diabetol. 2023 Jul 22;22(1):185.

Research Interest

Complication of diabetes, NAFLD, Obesity treatment

Remnant cholesterol and residual cardiovascular risk in obesity

Obesity is characterized by a distinct atherogenic dyslipidemia pattern—hypertriglyceridemia, low HDL-cholesterol, and a predominance of small, dense LDL particles—that is only partially captured by conventional LDL-cholesterol measurement. A substantial component of this residual risk is now attributed to remnant cholesterol, the cholesterol content carried by triglyceride-rich lipoproteins that accumulates when lipolysis is impaired in insulin-resistant, obese states.

Mendelian randomization studies have established that elevated remnant cholesterol is causally associated with increased risk of ischemic heart disease and all-cause mortality, independent of LDL-cholesterol, positioning it as a modifiable target rather than a mere biomarker. Because remnant particles are smaller and more numerous per unit cholesterol than LDL in hypertriglyceridemic states, standard LDL-C-focused risk assessment may substantially underestimate atherogenic burden in individuals with obesity, even when LDL-C appears well controlled.

Despite this mechanistic and epidemiological rationale, triglyceride-lowering trials have yielded inconsistent clinical outcomes: icosapent ethyl reduced cardiovascular events in REDUCE-IT, whereas the selective PPARα agonist pemafibrate failed to reduce cardiovascular events in PROMINENT despite substantial triglyceride and remnant cholesterol lowering, raising questions about which triglyceride-lowering mechanisms translate into outcome benefit.

This presentation will review the pathophysiology linking obesity, insulin resistance, and remnant lipoprotein accumulation; summarize epidemiological and genetic evidence for remnant cholesterol as a residual cardiovascular risk driver; critically appraise recent randomized trial evidence; and discuss the potential role of weight-loss-centered and incretin-based therapies in addressing remnant cholesterol beyond conventional lipid-lowering strategies. The overarching aim is to highlight remnant cholesterol as an underrecognized, obesity-relevant target for cardiovascular risk reduction that extends the therapeutic conversation beyond BMI and LDL-cholesterol alone.

CURRICULUM VITAE

Chang Hee Jung

Professor, University of Ulsan College of Medicine, Asan Medical Center, Republic of Korea

Education and Training

2002.02	Korea University, M.D., Medicine
2014.02	University of Ulsan, Ph.D., Internal Medicine

Employment and Position

2010-2012	Asan Medical Center, Clinical Fellow
2012-2017	Asan Medical Center, Assistant Professor
2017-2023	University of Ulsan College of Medicine, Asan Medical Center, Associate Professor
2018-2010	University of Virginia, VA, USA, Research Associate
2023-Current	University of Ulsan College of Medicine, Asan Medical Center, Professor

Important Publications

1. Jung HN, Cho YK, Kim MJ, Moon JY, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH. Defining Obesity Under the Lancet Commission Criteria: Metabolic Syndrome-Anchored Body Fat Percentage Thresholds in Korean Adults. Diabetes Obes Metab. 2026;28:5309-5317.
2. Jang MK, Jung CH*, Lee HS, Lee A, Jeoung SJ, Lee YH, Kim HJ, Kim SH, Yu SH, Chon S, Jeong IK*. Enavogliflozin Improves Non-invasive Hepatic Steatosis Indices in Patients With Type 2 Diabetes: A Pooled Analysis of Three Randomised Trials. Diabetes Obes Metab. 2026. Epub ahead of print (*Co-corresponding author)
3. Cho YK, Kim S, Kim MJ, Lee WJ, Kim YJ, Jung CH. New Users of Sodium-Glucose Cotransporter 2. Inhibitors Are at Low Risk of Prostate Cancer: A Nationwide Cohort Study. Diabetes Metab J. 2026;50:90-100.
4. Cho YK, Kim MJ, Kim EH, Lee MJ, Nam HJ, Lee WJ, Kim HK, Jung CH. Comparison of diagnostic criteria for. fatty liver disease in assessing cardiac dysfunction: a cross-sectional study. Hepatol Int. 2026;20:81-90.
5. Cho YK, Jung CH. Engineered nutrient-stimulated hormonal multi-agonists for precision targeting of obesity. and metabolic disorders. Clin Mol Hepatol. 2026;32:464-486.

Dual targeting of lipid and weight: emerging therapies and clinical strategies

Obesity and dyslipidemia frequently coexist and interact synergistically to accelerate atherosclerotic cardiovascular disease, metabolic dysfunction-associated steatotic liver disease, and type 2 diabetes. Although lowering low-density lipoprotein cholesterol remains the cornerstone of cardiovascular prevention, substantial residual risk persists because of excess adiposity, insulin resistance, elevated triglyceride-rich lipoproteins, and chronic inflammation. Therefore, integrated strategies targeting both body weight and atherogenic lipoproteins are increasingly important in contemporary cardiometabolic care.

Glucagon-like peptide-1 (GLP-1)-based therapies have transformed obesity management by producing clinically meaningful and sustained weight loss while improving multiple cardiovascular risk factors. GLP-1 receptor agonists and dual glucose-dependent insulinotropic polypeptide/GLP-1 receptor agonists reduce appetite, energy intake, visceral adiposity, blood pressure, glycemia, and systemic inflammation. They also produce favorable, although generally modest, changes in triglycerides, non-high-density lipoprotein cholesterol, and remnant cholesterol. Cardiovascular outcome trials further demonstrate that the benefits of GLP-1-based therapies extend beyond glycemic control and may complement the established benefits of lipid-lowering treatment.

This lecture will examine the mechanistic and clinical rationale for dual targeting of weight and lipids, with a particular focus on GLP-1-based pharmacotherapy. Emerging agents, including oral GLP-1 receptor agonists and GLP-1-based dual or triple agonists, will also be reviewed. Practical strategies for combining anti-obesity medications with statins, ezetimibe, proprotein convertase subtilisin/kexin type 9-targeted therapies, and triglyceride-lowering agents will be discussed. A phenotype-based approach integrating baseline cardiovascular risk, adiposity, lipid abnormalities, diabetes, fatty liver disease, and treatment tolerability may enable more effective and individualized reduction of cardiometabolic risk.

Symposium 22

Lp(a) beyond the Known: From Metabolic Pathways to Clinical Translation

Sep 12(Sat) 08:50–10:20 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Donghoon Choi (Yonsei University, Republic of Korea)
Keeho Song (Konkuk University, Republic of Korea)

08:50–09:10	**Oxidized phospholipids on lipoprotein(a): a key driver of atherothrombosis?** Sotirios Tsimikas (University of California, San Diego, USA)
09:10–09:30	**Does lipoprotein(a) promote thrombosis? evidence from genetics and population studies** Borge G. Nordestgaard (University of Copenhagen, Denmark)
09:30–09:50	**Epidemiology and cardiovascular events of Lp(a): what have we learned?** Sang-Rok Lee (Jeonbuk National University, Republic of Korea)
09:50–10:20	**Panel Discussion** Eu Jeong Ku (Seoul National University, Republic of Korea) Young Sup Byun (Inje University, Republic of Korea) Janghoon Lee (Kyungpook National University, Republic of Korea)

CURRICULUM VITAE

Sotirios Tsimikas

Professor of Medicine and Director of Vascular Medicine,
University of California San Diego School of Medicine, San Diego, USA

Education and Training

1988	University of Massachusetts School of Medicine, Worcester, MA, M.D., Medicine
1988-1991	University of Massachusetts Medical Center, Worcester, MA, Internship & Residency, Internal Medicine
1992-1995	University of California San Diego Medical Center, Fellowship, Cardiovascular Diseases
1994-1996	University of California San Diego, Specialized Center for Research on Arteriosclerosis and Molecular Medicine, Research Fellowship, Atherosclerosis & Molecular Medicine
1996-1997	University of California San Diego Medical Center, Fellowship, Interventional Cardiology
1980-1984	Boston College, Chestnut Hill, MA, B.S., Magna cum Laude, Chemistry

Employment and Position

2007-Present	University of California San Diego School of Medicine, Professor of Clinical Medicine
2002-Present	University of California San Diego, Division of Cardiovascular Medicine, Founding Director of Vascular Medicine
2003-2007	University of California San Diego School of Medicine, Associate Professor of Clinical Medicine
1997-2003	University of California San Diego Medical Center, Assistant Clinical Professor of Medicine

Oxidized phospholipids on lipoprotein(a): a key driver of atherothrombosis?

Lipoprotein(a) [Lp(a)] is a genetically determined and causal risk factor for atherosclerotic cardiovascular disease and calcific aortic valve stenosis (CAVS), but the mechanisms underlying its pathogenicity remain incompletely understood. A central hypothesis is that much of the cardiovascular risk attributed to Lp(a) reflects its role as the preferential carrier of pro-inflammatory oxidized phospholipids (OxPL).

OxPL are oxidation-specific epitopes generated during oxidative stress and are abundant in inflammatory tissues but largely absent from healthy tissues. Studies using the natural antibody E06, which recognizes the phosphocholine headgroup of OxPL, helped establish their presence in atherosclerotic lesions and subsequently demonstrated a strong relationship between circulating OxPL and Lp(a). Biochemical studies showed that Lp(a) preferentially binds OxPL, which are present both covalently on apolipoprotein(a) and within the lipid phase of the particle. Functionally, Lp(a)-associated OxPL promote arterial-wall inflammation and pro-inflammatory monocyte responses, while inhibition of OxPL attenuates these effects.

Clinical studies have consistently associated elevated OxPL-apoB with cardiovascular events and suggest that OxPL may account for an important component of Lp(a)-mediated cardiovascular risk. Lp(a) and OxPL are also associated with the prevalence, incidence, and progression of CAVS, providing a mechanistic rationale for ongoing trials of Lp(a)-lowering therapy in aortic stenosis.

These observations support a model in which Lp(a) combines three pathogenic components: an apoB-containing, pro-atherogenic particle; apo(a), with potential prothrombotic properties; and OxPL, which confer potent pro-inflammatory activity. Emerging Lp(a)-lowering therapies markedly reduce both Lp(a) and associated OxPL. Ongoing cardiovascular outcome trials will therefore test not only the Lp(a) hypothesis, but indirectly whether reducing its OxPL cargo translates into meaningful clinical benefit.

CURRICULUM VITAE

Borge G. Nordestgaard

Professor, Chief Physician,
Copenhagen University Hospital, University of Copenhagen, Denmark

Education and Training

1985	University of Copenhagen, Denmark, M.D., Clinical Chemistry and Cardiology
1990	University of Copenhagen, Denmark, DMSc, Medicine

Employment and Position

1999-2026	Copenhagen University Hospital, Denmark, Chief Physician
2005-2026	University of Copenhagen, Denmark, Professor

President European Atherosclerosis Society

Prof Nordestgaard studied and worked in Copenhagen, New York, and London.

He has for more than 40 years continued his interest in the pathogenesis, diagnosis and treatment of severe hypertriglyceridemia, familial hypercholesterolemia, hyperlipidemia, lipoprotein(a), remnant cholesterol, triglycerides, atherosclerosis, diabetes, and liver, renal, and cardiovascular disease, and has written extensively on these conditions.

He is chairing the Copenhagen General Population Study and is a steering committee member of the Copenhagen City Heart Study and five phase 3 cardiovascular intervention trials.

Prof. Børge G. Nordestgaard has supervised 75 Ph.D. students and 36 postdoctoral fellows, and has published 1050 original articles and 175 reviews, book chapters, consensus statements, & editorials. His H-index is 188 in Web of Science and he is listed among the top 0.1% researchers worldwide with the most highly cited papers.

Original articles include publications in NEJM, Lancet, JAMA, Nature, Science, baby Nature journals, BMJ, baby Lancet journals, Circulation, European Heart Journal, JACC and baby JAMA journals.

Does lipoprotein(a) promote thrombosis? evidence from genetics and population studies

Lipoprotein(a) is a causal risk factor for atherosclerotic cardiovascular disease (ASCVD=myocardial infarction, ischemic stroke, peripheral artery disease) and aortic valve stenosis. Plasma concentrations of lipoprotein(a) are >90% genetically determined. Multiple drugs in development reduce lipoprotein(a) by 80-98%; of these, four are currently in phase 3 trials aiming at reducing ASCVD, with results expected in 2026-2031. Lipoprotein(a) testing is currently increasing rapidly in many countries.

Most guidelines on dyslipidemia and prevention of cardiovascular disease world-wide now advise that lipoprotein(a) should be measured once in all adults, ideally jointly with the first lipid profile ordered. If previous lipid profiles were taken, then lipoprotein(a) should be included in the next lipid profile. Lipoprotein(a) should ideally be measured in nmol/L, and repeat measurements are relevant if drugs are given to lower lipoprotein(a) concentrations, initially at suspected acute coronary syndrome or stroke and again after one month, and some-times in women after menopause. For optimal clinical care to secure understanding of the patient's total cardiovascular risk, lipoprotein(a) concentration should be reported simultaneously with the standard lipid profile.

CURRICULUM VITAE

Sang-Rok Lee

Professor, Division of Cardiology, Jeonbuk National University Hospital, Republic of Korea

Education and Training

Feb. 1997	Chonnam National University Medical School, Korea, MD, Medicine
Feb. 2002	Chonnam National University Hospital, Korea, Internal Medicine
Feb. 2007	Chonnam National University Medical School, Korea, PhD, Medicine
Jun. 2007	Chonnam National University Hospital, Korea, Cardiology

Employment and Position

Dec. 2006-Mar. 2007	Jeonbuk National University Hospital, Korea, Clinical Professor
Mar. 2009-Apr. 2007	Jeonbuk National University Hospital, Korea, Full-time Lecturer
Feb. 2014-Apr. 2009	Jeonbuk National University Hospital, Korea, Assistant Professor
Sep. 2015-Aug. 2014	UC San Diego, USA, Visiting Scholor
Mar. 2019-Mar. 2014	Jeonbuk National University Hospital, Korea, Associate Professor
Present-Apr. 2019	Jeonbuk National University Hospital, Korea, Professor

Important Publications

1. Kim YS, Lee SR (corresponding). Successful Prasugrel Therapy for Recurrent Left Main Stent Thrombosis in a Clopidogrel Hyporesponder. Tex Heart Inst J 2015;42:483-6.
2. Jung LY, Lee SR (corresponding), Jung JM, Kim YS, Lee SH, Rhee KS, Chae JK, Lee DH, Kim DS, Kim WH, Ko JK. Rosuvastatin Reduces Blood Viscosity in Patients with Acute Coronary Syndrome. Korean Circ J 2016;46:147-53.
3. Lee SR (first author), Prasad A, Choi YS, Xing C, Clopton P, Witztum JL, Tsimikas S. The LPA Gene, Ethnicity, and Cardiovascular Events. Circulation 2017;135:251-63.
4. van Capelleveen JC, Lee SR (first author), Verbeek R, Kastelein JJP, Wareham NJ, Stroes ESG, Hovingh GK, Khaw KT, Boekholdt SM, Witztum JL, Tsimikas S. Relationship of lipoprotein-associated apolipoprotein C-III with lipid variables and coronary artery disease risk: The EPIC-Norfolk prospective population study. J Clin Lipidol 2018;12:1493-1501.
5. Liu T, Yoon WS, Lee SR (corresponding author). Recent Updates of Lipoprotein(a) and Cardiovascular Disease. Chonnam Med J 2021;57:36-43.

Epidemiology and cardiovascular events of Lp(a): what have we learned?

In recent years, epidemiological studies, genome-wide association studies, and Mendelian randomization studies have shown a strong association between increased levels of lipoproteins and both atherosclerotic cardiovascular disease (ASCVD) and calcific aortic valve stenosis (CAVS). Although international guidelines increasingly recognize Lp(a) as a risk-enhancing factor, thresholds for high Lp(a) in many Asian populations and treatment strategies remain undefined. Moreover, awareness, standardized isoform-insensitive measurement, and treatment strategies remain limited in Korea.

Based on large Korean cohorts, a 3-tiered classification is proposed from the Lp(a) Task Force of the Korean Society of Lipid and Atherosclerosis; normal (<30 mg/dL), borderline high (30-49 mg/dL), and high (≥50 mg/dL).

Novel therapeutics including antisense oligonucleotides, small interfering RNAs, and small molecular inhibitors have shown promising Lp(a)-lowering effects. In near future ongoing phase 3 trials will be reported. So, this presentation will provide structure, pathogenicity, prognostic evidences, and Korean epidemiologic data with summarizing global evidences. It will provide guidance in Korean healthcare system.

Symposium 23 – KSH–KSoLA Joint Symposium

Hypertension, Lipids, and Vascular Aging: Integrated Cardiovascular Risk Management

Sep 12(Sat) 13:00–14:00 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Kwang-il Kim (Seoul National University, Republic of Korea)
Jun Hwa Hong (Eulji University, Republic of Korea)

13:00–13:15	**Residual cardiovascular risk in hypertension despite optimal blood pressure control** Hyue Mee Kim (Chung-Ang University, Republic of Korea)
13:15–13:30	**Lipid management in hypertensive patients** Jaehoon Chung (Gachon University, Republic of Korea)
13:30–13:45	**Obesity, insulin resistance, and hypertension-related vascular injury** Mihye Seo (Soonchunhyang University, Republic of Korea)
13:45–14:00	**Panel Discussion** Eu Jeong Ku (Seoul National University, Republic of Korea) Hyo-Suk Ahn (The Catholic University of Korea, Republic of Korea) Han-Mo Yang (Seoul National University, Republic of Korea)

CURRICULUM VITAE

Hyue Mee Kim

Associate Professor, Chung-Ang University Hospital, Republic of Korea

Education and Training

2010.02	Chung-Ang University, Korea, M.D., Medicine
2025.02	Seoul National University, Korea, Ph.D., Internal Medicine

Employment and Position

2020-2024	Chung-Ang University, Korea, Assistant Professor
2025-Present	Chung-Ang University, Korea, Associate Professor

Important Publications

1. Kim HM, Lee H, Lim Y, Cho GY. Strain-Volume Loop Dynamics: A Novel Perspective on Left Ventricular Dysfunction. Korean Circ J. 2026 Jan 27. doi: 10.4070/kcj.2025.0331.
2. Hwang IC, Kim HM, Park J, Choi HM, Yoon YE, Cho GY. Machine-learning-derived phenotypes of hypertensive patients using multidimensional clinical and echocardiographic data including strain imaging. Eur Heart J Digit Health. 2026 Feb 9;7(2):ztag027.
3. Kim HM, Hwang IC, Park J, Choi HJ, Choi HM, Yoon YE, Cho GY. Impact of changes in left heart geometry on predicting new-onset atrial fibrillation in patients with hypertension. J Hypertens. 2025 Jan 1;43(1):120-127.
4. Kim HM, Kim HL, Kim MA, Lee HY, Park JJ, Choi DJ; on the behalf of the KorAHF Investigators. Sex differences in clinical characteristics and long-term outcome in patients with heart failure: data from the KorAHF registry. Korean J Intern Med. 2024 Jan;39(1):95-109.

Residual cardiovascular risk in hypertension despite optimal blood pressure control

Contemporary hypertension guidelines have progressively emphasized more intensive blood pressure (BP) control, and landmark trials such as SPRINT have demonstrated that intensive BP lowering reduces cardiovascular events. Nevertheless, substantial cardiovascular risk persists even among patients who achieve guideline-recommended BP targets, including the risk of myocardial infarction, stroke, heart failure, and atrial fibrillation. This residual risk highlights an important concept: achieving optimal BP control does not necessarily reverse the cumulative cardiovascular damage associated with longstanding hypertension.

Chronic exposure to elevated BP induces structural and functional changes throughout the cardiovascular system, including vascular remodeling and arterial stiffness, left ventricular hypertrophy and myocardial remodeling, and left atrial remodeling. These alterations may persist despite subsequent BP normalization and contribute to ongoing cardiovascular risk. Moreover, hypertension rarely exists in isolation but commonly clusters with dyslipidemia, insulin resistance, obesity, and chronic kidney disease. Atherogenic lipid burden and other metabolic risk factors may therefore further amplify residual risk, even when BP is adequately controlled.

This talk will review the epidemiology and mechanisms of residual cardiovascular risk in treated hypertension, with particular emphasis on the cumulative burden of BP exposure, cardiovascular target-organ damage, lipid abnormalities, and vascular aging. Emerging approaches to risk stratification, including global cardiovascular risk assessment and evaluation of subclinical vascular and cardiac damage, will also be discussed. An integrated approach that considers BP control together with atherogenic risk and established cardiovascular remodeling may allow more accurate identification of high-risk patients and ultimately help reduce cardiovascular events beyond achieving a single BP or LDL cholesterol target.

CURRICULUM VITAE

Jaehoon Chung

Professor, Gachon University, Republic of Korea

Education and Training

2010.02 Yeung-nam University, Korea, M.D, Medicine
2026.02 Dongguk University, Korea, Master of Internal Medicine

Employment and Position

2017-2024 National Medical Center, Clinical Doctor
2024-2026.06 Dongguk University Hospital, Assistant Professor
2026.07-Present Gil Medical Center, Associate Professor

Important Publications

1. Chung, J., Kim, H. L., Lim, W. H., Seo, J. B., Kim, S. H., Zo, J. H., & Kim, M. A. (2019). Association between invasively measured aortic pulse pressure and orthostatic hypotension in patients undergoing invasive coronary angiography. Journal of hypertension, 37(10), 1966-1973.
2. Chung, J., Kim, H. L., Lee, J. P., Lim, W. H., Seo, J. B., Kim, S. H., Zo, J. H., & Kim, M. A. (2020). Association of the Serum Osteoprotegerin Level With Target Organ Damage in Patients at High Risk of Coronary Artery Disease. Circulation journal : official journal of the Japanese Circulation Society, 85(1), 69-76.
3. Chung, J., Han, J. K., Yang, H. M., Park, K. W., Kang, H. J., Koo, B. K., Jeong, M. H., Kim, H. S., & investigators for Korea Acute Myocardial Infarction Registry (KAMIR) (2021). Long-term efficacy of vasodilating β-blocker in patients with acute myocardial infarction: nationwide multicenter prospective registry. The Korean journal of internal medicine, 36(Suppl 1), S62-S71.
4. Chung, J., Min, K. W., Son, B. K., Kim, D. H., & Kim, H. L. (2021). Association between histological severity of Helicobacter pylori infection and cardiovascular risk scores in the Korean population. Atherosclerosis, 333, 124-130.
5. Chung, J., Kim, H. L., Lim, W. H., Seo, J. B., Zo, J. H., Kim, M. A., & Kim, S. H. (2023). New onset diabetes mellitus and cardiovascular outcomes according to statin intensity in patients after drug-eluting stent implantation in Asian patients. Scientific reports, 13(1), 16061. https://doi.org/10.1038/s41598-023-42277-w

Awards and Honors

Research Interest
Atherosclerosis
Arterial stiffness
Lipid
Hypertension
Metabolic syndrome
Coronary artery disease

Lipid management in hypertensive patients

Hypertension and dyslipidemia commonly coexist and jointly amplify cardiovascular risk through shared vascular mechanisms. Evidence from major clinical trials supports statin therapy for cardiovascular risk reduction in hypertensive patients, including those without marked hypercholesterolemia. Current management should therefore integrate blood pressure and lipid control according to overall cardiovascular risk, with both targets reassessed in parallel.

CURRICULUM VITAE

Mihye Seo

Associate Professor, Division of Endocrinology, Department of Internal Medicine, Soonchunhyang University Hospital Seoul, Soonchunhyang University College of Medicine, Republic of Korea

Education & Training

Jeju National University School of Medicine, Korea, M.D., Medicine
Sungkyunkwan University College of Medicine, Korea, M.S., Internal Medicine
Sungkyunkwan University College of Medicine, Korea, Ph.D., Internal Medicine

Employment and Position

- Clinical Fellow-Division of Endocrinology and Metabolism, Department of Internal Medicine, Kangbuk Samsung Hospital, Sungkyunkwan University College of Medicine
- Assistant and Associate Professor-Division of Endocrinology and Metabolism, Department of Internal Medicine, Soonchunhyang University Hospital, Gumi, Soonchunhyang University College of Medicine
- Associate Professor

Research Interests

Diabetes, obesity, Cardiovascular disease, Dyslipidemia, Aging

Publication List

1. Seo M, Suh K, Park HK, Cho KW. From bench to bedside: adipose tissue fibrosis in obesity, anti-diabetic therapies, and bariatric surgery. Korean J Intern Med. 2026 Mar;41(2):210-229.
2. Seo M, Averia M, Julietta V, Soedono S, Joo EJ, Buedono E, Lee M, Kim MK, Hwang Y, Park HK, Suh K, Cho KW. Persistent adipose inflammation despite metabolic recovery reveals tissue-specific immunomodulation by tirzepatide. Endocrinol Metab. 2026 May 15 Epub ahead of print. (co-First author, co-Corresponding author)
3. Kim NH, Seo MH, Jung JH, Han KD, Kim MK, Kim NH. 2023 Diabetic Kidney Disease Fact Sheet in Korea. Diabetes Metab J. 2024 May;48(3):463-472. (co-First author)
4. Park SE, Seo MH, Cho JH, Kwon H, Kim YH, Han KD, Jung JH, Park YG, Rhee EJ, Lee WY. Dose-Dependent Effect of Smoking on Risk of Diabetes Remains after Smoking Cessation: A Nationwide Population-Based Cohort Study in Korea. Diabetes Metab J. 2021 Jul;45(4):539-546. (co-First author)
5. Seo M, Kim H, Noh H, Jeon JS, Byun DW, Kim SH, Kim HJ, Suh K, Park HK, Kwon SH. Effect of bariatric surgery on circulating and urinary mitochondrial DNA copy numbers in obesity with or without diabetes. BMJ Open Diabetes Res Care. 2020 Oct;8(1):e001372. (First author)

Obesity, insulin resistance, and hypertension-related vascular injury

Obesity has become the dominant modifiable driver of primary hypertension, and the vascular injury that follows is increasingly understood as a continuum rather than a set of separate complications. Dysfunctional, hypoxic, macrophage-infiltrated adipose tissue exports free fatty acids, leptin and inflammatory cytokines while withdrawing adiponectin. The resulting insulin resistance is not only metabolic but vascular: insulin loses its vasodilator action, and the endothelium shifts toward vasoconstriction, oxidative stress and impaired microvascular perfusion. Together with sympathetic and mineralocorticoid activation and renal sodium retention, these changes raise blood pressure. Sustained pressure then remodels the media through smooth muscle hypertrophy, elastin fragmentation and collagen crosslinking, producing aortic stiffening and early vascular aging. Finally, the adventitia and perivascular adipose tissue act as an outside-in effector of inflammation - a phenotype that is now measurable, and potentially modifiable, in patients.

This lecture examines obesity, insulin resistance, and hypertension-related vascular injury.

Symposium 24 – KSCVI-KSoLA Joint Symposium

Beyond Stenting: Contemporary Management of Residual Coronary Risk

Sep 12(Sat) 13:00-14:00 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Joon Hyung Doh (Inje University, Republic of Korea)
Sang Yeub Lee (Chung-Ang University, Republic of Korea)

13:00-13:15	**Impact of lipid-lowering therapy on plaque stabilization and regression** Jong Young Lee (Hallym University, Republic of Korea)
13:15-13:30	**Coronary microvascular dysfunction after PCI** Hyun Sung Joh (Seoul National University, Republic of Korea)
13:30-13:45	**Discordance between coronary artery calcium and coronary stenosis** Sanghoon Shin (Ewha Womans University, Republic of Korea)
13:45-14:00	**Panel Discussion** Yong Hyun Park (Pusan National University, Republic of Korea) Hyun-Woong Park (Chungnam National University, Republic of Korea) Jong Shin Woo (Kyung Hee University, Republic of Korea) Soo Young Lee (Chung-Ang University, Republic of Korea)

CURRICULUM VITAE

Jong Young Lee

Division of Cardiology, Hallym University Sacred Heart Hospital, Republic of Korea

Education

Feb. 1999	M.D., Yeungnam University College of Medicine
Feb. 2011	M.S., University of Ulsan College of Medicine
Feb. 2013	Ph.D., University of Ulsan College of Medicine

Impact of lipid-lowering therapy on plaque stabilization and regression

CURRICULUM VITAE

Hyun Sung Joh

Clinical Assistant Professor, Division of Cardiology, Department of Internal Medicine,
Seoul National University College of Medicine, Boramae Medical Center, Seoul, Republic of Korea

Education and Training

2012.03	Sungkyunkwan University, Korea, M.D., Internal Medicine

Employment and Position

2013-2017	Samsung Medical Center, Residency in Internal Medicine
2020-2022	Samsung Medical Center, Clinical Fellowship
2022-Now	Boramae Medical Center, Clinical Assistant Professor

Coronary microvascular dysfunction after PCI

CURRICULUM VITAE

Sanghoon Shin

Clinical Associate Professor, Division of Cardiology, Department of Internal Medicine, Ewha Womans University Seoul Hospital, Ewha Womans University College of Medicine, Seoul, Republic of Korea

Education and Training

1999.03-2005.02	Yonsei University College of Medicine, Seoul, Korea, M.D., Medicine
2006.03-2010.02	Severance Hospital, Yonsei University College of Medicine, Seoul, Korea, Residency, Internal Medicine
2007.09-2011.02	Yonsei University Graduate School, Seoul, Korea, M.S., Medicine
2010.03-2011.02	Severance Hospital, Yonsei University College of Medicine, Seoul, Korea, Clinical Fellowship, Cardiology
2011.03-2013.02	Severance Hospital, Yonsei University College of Medicine, Seoul, Korea, Subspecialty Fellowship, Interventional Cardiology
2012.09-2024.09	Yonsei University Graduate School, Seoul, Korea, Ph.D., Medicine

Employment and Position

2013.03-2014.04	Severance Hospital, Yonsei University College of Medicine, Seoul, Korea, Assistant Clinical Professor
2014.05-2019.01	National Health Insurance Service Ilsan Hospital, Goyang, Korea, Clinical Professor
2019.02-Present	Ewha Womans University Seoul Hospital, Seoul, Korea, Associate Professor

Important Publications

1. Han JK, ..., Shin S. Short-Term Dual Antiplatelet Therapy After Complex Percutaneous Coronary Intervention With Third-Generation Drug-Eluting Stents: A Post Hoc Analysis of the HOST-IDEA Trial. Circ Cardiovasc Interv. 2025. (Corresponding author)
2. Lee YJ, Shin S, et al. Ticagrelor monotherapy for acute coronary syndrome: an individual patient data meta-analysis of the TICO and T-PASS trials. Eur Heart J. 2024;45(30). (Co-first author)

Discordance between coronary artery calcium and coronary stenosis

Coronary artery calcium (CAC) scoring is the most widely used non-invasive marker of subclinical atherosclerosis, yet it is often read as a surrogate for luminal stenosis. CAC quantifies only the calcified component of total plaque burden; stenosis reflects the failure of compensatory remodeling, and ischemia is a further step removed. Discordance between calcium and stenosis is therefore biology, not measurement error.

Discordance runs in both directions. Among symptomatic patients, obstructive disease despite a zero score is uncommon - about 3.5% with 50% or greater stenosis in CONFIRM - but far from negligible in higher-prevalence settings, reaching 19% among patients referred for invasive angiography in CORE64. Its determinants are predictable: young age (a zero score in roughly half of patients under 40 with obstructive disease, versus a small minority over 70), male sex, smoking, and diabetes. Calcification is a late, stabilizing event, so the plaques that cause events - lipid-rich, positively remodeled, low-attenuation - may precede any detectable calcium. Conversely, in population cohorts such as SCAPIS, most individuals with a score of 400 or higher have no obstructive lesion yet carry substantial event risk; and heavy calcification degrades CT stenosis assessment through blooming artifact, generating reader-made discordance that physiologic or intracoronary assessment is needed to resolve.

The clinical message aligns with the theme of this session: residual coronary risk resides in plaque burden rather than in the lumen. Non-obstructive but calcium-laden disease is a treatment target, not a reassuring result. CAC should be used neither as a stand-alone gatekeeper nor discarded, but folded into clinical likelihood estimation, as in the CAC-weighted clinical likelihood model endorsed by the 2024 ESC chronic coronary syndrome guideline.

This lecture reviews the mechanisms and determinants of CAC-stenosis discordance in both directions and proposes a practical, age- and risk-factor-stratified framework for interpreting the calcium score in contemporary practice.

Symposium 25 – KDA-KSoLA Joint Symposium

Integrated Cardiometabolic Risk Management in Diabetes

Sep 12(Sat) 13:00-14:00 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Sung Rae Kim (The Catholic University of Korea, Republic of Korea)
Hyuk-Sang Kwon (The Catholic University of Korea, Republic of Korea)

13:00-13:15 **Hypertriglyceridemia and remnant cholesterol in diabetes**
Sung Hee Choi (Seoul National University, Republic of Korea)

13:15-13:30 **Arterial stiffness and vascular aging in diabetes**
Hack-Lyoung Kim (Seoul National University, Republic of Korea)

13:30-13:45 **Coronary artery calcium and subclinical atherosclerosis in diabetes**
Janghoon Lee (Kyungpook National University, Republic of Korea)

13:45-14:00 **Panel Discussion**
Joon Ho Moon (Seoul National University, Republic of Korea)
Ki-Hyun Jeon (Seoul National University, Republic of Korea)
A Ram Hong (Chonnam National University, Republic of Korea)

CURRICULUM VITAE

Sung Hee Choi

Professor, Internal Medicine, Endocrinology & Metabolism,
Seoul National University Bundang Hospital, Seoul National University College of Medicine,
Republic of Korea

Education

1991-1997	Yonsei University College of Medicine, M.D
1998-2001	Yonsei University College of Medicine, Internal Medicine, MSc
2002-2006	Yonsei University College of Medicine, Medical Science, Ph.D

Professional (Employment / Work) Experiences

2017-Current	Seoul National University College of Medicine & SNU Bundang Hospital, Professor
2004-2017	Seoul National University College of Medicine & SNU Bundang Hospital, Assistant, Associate Professor
2009-2011	Columbia University, NYC, NY, USA, Research Scholar
1997-2002	Yonsei University College of Medicine, Severance Hospital, Intern, Residency, Clinical Fellow
2002	Schwabinger Krankenhaus, Munich, Germany, Clinical Fellow

Publications

1. Single-cell landscape of human adipose tissue reveals THBS1 as a central driver of fibro-inflammation in metabolic disease. 2026 Aug, accepted. Nat. Commun.
2. Long-term risk of offspring type 1 and type 2 diabetes following maternal gestational diabetes mellitus: a nationwide birth cohort study with 10-year follow-up, BMC med, 2026 Mar 6;24(1):237.
3. Diacerein improves liver fibrosis, steatosis, and atherosclerosis in ApoE knockout mice, J Mol Med 2026 Mar 11;104(1):50.
4. Diabetes Fact Sheet 2025: Special Edition on Diabetes with Obesity and in Pregnancy, Diab Metab Journal 2026;50(2):255-266.
5. Diabetes in Pregnancy in Korea: Prevalence, Clinical Characteristics, and Postpartum Comorbidities, Diab Metab Journal 2026;50(2):280-290.

Awards & Honors

2009, 2022	Excellent Research Award, Seoul National University & SNU College of Medicine
2015	Korean Endocrine Society, The Best Research Award (연구본상)
2022	Seoul National University Bundang Hospital, The best academic awards (불곡의학상 최우수상)

Hypertriglyceridemia and remnant cholesterol in diabetes

Hypertriglyceridemia is a common feature of diabetic dyslipidemia and contributes to residual cardiovascular risk even after optimal LDL cholesterol lowering. Increasing evidence suggests that this risk is largely mediated by triglyceride-rich lipoproteins and their cholesterol content, referred to as remnant cholesterol. Remnant particles can penetrate the arterial wall and promote atherosclerosis through cholesterol accumulation and inflammatory pathways. In people with type 2 diabetes, insulin resistance, increased hepatic VLDL production, and impaired clearance of triglyceride-rich lipoproteins further increase remnant cholesterol levels. Recent epidemiological studies, including large nationwide Korean cohorts, have demonstrated that elevated remnant cholesterol is associated with cardiovascular events independently of LDL cholesterol and conventional risk factors. This presentation will review the pathophysiology and clinical significance of hypertriglyceridemia and remnant cholesterol in diabetes, highlight recent evidence from Korean populations, and discuss practical approaches and emerging therapeutic strategies for reducing triglyceride-rich lipoprotein-related residual cardiovascular risk.

CURRICULUM VITAE

Hack-Lyoung Kim

Associate Professor, Division of Cardiology, Boramae Medical Center, Seoul, Republic of Korea

Education and Training

2003.02	Chonnam National University College of Medicine, Korea, M.D., Medicine
2011.08	Seoul National University College of Medicine, Korea, Ph.D., Internal Medicine

Employment and Position

2004-2008	Seoul National University Hospital, Seoul, Korea, Residency in Internal Medicine
2008-2011	JSA and Armed Forces Seoul Hospital, Korea, Military Medical Officer, Captain
2011-2012	Seoul National University Hospital, Seoul, Korea, Clinical Fellow in Cardiology
2012-Present	Boramae Medical Center, Seoul, Korea, Associate professor in Cardiology

Important Publications

1. Kim HL, et al. Comparisons of prognostic value between brachial-ankle pulse wave velocity and estimated pulse wave velocity. Hypertens Res. 2026 Jul;49(7):2038-2046.
2. Kim HL, et al. The variability of pulse wave velocity and long-term cardiovascular outcomes. Am J Hypertens. 2026 Aug 1;39(8):970-978.
3. Jeon BK, Kim HL, et al. Prognostic Value of Brachial-Ankle Pulse Wave Velocity in the Prediction of Cardiovascular Events: Comparison with Brachial Pulse Pressure. J Clin Med. 2025 Dec 9;14(24):8724.
4. Kim HL, et al. Invasively measured aortic pulse pressure and long-term prognosis in patients undergoing invasive coronary angiography: a prospective observational study. J Hypertens. 2026 Feb 1;44(2):354-359.
5. Kim HL, et al. Arterial Stiffness and Histologically Confirmed Helicobacter pylori Infection in Young Adults. Helicobacter. 2025 Jul-Aug;30(4):e70067.

Arterial stiffness and vascular aging in diabetes

Diabetes mellitus is a major accelerator of vascular aging and is strongly associated with premature cardiovascular disease. Chronic hyperglycemia, insulin resistance, oxidative stress, inflammation, endothelial dysfunction, and accumulation of advanced glycation end-products contribute to progressive structural and functional changes in the arterial wall. These alterations promote collagen cross-linking, elastin fragmentation, vascular calcification, and impaired vasomotor function, ultimately leading to increased arterial stiffness. As arterial stiffness progresses, the buffering capacity of large arteries declines, pulse wave velocity increases, and excessive pulsatile energy is transmitted to the heart and vulnerable microvascular beds, including the kidneys and brain. Accordingly, increased arterial stiffness in patients with diabetes is closely associated with both macrovascular and microvascular complications, including coronary artery disease, heart failure, stroke, chronic kidney disease, retinopathy, and cognitive impairment. Importantly, arterial stiffness reflects cumulative vascular injury and may provide information beyond conventional cardiovascular risk factors and glycemic indices alone. Patients with similar levels of glycated hemoglobin may exhibit markedly different degrees of vascular aging and cardiovascular risk. Pulse wave velocity, particularly carotid-femoral and brachial-ankle pulse wave velocity, offers a practical approach to assessing arterial stiffness and biological vascular age. Incorporating arterial stiffness into cardiovascular risk assessment may help identify individuals with accelerated vascular aging who could benefit from earlier and more intensive preventive strategies. In addition, improving blood pressure control, glycemic status, lipid levels, body weight, and lifestyle factors may favorably influence arterial stiffness. Future studies should determine whether arterial stiffness-guided management can improve clinical outcomes. Integrating vascular aging markers into diabetes care may provide a useful framework for more personalized cardiovascular prevention and precision risk stratification.

CURRICULUM VITAE

Janghoon Lee

Professor, Kyungpook National University Hospital, Republic of Korea

Education and Training

1999.02	Kyungpook National University, Korea, M.D, Medicine
2005.02	Kyungpook National University, Korea, Ph.D, Master of Science
2007.03	Korean Ministry of Health & Welfare, Korea, Korean Board of Internal Medicine, Internal Medicine

Employment and Position

2007-2011	Kyungpook National University Hospital, Fellowship
2011-2015	Kyungpook National University Hospital, Assistant Professor
2015-2020	Kyungpook National University Hospital, Associate Professor
2020-	Kyungpook National University Hospital, Professor
2020-	Daegu-Gyeongbuk Regional Cardiocerebrovascular Center, Chief of Cardiovascular Center

Coronary artery calcium and subclinical atherosclerosis in diabetes

Background: Diabetes substantially increases atherosclerotic cardiovascular disease (ASCVD) risk, but cardiovascular risk within diabetes is heterogeneous. CAC measured by noncontrast computed tomography is a reproducible marker of calcified coronary atherosclerotic burden and has emerged as one of the strongest imaging-based predictors of future coronary and cardiovascular events. [8-15]

Observations: Hyperglycemia, advanced glycation end products, oxidative stress, inflammation, diabetic dyslipidemia, vascular smooth-muscle-cell osteogenic differentiation, cellular senescence, and CKD-associated mineral abnormalities contribute to accelerated vascular calcification in diabetes. CAC nevertheless varies from zero to extremely high values, reflecting marked heterogeneity in cumulative atherosclerotic exposure. Standard Agatston scoring identifies calcified lesions using a threshold of approximately 130 Hounsfield units and integrates lesion area and peak density; CAC volume, mass, density, percentile, and vessel-specific measures provide complementary information. [8,9,22-29]

Across observational cohorts—including MESA, PREDICT, Diabetes Heart Study, and large clinical registries—CAC provides strong, graded prognostic information in diabetes beyond conventional risk factors. CAC=0 identifies low near-term risk but does not confer the prolonged "warranty period" observed in many people without diabetes; diabetes accelerates conversion from CAC=0 to incident CAC. Conversely, CAC ≥ 300-400 and especially ≥1,000 identifies a phenotype with high absolute event risk. [12-20]

Clinical implications: Current guidelines converge on selective rather than universal CAC testing. The 2026 ACC/AHA dyslipidemia guideline explicitly integrates CAC into a "calculate-personalize-reclassify" framework while retaining diabetes as an indication for LDL-lowering treatment. The 2025 ESC/EAS focused update classifies increased CAC or imaging-detected subclinical coronary atherosclerosis as a risk modifier in moderate-risk individuals. The 2023 ESC diabetes guideline uses SCORE2-Diabetes for risk stratification in appropriate patients. KSoLA recognizes CAC as a risk modifier, while ADA 2026 considers CAC reasonable for risk assessment in adults with diabetes aged ≥40 years but recommends against routine screening for asymptomatic CAD. [1-7]

Conclusions: CAC is most useful in diabetes as a marker of *atherosclerotic disease burden and residual risk*, particularly when therapeutic uncertainty remains despite conventional clinical risk assessment. CAC=0 can support a less aggressive approach to optional therapies but generally should not justify withdrawal of foundational statin treatment. CAC ≥ 300-400 should prompt reconsideration of whether the patient deserves a prevention strategy closer to that used for very-high-risk primary prevention, and CAC ≥1,000 should be considered an extreme-risk phenotype. Definitive randomized trials comparing CAC-guided versus guideline-only preventive strategies in diabetes, particularly in Asian populations, remain a major unmet need.

Keywords: Coronary artery calcium, Diabetes mellitus, Subclinical atherosclerosis, Cardiovascular risk, Statin, PCSK9 inhibitor, Aspirin, GLP-1 receptor agonist, SGLT2 inhibitor, Computed tomography, Precision prevention

Symposium 26

Integrated Cardiovascular-kidney-metabolic Care: A Multidisciplinary Approach to CKM Syndrome

Sep 12(Sat) 14:10-15:40 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Yong-Jae Kim (Kim's Neurology Clinic, Republic of Korea)
Sung Kee Ryu (Ewha Womans University, Republic of Korea)

14:10-14:30	**Cardiologist perspective: implementing novel cardiometabolic therapies in CVD** Donna Shu-Han Lin (Shin Kong Wu Ho-Su Memorial Hospital, Taiwan)
14:30-14:50	**Nephrologist's perspective: kidney protection strategies to improve cardio-kidney outcomes** Jong Hyun Jhee (Yonsei University, Republic of Korea)
14:50-15:10	**Endocrinologist perspective: optimizing combination therapy in complex CKM patients** Jae-Seung Yun (The Catholic University of Korea, Republic of Korea)
15:10-15:40	**Panel Discussion** Jaeseok Kim (Yonsei University, Republic of Korea) Min Kyung Lee (Hanyang University, Republic of Korea) Min-Ho Lee (Sahmyook Medical Center, Republic of Korea)

CURRICULUM VITAE

Donna Shu-Han Lin

Attending Physician, Division of Cardiology, Deputy Director of Internal Medicine Education, Shin Kong Wu Ho-Su Memorial Hospital, Taipei, Taiwan

Education and Training

2008-2015	National Yang-Ming University, Taipei, Taiwan, M.D., Medicine
2015-2016	Taipei Veterans General Hospital, Taipei, Taiwan, Post-Graduate Year Residency, General Medicine
2016-2019	National Taiwan University Hospital, Taipei, Taiwan, Residency, Internal Medicine
2019-2021	National Taiwan University Hospital, Taipei, Taiwan, Fellowship, Cardiovascular Medicine

Employment and Position

2023.04-Present	Shin Kong Wu Ho-Su Memorial Hospital, Taipei, Taiwan, Attending Physician, Division of Cardiology; Deputy Director of Internal Medicine Education (2026-Present)
2021-2023	National Taiwan University Hospital, Hsin-Chu Branch, Taiwan, Attending Physician, Division of Cardiology
2019-2021	National Taiwan University Hospital, Taipei, Taiwan, Fellow, Division of Cardiology
2016-2019	National Taiwan University Hospital, Taipei, Taiwan, Resident, Department of Internal Medicine

Important Publications

1. Lin DS-H, Lo H-Y, Lee J-K. Revisiting glycemic control targets: survival outcomes by HbA1c levels among patients with diabetes under contemporary antidiabetic therapy. J Am Heart Assoc. 2026;15(10):e046125.
2. Lin DS-H, Lee J-K, Huang K-C, Lin T-T, Lo H-Y. Effects of sodium-glucose cotransporter 2 inhibitors on cardiovascular and renal outcomes in people with diabetes and advanced chronic kidney disease. Diabetologia. 2024;67(11):2459-2470.

Cardiologist perspective: implementing novel cardiometabolic therapies in CVD

The cardiometabolic pharmacopoeia has expanded faster than our ability to deliver it. Incretin-based therapies, SGLT2 inhibitors, non-steroidal mineralocorticoid receptor antagonists and a widening class of lipid-modifying agents all reduce cardiovascular events — yet almost every landmark outcome trial enrolled patients who already had clinical disease. Viewed through the cardiovascular-kidney-metabolic (CKM) framework, the evidence base is inverted relative to the population it must serve: in a Taiwanese cohort of more than 500,000 adults, only 5.7% occupy CKM stages 3-4, where those trials were conducted, while roughly two-thirds sit at stages 1-2.

This lecture asks what is genuinely proven earlier in the continuum. Analyses restricted to participants without established atherosclerotic disease show that incretins and SGLT2 inhibitors lose their effect on major adverse cardiovascular events, while retaining robust reductions in heart failure hospitalisation and in kidney function decline. At CKM stage 2, in other words, these agents do not prevent atherothrombosis; they prevent the transition to stages 3 and 4. Lipid lowering remains the only class with large, replicated, positive primary-prevention outcome trials, and the trials that failed — in weight loss and in triglyceride reduction — are examined for what they reveal about apolipoprotein B as the causal mediator. CKM stage 3 is then dissected into its constituent limbs, of which only very-high-risk chronic kidney disease is adequately evidenced.

The second half addresses implementation. Registry and randomised data show that identification converts poorly into prescription, and prescription poorly into sustained treatment — a pattern reproduced in our own institutional CKM labelling and bidirectional referral programme. Finally, local reimbursement architecture, illustrated with Taiwan's National Health Insurance criteria, determines whether guideline-directed cardiometabolic therapy is deliverable at all: a consideration frequently absent from international guidance.

CURRICULUM VITAE

Jong Hyun Jhee

Associate Professor, Gangnam Severance Hospital Yonsei University, Republic of Korea

Education and Training

2011.02	Yonsei University, Korea, M.D., Medical School
2019.02	Yonsei University, Korea, Ph.D., Medical Science

Employment and Position

2012-2016	Internal Medicine/Severance Hospital, Resident
2016-2018	Nephrology/Severance Hospital, Fellow
2018-2019	Nephrology/Inha University Hospital, Assistant Professor
2020-2025	Nephrology/Gangnam Severance Hospital, Assistant Professor
2026-	Nephrology/Gangnam Severance Hospital, Associate Professor

Important Publications

1. Jhee JH, Ha KH, Lee H et al. Cumulative Cardiovascular Health Score Through Young Adulthood and Cardiovascular and Kidney Outcomes in Midlife. JAMA Cardiol. 2025 Oct 1:e253269. doi: 10.1001/jamacardio.2025.3269.
2. Jhee JH, Lee HH, Lee H et al. Blood Pressure and Cardiovascular Outcomes in Adults With Diabetes and Chronic Kidney Disease. J Am Heart Assoc. 2025 Oct 7;14(19):e042966. doi: 10.1161/JAHA.125.042966.

Nephrologist's perspective: kidney protection strategies to improve cardio-kidney outcomes

Chronic kidney disease (CKD) is both a major component and a powerful risk amplifier of cardiovascular-kidney-metabolic (CKM) syndrome. Progressive loss of kidney function, albuminuria, hypertension, diabetes, obesity, and cardiovascular disease interact bidirectionally, creating a cycle in which kidney dysfunction substantially increases cardiovascular morbidity and mortality. Therefore, kidney protection should be considered a central component of integrated CKM care rather than an isolated renal treatment strategy.

The therapeutic landscape of CKD has evolved from single-disease management toward comprehensive cardiorenal risk reduction. Renin-angiotensin system inhibitors remain a fundamental therapy, particularly in albuminuric CKD, while sodium-glucose cotransporter-2 (SGLT2) inhibitors provide robust kidney and cardiovascular protection across a broad spectrum of patients with CKD. In patients with type 2 diabetes and persistent albuminuria, the nonsteroidal mineralocorticoid receptor antagonist, finerenone provides additional cardiorenal benefit through complementary anti-inflammatory and antifibrotic pathways. Glucagon-like peptide-1 receptor agonists further expand this strategy by targeting obesity, glycemic control, atherosclerotic cardiovascular risk, and kidney outcomes.

Importantly, multidrug therapy does not necessarily imply simultaneous initiation. Most pivotal CKD trials evaluated therapies as add-on treatment to established background therapy, and combining multiple agents may amplify early changes in estimated glomerular filtration rate, potassium, blood pressure, and volume status. These issues are particularly relevant in older or frail patients, advanced CKD, and those with multimorbidity or polypharmacy. Thus, treatment should be individualized according to kidney and cardiovascular risk, albuminuria, eGFR, metabolic phenotype, hemodynamic tolerance, and safety.

A practical approach is risk-based accelerated sequential therapy: identify patients at high risk of kidney failure and cardiovascular events, establish appropriate foundational therapy, and rapidly layer complementary kidney-protective agents with structured monitoring. Such an approach may minimize therapeutic inertia while preserving safety and treatment adherence.

Ultimately, optimal kidney protection in CKM syndrome requires moving beyond a kidney-centric perspective toward coordinated, multidisciplinary management of shared cardiovascular, renal, and metabolic risk. In this lecture, we will discuss current evidence and practical strategies for integrating kidney-protective therapies to improve cardio-kidney outcomes in patients across the spectrum of CKM syndrome.

CURRICULUM VITAE

Jae-Seung Yun

Professor, Division of Endocrinology and Metabolism, Department of Internal Medicine,
St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Republic of Korea

Education and Training

2000-2006	The Catholic University of Korea, College of Medicine, Korea, M.D., Medicine
2008-2010	The Catholic University of Korea, Korea, M.S., Medical Science
2013-2016	The Catholic University of Korea, Korea, Ph.D., Medical Science

Employment and Position

2016-2018	St. Vincent's Hospital, The Catholic University of Korea, Assistant Professor
2019-2022	St. Vincent's Hospital, The Catholic University of Korea, Associate Professor
2020-2021	Perelman School of Medicine, University of Pennsylvania, USA, Visiting Professor
2023-Present	St. Vincent's Hospital, The Catholic University of Korea, Professor
2023-Present	Korean Diabetes Association, Director, Digital Health Committee

Important Publications

1. Yu HJ, Hong D, Kim K, Heo JH, Cho DH, Hashimoto Y, Yun JS. Optimizing physical activity strategies for older adults with diabetes. Diabetes Metab J. 2025;49(6):1178-97.
2. Yun JS, Jung JH, Kim B, Kim K, Ahn YB, Kim SY, et al. Age and sex differences of cardiovascular and mortality risks in Korean type 2 diabetes and prediabetes: an 8 million Korean nationwide population-based cohort study. Diabetes Res Clin Pract. 2025;227:112401.
3. Kim K, Kim B, Lee K, Ahn YB, Ko SH, Choi SH, Han K, Yun JS. Older adults with diabetes in Korea: latest clinical and epidemiologic trends. Diabetes Metab J. 2025;49:183-93.

Endocrinologist perspective: optimizing combination therapy in complex CKM patients

Cardiovascular-kidney-metabolic (CKM) syndrome reframes obesity, type 2 diabetes, chronic kidney disease, and cardiovascular disease as connected manifestations of a single continuum rather than as separate diagnoses. The burden is already substantial in Korea. In nationally representative survey data, stage 2 is the most common stage, accounting for more than 40% of adults, and the prevalence of the advanced stages 3 and 4 rose significantly over an eleven-year period while stage 0 declined. In June 2026, the ACC, AHA, ADA, and ASN released the first joint guideline dedicated to CKM syndrome.

Four elements reshape everyday practice. CKM staging is now recommended across the life course, for youths as well as adults, to tailor therapy to absolute risk and to promote stage regression. The PREVENT equations quantify 10- and 30-year risk for individuals in stages 0 to 3, and a predicted 10-year cardiovascular risk of 20% or higher serves as one criterion for stage 3. Body mass index and waist circumference are both used to characterize adiposity, and weight management is positioned as a foundational intervention. Interdisciplinary care models with a designated coordination point person are recommended when diabetes, kidney disease, and cardiovascular disease overlap.

Lipid management remains central to this framework. Atherogenic dyslipidemia appears early in the CKM continuum, and residual risk after LDL cholesterol targets are met deserves particular attention when kidney function is impaired. This lecture reviews the pathophysiology and stage-based evaluation of CKM syndrome from an endocrinologist's perspective, considers how agents with proven cardiovascular and kidney protection fit each stage, and examines the practical barriers to delivering integrated rather than siloed care in Korea, including referral pathways, shared protocols, and digital tools for risk stratification and remote monitoring.

Symposium 27

AI-driven Innovations in Atherosclerosis

Sep 12(Sat) 14:10-15:40 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Yongseek Park (Kyung Hee University, Republic of Korea)
Hyun Jae Kang (Seoul National University, Republic of Korea)

14:10-14:28	**AI-CVD: opportunistic testing of coronary calcium and beyond** Nathan D. Wong (University of California, Irvine, USA)
14:28-14:46	**A+4DCT: angio enhanced dynamic CT for non-invasive functional diagnosis of coronary artery disease** Joon Sang Lee (Yonsei University, Republic of Korea)
14:46-15:04	**Application of AI to coronary angiography** Si-Hyuck Kang (Seoul National University, Republic of Korea)
15:04-15:22	**AI-based retinomics: cardiovascular risk prediction using retinal imaging** Sahil Thakur (Medical Affairs, Mediwhale Inc., Singapore)
15:22-15:40	**Panel Discussion** Kyu-Yong Ko (Inje University, Republic of Korea) Hee-Dong Kim (Soonchunhyang University, Republic of Korea) Kwan Yong Lee (The Catholic University of Korea, Republic of Korea)

CURRICULUM VITAE

Nathan D. Wong

Professor and Director, Heart Disease Prevention Program,
Mary and Steve Wen Cardiovascular Division and Co-Director,
Center for Global Cardiometabolic Health and Nutrition
Past President, American Society for Preventive Cardiology and
Pacific Lipid Association Chapter of the National Lipid Association
University of California, Irvine School of Medicine, Irvine, USA

Education and Training

1985	Yale University, MPH, Epidemiology
1987	Yale University, PhD, Epidemiology

Employment and Position

1988–Present	University of California, Irvine School of Medicine, Professor

Important Publications

1. Wong ND. Epidemiological studies of CHD and the evolution of preventive cardiology. Nat Rev Cardiol. 2014 May;11(5):276-89. doi: 10.1038/nrcardio.2014.26. Epub 2014 Mar 25. PMID: 24663092 (IF=41.7).
2. Wong ND, Zhao Y, Patel R, Patao C, Malik S, Bertoni AG, Correa A, Folsom AR, Kachroo S, Mukherjee J, Taylor H, Selvin E. Cardiovascular Risk Factor Targets and Cardiovascular Disease Event Risk in Diabetes: A Pooling Project of the Atherosclerosis Risk in Communities Study, Multi-Ethnic Study of Atherosclerosis, and Jackson Heart Study. Diabetes Care. 2016; 39(5):668-76 (IF=16.6).
3. Wong ND, Sattar N. Cardiovascular risk in diabetes mellitus: epidemiology, assessment and prevention. Nat Rev Cardiol. 2023; 20(10):685-695 (IF=41.7).

AI-CVD: opportunistic testing of coronary calcium and beyond

Coronary artery calcium (CAC) is the most powerful of subclinical atherosclerosis measures for the prediction of atherosclerotic cardiovascular disease (ASCVD). CAC testing in recent years has been incorporated into cardiovascular disease prevention guidelines for ASCVD risk stratification and most recently even to recommend specific goals for LDL-C control. While there has been an increase in CAC testing utilizing gated CT scans, the availability of AI techniques in recent years for measuring CAC has provided significant opportunities for assessing CAC in the much larger population received non-gated CT chest CT scans for other indications such as lung cancer screening. A recent American Heart Association statement further describes the emerging and valuable role such opportunistic assessment of CAC has in cardiovascular risk assessment. Moreover, AI technology enables efficient and rapid assessment of a wide range of other cardiac and non-cardiac measures from either the gated or non-gated CT scan, including other vascular calcification such as aortic and mitral valve calcium, thoracic aortic calcium, bone mineral density, left and right ventricular and atrial volumes, myosteatosis, and liver attenuation/fat. Moreover, recent newer generation AI-CAC (Agatston 2.0) offers the ability to identify earlier borderline or semi-calcified plaque that adds to ASCVD risk prediction, even when traditional CAC scores are 0. Emerging and recently published evidence documents how individual and multiple measures of these imaging parameters improve prediction of cardiovascular events beyond existing risk factors and scores (e.g., PREVENT, CHARGE-AF), as well as risks of specific cardiovascular outcomes (including heart failure, stroke, and atrial fibrillation). Moreover, AI assessments from CT angiography offer the ability to quantitate calcified and non-calcified plaque which is also helpful to assess ASCVD risk. These advancements hold promise for personalizing therapeutic approaches to best address risks in the vulnerable patient.

CURRICULUM VITAE

Joon Sang Lee

Professor, School of Mechanical Engineering, Yonsei University, Seoul, Republic of Korea

Education and Training

1999-2004	Iowa State University, Ames, IA, USA, Ph.D., Mechanical Engineering

Employment and Position

2009-Present	School of Mechanical Engineering, Yonsei University, Korea, Assistant/Associate/Full Professor
2018-Present	College of Medicine and College of Dentistry, Yonsei University, Korea, Adjunct Professor
2023-Present	The M.E.N.D. BioSimulator Inc., Korea, CEO
2023-Present	Ministry of Education / Ministry of Justice, Korea, Member of the Education Internationalization Capacity Accreditation Committee
2022-Present	Journal of Mechanical Science and Technology, Korea, Associate Editor
2024-2025	Biomedical Engineering Society for Circulation, Korea, President
2024-2025	Yonsei Enterprise Support Foundation, Yonsei University, Korea, Director
2020-2023	Office of International Affairs, Yonsei University, Korea, Associate Vice President
2019-2021	Korea Institute of Civil Engineering and Building Technology, Korea, Visiting Professor
2018-2019	Samsung Electronics, Korea, Technical Advisory Professor
2016-2020	College of Engineering, Yonsei University, Korea, Associate Dean
2004-2009	Department of Mechanical Engineering, Wayne State University, USA, Assistant Professor
2002-2003	Whirlpool Co., USA, Engineer

A+4DCT: angio enhanced dynamic CT for non-invasive functional diagnosis of coronary artery disease

CT-derived fractional flow reserve (CT-FFR) is a widely used noninvasive method for assessing the functional significance of coronary artery stenosis, but its accuracy is limited by two inherent factors. First, the limited spatial resolution of CT and blooming artifacts around calcified lesions hinder precise delineation of the true luminal boundary, so the stenosis severity and FFR values derived from CT-based segmentation may deviate from invasive clinical measurements. Second, conventional CT-FFR is computed on a static single-phase geometry, whereas the coronary arteries continuously deform over the cardiac cycle, causing the pressure drop to fluctuate. An FFR evaluated at a single time point may therefore overlook transient FFR reductions occurring at specific cardiac phases.

To address these limitations, we present A+4DCT, an angio-enhanced framework for dynamic FFR assessment. First, to restore luminal accuracy, the framework leverages invasive angiography, which provides complementary information on the coronary lumen, particularly where CT segmentation is compromised by calcification. During model development, invasive coronary angiography is used as a supervisory reference to improve the accuracy of coronary lumen reconstruction from CT, particularly in regions affected by calcification and blooming artifacts. Importantly, angiography serves only as a training reference and is not required at inference. Once trained, the A+4DCT framework is designed to generate lumen-accurate dynamic coronary geometry from a single-phase CT alone. Second, to incorporate cardiac dynamics, the lumen-corrected single-phase CT generated by the trained model is used to synthesize the remaining cardiac phases. The 4DCT model, trained with combined vision and motion losses, captures the nonlinear deformation of the coronary arteries throughout the cardiac cycle. Then, phase-wise CFD on the synthesized full-cycle geometry yields dynamic FFR.

The single-phase 4DCT model achieved a peak signal-to-noise ratio (PSNR) of 38 dB and a structural similarity index measure (SSIM) of 0.974, and in a preliminary case with an invasive FFR of 0.70, the framework yielded a phase-averaged dynamic FFR of 0.77, demonstrating the feasibility of the proposed pipeline. Ultimately, a physics-informed neural operator (PINO), trained on CFD-generated flow fields, will enable direct prediction of dynamic 4DCT-FFR from a single end-diastolic CT, eliminating the need for both invasive angiography at inference and case-specific CFD simulation. This approach is therefore expected to reduce radiation exposure and improve the reliability and accessibility of noninvasive functional diagnosis of coronary artery disease.

CURRICULUM VITAE

Si-Hyuck Kang

Associate Professor, Cardiology (Interventional Cardiology),
Seoul National University Bundang Hospital, Republic of Korea

Education and Training

2015.03-Present	Seoul National University, Korea, Ph.D., Medicine (Cardiology)
2009.03-2014.02	Seoul National University, Korea, M.M.Sc., Medical Science
2001.03-2005.02	Seoul National University College of Medicine, Korea, M.D., Medicine
1998.03-2001.02	Seoul National University, Korea, Premedical, Premedical Course

Employment and Position

2022-Present	Seoul National University Bundang Hospital, Seongnam, Korea, Associate Professor, Cardiology
2016-2022	Seoul National University Bundang Hospital, Seongnam, Korea, Assistant Professor, Cardiology
2020-2022	Google Health, Palo Alto, CA, USA, Visiting Researcher
2013-2016	Seoul National University Hospital, Seoul, Korea, Clinical Fellow, Cardiology
2011-2013	Ministry of National Defense, Seoul, Korea, Medical Officer
2010-2011	UN Command Security Battalion, Joint Security Area, Korea, Medical Officer
2006-2010	Seoul National University Hospital, Seoul, Korea, Resident, Internal Medicine
2005-2006	Seoul National University Hospital, Seoul, Korea, Intern

Important Publications

1. Kim Y, Yoon HJ, Suh J, Kang SH, et al. Artificial Intelligence-Based Fully Automated Quantitative Coronary Angiography vs Optical Coherence Tomography-Guided PCI (FLASH Trial). JACC Cardiovasc Interv. 2025;18(2):187-197.
2. Choi H, Kang SH, et al. Lipid-Lowering Efficacy of Combination Therapy With Moderate-Intensity Statin and Ezetimibe Versus High-Intensity Statin Monotherapy: A Randomized, Open-Label, Non-Inferiority Trial From Korea. J Lipid Atheroscler. 2023;12(3):277-289.
3. Moon IT, Kim SH, Chin JY, Park SH, Yoon CH, Youn TJ, Chae IH, Kang SH. Accuracy of Artificial Intelligence-Based Automated Quantitative Coronary Angiography Compared to Intravascular Ultrasound. JMIR Cardio. 2023;7:e45299.

Application of AI to coronary angiography

Coronary angiography remains the gold standard for diagnosing and treating cardiovascular diseases, including atherosclerosis. However, the continuous concern regarding cumulative radiation exposure to both patients and healthcare professionals necessitates innovative solutions. Recent advancements in generative artificial intelligence (AI) offer a transformative approach to overcoming these clinical limitations, maximizing both the safety and efficiency of interventions.

This presentation will explore the latest achievements in applying AI to coronary angiography, focusing on deep learning models designed to optimize image quality and procedural safety. A key highlight is the development and clinical validation of Angio-FILM, a novel frame interpolation model that synthesizes high-frame-rate (15 FPS) videos from low-frame-rate (7.5 FPS) inputs. Crucially, Angio-FILM reduces radiation exposure by half while strictly preventing "clinical hallucinations," ensuring the diagnostic integrity and reliability of the synthesized images.

Furthermore, the lecture will introduce generative AI-based super-resolution technology, which elevates standard fluoroscopic images to cine-quality sequences, promising a more dramatic reduction in radiation doses without compromising visual clarity. We will also discuss the recent outcomes of AngioDINO, an AI-driven foundation model that meticulously analyzes vascular structures and atherosclerotic lesions to assist in clinical decision-making.

By accurately visualizing lesions without anatomical distortion, these innovative AI-based image enhancement and analysis technologies serve as vital decision-support tools for operators. This session will highlight how AI is shifting the paradigm of coronary interventions and discuss future directions for seamlessly integrating these models into real-world clinical practice, ultimately fostering a safer and more optimized procedural environment.

CURRICULUM VITAE

Sahil Thakur

Adjunct Research Fellow, Duke NUS Medical School, Singapore

Education and Training

2009-2015	Panjab University, India, MBBS, Medicine
2015-2018	Panjab University, India, M.D., Ophthalmology
2020-2024	Duke NUS Medical School, Singapore, Ph.D., Translational Medicine
2023-2025	Quantic School of Business and Technology, USA, MBA, Business

Employment and Position

2018-2024	Singapore Eye Research Institute, Research Fellow
2024-Current	Mediwhale, South Korea, Head of Medical Affairs
2025-Current	Duke NUS Medical School, Singapore, Adjunct Research Fellow
2025-Current	Singapore Eye Research Institute, Adjunct Research Fellow

AI-based retinomics: cardiovascular risk prediction using retinal imaging

Cardiovascular disease (CVD) remains the leading cause of death worldwide, yet current risk assessment strategies rely heavily on traditional clinical risk factors and laboratory investigations that may limit access to timely screening. Recent advances in artificial intelligence (AI) and retinal imaging have introduced retinomics, an emerging field that leverages retinal photographs as a non-invasive source of systemic cardiovascular information. The retina provides a unique window into the body's microvascular health, enabling AI algorithms to detect vascular and structural changes associated with cardiovascular risk. This presentation will review the scientific basis of AI-driven retinal analysis for cardiovascular risk prediction and discuss its role alongside conventional risk assessment tools. Evidence from large population cohorts and external validation studies will be presented, demonstrating the ability of retinal AI models to identify individuals at elevated risk of major adverse cardiovascular events, often without requiring blood tests or specialized imaging. The presentation will also examine how retinal biomarkers complement established risk scores by improving risk stratification and identifying high-risk individuals who may otherwise remain undetected. Practical considerations for clinical implementation will be discussed, including workflow integration within primary care, cardiology, diabetes, and ophthalmology settings. The talk will highlight the advantages of retinal AI as a rapid, scalable, and accessible screening tool that can support preventive cardiovascular care, particularly in resource-limited environments. Current challenges, including validation across diverse populations, regulatory considerations, model interpretability, and integration into existing healthcare systems, will also be addressed. By exploring the intersection of ophthalmology, artificial intelligence, and cardiovascular medicine, this session will provide clinicians with an overview of how AI-based retinomics is reshaping cardiovascular screening and its potential to advance earlier detection, personalized risk assessment, and precision prevention strategies.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Symposium 28

Primary Prevention in Cardiovascular Disease: Beyond Traditional Risk Factors

Sep 12(Sat) 14:10–15:40 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Bon-kwon Koo (Seoul National University, Republic of Korea)
Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea)

14:10–14:30	**Lipid management in primary prevention: who, when, and how aggressive?** Yeoree Yang (The Catholic University of Korea, Republic of Korea)
14:30–14:50	**Every minute counts: exercise snacks for better health and life's essential 8** SoJung Lee (Kyung Hee University, Republic of Korea)
14:50–15:10	**Advancing cardiovascular management through vaccination** Jae Hyoung Park (Korea University, Republic of Korea)
15:10–15:40	**Panel Discussion** Mihye Seo (Soonchunhyang University, Republic of Korea) Hyun Ju You (Seoul National University, Republic of Korea) Jun Hwa Hong (Eulji University, Republic of Korea)

CURRICULUM VITAE

Yeoree Yang

Clinical Assistant Professor,
Division of Endocrinology & Metabolism, Department of Internal Medicine,
Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea
Vice Director, Catholic Smart Health Care Center, The Catholic University of Korea, Republic of Korea

Education and Training

2004-2010	The Catholic University of Korea, M.D., Medicine
2015-2023	The Catholic University of Korea, Ph.D., Internal Medicine

Employment and Position

2018-Present	Division of Endocrinology & Metabolism, Department of Internal Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Clinical Assistant Professor
2019-Present	Catholic Smart Health Care Center, The Catholic University of Korea, Vice Director

Lipid management in primary prevention: who, when, and how aggressive?

Primary prevention is where lifelong cardiovascular protection is largely determined, yet it remains the setting in which we are least certain whom to treat, when to begin, and how far to lower. Earlier guidelines and the current Korean guideline assign risk categories from clinical comorbidities and the number of major risk factors, then use the LDL-C level to decide when to treat and how far to lower it. Recent guidelines are more layered: the 2026 AHA/ACC guideline sets statin intensity and the LDL-C goal according to 10-year risk estimated with the PREVENT-ASCVD equations, and further recommends a 30-year estimate in adults aged 30 to 59 years whose 10-year risk is low. Atherosclerosis develops over decades, and 10-year risk is typically low in younger adults, in whom only a longer horizon makes that risk visible.

This reframes when to begin. What a single measurement cannot convey is duration of exposure: cumulative LDL-C burden from young adulthood predicts coronary events independently of midlife levels, and lifelong exposure to lower LDL-C is associated with greater benefit than treatment started in middle age. The guideline reflects this by advising early consideration of pharmacotherapy in youth with familial hypercholesterolemia and in young adults with LDL-C ≥160 mg/dL or a strong family history of premature ASCVD. Where risk remains ambiguous, risk enhancers — elevated Lp(a), high polygenic risk, hsCRP, apolipoprotein B — help personalize the estimate, and coronary artery calcium can move the decision in either direction.

How aggressively to treat is defined by goals, but also by how much exposure remains across expected life expectancy. Cumulative burden is a useful concept yet hard to apply: historical values are seldom available, fluctuations are difficult to quantify, and no universal cutoff exists. What follows is a personalized approach — early screening, periodic reassessment, family history, genetic and long-term risk estimation — moving toward lower, earlier, and for longer, with greater emphasis on primordial prevention.

CURRICULUM VITAE

SoJung Lee

Full Professor, Kyung Hee University, Republic of Korea

Education and Training

2005	Queen's University (Canada), Ph.D., Obesity, Physical Activity, Health
2001	University of Manitoba (Canada), M.Sc., Exercise Physiology
1996	Leisure and Recreation, Ewha Womans University (South Korea), B.Sc., Leisure and Recreation

Employment and Position

2017.03-Present	Division of Sports Medicine & Science, Graduate School of Physical Education, Kyung Hee University, Full Professor
2015.07-2017.06	Division of Weight Management and Wellness, UPMC Children's Hospital of Pittsburgh, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA, Associate Professor of Pediatrics with Tenure
2007.03-2015.06	Division of Weight Management and Wellness, UPMC Children's Hospital of Pittsburgh, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA, Assistant Professor of Pediatrics
2005.02-2007.02	Division of Weight Management and Wellness, UPMC Children's Hospital of Pittsburgh, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA, Post-Doctoral Research Associate

Important Publications

1. Park J, Lee S, Yun J, Cho S, Lee S*. Feasibility and Effects of Exercise Snacks on Fitness and Body Composition in Women with Overweight or Obesity: A Randomized Controlled Trial. J Phys Act Health. 2026. In Press. * Corresponding author.
2. Mirzadeh P, Lee S*, Arslanian S, Kuk JL. Individual Variability in Insulin Sensitivity, Visceral Fat, and Cardiorespiratory Fitness in Response to Exercise in Adolescents with Obesity. Nutr Metab Cardiovasc Dis. 2026 Jun;36(6):104615. * Corresponding author.
3. Kim S, Cho S, Lee S*. Effects of Whole-body Vibration Exercise on Cardiorespiratory fitness, Muscular Strength and Body Fat in Women with Overweight and Obesity: A Randomized Pilot Study. Nutr Metab Cardiovasc Dis. 2025 Jun;35(6):104019. * Corresponding author.

Every minute counts: exercise snacks for better health and life's essential 8

In recent years, physical activity levels among Korean adults have continued to decline, while sedentary behavior has steadily increased. The World Health Organization recommends that adults should engage in at least 150 minutes of moderate-intensity aerobic physical activity or 75 minutes of vigorous-intensity aerobic physical activity per week to improve fitness and prevent chronic diseases. However, according to the National Survey, 32.2% of Korean adults reported no participation in physical activity, with lack of time (57.5%) and limited access to sports facilities (35.0%) identified as major barriers to physical activity.

Against this backdrop, "exercise snacks" have recently gained attention as a practical exercise strategy for reducing prolonged sedentary behavior and improving metabolic health. Although a standardized protocol for exercise snacks has not yet been established, they generally refer to brief bouts of high-intensity physical activity, typically lasting 1-2 minutes, performed intermittently several times throughout the day. As these activities can be completed in a short period of time, exercise snacks may represent a feasible and accessible exercise strategy for adults who face substantial time constraints.

This lecture will introduce the concept and practical application of exercise snacks and review the effects of exercise snacks on cardiorespiratory fitness, body composition and metabolic risk factors based on the recent evidence. In particular, from a practical exercise prescription perspective in the management of obesity, we will discuss how exercise snacks may be incorporated to address not only weight loss but also improvements in cardiorespiratory fitness and overall metabolic health.

CURRICULUM VITAE

Jae Hyoung Park

Clinical Professor, Korea University Anam Hospital, Republic of Korea

Education and Training

1999.02	Korea University, M.D., Internal Medicine
2012.02	Korea University, Korea, Ph.D., Internal Medicine

Employment and Position

2007-2008	Asan Medical Center, Seoul, Korea, Clinical Fellow
2008-	Korea University Medical Center, Korea, Clinical Professor

Important Publications

1. Park JH, Kim JS, Ahn CM, et al. Prospective partially randomized comparison of clopidogrel loading versus maintenance dosing to prevent periprocedural myocardial infarction after stenting for stable angina pectoris: Results from the "Method of Clopidogrel Pre-treatment Undergoing Conventional Coronary Angiogram in Angina Patients (MECCA)" study [published online ahead of print, 2020 Jun 26]. Int J Clin Pharmacol Ther. 2020;10.5414/CP203644. doi:10.5414/CP203644

Advancing cardiovascular management through vaccination

Contracting influenza triggers a systemic inflammatory response and a "cytokine storm," leading to elevated levels of interleukin-6 (IL-6) and TNF-α. This systemic inflammation impairs vascular endothelial function and promotes the inflammatory destabilization of atherosclerotic plaques; furthermore, it increases the risk of thrombosis due to platelet activation and elevated coagulation factors. Such changes can precipitate vascular events—such as acute myocardial infarction or stroke—and cause acute exacerbation of pre-existing heart failure. Consequently, as influenza infection can trigger recurrent cardiovascular events in patients with acute coronary syndromes, vaccination serves as a crucial component of secondary prevention.

According to the 2025 European Society of Cardiology (ESC) clinical consensus statement on the prevention of cardiovascular disease, recommendations include—in addition to annual influenza vaccination—pneumococcal vaccination every 5 to 10 years, RSV vaccination approximately every two years, and at least one lifetime dose of the shingles vaccine.

Similarly, the 2025 American College of Cardiology (ACC) expert consensus statement on adult vaccination for cardiovascular disease management recommends annual influenza vaccination for individuals aged 6 months and older, at least one pneumococcal vaccination for those aged 19 and older, COVID-19 vaccination for those aged 6 months and older, RSV vaccination for those aged 60 and older, and two doses of the recombinant shingles vaccine for those aged 50 and older.

Like influenza, shingles increases inflammation and the risk of developing cardiovascular disease; conversely, patients with cardiovascular disease are more prone to developing shingles.

In summary, for patients with cardiovascular disease, four major vaccinations are recommended for those aged 50 and older (influenza, pneumococcal, shingles, and COVID-19), while five are recommended for those aged 60 and older (adding the RSV vaccine).

Symposium 29 – KES-KSoLA Joint Symposium

Lipid Metabolism Across Endocrine Disorders: From Mechanism to Clinical Practice

Sep 12(Sat) 14:10-15:40 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Sung Hee Choi (Seoul National University, Republic of Korea)
Sun Wook Cho (Seoul National University, Republic of Korea)

14:10-14:30	**Diabetic dyslipidemia: targeting residual risk beyond LDL-C lowering** Kyung-Soo Kim (CHA University, Republic of Korea)
14:30-14:50	**Subclinical thyroid dysfunction: when should we treat for cardiovascular and metabolic health?** Eu Jeong Ku (Seoul National University, Republic of Korea)
14:50-15:10	**Atherogenic dyslipidemia in women: PCOS, menopause, and cardiovascular risk** Jeong Rang Park (Gyeongsang National University, Republic of Korea)
15:10-15:40	**Panel Discussion** Dae-Young Kim (Inha University, Republic of Korea) Seo Young Sohn (Inje University, Republic of Korea) You-Bin Lee (Sungkyunkwan University, Republic of Korea)

CURRICULUM VITAE

Kyung-Soo Kim

Professor, Division of Endocrinology and Metabolism, Department of Internal Medicine,
CHA Bundang Medical Center, CHA University School of Medicine, Republic of Korea

Education and Training

2004	CHA University, College of Medicine, Korea, M.D.
2020	CHA University School of Medicine, Korea, Ph.D., Medicine

Employment and Position

2014-2020	Division of Endocrinology and Metabolism, Department of Internal Medicine, CHA Bundang Medical Center, CHA University School of Medicine, Korea, Assistant Professor
2020-2025	Division of Endocrinology and Metabolism, Department of Internal Medicine, CHA Bundang Medical Center, CHA University School of Medicine, Korea, Associate Professor
2025-	Division of Endocrinology and Metabolism, Department of Internal Medicine, CHA Bundang Medical Center, CHA University School of Medicine, Korea, Professor

Important Publications

1. Kim KS, Kim B, Han K. Big Data Research for Diabetes-Related Diseases Using the Korean National Health Information Database. Diabetes Metab J 2025;49:13-21.
2. Kim KS, Hong S, Han K, Park CY. Association of non-alcoholic fatty liver disease with cardiovascular disease and all cause death in patients with type 2 diabetes mellitus: nationwide population based study. BMJ 2024;384:e076388.
3. Park J, Jung JH, Park H, Song YS, Kim SK, Cho YW, Han K, Kim KS. Association between exercise habits and incident type 2 diabetes mellitus in patients with thyroid cancer: nationwide population-based study. BMC Med 2024;22:251. (Corresponding author)
4. Kim KS, Hong S, Ahn HY, Park CY. Metabolic dysfunction-associated fatty liver disease and mortality: a population-based cohort study. Diabetes Metab J 2023;47:220-231.
5. Kim KS, Hong S, Han K, Park CY. Fenofibrate add-on to statin treatment is associated with low all-cause death and cardiovascular disease in the general population with high triglyceride levels. Metabolism. 2022;137:155327.

Awards and Honors

2025	Songwon Medical Scientist Award, Korean Endocrine Society
2025	Young Investigator Award, Korean Diabetes Association
2022	Hyangseol Young Investigator Award, Korean Endocrine Society

Research Interest

Diabetes mellitus, Insulin resistance, Fatty liver disease, Lipid metabolism, Endocrine disorder

Diabetic dyslipidemia: targeting residual risk beyond LDL-C lowering

Individuals with type 2 diabetes have an increased risk of atherosclerotic cardiovascular disease, partly driven by mixed dyslipidemia. Reduction of LDL-cholesterol is the primary therapeutic target and is strongly associated with cardiovascular risk reduction. However, despite effective lipid-lowering therapies, lipid target attainment remains suboptimal, and residual atherogenic risk persists in many patients with diabetes. In this talk, I will present about diabetic dyslipidemia: targeting residual risk beyond LDL-C lowering.

CURRICULUM VITAE

Eu Jeong Ku

Associate Professor,
Seoul National University Hospital Healthcare System Gangnam Center, Republic of Korea

Education and Training

2008.02	Kyoungpook National University College of Medicine, Daegu, Korea, M.D., Medicine
2013.08	Seoul National University College of Medicine, Seoul, Korea, M.S., Clinical Medical Sciences
2021.02	Seoul National University College of Medicine, Seoul, Korea, Ph.D., Translational Medicine

Employment and Position

2015-2023	Chungbuk National University Hospital, Professor
2023-	Seoul National University Hospital Healthcare System Gangnam Center, Professor

Important Publications

1. Ku EJ, et al. Osteoporotic fracture risk in hypoparathyroidism following total thyroidectomy: a retrospective nationwide cohort study. Eur J Endocrinol. 2025 Jun 30;193(1):39-47.
2. Ku EJ, et al. Deep Learning-Based Adrenal Gland Volumetry for the Prediction of Diabetes. Endocrinol Metab (Seoul). 2025 Dec;40(6):991-1001.
3. Ku EJ, et al. Long-term Nonskeletal Complications in Patients With Thyroid Cancer and Hypoparathyroidism Post Total Thyroidectomy. J Clin Endocrinol Metab. 2025 Nov 18;110(12):3538-3545.
4. Ku EJ, et al. Risk of Osteoporotic Fractures among Patients with Thyroid Cancer: A Nationwide Population-Based Cohort Study. Endocrinol Metab (Seoul). 2025 Apr;40(2):225-235.
5. Yoo WS and Ku EJ, et al. Incidence of Endocrine-Related Dysfunction in Patients Treated with New Immune Checkpoint Inhibitors: A Meta-Analysis and Comprehensive Review. Endocrinol Metab (Seoul). 2023 Dec;38(6):750-759.

Subclinical thyroid dysfunction: when should we treat for cardiovascular and metabolic health?

Subclinical thyroid dysfunction — subclinical hypothyroidism (SCHypo) and subclinical hyperthyroidism (SCHyper) — is defined biochemically by an abnormal thyroid-stimulating hormone (TSH) level with normal free thyroxine, and is frequently detected incidentally during health screening. Both conditions have been associated with adverse cardiovascular and metabolic outcomes, yet the evidence for benefit from treatment differs substantially between them, making the decision of when, and whether, to intervene a persistent clinical challenge.

For SCHypo, recent large-scale randomized trials (TRUST, IEMO) and meta-analyses have found no consistent benefit of levothyroxine for quality of life, cognitive function, or cardiovascular outcomes, particularly in older adults. This evidence underpinned the 2019 BMJ Rapid Recommendation against routine treatment and the 2023 Korean Thyroid Association (KTA) guidelines, which introduced a region-specific TSH reference range (upper limit 6.8 mIU/L, based on national survey data) and stratified management by severity (mild vs. severe, cutoff 10.0 mIU/L) and age (<70 vs. ≥70 years). Treatment is generally reserved for adults under 70 with severe SCHypo and concurrent coronary artery disease, heart failure, or dyslipidemia, while elderly patients are managed with observation regardless of severity.

For SCHyper, the risk-benefit calculus differs: evidence more consistently supports associations between TSH suppression and atrial fibrillation, heart failure, and accelerated bone loss, particularly when TSH is persistently low. Treatment thresholds in this setting are similarly shaped by the degree of TSH suppression, patient age, and underlying cardiovascular or skeletal risk, but with a comparatively stronger rationale for intervention in higher-risk subgroups.

This presentation will cover the parallel evidence bases for SCHypo and SCHyper, contrasting their differing risk-benefit profiles, and propose a unified, risk-stratified framework, anchored in cardiovascular and metabolic endpoints, for deciding when treatment of subclinical thyroid dysfunction is indicated.

CURRICULUM VITAE

Jeong Rang Park

Division of Cardiology, Gyeongsang National University Hospital,
Professor, Division of Cardiology, Department of Internal Medicine,
Gyeongsang National University School of Medicine, Jinju, Republic of Korea

Education

2012	Ph.D. in Internal Medicine, Graduate School, Gyeongsang National University School
2005	Master in Internal Medicine, Graduate School, Gyeongsang National University School
2000	MD, in Medicine, Gyeongsang National University School of Medicine

Medical Training

2017	Research Fellow, Echocardiography Laboratory Cardiovascular Diseases, Mayo Clinic,
2009	Cardiovascular Imaging Fellow, Cardiovascular Imaging Center, Cardiac and Vascular Center, Samsung Medical Center, Sungkyunkwan University School of Medicine
2005	Clinical Fellow, Cardiology, Gyeongsang National University Hospital
2001-2004	Internal Medicine Resident, Gyeongsang National University Hospital

Professional Memberships

Korean Society of Internal Medicine
Korean Society of Cardiology
Korean Society of Echocardiography

Atherogenic dyslipidemia in women: PCOS, menopause, and cardiovascular risk

The 2026 dyslipidemia guideline emphasizes the importance of considering female-specific reproductive risk factors, including polycystic ovary syndrome (PCOS) and menopause, for more comprehensive cardiovascular (CV) risk assessment and management. PCOS, the most common endocrine disorder among women of reproductive age, is often overlooked from a cardiovascular perspective because it predominantly affects young women. This review aims to explore the mechanisms underlying the increased cardiometabolic and atherogenic risk associated with PCOS. In addition, we discuss the hormonal and metabolic changes occurring during the menopausal transition and their impact on dyslipidemia and cardiovascular risk.

Polycystic ovary syndrome (PCOS) is characterized by insulin resistance, hyperandrogenism, and frequently increased adiposity. In 2026, PCOS was officially renamed polyendocrine metabolic ovarian syndrome (PMOS), reflecting its multisystem nature and emphasizing metabolic abnormalities as an integral component of the condition. These metabolic disturbances promote an atherogenic lipid profile; consequently, PCOS may represent an early-life marker of increased lifetime cardiometabolic risk.

During the menopausal transition, declining estrogen levels, redistribution of adiposity toward a visceral pattern, and worsening insulin resistance lead to adverse changes in lipid metabolism. Reduced hepatic LDL clearance and altered lipoprotein metabolism contribute to increases in LDL cholesterol and apoB-containing lipoprotein burden. Premature and early menopause are additionally associated with increased cardiovascular risk and may serve as important risk-enhancing factors.

Recognizing female-specific risk factors may enable earlier identification and management of women at increased lifetime cardiovascular risk.

Symposium 30

Neural and Genetic Control of Eating Behavior and Cardiometabolic Health

Sep 12(Sat) 15:40–17:10 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Yang Ha Kim (Ewha Womans University, Republic of Korea)
Min-Jeong Shin (Korea University, Republic of Korea)

15:40–16:00 **Dietary and social determinants of the human microbiome: implications for metabolic health**
Soyoung Kwak (Kyungpook National University, Republic of Korea)

16:00–16:20 **Understanding gene–diet interactions in cardiometabolic health: genetic risk scores and their modification by diet in Korean and UK biobank cohorts**
Sunmin Park (Hoseo University, Republic of Korea)

16:20–16:40 **Interaction effects of genetic variants and dietary intake on the development of metabolic syndrome**
Dayeon Shin (Ewha Womans University, Republic of Korea)

16:40–17:10 **Panel Discussion**
Mogan Gim (Hankuk University of Foreign Studies, Republic of Korea)
Jeong-Min Kim (Seoul National University, Republic of Korea)
Seung-Hwan Lee (The Catholic University of Korea, Republic of Korea)

CURRICULUM VITAE

Soyoung Kwak

Assistant Professor, Kyungpook National University, School of Dentistry,
Department of Microbiology and Immunology, Republic of Korea

Education and Training

2016.02	Korea University, Korea, B.A., Food and Nutrition
2021.02	Korea University, Korea, Ph.D., Biomedical Science and Food Science

Employment and Position

2021.07–2026.01	New York University, USA, Postdoctoral Researcher

Important Publications

1. Kim EK, Oh SY, Choi SY, Kim TL, Lee HJ, Kwak S, Hong SH. Association of MTUS1 with cisplatin response in head and neck squamous cell carcinoma: a retrospective cohort analysis of The Cancer Genome Atlas data. J Yeungnam Med Sci. 2026;43:35
2. Ahn J, Halloran Z, Beggs DB, Wu F, Duan Y, Hwang H, Zhu Z, Flores V, Albergamo V, Kwak S, Kim S, Mei S, Đoàn LN, Yi SS, Li H, Hayes RB. The Food and Microbiome Longitudinal Investigation (FAMiLI) Study: an Asian American (AsA) Enriched Multi-ethnic Environmental Cohort. Cancer Epidemiol Biomarkers Prev. 2026 Jun 29
3. Meng Y, Wu F, Kwak S, Wang C, Usyk M, Freedman ND, Huang WY, Um CY, Gonda TA, Oberstein PE, Li H, Hayes RB, Ahn J. Oral Bacterial and Fungal Microbiome and Subsequent Risk for Pancreatic Cancer. JAMA Oncol. 2025 Nov 1; 11(11):1331-1340
4. Kwak S, Wang C, Usyk M, Wu F, Freedman ND, Huang WY, McCullough ML, Um CY, Shrubsole MJ, Cai Q, Li H, Ahn J, Hayes RB. Oral Microbiome and Subsequent Risk of Head and Neck Squamous Cell Cancer. JAMA Oncol. 2024 Nov 1;10(11):1537-1547
5. Kwak S, Usyk M, Beggs D, Choi H, Ahdoot D, Wu F, Maceda L, Li H, Im EO, Han HR, Lee E, Wu A, Hayes R, Ahn J. Sociobiome - Individual and neighborhood socioeconomic status influence the gut microbiome in a multi-ethnic population in the US. NPJ Biofilms Microbiomes. 2024 Mar 11;10(1):19

Dietary and social determinants of the human microbiome: implications for metabolic health

The human microbiome in increasingly recognized as a dynamic interface between environmental exposures and human health. While host genetics contributes to microbial variation, modifiable factors including diet, social environment, and socioeconomic conditions may play equally important roles in shaping microbial communities. Understanding these population-level determinants may provide insights into biological pathways linking environmental exposures to chronic diseases.

In this lecture, I will present findings from population-based studies examining dietary and social determinants of the gut and oral microbiome. Our studies suggest that dietary acculturation is associated with variation in gut microbial composition, highlighting the potential impact of changing dietary patterns on the microbiome. We also observed greater similarity in gut microbiota profiles among individuals sharing a living environment, suggesting the common environmental exposures, including shared dietary behaviors, may contribute to microbial convergence. Beyond the household environment, socioeconomic factors were also associated with variation in microbial diversity and specific microbial features, supporting the role of broader social conditions in shaping the human microbiome.

We further examined whether socially patterned microbial variation may have implications for disease risk. In prospective studies of the oral microbiome, several microbial species previously reported to be associated with a lower risk of head and neck cancer were less abundant among individuals with lower educational attainment. One of these protective microbial species partially mediated the association between educational attainment and subsequent cancer risk, providing an example of how socially patterned differences in the microbiome may contribute to health disparities.

Together, these findings highlight the human microbiome as a potential biological interface connecting dietary and social environments with downstream health outcomes. A better understanding of these relationships may help clarify how environmental and behavioral factors contribute to metabolic and other chronic diseases and may ultimately inform population-based prevention strategies.

CURRICULUM VITAE

Sunmin Park

Dept. of Food & Nutrition, Hoseo University, Republic of Korea

Education

1992	Ph.D. : Ohio State University, Department of Human Nutrition
1994	Master : Ohio State University, Department of Applied Statistics
1995	AP4 program : Ohio State University, Department of Human Nutrition (Registered dietician in USA)
1987	Master : Ewha Womans University, Department of Food and Nutrition
1985	Bachelor : Ewha Womans University, Department of Food and Nutrition

Academic Experience

2023-2024, 2026-Current	Journal of Medicinal Foods, Chief of editor
2015-Present	Korean Academy of Science and Technology, Member
1996-Present	Hoseo University, Dept. of Food and Nutrition, Professor
1992-1995	Ohio State University, Researcher
2002-2003	Harvard Medical School Joslin Diabetes Center Dept of Cellular and molecular Physiology, Senior Researcher
2000	UCSD Medical School, Dept. of Endocrinology, visiting researcher
1999	Tokyo Pharmacy University, Dept. of Medicinal plant, visiting researcher

Selected Publications

1. Gender-Specific Associations of Diet Quality and Dietary Patterns with Metabolic Syndrome in Korean Adults in KNHANES VI. Nutrition & Metabolism 23:68, 2026.
2. Autophagy-Mitophagy Genetic Risk Score and Gene-Lifestyle Interactions Modulate Chronic Low-Grade Systemic Inflammation in Asian and European Cohorts. Int J Mol Sci 27(7), 3062, 2026.

Understanding gene-diet interactions in cardiometabolic health: genetic risk scores and their modification by diet in Korean and UK biobank cohorts

Cardiometabolic and atherosclerotic disease risk is shaped jointly by common genetic variants and dietary exposure. Unlike rare monogenic mutations, common variants confer probabilistic risk that is often modifiable by diet. This talk presents studies from our group, based on the Korean Genome and Epidemiology Study (KoGES) and UK Biobank, using genetic risk scores (GRS) to examine how polygenic risk interacts with diet and lifestyle across inflammation, lipid metabolism, and skeletal muscle traits relevant to cardiometabolic and atherosclerotic risk. A GRS built from common variants in autophagy-mitophagy pathway genes was associated with chronic low-grade systemic inflammation and metabolic syndrome in both KoGES and UK Biobank (odds ratio 1.91 and 1.62, respectively). This genetic risk was modified by diet. Among individuals with high vegetable and fruit intake, inflammation prevalence remained stable across GRS strata, whereas prevalence rose sharply with GRS under low intake. This pattern extended to atherosclerosis-relevant lipid traits. A GRS for blood pressure interacted with nutrient intake, and GRSs for LDL cholesterol and triglycerides showed diet-dependent effects across KoGES and UK Biobank, relevant to PCSK9 inhibitor response. The framework also extended to skeletal muscle mass, a trait mechanistically linked to insulin resistance and lipid handling. A GRS for low lower-limb muscle mass, including IL5RA and ADAMTSL3 variants, identified older adults at elevated risk, and adequate protein and vitamin D intake attenuated this genetic risk, with ADAMTSL3 offering a mechanistic link through reduced vitamin D3 binding affinity. At the single-variant level, the BDNF Val66Met polymorphism interacted with energy intake and dietary bioactive compounds to influence obesity risk, illustrating gene-diet interaction at a molecular scale. Collectively, these findings show that genetic risk scores capture risk that is modifiable by diet quality, not fixed, across inflammatory, lipid, and muscle phenotypes. This supports extending GRS-based risk stratification into precision nutrition strategies for atherosclerosis prevention.

CURRICULUM VITAE

Dayeon Shin

Associate Professor, Department of Nutritional Science and Food Management,
Ewha Womans University, Republic of Korea

Education and Training

2016.06	Michigan State University, R.D., Dietetic Intern
2015.12	Michigan State University, Ph.D., Human Nutrition
2012.08	Michigan State University, M.S., Human Nutrition
2009.02	Ewha Womans University, B.S., Nutritional Science and Food Management

Employment and Position

2016-2017	University of North Dakota, Assistant Professor (Tenure Track)
2017-2018	Syracuse University, Assistant Professor (Tenure Track)
2019-2026	Inha University, Assistant/Associate Professor (Tenure Track)
2026-Present	Ewha Womans University, Associate Professor (Tenure Track)

Important Publications

1. Kim SH, Kim H, Rebholz CM, Shin D. Association between the inflammatory potential of the diet and incidence of metabolic syndrome in middle-aged adults. Clinical Nutrition. 2026:106697. https://doi.org/10.1016/j.clnu.2026.106697
2. Kim SH, Kim H, Shin D. Adherence to the Dietary Approaches to Stop Hypertension score and cardiovascular disease risk in Korean adults: a prospective cohort study using marginal structural models. American Journal of Clinical Nutrition. 2026;123:101297. https://doi.org/10.1016/j.ajcnut.2026.101297
3. Cha J, Ko SH, Shin D. Causal effect of kimchi intake on HDL-cholesterol levels in middle aged Korean men: a two-sample Mendelian randomization analysis. Genes & Nutrition. 2025;20:23. https://doi.org/10.1186/s12263-025-00785-6

Interaction effects of genetic variants and dietary intake on the development of metabolic syndrome

Metabolic syndrome (MetS) is a growing public health burden among Korean middle-aged adults, shaped by complex interactions between genetic susceptibility and dietary behavior. Because the Korean diet is characteristically carbohydrate-rich, gene-diet interactions may shape cardiometabolic risk in ways not captured by genetic main effects alone. Drawing on the community-based Korean Genome and Epidemiology Study (KoGES), this presentation synthesizes a series of studies that dissect the genetic architecture of MetS across three levels of resolution—single-nucleotide polymorphisms (SNPs), copy number variation (CNV), and polygenic risk scores (PRS)—each examined through the lens of dietary intake.

At the single-variant level, dietary carbohydrate and other Korean food exposures modified the associations between variants in *AMY1*, *SLC35F3*, *VIPR2*, *FHIT*, and bitter-taste-receptor genes, and the incidence of type 2 diabetes and MetS. At the structural level, higher genome-wide CNV burden was associated with earlier MetS onset, and a carbohydrate-dependent interaction between *AMY1A/AMY2B* CNV and *APOA5* rs651821 elevated the odds of hypertriglyceridemia—linking amylase gene dosage, triglyceride-regulating variants, and dietary starch. At the polygenic level, PRS interacted with the oxidative balance score, red and processed meat intake, and seaweed consumption to modify the incidence of MetS, hyperuricemia, and inflammation. Complementary two-sample Mendelian randomization analyses supported causal effects of kimchi on HDL-cholesterol and of coffee on MetS, while metabolomic profiling and machine-learning models integrating genetic and nutritional factors improved MetS prediction and revealed metabolite-nutrient interactions.

Together, these findings demonstrate that the genetic contribution to MetS is consistently diet-contingent, with dietary carbohydrate emerging as a recurring effect modifier in this population. This evidence supports a precision-nutrition framework in which genotype-informed dietary guidance may enhance the prevention of MetS in Korean adults.

Symposium 31

From Cells to Systems: Integrated Control of Cardiometabolic Health

Sep 12(Sat) 15:40–17:10 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Goo Taeg Oh (Ewha Womans University, Republic of Korea)
Young Mi Park (Ewha Womans University, Republic of Korea)

15:40–16:00	**Non-inflammatory function of dendritic cell in the regulation of obesity-associated heart failure** Kae Won Cho (Soonchunhyang University, Republic of Korea)
16:00–16:20	**Hepatic lipotoxicity and fibrotic remodeling in metabolic liver disease** KyeongJin Kim (Inha University, Republic of Korea)
16:20–16:40	**From lipidomics to systems biology: decoding metabolic networks** Guanghou Shui (Chinese Academy of Sciences, China)
16:40–17:10	**Panel Discussion** Yoo-Wook Kwon (Seoul National University, Republic of Korea) Hyue Mee Kim (Chung-Ang University, Republic of Korea) Kyung-Sun Heo (Chungnam National University, Republic of Korea)

CURRICULUM VITAE

Kae Won Cho

Soonchunhyang Institute of Medi-bio Science (SIMS),
Soonchunhyang University, Republic of Korea

Educational Background

2001-2006	Purdue University, Ph.D
1997-1999	Korea University, MA
1993-1997	Korea University, BS

Professional Experience

2023-Present	SoonChunHyang University, Professor
2018-2023	SoonChunHyang University, Associate Professor
2014-2018	SoonChunHyang University, Assistant Professor
2011-2014	University of Michigan Medical School, Research Investigator
2006-2011	University of Michigan Medical School, Postdoctoral Fellow

Research Interests

Immunometabolism, Obesity, Adipose Tissue, Macrophages, Dendritic Cells

Publications

1. Soedonon S, Vo DHN, Chang J, Sharlene, Bayona PW, Kim S, Hong JY, Cho KW (2025) Depletion of myeloid-derived zbtb46+ cells improves glycemic control in obesity via the DPP4/GLP-1 pathways J. Adv. Res. S2090-1232(25)00828-8
2. Yoo HJ, Vo DHN, Kang SE, Lee SJ, Soedono S, Joo EJ, Averia M, Song YW, Cho KW (2025) Targeting the ApoB100-ENO1 interaction with engineered peptdies attenuates atherosclerotic inflammation and plaque progression. Transl. Res.. 285-286;28-39
3. Soedono S, Sharlene S, Vo DHN, Averia M, Rosalie EE, Lee YK, Cho KW (2024) Obese visceral adipose dendritic cells downregulate regulatory T cell development through IL-33. Front. Immunol. 15,1334651
4. Han HS, Kim SG, Kim YS, Jang SH, Kwon Y, Choi D, Huh T, Moon E, Ahn E, Seong JK, Kweon HS, Hwang GS, Lee DH, Cho KW*, Koo SH* (2022) A novel role of CRTC2 in promoting nonalcoholic fatty liver disease. Mol Metab 55: 101402
5. Cho KW, Morris DL, Delproposto JL, Geletka LM, Zamarron B, Marintez-Santibanez G, Meyer KA, Singer K, O'Rourke RW, Lumeng CN (2014) An MHC II-Dependent Activation Loop between Adipose tissue Macrophages and CD4+ T cells Controls Obesity-Induced Inflammation. Cell Reports 9 (2): 605-617

Non-inflammatory function of dendritic cell in the regulation of obesity-associated heart failure

Dendritic cells (DCs) are traditionally recognized for their canonical roles in antigen presentation and the initiation of tissue inflammation. However, their physiological contributions beyond classical immunity—particularly in the context of obesity-induced cardiovascular dysfunction—are not fully understood. This presentation will highlight the emerging, non-inflammatory functions of adipose tissue dendritic cells (ATDCs) and their critical involvement in the regulation of obesity-associated heart failure. Our recent findings reveal that ATDCs possess a profound regulatory capacity that operates independently of classical inflammatory pathways. Specifically, we demonstrate that DCs modulate metabolic and hormonal axes, such as the DPP4/GLP-1 pathway, to influence systemic physiology. In this session, we will expand upon these findings to discuss how these DC-mediated, non-inflammatory signals communicate with the cardiovascular system. Ultimately, this presentation aims to provide a broader perspective on neuro-immune-metabolic crosstalk, suggesting that targeting the non-inflammatory pathways of dendritic cells could offer a novel therapeutic avenue for preventing or treating heart failure in obese patients.

CURRICULUM VITAE

KyeongJin Kim

Associate Professor, Inha University, Republic of Korea

Education and Training

2004.02	Pusan National University, Korea, B.S., Molecular Biology
2006.02	Pusan National University, Korea, M.S., Molecular Biology
2010.02	Pusan National University, Korea, Ph.D., Molecular Biology

Employment and Position

2010-2013	Sungkyunkwan University, Korea, Postdoctoral Fellow
2013-2019	Columbia University, USA, Postdoctoral/Associate Research Scientist
2019-Present	Inha University, Assistant/Associate Professor
2025-Present	Inha University Hospital, Adjunct Professor

Important Publications

1. Jeong Y, Oh AR, Jung YH, Jung KH, Lee S, Carrer M, Lee SB, Valenti L, Pajvani UB, Kim K*. Hepatocyte KCTD17-mediated SERPINA3 inhibition determines liver fibrosis in metabolic dysfunction-associated steatohepatitis. Experimental & Molecular Medicine. 2025 Aug;57(8):1673-1685.
2. Jung YH#, Lee YJ#, Dao T, Jung KH, Yu J, Oh AR, Jeong Y, Gi H, Kim YU, Ryu D, Carrer M, Pajvani UB, Lee SB*, Hong SS*, Kim K*. KCTD17-mediated Ras stabilization promotes hepatocellular carcinoma progression. Clinical and Molecular Hepatology. 2024 Oct;30(4):895-913.
3. Jeong Y#, Oh AR#, Jung YH#, Gi H, Kim YU, Kim K*. Targeting E3 ubiquitin ligases and their adaptors as a therapeutic strategy for metabolic diseases. Experimental & Molecular Medicine. 2023 Oct;55(10):2097-2104.
4. Oh AR#, Jeong Y#, Yu J, Tam DHM, Kang JK, Jung YH, Im SS, Lee SB, Ryu D, Pajvani UB*, Kim K*. Hepatocyte Kctd17 inhibition ameliorates glucose intolerance and hepatic steatosis caused by obesity-induced Chrebp stabilization. Gastroenterology. 2023 Mar;164(3):439-453.
5. Kim K*, Kang JK, Jung YH, Lee SB, Rametta R, Dongiovanni P, Valenti L, Pajvani UB. Adipocyte PHLPP2 inhibition prevents obesity-induced fatty liver. Nature Communications. 2021 Mar 23;12(1):182.

Awards and Honors

2022.02	The Green Ribbon Lecture Award from the Korea Society for Molecular and Cellular Biology

Hepatic lipotoxicity and fibrotic remodeling in metabolic liver disease

Metabolic dysfunction-associated steatohepatitis (MASH) is a leading cause of chronic liver disease. Available therapies show inconsistent results on fibrosis, likely due to heterogeneity in disease trajectory or incomplete understanding of molecular determinants. Here, we identified increased *KCTD17* (potassium channel tetramerization domain-containing protein 17) levels in patients with MASH, and MASH diet-induced rodents, that showed an inverse correlation with expression of serine protease inhibitor a3k (*SERPINA3* or *Serpina3k* in mice). KCTD17 depletion increased *SERPINA3* and reduced liver fibrosis in mice by inhibiting Par2 (protease-activated receptor 2)/TGFb (transforming growth factor beta)-mediated activation of hepatic stellate cells (HSCs). Mechanistically, Kctd17 regulates *Serpina3k* expression by facilitating ubiquitin-mediated degradation of Zbtb7b (zinc finger and BTB domain-containing protein 7b), which in turn diminishes Serpina3k secretion. As such, pharmacological inhibition of Kctd17 effectively reverses MASH-induced liver fibrosis. In summary, these findings underscore the therapeutic potential of targeting KCTD17 for the treatment of MASH-induced liver fibrosis and reveal a previously unrecognized KCTD17-SERPINA3 axis in disease progression. Beyond liver fibrosis, we further discuss the emerging role of SERPINA3 as a hepatokine regulated by obesity, which contributes to glucose homeostasis and may participate in adipose tissue remodeling during obesity.

CURRICULUM VITAE

Guanghou Shui

Principal Investigator, Guangzhou National Laboratory, Guangzhou, China

Education and Training

1989.09-1993.07	Nanjing Tech University, China, Bachelor of Engineering, Industrial Analysis
2000.07-2004.09	National University of Singapore, Singapore, Ph.D., Chemistry
2004.09-2008.03	Department of Biochemistry, National University of Singapore, Singapore, Postdoctoral Fellow, Biochemistry & Lipidomics

Employment and Position

1993.07-2000.06	Southwest Research & Design Institute of Chemical Industry, Ministry of Chemical Industry, China, Senior Research Engineer
2008.08-2012.12	Institute of Life Sciences, National University of Singapore, Singapore, Research Fellow
2013.01-Present	Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, China, Principal Investigator
To be Employed Full-Time	Guangzhou National Laboratory, Guangzhou, China, Principal Investigator

Important Publications

1. Sin Man Lam1,2,11, Zehua Wang1,11, Jin-Wen Song3,11, Yue Shi1,4,11, Wen-Yue Liu,11, Lin-Yu Wan3, Kai-bo Duan6, Gek Huey Chua2, Yingjuan Zhou2, Guibin Wang7, Xiahe Huang1, Yingchun Wang1,4, Fu-Sheng Wang 3,*, Ming-Hua Zheng 8,9,10,*, and Guanghou Shui 1,4,12,*.Non-invasive lipid panel of MASLD fibrosis transition underscores the role of lipoprotein sulfatides in hepatic immunomodulation. Cell Metabolism, 2025, 37(1):69-86.E7. SCI, Q1, IF=37.0, Corresponding Author
2. Chen X#, Li J#, Gao Z, Yang Y, Kuang W, Dong Y, Chua GH, Huang X, Jiang B, Tian H, Wang Y, Huang X, Li Y, Lam SM*, Shui G*. Endogenous ceramide phosphoethanolamine modulates circadian rhythm via neural-glial coupling in Drosophila. National Science Review, 2022, 9(12): nwac148. SCI, Q1, IF=20.7, Corresponding Author
3. Lam SM#, Zhang C#, Wang Z#, Ni Z#, Zhang S, Yang S, Huang X, Mo L, Li J, Lee B, Mei M, Huang L, Shi M, Xu Z, Meng FP, Cao WJ, Zhou MJ, Shi L, Chua GH, Li B, Cao J, Wang J, Bao S, Wang Y, Song JW*, Zhang F*, Wang FS*, Shui G*. A multi-omics investigation on the composition and function of extracellular vesicles along the temporal trajectory of COVID-19. Nature Metabolism, 2021, 3(7):909-922. SCI, Q1, IF=20.8, Corresponding Author
4. Wang X#, Lam SM#, Cao M, Wang T, Wang Z, Yu M, Li B, Zhang H, Ping F, Song G, Feng K, Zhang Q, Xu J, Zhou L, Deng M, Zhai X, Xiao X*, Shui G*. Localized increases in CEPT1 and ATGL elevate plasmalogen phosphatidylcholines in HDLs contributing to atheroprotective lipid profiles in hyperglycemic GCK-MODY. Redox Biology, 2021, 40:101855. SCI, Q1, IF=11.4, Corresponding Author

From lipidomics to systems biology: decoding metabolic networks

The diversity and complexity of lipidome in biological samples have stimulated innovation and improvement in various analytical techniques to meet the needs of countless research and applications in biomedical fields. The development of lipidomics research in the past 20 years is mainly attributed to the advancement of related technologies, especially mass spectrometry, and the emergence and development of various innovative methods. Multi-levels and quantitative lipidomics, and systematic screening of functional lipids have gradually become one of the mainstream directions in biomedical research, providing important clues for the metabolic decoding of multiple human diseases. The decoding of functional lipids is expected to provide new tools for early diagnosis, classification, and prognosis of related diseases, as well as new strategies for early intervention, precision treatment, and exploration of new targets.

Symposium 32

Environmental Cardiovascular Risk

Sep 12(Sat) 15:40-17:10 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Sun Ha Jee (Yonsei University, Republic of Korea)
Oh-Yoen Kim (Dong-A University, Republic of Korea)

15:40-16:00	**Air pollution and cardiovascular disease** Sera Kim (Korea Environment Institute, Republic of Korea)
16:00-16:20	**Microplastics and cardiovascular health: current epidemiological evidence and challenges** Dong-Wook Lee (Seoul National University, Republic of Korea)
16:20-16:40	**Climate change and cardiometabolic disease** Whanhee Lee (Korea University, Republic of Korea)
16:40-17:10	**Panel Discussion** Si-Hyuck Kang (Seoul National University, Republic of Korea) Jong Young Lee (Hallym University, Republic of Korea) Jun Young Hong (Yonsei University, Republic of Korea)

CURRICULUM VITAE

Sera Kim

Research Fellow, Ph.D., Korea Environment Institute, Republic of Korea

Education and Training

2019.08	Korea University, Korea, M.P.H., Environmental Epidemiology
2024.02	Korea University, Korea, Ph.D., Environmental Epidemiology

Employment and Position

2024-2026	Korea University, Korea, Postdoctoral Researcher
2026-Present	Korea Environment Institute, Korea, Research Fellow

Important Publications

1. Kim, S., Kim, Y., Park, J., Kim, R., Lee, W., Bell, M. L., & Lee, J. T. (2026). Leave no one behind: a call to include people with disabilities in climate change and health research. The Lancet Planetary Health, 10(3).
2. Kim, S., & Lee, J. T. (2025). Intersectional inequalities in heat-related mortality risks in South Korea. Environmental Research Letters, 20(10), 104018.
3. Byun, G., Kim, S., Choi, Y., Kim, A., Team, A. C., Lee, J. T., & Bell, M. L. (2024). Long-term exposure to PM2.5 and mortality in a national cohort in South Korea: effect modification by community deprivation, medical infrastructure, and greenness. BMC Public Health, 24(1), 1266.
4. Kim, S., Byun, G., & Lee, J. T. (2023). Association between non-optimal temperature and cardiovascular hospitalization and its temporal variation at the intersection of disability. Science of the Total Environment, 904, 166874.
5. Kim, S., & Lee, J. T. (2022). Short-term exposure to PM10 and cardiovascular hospitalization in persons with and without disabilities: invisible population in air pollution epidemiology. Science of The Total Environment, 848, 157717.

Research Interest

Environmental epidemiology (Air pollution, climate change), Environmental health policy

Air pollution and cardiovascular disease

This talk approaches the topic from the perspective of an environmental epidemiologist, focusing on the evidence linking air pollution to cardiovascular disease and its implications for clinical practice.

Ambient air pollution, primarily fine particulate matter ($PM_{2.5}$), is now the leading environmental contributor to global cardiovascular morbidity and mortality, with the majority of pollution-attributable deaths arising from atherosclerotic cardiovascular disease. Cardiovascular disease occupies a dual position in this literature: it is both a primary outcome of air pollution exposure and a marker of vulnerability that amplifies pollution-related risk. Nationwide cohort studies based on South Korea's National Health Insurance Service database have consistently linked long-term $PM_{2.5}$ exposure to incident ischemic heart disease, myocardial infarction, heart failure, and stroke, while studies show that short-term spikes in $PM_{2.5}$ can also acutely trigger myocardial infarction and stroke, particularly among older adults. Existing studies further show that individuals with pre-existing cardiovascular, renal, or metabolic disease experience substantially larger air pollution-associated risks than the general population. Accountability studies following industrial and power plant closures provide some of the most direct causal evidence available. For example, closure of a coking plant in Pittsburgh was followed by an immediate and sustained reduction in cardiovascular emergency visits.

Despite the 2021 tightening of WHO guidelines, over 90% of the global population continues to live in areas exceeding recommended $PM_{2.5}$ levels, leaving a substantial policy-implementation gap. In this interim, clinicians managing lipid and atherosclerotic diseases are well positioned to identify high-risk patients, incorporate environmental exposure into risk counseling, and partner with environmental epidemiologists to close remaining evidence gaps.

CURRICULUM VITAE

Dong-Wook Lee

Assistant Professor, Seoul National University College of Medicine, Republic of Korea

Education and Training

2007-2013	Seoul National University College of Medicine, Korea, M.D., Medicine
2014-2016	Seoul National University College of Medicine, Korea, M.Sc., Preventive Medicine
2016-2021	Seoul National University College of Medicine, Korea, Ph.D., Preventive Medicine

Employment and Position

2014-2018	Seoul National University College of Medicine, Korea, Resident, Occupational and Environmental Medicine
2018-2021	Seoul National University College of Medicine, Korea, Research Instructor, Preventive Medicine
2021-2023	Seoul National University Hospital, Korea, Clinical Assistant Professor, Public Healthcare Center
2021-2023	Seoul National University, Korea, Adjunct Assistant Professor, Preventive Medicine
2023-2026	Inha University Hospital, Korea, Clinical Assistant Professor, Occupational and Environmental Medicine
2026-Present	Seoul National University, Korea, Assistant Professor, Preventive Medicine

Important Publications

1. Lee DW, et al. Further research on impact of microplastics on children's health is essential to protecting future generations. Clinical and Experimental Pediatrics. 2025;68.
2. Lee DW (Corresponding Author), et al. Long-Term Exposure to Air Pollution and Incident Alzheimer's Disease Among Older Adults: Evidence from a Large-Scale Retrospective Cohort in Korea. Environmental Science and Pollution Research. 2025;32.

Microplastics and cardiovascular health: current epidemiological evidence and challenges

Micro- and nanoplastics (MNPs) are detected not only in environmental media, food, and air, but also in a variety of human biospecimens including blood, placenta, lung, and vascular tissue, and interest in human exposure and its health effects is rapidly increasing. In particular, the 2024 *New England Journal of Medicine* study reporting an association between the detection of MNPs within carotid atherosclerotic plaque and the incidence of cardiovascular events marked an important turning point, extending the discussion beyond its traditional focus on experimental toxicology into the domains of human disease-risk assessment and epidemiological research.

Recently, a growing body of studies has quantified MNPs in diverse biospecimens—including blood, feces, respiratory samples, reproductive tissues, and vascular lesions—and has explored their associations with inflammation, coagulation, cardiovascular disease, the gut microbiota, reproductive health, and neurological health indicators. To date, however, human studies have generally been limited by small sample sizes, a predominance of cross-sectional designs, and challenges relating to the temporal representativeness of exposure, control of confounding, the possibility of reverse causation, and the interpretive gap between preclinical stages and overt clinical disease.

This presentation reviews recent trends in MNPs epidemiology from two perspectives: the assessment of associations with disease and clinical indicators, and biospecimen-based exposure assessment. Biological exposure assessment, in particular, represents the central bottleneck in current MNPs health-effects research. Key challenges include the differing limits of detection across analytical methods such as μ-FTIR, μ-Raman, LDIR, and Py-GC/MS; discrepancies between particle-number and mass concentrations; the difficulty of detecting nanoplastics; losses during sample preparation; the control of external contamination; and the absence of reference materials and standardized protocols.

This presentation discusses the epidemiological evidence accumulated to date, the accompanying methodological challenges, and future directions for MNPs health-effects research.

CURRICULUM VITAE

Whanhee Lee

Associate Professor, School of Health Policy and Management,
Korea University, Republic of Korea

Education and Training

2015.09–2019.02	Seoul National University, Korea, Ph.D., Public Health (Biostatistics)
2013.09–2015.08	Seoul National University, Korea, M.P.H., Public Health (Biostatistics)
2009.03–2013.08	Sungkyunkwan University, Korea, B.S., Statistics

Employment and Position

2026.09–Present	Korea University, Korea, Associate Professor
2025.03–2026.08	Pusan National University, Korea, Associate Professor
2022.09–2025.02	Pusan National University, Korea, Assistant Professor
2021.09–2022.02	Yale University, USA, Postdoctoral Fellow
2020.10–2021.08	Yale University, USA, Postdoctoral Associate

Important Publications

1. Ahn S, Yoon H, Al-Aly Z, Bell M, Kim S, Lee W*. Atmospheric stressors and kidney diseases. Nature Reviews Nephrology. 2026; 22 (487-498).
2. Park J, Kim A, Al-Aly Z, Ebi KL, Kim H, Lee W*. Heat and hospitalization risks among people with disabilities in South Korea. Nature Communications. 2025;16(1).
3. Park D, Kim A, Bell ML, Kim H, Lee W*. Heat and hospital admission via the emergency department for people with intellectual disability, autism, and mental disorders in South Korea: a nationwide, time-stratified, case-crossover study. Lancet Psychiatry. 2024;11(5).

Climate change and cardiometabolic disease

Over the past decade, my research program has quantified how climate-related exposures—ambient heat, ozone, and particulate air pollution—affect cardiometabolic and renal health, drawing on nationwide Korean health insurance data, US Medicare cohorts, and the Multi-Country Multi-City (MCC) Collaborative Research Network.

For cardiometabolic outcomes, our multi-country analyses demonstrated robust associations between extreme temperatures and cause-specific cardiovascular mortality across 27 countries, and showed that heat-related cardiorespiratory mortality is further modified by concurrent air pollution across 482 cities. Nationally, we quantified excess out-of-hospital cardiac arrests attributable to ambient temperature and projected substantial increases in heat-related stroke mortality across East Asian cities under warming scenarios. Turning to ozone, we identified nationwide associations between short-term warm-season ozone exposure and cardiovascular mortality, revealing previously unrecognized high-risk subgroups, and linked ambient ozone to sudden cardiac arrest incidence. For particulate matter, we showed that the onset of cardiovascular and metabolic disease substantially modifies the long-term PM2.5-mortality association, and that Asian dust-derived particles trigger ST-elevation myocardial infarction.

For kidney disease, we demonstrated that long-term exposure to high perceived temperature—and, conversely, to cold winter conditions—increases mortality among patients with chronic kidney disease, and that ozone exposure adversely affects kidney transplant outcomes. Using the US Medicare cohort, we established that long-term air pollution exposure increases the risk of acute kidney injury and of first hospitalization for kidney and urinary system diseases, while short-term exposure carries a quantifiable attributable burden; a six-country analysis further revealed that AKI-related mortality risk fluctuates for up to four weeks after exposure.

Synthesized in our recent review of atmospheric stressors and kidney disease, these findings indicate that cardiometabolic and renal conditions should be treated as priority targets for climate-health adaptation, with high-risk populations explicitly identified to guide clinical and policy responses.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Joint Symposia

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

IHA-KSoLA Joint Symposium

Optimizing Lipid-Lowering Strategies: Facing Challenges in Asian High-Risk Patients

Sep 10(Thu) 14:30-16:00 | Room 3 (3F, Conrad Seoul)

CHAIRPERSONS : Ick Mo Chung (Ewha Womans University, Republic of Korea)
Ade Meidian Ambari (University of Indonesia, Indonesia)

14:30-14:50	**The race to the bottom: intensive LDL-C lowering in very high-risk Asian patients** Ade Meidian Ambari (University of Indonesia, Indonesia)
14:50-15:10	**Combination therapy in high-risk patients: early vs stepwise approach** Han Na Jung (Hallym University, Republic of Korea)
15:10-15:30	**Practical approaches to statin intolerance: from clinical trials to real-world solutions** Jung-Joon Cha (Korea University, Republic of Korea)
15:30-16:00	**Panel Discussion** Kyung An Kim (The Catholic University of Korea, Republic of Korea) Hyeonju Jeong (Hanyang University, Republic of Korea) Hun Jee Choe (Hallym University, Republic of Korea)

CURRICULUM VITAE

Ade Meidian Ambari

University of Indonesia, Indonesia

Current Positions

- President, Indonesian Heart Association, 2025-2028
- Cardiologist specializing in Cardiovascular Prevention and Rehabilitation, National Cardiovascular Center Harapan Kita Hospital, Jakarta, Indonesia
- Faculty Member, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia
- Cardiologist, Jakarta Heart Center, Jakarta, Indonesia
- Cardiologist, Ciputra Cardiac Center Hospital, Jakarta, Indonesia
- Chair, JEDI Committee, Asian Heart Society

Education and Training

- Medical Degree, Faculty of Medicine, Universitas Indonesia, 2001
- Specialist in Cardiology and Vascular Medicine, Universitas Indonesia, 2012
- Ph.D. in Cardiovascular Research, Utrecht University, Utrecht, the Netherlands

Professional and Organizational Experience

- President-Elect, Indonesian Heart Association, 2022-2025
- Chair, Division of Cardiovascular Prevention and Rehabilitation, National Cardiovascular Center Harapan Kita Hospital
- National Board Member, Exercise Is Medicine Indonesia
- Advisory Board Member, Exercise Is Medicine Indonesia National Center
- Vice Secretary-General, Non-Communicable Disease Alliance Indonesia
- Member, Indonesian Heart Foundation

Areas of Expertise

- Cardiovascular prevention
- Cardiac rehabilitation
- Secondary prevention of cardiovascular disease
- Cardiovascular risk-factor management
- Exercise-based cardiac rehabilitation
- External counterpulsation therapy

Professional Fellowships

- Fellow of the Indonesian Heart Association (FIHA)
- Fellow of the ASEAN College of Cardiologists (FAsCC)
- Fellow of the Japanese Circulation Society (FJCS)
- Fellow of the European Society of Cardiology (FESC)

The race to the bottom: intensive LDL-C lowering in very high-risk Asian patients

CURRICULUM VITAE

Han Na Jung

Clinical Assistant Professor, Hallym University, Republic of Korea

Education and Training

2017.02	Korea University, Korea, MD, Medicine
2022.02	University of Ulsan, Korea, MS, Internal Medicine
2026.02	University of Ulsan, Korea, PhD, Internal Medicine
2021.02	Asan Medical Center, Korea, Resident, Internal Medicine
2023.02	Asan Medical Center, Korea, Clinical Fellow, Endocrinology and Metabolism
2024.02	Hallym University Sacred Heart Hospital, Korea, Clinical Fellow, Endocrinology and Metabolism

Employment and Position

2024-	Hallym University Sacred Heart Hospital, Korea, Clinical Assistant Professor

Important Publications

1. Jung HN (co-first), Cho YK, Kim MJ, Moon JY, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH. Defining Obesity Under the Lancet Commission Criteria: Metabolic Syndrome-Anchored Body Fat Percentage Thresholds in Korean Adults. Diabetes Obes Metab. 2026 Jun;28(6):5309-5317.
2. Moon JH, Jung HN (co-first), Kim B, Ko SH, Kwak SH, Han KD, Choi SH. Diabetes in Pregnancy in Korea: Prevalence, Clinical Characteristics, and Postpartum Comorbidities. Diabetes Metab J. 2026 Mar;50(2):280-290.
3. Moon JH, Jung HN (co-first), Kim B, Kim J, Jung YM, Kim HJ, Park JY, Oh TJ, Kwak SH, Han KD, Choi SH. Long-term risk of offspring type 1 and type 2 diabetes following maternal gestational diabetes mellitus: a nationwide birth cohort study with 10-year follow-up. BMC Med. 2026 Mar 6;24(1):237.
4. Jung HN (co-first), Heo JH, Kim MJ, Han KD, Kang JG, Lee SJ. Remnant Cholesterol as an Independent Cardiovascular Risk Factor in Young Adults: An Age-Stratified Cohort Study. J Am Coll Cardiol. 2025 Nov 25;86(21):2047-2058.

Combination therapy in high-risk patients: early vs stepwise approach

Lowering low-density lipoprotein cholesterol (LDL-C) remains the cornerstone of atherosclerotic cardiovascular disease prevention, yet a large proportion of high-risk and very high-risk patients still fail to reach recommended goals under the conventional stepwise paradigm, in which high-intensity statin monotherapy is uptitrated first and a second agent is added only after a target has been missed. This lecture examines whether early, upfront combination therapy should displace that sequential approach in high-risk patients, with particular attention to the Asian context. The rationale for early combination therapy draws on several interrelated considerations, including the cumulative nature of LDL-C exposure, in which time spent above goal adds to atherosclerotic burden; the faster and more reliable goal attainment that combination regimens offer relative to progressive statin uptitration; and the reduction of clinical inertia, the well-documented tendency for intensification to stall once initial therapy is prescribed. Randomized trials establish that statin plus ezetimibe combination is at least as effective as, and better tolerated than, high-intensity statin monotherapy, while observational cohorts consistently link earlier combination with fewer downstream events. These considerations carry added weight in East Asian patients. Distinct pharmacokinetics and genetic transporter variants produce higher statin exposure, comparable LDL-C reduction at lower doses, and a greater burden of dose-dependent intolerance, all of which blunt the incremental value of maximizing statin intensity and favor adding a complementary mechanism early. Importantly, no randomized trial has yet compared upfront combination against stepwise escalation using cardiovascular endpoints; the present case for early combination treatment is therefore mechanistic and surrogate-based rather than a matter of direct outcome evidence. This talk will weigh that evidence gap and outline which patients and clinical settings are most likely to benefit from an early combination strategy in Asian high-risk practice.

CURRICULUM VITAE

Jung-Joon Cha

Clinical Associate Professor, Korea University Anam Hospital,
Korea University College of Medicine, Republic of Korea

Education and Training

	Gwangju Institute of Science and Technology, Korea, Ph.D., Cardiovascular Cell Aging / Biomedical Engineering
	Yonsei University College of Medicine, Korea, Master's Degree, Medicine / Cardiology
2009	Yonsei University Wonju College of Medicine, Korea, M.D., Medicine
2010-2014	Yonsei University Gangnam Severance Hospital, Korea, Residency, Internal Medicine
2018-2019	Yonsei University Severance Hospital, Korea, Fellowship, Cardiology
2019-2020	Yonsei University Severance Hospital, Korea, Interventional Fellowship, Interventional Cardiology

Employment and Position

2024-Present	Korea University Anam Hospital, Korea University College of Medicine, Clinical Associate Professor
2020-2024	Korea University Anam Hospital, Korea University College of Medicine, Clinical Assistant Professor

Important Publications

1. Cha JJ, Shin Y, Joo HJ, et al. Metabolic and Safety Outcomes of Statin-Ezetimibe Versus High-Intensity Statin in Older Patients. J Am Heart Assoc. 2026;15(13):e048711. doi: 10.1161/JAHA.125.048711.
2. Cha JJ, Hong SJ. Diagnosis and Management of Statin-Associated Muscle Symptoms. Korean Circ J. 2025;55:957-968. doi: 10.4070/kcj.2025.0285.
3. Cha JJ, Kim JH, Hong SJ, et al. Safety and efficacy of moderate-intensity statin with ezetimibe in elderly patients with atherosclerotic cardiovascular disease. J Intern Med. 2025;297:400-408. doi: 10.1111/joim.20029.
4. Cha JJ, Hong SJ, Lim S, et al. Comparison of Statin With Ezetimibe Combination Therapy Versus Statin Monotherapy for Primary Prevention in Middle-Aged Adults. Korean Circ J. 2024;54:534-544. doi: 10.4070/kcj.2024.0036.
5. Lee SJ, Cha JJ, Choi WG, et al. Moderate-Intensity Statin With Ezetimibe Combination Therapy vs High-Intensity Statin Monotherapy in Patients at Very High Risk of Atherosclerotic Cardiovascular Disease. JAMA Cardiol. 2023;8(9):853-858. doi: 10.1001/jamacardio.2023.2222.

Awards and Honors

1. Best Presenter, Korean Society of Interventional Cardiology (KSIC), 2025
2. Outstanding Reviewer Award, Korean Circulation Journal, 2024
3. Best Abstract, Korean Vascular Research Working Group (KOVAS), 2024

Practical approaches to statin intolerance: from clinical trials to real-world solutions

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

JAS-KSoLA Joint Symposium

Comprehensive Lipid Management in East Asians: Integrating LDL-C Optimization, Residual Risk, and Genetic Factors

Sep 10(Thu) 16:10-17:40 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Sung Rae Kim (The Catholic University of Korea, Republic of Korea)
Jae Hyoung Park (Korea University, Republic of Korea)

16:10-16:28	**Do East Asians need lower statin intensity? ethnic differences in LDL-C response and safety** Eun Jeong Cho (Chung-Ang University, Republic of Korea)
16:28-16:46	**Why TG is important in Asians?** Daisaku Masuda (Rinku General Medical Center, Japan)
16:46-17:04	**Genetic stratification and treatment response in Korean severe hypertriglyceridemia: a prospective multicenter registry study** Hun Jee Choe (Hallym University, Republic of Korea)
17:04-17:22	**Chylomicronemia: past, present and future** Hiroaki Okazaki (Teikyo University, Japan)
17:22-17:40	**Panel Discussion** Osung Kwon (The Catholic University of Korea, Republic of Korea) Gyu Chul Oh (The Catholic University of Korea, Republic of Korea) Dae Young Cheon (Hallym University, Republic of Korea)

CURRICULUM VITAE

Eun Jeong Cho

Clinical Professor, Chung-Ang University College of Medicine,
Chung-Ang University Gwangmyeong Hospital, Republic of Korea

Education and Training

1995.03-1999.02	Yeungnam University, Korea, M.D., Pharmacy
2000.03-2006.02	Chung-Ang University, Korea, Bachelor's, Medicine
2007.09-2009.08	Chung-Ang University, Korea, Master's, Internal Medicine
2010.09-2012.08	Chung-Ang University, Korea, Ph.D., Internal Medicine
2006.03-2007.02	Chung-Ang University Hospital, Internship, Medicine
2007.03-2011.02	Chung-Ang University Hospital, Residency, Internal Medicine

Employment and Position

2012.03-2015.02	Samsung Medical Center, Korea, Fellowship
2015.03-2019.07	National Cancer Center, Korea, Staff
2019.07-2022.02	Hallym University Medical Center, Korea, Professor
2022.03-Present	Chung-ang University Gwangmyeong Medical Center, Professor

Important Publications

1. Sex-Specific and Time-Dependent Outcomes After TAVR Versus SAVR, A Meta-Analysis of Randomized Trials, JACC Adv. 2026;5:102856.
2. Feasibility of Distal Radial Access in the Use of a Large-Bore Sheath for Percutaneous Coronary Intervention: Real-World Evidence From the Nationwide Cohort, Korean Circ J. 2026 Dec;56(12):e89.
3. Arterial stiffness index and blood pressure phenotypes in patients undergoing percutaneous coronary intervention: prognostic implications for ischemic and bleeding events, Clin Hypertens. 2026 Apr 1;32:e15.
4. Temporal Trend of Cardiovascular Disease Burden Among Cancer Patients Between 2005 and 2022: Nationwide Population-Based Cohort Study in South Korea, Korean Circ J. 2026;56:e53.
5. Local anesthesia alone versus with sedation for transcatheter aortic valve implantation: a systematic review and meta analysis, Clinical Research in Cardiology.

Do East Asians need lower statin intensity? ethnic differences in LDL-C response and safety

Statins remain the cornerstone of lipid-lowering therapy for the prevention of atherosclerotic cardiovascular disease (ASCVD). Current treatment guidelines generally recommend statin intensity according to a patient's cardiovascular risk rather than ethnicity. However, accumulating evidence suggests that East Asian populations may exhibit greater low-density lipoprotein cholesterol (LDL-C) reduction at equivalent statin doses and may have distinct pharmacokinetic and pharmacodynamic responses compared with Western populations. These observations have raised an important clinical question: Do East Asians require lower statin intensity to achieve optimal cardiovascular benefit while minimizing adverse effects?

This lecture will review the evidence regarding ethnic differences in statin response, including mechanisms underlying enhanced LDL-C lowering in East Asians, the influence of genetic and environmental factors, and differences in statin metabolism.

This lecture will address whether lower-intensity statin therapy is sufficient for East Asian patients or whether treatment should remain guided primarily by LDL-C reduction and overall ASCVD risk rather than ethnicity alone. This lecture aims to examine practical recommendations for statin therapy tailored to the East Asian population based on current international guidelines and published research findings.

CURRICULUM VITAE

Daisaku Masuda

Deputy Director, Sumitomo Mitsui Banking Corporation/Rinku General Medical Center, Japan

Education and Training

1999.03	Osaka University School of Medicine, M.D., Medicine
2004.04-2009.03	Osaka University Graduate School of Medicine, Ph.D., Medicine

Employment and Position

1999.04-2000.03	Osaka University Hospital (Department of Molecular Medicine), Medical Intern
2000.04-2003.03	National Hospital Organization Kure Medical Center, Resident (Internal Medicine & Cardiology)
2009.04-2013.10	Osaka University Graduate School of Medicine (Department of Cardiovascular Medicine), Specially Appointed Assistant Professor
2018.04-2026.03	Rinku General Medical Center, Director, Department of Cardiology, Director, Center for Advanced Medical Development, Director, Doctor Support Center, Director, Dementia Care Center, Deputy Director, Health Management Center, Occupational Physician
2026.04-Present	Sumitomo Mitsui Banking Corporation, Deputy Director, Osaka Health Support Center Rinku General Medical Center, Department of Cardiology

Important Publications

1. Masuda D, Kajinami K, Sakayoshi N, Yokota D, Nagamine M, Morikawa-Isogai Y, Yamashita S. Long-term Safety and Efficacy of Bempedoic Acid in Japanese Patients with Hypercholesterolemia: the CLEAR-J LONG. J Atheroscler Thromb. 2025 Nov 28. doi: 10.5551/jat.65947. Online ahead of print.
2. Masuda D, Okada T, Sairyou M, Takafuji K, Ohama T, Koseki M, Nishida M, Sakata Y, Yamashita S. Proteomic Analysis of Human Chylomicron Remnants Isolated by Apolipoprotein B-48 Immunoprecipitation. J Atheroscler Thromb. 2025 Feb 1;32(2):226-238. doi: 10.5551/jat.64920.
3. Fujiyoshi A, Kohsaka S, Hata J, Hara M, Kai H, Masuda D, et al.; Japanese Circulation Society Joint Working Group. JCS 2023 Guideline on the Primary Prevention of Coronary Artery Disease. Circ J. 2024;88(5):763-842. doi: 10.1253/circj.CJ-23-0285.
4. Masuda D. Triglyceride Level and Cardiovascular Risk Reduction Using Pemafibrate Compared with Fibrates. J Atheroscler Thromb.;30(5):429-431. doi: 10.5551/jat.ED216.
5. Okamura T, Tsukamoto K, Arai H, Fujioka Y, Masuda D, et al. Japan Atherosclerosis Society (JAS) Guidelines for Prevention of Atherosclerotic Cardiovascular Diseases 2022. J Atheroscler Thromb. 2024;31(6):641-853. doi: 10.5551/jat.GL2022.

Why TG is important in Asians?

The prevalence of hypertriglyceridemia has been steadily increasing among Asian populations, including Japanese individuals. Despite established interventions for hypercholesterolemia, the incidence of cardiovascular events has been increasing. The co-occurrence of hypercholesterolemia and hypertriglyceridemia is strongly driven by worsening metabolic profiles associated with rising prevalences of obesity and insulin resistance in Asians. In patients with hypertriglyceridemia, the atherogenicity of vessels is correlated with elevated remnant lipoproteins and an increase in small, dense low-density lipoprotein (sdLDL) particles. In the modern clinical era, where low-density lipoprotein cholesterol (LDL-C) can be effectively controlled using potent statins and other novel lipid-lowering therapies, targeting hypertriglyceridemia as a key component of residual cardiovascular risk has become paramount. This session will address the clinical significance of hypertriglyceridemia in Asian populations, focusing on the pathophysiology of atherogenic dyslipidemia, diagnostic evaluation of remnant lipoproteins, and evidence-based therapeutic strategies designed to mitigate residual cardiovascular risk beyond traditional cholesterol lowering.

CURRICULUM VITAE

Hun Jee Choe

Assistant Professor, Hallym University, Republic of Korea

Education and Training

2016.02	Seoul National University, Korea, M.D., Medicine
2023.02	Seoul National University, Korea, Ph.D., Internal Medicine

Employment and Position

2016–2017	Seoul National University Hospital, Korea, Intern doctor
2017–2020	Seoul National University Bundang Hospital, Resident, Internal Medicine
2020–2022	Seoul National University Hospital, Korea, Fellow
2022–2023	Seoul National University Hospital, Korea, Research Fellow
2023–2026	Hallym University Dongtan Sacred Heart Hospital, Clinical Assistant Professor
2026–	Hallym University, Korea, Assistant Professor

Important Publications

1. Choe HJ, Lee JS, Park JY, Lee SA, Park YJ, Chung SS, Park KS. SENP2 regulates UCP1-dependent thermogenesis in brown adipocytes via deSUMOylation of ERRα. Exp Mol Med. 2025 Jun;57(6):1283-1293. DOI: 10.1038/s12276-025-01458-5. PMID: 40579429.
2. Choe HJ*, Rhee EJ*, Won JC*, Park KS, Lee WY, Cho YM. Effects of Patient-Driven Lifestyle Modification Using Intermittently Scanned Continuous Glucose Monitoring in Patients With Type 2 Diabetes: Results From the Randomized Open-label PDF Study. Diabetes Care. 2022;45:2224-2230. doi: 10.2337/dc22-0764
3. Choe HJ*, Moon JH*, Kim W*, Koo BK, Cho NH. Steatotic liver disease predicts cardiovascular disease and advanced liver fibrosis: A community-dwelling cohort study with 20-year follow-up. Metabolism. 2024 Apr;153:155800. doi: 10.1016/j.metabol.2024.155800. Epub 2024 Jan 22. PMID: 38266957.

Genetic stratification and treatment response in Korean severe hypertriglyceridemia: a prospective multicenter registry study

Severe hypertriglyceridemia (HTG), commonly defined as fasting triglycerides ≥885 mg/dL, carries a substantial risk of acute pancreatitis and spans a heterogeneous spectrum. At one end lies familial chylomicronemia syndrome (FCS), a rare autosomal recessive disorder of the lipoprotein lipase pathway; at the other, the far more common multifactorial chylomicronemia syndrome (MCS), in which polygenic susceptibility combines with diabetes, obesity, alcohol, medications, and diet. Distinguishing the two carries real therapeutic consequences, yet prospective Asian data integrating genotype with longitudinal treatment response remain scarce.

The Korean Severe Hypertriglyceridemia (KSHT) Registry is a prospective multicenter cohort established under the Korean Society of Lipid and Atherosclerosis. Adults with severe HTG are enrolled at university-affiliated hospitals nationwide, with systematic collection of clinical phenotype, secondary contributors, targeted genetic testing, medication exposure, and longitudinal triglyceride response. This lecture presents findings from the first 100 patients enrolled.

The cohort was dominated by middle-aged men with extreme triglyceride elevation, a high burden of diabetes and other reversible secondary contributors, and a frequent history of acute pancreatitis, yet without the degree of obesity typical of Western chylomicronemia cohorts. Sequencing of the canonical lipolysis genes identified biallelic variants consistent with FCS in only a small minority, whereas monoallelic pathogenic variants and variants of uncertain significance were considerably more common. Most patients achieved substantial triglyceride reduction within six months, indicating that metabolically reversible disease predominated, although a small subgroup remained in the chylomicronemia range and responses among the genetically defined cases were strikingly divergent.

I will place these observations alongside existing FCS clinical scoring systems, the recently proposed concept of persistent chylomicronemia, and emerging APOC3-directed therapies, and argue that severe HTG is best approached as a dynamic phenotype integrating metabolic context, genotype, and treatment response.

CURRICULUM VITAE

Hiroaki Okazaki

Professor, Department of Internal Medicine, Teikyo University Hospital, Mizonokuchi, Japan

Education and Training

1997	University of Tokyo, Japan, M.D., Faculty of Medicine
2003	Graduate School of Medicine, University of Tokyo, Japan, Ph.D., Department of Metabolic Diseases

Employment and Position

1997-1998	The University of Tokyo Hospital, Tokyo, Japan, Resident
1998-1999	Kanto Central Hospital of the Mutual Aid Association of Public School Teachers, Tokyo, Japan, Resident
2005-2011	Department of Molecular Genetics, University of Texas Southwestern Medical Center at Dallas, USA, Postdoctoral Research Fellow
2011-2014	Molecular Medicinal Sciences on Metabolic Regulation, Department of Diabetes and Metabolic Diseases, Graduate School of Medicine, The University of Tokyo, Japan, Project Associate Professor
2014-2021	Department of Diabetes and Metabolic Diseases, The University of Tokyo Hospital, Japan, Assistant Professor
2022-2025	Division of Endocrinology and Metabolism, Department of Internal Medicine, Jichi Medical University, Japan, Associate Professor
2025-	Department of Internal Medicine, Teikyo University Hospital, Mizonokuchi, Japan, Professor

Important Publications

1. Masaki Tanaka, Sachiko Okazaki, Manabu Takahashi, Tetsuji Wakabayashi, Satoru Takase, Hiroyuki Ishiura, Jun Mitsui, Shoji Tsuji, Shintaro Yanagimoto, Toshimasa Yamauchi, and Hiroaki Okazaki. Vitamin K1 as a screening marker to facilitate the genetic diagnosis of class I familial hypobetalipoproteinemia: A prospective cohort study with a systematic review analysis. J Clin Lipidol. 2026 Jun;S1933-2874(26)00406-X. doi:10.1016/j.jacl.2026.06.013 PubMed PMID: 42399142.

Chylomicronemia: past, present and future

Primary chylomicronemia (PCM) is a disease defined by an accumulation of chylomicrons (CM) in the plasma. PCM devastatingly affects patients' quality of life through strict dietary restriction and, in the face of severe hypertriglyceridemia (HTG), repeated attacks of acute pancreatitis (AP), so-called HTG-induced AP (HTG-AP). As earlier implementation of dietary and drug therapy results in better management of HTG and AP throughout life, early diagnosis is warranted. However, PCM is often misdiagnosed or diagnosed after multiple AP attacks. Increasing awareness of PCM among doctors, medical staff, patients, and the general public will help improve the disease course. The Japan Atherosclerotic Society, in collaboration with the Committee on Primary Dyslipidemia under the Research Program on Rare and Intractable Disease of the Ministry of Health, Labour and Welfare of Japan, has been working on these issues. We proposed simple diagnostic criteria to improve diagnostic rates, implemented ongoing registry research to better understand the natural history of PCM in Japan (PROLIPID study), and published an online guide for patients and their families as well as comprehensive online clinical reviews of PCM for doctors and medical staff. The clinical reviews were published in Japanese and English to better increase disease awareness and facilitate clinical decision-making by covering a wide range of topics, such as pregnancy-induced HTG-AP and PCM caused by anti-GPIHBP1 autoantibodies. Notably, PCM due to genetic causes or autoantibodies is at a higher risk of HTG-AP than PCM without such causes, and, recently, genetic tests for PCM have begun to be covered by Japanese health insurance. With new therapeutic modalities approaching, patients with PCM may increasingly benefit from better screening, early diagnosis, and risk stratification. The past, present, and future of PCM will be discussed along with the presentation of Japanese cases of PCM.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

VAS-KSoLA Joint Symposium

Residual Cardiovascular Risk Beyond LDL-C: The Emerging Role of Lipoprotein(a) in Asia

Sep 11(Fri) 13:45-15:15 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Donghoon Choi (Yonsei University, Republic of Korea)
Kyung Woo Park (Seoul National University, Republic of Korea)

13:45-14:05	**Lipoprotein(a) in Korean patients: real-world data, cardiovascular risk, and clinical implications** Sang Min Park (Eulji University, Republic of Korea)
14:05-14:25	**Lipoprotein(a) and coronary atherosclerosis in Vietnam: real-world data and emerging cardiovascular risk** Anh Tien Hoang (Hue University, Vietnam)
14:25-14:45	**Measuring Lipoprotein(a) in clinical practice: assay challenges, standardization, and interpretation** Sang-Guk Lee (Yonsei University, Republic of Korea)
14:45-15:15	**Panel Discussion** Jaehyun Bae (Hallym University, Republic of Korea) Heung Yong Jin (Jeonbuk National University, Republic of Korea) Jung-Kyu Han (Seoul National University, Republic of Korea)

CURRICULUM VITAE

Sang Min Park

Cardiology Division, Department of Internal Medicine,
Nowon Eulji Medical Center, Eulji University College of Medicine, Seoul, Republic of Korea

Degrees, Training & Employment

2008–2009	Fellowship, Dept. of Cardiology, Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea
2010–2019	Masters and Ph.D., Department of Medicine, the Graduate School of Yonsei University, Seoul, Republic of Korea
2013–2020	Assistant professor, Dept. of Cardiology, Chuncheon Sacred Heart Hospital, Hallym University College of Medicine
2013–Present	Expert, Medical Device Act, Ministry of Food and Drug Safety, government of Republic of Korea
2020–2023	Associate professor, Nowon Eulji Medical Center, Eulji University School of Medicine
2024–Present	Professor, Nowon Eulji Medical Center, Eulji University School of Medicine

Lipoprotein(a) in Korean patients: real-world data, cardiovascular risk, and clinical implications

Lipoprotein(a) [Lp(a)] is a predominantly genetically determined, causal risk factor for atherosclerotic cardiovascular disease (ASCVD). Although its clinical importance is increasingly recognized, real-world evidence in Asian populations has been relatively limited. Recent Korean data provide important insights into the distribution of Lp(a), its association with cardiovascular outcomes, and its implications for clinical practice.

In a large Korean health-screening cohort of 275,430 adults, the median Lp(a) concentration was 18.5 mg/dL, and 12.8% had Lp(a) ≥50 mg/dL. Elevated Lp(a) was independently associated with cardiovascular mortality, including among individuals with different levels of LDL cholesterol. In patients with Lp(a) ≥50 mg/dL, cardiovascular mortality was approximately 80% higher than in those with lower concentrations.[1] More recent Korean data further demonstrate that elevated Lp(a) is not a homogeneous risk state: among 2,355 patients with Lp(a) ≥50 mg/dL, distinct clinical phenotypes showed substantially different rates of major adverse cardiovascular events despite similar Lp(a) concentrations.[2] These findings suggest that Lp(a) should be interpreted within the context of overall cardiovascular risk rather than as an isolated biomarker.

The Korean Society of Lipid and Atherosclerosis Lp(a) Task Force recently proposed a three-tier classification: normal (<30 mg/dL), borderline high (30–49 mg/dL), and high (≥50 mg/dL), while emphasizing standardized, isoform-insensitive measurement.[3] Current international guidance recommends measuring Lp(a) at least once in adulthood.[4] In patients with elevated Lp(a), aggressive management of modifiable risk factors—particularly LDL-C, blood pressure, diabetes, smoking, and lifestyle—remains the cornerstone of risk reduction. Meanwhile, antisense oligonucleotides and small-interfering RNA therapies targeting Lp(a) are being evaluated in late-stage clinical trials.

In conclusion, Lp(a) has emerged as an important component of residual cardiovascular risk in Korean patients. Integrating Lp(a) measurement with comprehensive risk assessment may facilitate earlier identification of high-risk individuals and provide a foundation for personalized preventive strategies.

Table 1. Practical interpretation of Lp(a) in Korean patients

Lp(a) level	Interpretation	Clinical implication
<30 mg/dL	Normal	Routine ASCVD risk assessment
30–49 mg/dL	Borderline high	Consider overall risk and risk-enhancing factors
≥50 mg/dL	High	Intensify global ASCVD risk-factor management; consider family testing
≥100 mg/dL	Very high	Particularly high residual risk; aggressive risk-factor modification and consideration of future Lp(a)-lowering therapies

The proposed Korean classification is based on the 2026 Korean Society of Lipid and Atherosclerosis position paper.[3]

References: 1. Kim BJ, et al. Lipoprotein(a)-related cardiovascular and all-cause mortalities in Korean adults. Eur J Prev Cardiol. 2023;30(4):308-317. 2. Joo HJ, Hong SJ, Yu CW, Shin SY, Kim EJ. Phenotypic clustering identifies heterogeneous cardiovascular risk among patients with elevated lipoprotein(a). Sci Rep. 2026;16 (1):24477. 3. Jang Y, Lee JH, Lee SG, Choe HJ, Park SM, Jeong IK, et al. A Position Paper on Lipoprotein(a) From the Lipoprotein(a) Task Force of the Korean Society of Lipid and Atherosclerosis: Current Evidence, Clinical Applications, and Future Directions. J Lipid Atheroscler. 2026;15(1):2-25. 4. Kronenberg F, Mora S, Stroes ESG, et al. Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: a European Atherosclerosis Society consensus statement. Eur Heart J. 2022;43(39):3925-3946.

CURRICULUM VITAE

Anh Tien Hoang

Professor of Cardiology, Hue University of Medicine and Pharmacy, Vietnam
Vice President of Vietnam Atherosclerosis Society, Vietnam
Professor, Director of Cardiovascular Center,
Hue University of Medicine and Pharmacy Hospital, Vietnam

Education and Training

1997-2003	Hue University of Medicine and Pharmacy, Vietnam, M.D., General Medicine
2004-2006	Hue University of Medicine and Pharmacy, Vietnam, M.Sc., Internal Medicine
2007-2011	Hue University of Medicine and Pharmacy, Vietnam, Ph.D., Cardiology
2015	Hue University of Medicine and Pharmacy, Vietnam, Assoc. Professor, Cardiology
2025	Hue University of Medicine and Pharmacy, Vietnam, Professor, Cardiology

Employment and Position

2004-Present	Hue University of Medicine and Pharmacy, Lecturer → Senior Lecturer, Dept. of Internal Medicine
2010-2022	Hue Univ. of Medicine and Pharmacy Hospital, Deputy Head, Cardiology Department
2019-Present	Hue University of Medicine and Pharmacy, Deputy Head, Dept. of Internal Medicine
2022-Present	Hue Univ. of Medicine and Pharmacy Hospital, Head, Cardiology Department
Present	Hue Univ. of Medicine and Pharmacy Hospital, Vice Director, Cardiovascular Centre

Important Publications

1. Dang HNN, Luong TV, Pham NC, Tran HTN, Hoang AT (corresponding), Ho BA, Doan TC, Nguyen HM. Double-Kissing Crush Versus Provisional Stenting for Coronary Bifurcations: A 1-Year Follow-up Study in Vietnam. J Soc Cardiovasc Angiogr Interv (JSCAI). 2025. doi:10.1016/j.jscai.2024.102500. [Q2 · Corresponding author]

Lipoprotein(a) and coronary atherosclerosis in Vietnam: real-world data and emerging cardiovascular risk

Background: Low-density lipoprotein cholesterol (LDL-C) is the primary causal driver of atherosclerotic cardiovascular disease (ASCVD), yet substantial residual risk persists despite optimal LDL-C control. Lipoprotein(a) [Lp(a)], a genetically determined LDL-like particle, is an independent causal risk factor for coronary artery disease, myocardial infarction, ischemic stroke, and calcific aortic valve stenosis. Current ESC/EAS, AHA/ACC, and NLA guidelines recommend at least one lifetime Lp(a) measurement and recognize elevated Lp(a) as a risk-enhancing factor. Although East Asians generally have lower Lp(a) levels than Western populations, elevated Lp(a) remains associated with ASCVD risk. Vietnam-specific evidence is limited.

Objective: To summarize emerging Vietnamese real-world data on Lp(a), compare them with regional Asian evidence, and evaluate their implications for current lipid management guidelines.

Real-world Vietnamese data. In patients undergoing coronary angiography (n=138), Lp(a) ≥30 mg/dL independently predicted severe coronary stenosis (Gensini >40), with an optimal cut-off of 30.6 mg/dL (AUC 0.869). In acute myocardial infarction (n=199), Lp(a) ≥ 50 mg/dL was associated with NSTEMI presentation. Among 165 asymptomatic cardiologists screened at the VAS Congress 2024, 12.1% had Lp(a) ≥125 nmol/L, including 8% who had already achieved LDL-C and non-HDL-C targets, highlighting persistent residual risk.

Regional and guideline context. Korean and pan-Asian studies similarly demonstrate LDL-C-independent associations between elevated Lp(a) and cardiovascular mortality. These findings support universal one-time Lp(a) testing, consistent with ESC/EAS recommendations.

Conclusions: Vietnamese real-world evidence confirms elevated Lp(a) as an independent marker of coronary atherosclerosis severity and cardiovascular risk, consistent with Asian and international data. Routine lifetime Lp(a) measurement, intensified risk-factor control, and establishment of standardized national Lp(a) registries should be prioritized as selective Lp(a)-lowering therapies become available.

CURRICULUM VITAE

Sang-Guk Lee

Professor, Yonsei University College of Medicine, Republic of Korea

Education and Training

2005.02	Yonsei University, Korea, M.D, Medicine
2017.08	Yonsei University, Korea, Ph.D, Laboratory Medicine

Employment and Position

2013-2014	Yonsei University College of Medicine, Clinical and Research Fellow
2014-2020	Yonsei University College of Medicine, Assistant Professor
2020-2024	Yonsei University College of Medicine, Associate Professor
2025-	Yonsei University College of Medicine, Professor

Important Publications

1. Lee KS, Lee YH, Lee SG. Alanine to glycine ratio is a novel predictive biomarker for type 2 diabetes mellitus. Diabetes, obesity & metabolism. 2024;26(3):980-8.
2. Yun SY, Rim JH, Kang H, Lee SG, Lim JB. Associations of LDL Cholesterol, Non-HDL Cholesterol, and Apolipoprotein B With Cardiovascular Disease Occurrence in Adults: Korean Genome and Epidemiology Study. Annals of laboratory medicine. 2023;43(3):237-43.
3. Ahn S, Lee SH, Chung KS, Ku NS, Hyun YM, Chun S, et al. Development and validation of a novel sepsis biomarker based on amino acid profiling. Clinical nutrition. 2021;40(6):3668-76.
4. Kim SR, Lee SG, Kim SH, Kim JH, Choi E, Cho W, et al. SGLT2 inhibition modulates NLRP3 inflammasome activity via ketones and insulin in diabetes with cardiovascular disease. Nature communications. 2020;11(1):2127.
5. Nam HS, Ha J, Ji D, Kwon I, Lee HS, Han M, et al. Elevation of the Gut Microbiota Metabolite Trimethylamine N-Oxide Predicts Stroke Outcome. Journal of stroke. 2019;21(3):350-2.

Measuring Lipoprotein(a) in clinical practice: assay challenges, standardization, and interpretation

The LPA gene contains a highly variable kringle IV type 2 (KIV2) domain that can be repeated more than 40 times, resulting in marked apo(a) size polymorphism with molecular weights ranging from approximately 250 to 800 kDa. A variety of immunoassay-based Lp(a) tests are commercially available. Most antibodies used in immunoturbidimetric or nephelometric assays are polyclonal and recognize epitopes within the repetitive KIV2 region of apo(a). Because these antibodies may bind multiple identical epitopes on a single apo(a) molecule, direct molar quantification of Lp(a) presents a unique analytical challenge.

Although most commercially available Lp(a) assays use antibodies that recognize the repetitive KIV2 structure and are therefore intrinsically apo(a) isoform dependent, analytical performance is determined not only by antibody specificity but also by assay calibration. As demonstrated by recent investigations, assays employing multiple independent calibrators with representative apo(a) isoform distributions can substantially reduce isoform-dependent bias, whereas assays using serial dilutions from a single calibrator are more susceptible to underestimating high Lp(a) concentrations associated with low-molecular-weight apo(a) isoforms and overestimating low concentrations associated with high-molecular-weight isoforms. Consequently, many contemporary assays achieve clinically acceptable isoform independence despite using antibodies targeting repetitive KIV2 region by employing multiple independent calibrators.

The availability of universally accessible reference materials assigned by molar-based reference measurement procedures will be essential for further harmonization and standardization of Lp(a) measurement. Although the development of antibodies recognizing unique, non-repetitive apo(a) epitopes remains technically challenging, advances in calibration strategies and use of reference materials for calibrator value assignment demonstrate that clinically reliable molar quantification is achievable even with existing antibody technologies. Ultimately, the goal of standardized molar reporting of Lp(a) should remain a priority for the laboratory medicine community.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

TSLA-KSoLA Joint Symposium

Contemporary Management of Residual Cardiovascular Risk in Asia

Sep 12(Sat) 08:50-10:20 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : **Sang-Hyun Kim** (Seoul National University, Republic of Korea)
Chin-Chou Huang (Taipei Veterans General Hospital, Taiwan)

08:50-09:10	**The emerging role of ApoB and triglyceride-rich lipoproteins** **Sin Yung Woo** (Konyang University, Republic of Korea)
09:10-09:30	**Beyond LDL-C: targeting inflammation and lipoprotein(a) to address residual cardiovascular risk in Asia** **Ping-Yen Liu** (National Cheng Kung University, Taiwan)
09:30-09:50	**Cardiometabolic residual risk in Asia: diabetes, metabolic dysfunction, and vascular aging** **Minkwan Kim** (Yonsei University, Republic of Korea)
09:50-10:20	**Panel Discussion** **Young-Sang Lyu** (Chosun University, Republic of Korea) **Hong Seok Lim** (Ajou University, Republic of Korea) **Hansaem Jung** (Jeong Hansaem Internal Medicine Clinic, Republic of Korea)

CURRICULUM VITAE

Sin Yung Woo

Assistant Professor, Konyang University College of Medicine, Republic of Korea

Education and Training

2022.02	Konyang University, Korea, M.D., Internal Medicine

Employment and Position

2022-2023	Yonsei University Severance Hospital, Clinical Fellow
2023-2026	Konyang University, Clinical Assistant Professor
2026-	Konyang University, Assistant Professor

The emerging role of ApoB and triglyceride-rich lipoproteins

Despite effective low-density lipoprotein cholesterol (LDL-C) reduction with statin therapy, significant residual cardiovascular risk persists. Emerging evidence highlights a major paradigm shift toward Apolipoprotein B (ApoB) and triglyceride-rich lipoproteins (TRLs), along with their remnants, as primary drivers of atherogenesis. TRL remnants deliver up to fourfold more cholesterol per particle to the arterial wall compared to LDL, promoting endothelial dysfunction, oxidative stress, and pro-coagulant cascades.

Discordance analysis demonstrates that elevated ApoB or non-HDL-C levels predict myocardial infarction and atherosclerotic cardiovascular events far more reliably than LDL-C alone, particularly as achieved LDL-C levels decline. Biologically, ApoB acts as a direct ligand for scavenger receptors (SR-A, LOX-1) on macrophages and vascular cells, accelerating plaque development. Concurrently, Apolipoprotein C-III (ApoC-III) regulates TRL clearance by inhibiting lipoprotein lipase (LPL) and triggering vascular inflammation via monocyte activation and NLRP3 inflammasome pathways. ApoC-III elevation is closely linked to insulin resistance, type 2 diabetes, and diabetic microvascular complications.

Clinically, these insights necessitate a re-evaluation of hypertriglyceridemia management, supporting lower thresholds for optimal triglyceride levels and establishing explicit ApoB treatment targets. Furthermore, therapeutic innovation has ushered in a new era: novel GalNAc-conjugated antisense oligonucleotides (ASOs) and small interfering RNAs (siRNAs)—such as Olezarsen, Plozasiran, and Zodasiran—effectively target ApoC-III and ANGPTL pathways. These targeted interventions offer potent, infrequent dosing schedules that substantially suppress remnant lipoproteins. Shifting clinical focus from traditional LDL-C metrics to ApoB and TRL-mediated pathways provides a necessary framework for addressing residual atherogenic risk.

CURRICULUM VITAE

Ping-Yen Liu

Professor / Attending Physician,
National Cheng Kung University / National Cheng Kung University Hospital, Taiwan

Education and Training

1987-1994	Kaohsiung Medical University, Kaohsiung, Taiwan, M.D., Internal Medicine
2001-2005	National Cheng Kung University, Tainan, Taiwan, Ph.D., Institute of Clinical Medicine

Employment and Position

2019-2023	Institute of Clinical Medicine, National Cheng Kung University, Tainan, Taiwan, Director
2019-2023	Clinical Medicine Research Center, National Cheng Kung University Hospital, Tainan, Taiwan, Director
2021-2024	Taiwan Society of Lipids & Atherosclerosis, Taipei, Taiwan, Secretary General
2023-2026	Internal Medicine, National Cheng Kung University Hospital, Tainan, Taiwan, Director
2024-Present	Taiwan Society of Lipids & Atherosclerosis, Taipei, Taiwan, President

Important Publications

1. Huang MS, Wang CS, Chiang JH, Liu PY, Tsai WC: Automated Recognition of Regional Wall Motion Abnormalities Through Deep Neural Network Interpretation of Transthoracic Echocardiography. Circulation 2020/10;142(16):1510-1520.
2. Chang HY, Hsu HC, Fang YH, Liu PY*, Liu YW: Empagliflozin attenuates doxorubicin-induced cardiotoxicity by inhibiting the JNK signaling pathway. Biomedicine & Pharmacotherapy 2024/07;176:116759 (co-Corresponding author)
3. Chen PW, Tseng BY, Yang ZH, Yu CH, Lin KT, Chen JN, Liu PY*: Deep learning model for diagnosis of venous thrombosis from lower extremity peripheral ultrasound imaging. iScience 2024/11;27(12):111318.

Beyond LDL-C: targeting inflammation and lipoprotein(a) to address residual cardiovascular risk in Asia

Despite remarkable advances in LDL-C lowering therapies, a substantial burden of residual cardiovascular risk persists in patients with established or high-risk atherosclerotic cardiovascular disease (ASCVD). Beyond traditional lipid management, chronic vascular inflammation and elevated lipoprotein(a) [Lp(a)] have emerged as two major and interrelated drivers of residual risk, contributing to atherosclerosis progression, plaque instability, and thrombotic events. Recent evidence has further highlighted the synergistic interaction between inflammatory pathways and Lp(a), supporting a more comprehensive approach to cardiovascular risk reduction.

In Asia, increasing awareness of Lp(a) has led to important regional initiatives aimed at improving early detection and personalized prevention. The Taiwan Society of Lipid and Atherosclerosis (TSLA) has actively promoted a "Once-in-a-Lifetime Lp(a) Testing" strategy in alignment with international recommendations, while fostering regional consensus on standardized measurement, clinical interpretation, and integration of Lp(a) into routine cardiovascular risk assessment. These efforts represent an important step toward precision cardiovascular medicine across Asian populations.

This lecture will review the evolving concepts of residual cardiovascular risk beyond LDL-C, focusing on the interplay between inflammation and Lp(a), emerging clinical evidence, and practical implications for patient management. Particular emphasis will be placed on the rapidly advancing landscape of RNA-based Lp(a)-lowering therapies—including antisense oligonucleotides and small interfering RNA (siRNA) agents—which have demonstrated unprecedented reductions in circulating Lp(a) concentrations and may redefine future preventive strategies. Finally, perspectives on implementing comprehensive residual risk assessment and personalized lipid management in Asia will be discussed, highlighting opportunities for regional collaboration to further improve cardiovascular outcomes.

CURRICULUM VITAE

Minkwan Kim

Assistant Professor, Yonsei University College of Medicine, Republic of Korea

Education and Training

2010.02	Dong-A University, Korea, M.D., Medicine
2014.02	Graduate School, Dong-A University, Korea, M.S., Internal Medicine
2018.02	Graduate School, Dong-A University, Korea, Ph.D., Internal Medicine
2015.02	Dong-A University, Korea, Internal Medicine
2020.02	Seoul National University Hospital, Korea, Cardiology Fellowship
2026.02	University of Pennsylvania, USA, Visiting Scholar

Employment and Position

2020-Present	Yongin Severance Hospital / Yonsei University College of Medicine, Korea, Assistant Professor

Important Publications

1. Kim M, Tavolinejad H, Sarmiento Bustamante M, Segers P, Neirynck RE, De Meyer T, De Buyzere ML, Rietzschel E, Chirinos JA. Proteomic Risk Score for Prediction of Incident Hypertension. Hypertension. 2026;83(4):e26321. (First author)
2. Kim M, Yoo J, Baik M, Kim J, Jung IH. Clinical Usefulness of Left Ventricular Global Longitudinal Strain as a Predictor of Prognosis in Patients With Acute Ischemic Stroke (GLS-STROKE Study). J Am Heart Assoc. 2025:e042800. (First author)

Cardiometabolic residual risk in Asia: diabetes, metabolic dysfunction, and vascular aging

Despite decades of progress in lowering LDL-cholesterol and blood pressure, a substantial burden of atherosclerotic cardiovascular disease persists among treated patients. This residual cardiovascular risk is increasingly cardiometabolic in origin, and nowhere is this more consequential than in Asia, where more than half of global cardiovascular deaths now occur. This lecture examines three interrelated drivers of cardiometabolic residual risk in Asian populations: diabetes, metabolic dysfunction, and vascular aging.

Asia is the epicenter of the global diabetes epidemic. Asian patients develop diabetes at younger ages and at lower body-mass index than Western counterparts, extending lifetime glycemic exposure. Extended follow-up of UKPDS demonstrates a near-lifelong legacy effect of early glycemic control, whereas cardiovascular outcome trials of SGLT2 inhibitors and incretin-based therapies, including SELECT and SURPASS-CVOT, with consistent benefits in Asian subgroups, have made dysglycemia and adiposity modifiable components of residual risk.

Metabolic dysfunction extends beyond glucose. The redefinition of fatty liver disease as MASLD explicitly anchors hepatic steatosis to cardiometabolic risk; MASLD, frequently a lean phenotype in Asians, independently predicts cardiovascular events. Insulin-resistance measures such as the triglyceride-glucose index offer pragmatic risk stratification, and triglyceride-rich lipoproteins and remnant cholesterol mechanistically link metabolic dysfunction to atherogenesis, although PROMINENT cautions that triglyceride lowering without apoB reduction confers no outcome benefit.

Vascular aging integrates these cumulative exposures. Early vascular aging, or accelerated large-artery stiffening relative to chronological age, is both a consequence of cardiometabolic disease and an independent predictor of events beyond conventional risk factors. Large-artery stiffness impairs ventricular-arterial coupling and microvascular integrity, and in Asian practice it can be readily assessed with standardized brachial-ankle pulse wave velocity and the cardio-ankle vascular index.

The lecture concludes with a practical framework for the contemporary management of residual cardiovascular risk in Asia: early and durable glycemic control, systematic hepatic and metabolic phenotyping, vascular age assessment for refined risk stratification, and emerging inflammation- and lipid-targeted therapies, emphasizing opportunities and evidence gaps specific to Asian populations.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

MSA-KSoLA Joint Symposium

From Diversity to Action: Ethnicity, Genetics, and Closing the Treatment Gap in Asian Dyslipidemia

Sep 12(Sat) 08:50-10:20 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Sang Ho Jo (Hallym University, Republic of Korea)
Hack-Lyoung Kim (Seoul National University, Republic of Korea)

08:50-09:10	**Dyslipidemia across ethnicities in Malaysia: who is at risk, and who is being left behind?** Nor Ashikin Md Sari (University of Malaya, Malaysia)
09:10-09:30	**Genetic architecture of dyslipidemia and cardiovascular risk in East Asians: from monogenic variants to polygenic risk** Soo Heon Kwak (Seoul National University, Republic of Korea)
09:30-09:50	**Epidemiological trends of dyslipidemia in Korea and their implications for Asian populations** Hokyou Lee (Yonsei University, Republic of Korea)
09:50-10:20	**Panel Discussion** Ji Hyun Kim (The Catholic University of Korea, Republic of Korea) Joon Ho Moon (Seoul National University, Republic of Korea) Junghwan Park (Hanyang University, Republic of Korea)

CURRICULUM VITAE

Nor Ashikin Md Sari

Consultant Cardiologist / Doctor, University of Malaya, Malaysia

Education and Training

2002	University of Malaya, MBBS, Medicine
2010	University of Malaya, MMED, Internal Medicine

Employment and Position

2002-2010	University Malaya, Lecturer
2010-Present	University Malaya, Senior Lecturer

Important Publications

1. Ding, C. C. A., Dokos, S., Bakir, A. A., Zamberi, N. J., Liew, Y. M., Chan, B. T., Md Sari, N. A., Avolio, A., & Lim, E. (2024). Simulating impaired left ventricular-arterial coupling in aging and disease: a systematic review. Biomedical Engineering Online, 23(1), 24. https://doi.org/10.1186/s12938-024-01206-2
2. Ooi, J. H., Lim, R., Seng, H., Tan, M. P., Goh, C. H., Lovell, N. H., Argha, A., Beh, H. C., Md Sari, N. A., & Lim, E. (2024). Non-invasive parameters of autonomic function using beat-to-beat cardiovascular variations and arterial stiffness in hypertensive individuals: a systematic review. Biomedical Engineering Online, 23(1), 23. https://doi.org/10.1186/s12938-024-01202-6
3. Chuah, S. H., Sari, N. A. M., Chew, B. T., Tan, L. K., Chiam, Y. K., Chan, B. T., Lim, E., Aziz, Y.F.A. & Liew, Y. M. (2020). Phenotyping of hypertensive heart disease and hypertrophic cardiomyopathy using personalized 3D modelling and cardiac cine MRI. Physica Medica, 78, 137-149. https://doi.org/10.1002/jmri.28915
4. Chuah, S. H., Tan, L. K., Md Sari, N. A., Chan, B. T., Hasikin, K., Lim, E., Ung, N.M., Abdul Aziz, Y.F., Jayabalan, J.& Liew, Y. M. (2024). Remodeling in Aortic Stenosis With Reduced and Preserved Ejection Fraction: Insight on Motion Abnormality Via 3D+ Time Personalized LV Modeling in Cardiac MRI. Journal of Magnetic Resonance Imaging, 59(4), 1242-1255. https://doi.org/10.1016/j.ejmp.2020.08.022
5. Low, S. C., Sari, N. A. M., Tan, C. Y., Ahmad-Annuar, A., Wong, K. T., Law, W. C., ... Goh, K. J. (2021). Hereditary transthyretin amyloidosis in multi-ethnic Malaysians. Neuromuscular Disorders, 31(7), 642-650. doi: 10.1016/j.nmd.2021.03.008

Awards and Honors

1. Fellowship National Heart Association Malaysia (FNHAM)
2. Fellowship of ASEAN Federation of Cardiology (FAsCC)

Research Interest

Lipids, Cardiovascular Magnetic Resonance, Heart Failure, Hypertension, Cardiac amyloidosis

Dyslipidemia across ethnicities in Malaysia: who is at risk, and who is being left behind?

CURRICULUM VITAE

Soo Heon Kwak

Professor, Seoul National University, Republic of Korea

Education and Training

1996-2002	Seoul National University, College of Medicine, M.D., Medicine
2009-2012	Seoul National University, Graduate School, Ph.D., Internal Medicine

Employment and Position

2015-2021	Seoul National University Hospital, Associate Professor
2021-	Seoul National University Hospital, Professor

Important Publications

1. Lee H, Christie HE, Lee JS, Kwon WI, Moon MK, Choi SH, Lee DH, Lee S, Lee H, Kho M, Boscolo F, Dalla Man C, Cho NH, Choi J, Lee J, Park J, Jo Y, Lee S, Vella A, Kwak SH, Kim JI, Park KS. The Gain-of-Function GLP1R R131Q Variant Preserves β-Cell Function and Enhances Response to GLP-1 Receptor Agonists. Diabetes Care. 2026 Aug 6:dc260662. doi: 10.2337/dc26-0662. Epub ahead of print. PMID: 42561183.
2. Choi J, Lee H, Kuang A, et al. Genome-Wide Polygenic Risk Score Predicts Incident Type 2 Diabetes in Women With History of Gestational Diabetes. Diabetes Care. Published online June 28, 2024. doi:10.2337/dc24-0022
3. Lee H, Choi J, Kim JI, et al. Higher Genetic Risk for Type 2 Diabetes Is Associated With a Faster Decline of β-Cell Function in an East Asian Population. Diabetes Care. 2024;47(8):1386-1394. doi:10.2337/dc24-0058
4. Kwak SH, Hernandez-Cancela RB, DiCorpo DA, et al. Time-to-Event Genome-Wide Association Study for Incident Cardiovascular Disease in People With Type 2 Diabetes. Diabetes Care. 2024;47(6):1042-1047. doi:10.2337/dc23-2274
5. Kwak SH, Srinivasan S, Chen L, et al. Genetic architecture and biology of youth-onset type 2 diabetes. Nat Metab. 2024;6(2):226-237. doi:10.1038/s42255-023-00970-0

Genetic architecture of dyslipidemia and cardiovascular risk in East Asians: from monogenic variants to polygenic risk

Dyslipidemia results from a spectrum of genetic susceptibility ranging from highly penetrant monogenic disorders to the cumulative effects of numerous common variants. Understanding this continuum is particularly important in East Asian populations, whose allele frequencies, linkage disequilibrium patterns, and genetic architecture may differ substantially from those of European populations that have dominated prior genetic studies.

Familial hypercholesterolemia (FH) is a prototypic monogenic dyslipidemia, most commonly caused by pathogenic variants in *LDLR*, *APOB*, or *PCSK9*. Early recognition through clinical assessment, genetic testing, and cascade screening enables timely, intensive LDL-cholesterol lowering and may substantially reduce lifelong atherosclerotic cardiovascular disease risk. Korean FH data indicate considerable genetic and phenotypic heterogeneity, supporting the need for population-specific diagnostic and screening strategies.

Beyond monogenic disease, large-scale East Asian genomic studies have expanded the understanding of common and rare variants associated with lipid traits. A meta-analysis of Korean and Japanese biobank data involving 288,137 East Asian individuals identified numerous previously unrecognized associations with lipid and other metabolic traits. Rare functional variants, although contributing relatively little to population-level trait variance, can have large effects in individual carriers and may alter risk estimates based solely on common-variant polygenic scores. Many of these variants are uncommon or absent in European populations, emphasizing the limitations of directly transferring European-derived genetic evidence to East Asian populations.

Polygenic risk scores offer an opportunity to refine cardiovascular risk stratification among individuals without a pathogenic monogenic variant and to explain phenotypic variability among FH carriers. Their clinical implementation, however, requires ancestry-matched discovery data, robust validation, clear interpretation, and integration with LDL-cholesterol levels, family history, and conventional cardiovascular risk factors.

This presentation will review the genetic spectrum of dyslipidemia in East Asians and discuss how monogenic testing and polygenic risk assessment may be incorporated into more precise, equitable prevention of cardiovascular disease.

CURRICULUM VITAE

Hokyou Lee

Associate Professor of Preventive Medicine, Yonsei University, Republic of Korea

Education and Training

2008.05	University of California, Berkeley, CA, USA, BS, Chemistry
2013.02	Yonsei University College of Medicine, Seoul, Korea, MD, Medicine
2022.02	Yonsei University, Seoul, Korea, PhD, Preventive Medicine

Employment and Position

2013-2018	Yonsei University Severance Hospital, Intern/Resident, Internal Medicine
2018-2022	Yonsei University College of Medicine, Fellow, Preventive Medicine
2022-2024	Yonsei University College of Medicine, Assistant Professor, Preventive Medicine
2024-Present	Yonsei University College of Medicine, Associate Professor, Preventive Medicine

Important Publications

1. Lee H, Inoue K, Son D, Mori Y, Yang E, Yano Y, Kim HC, Kazi DS, Lloyd-Jones DM, Khan SS. Evaluation of the PREVENT Equations in a Nationwide Cohort of 7.7 Million Korean Adults. J Am Coll Cardiol 2025;86(23):2395-2398.
2. Lee H, Huang X, Khan SS, Son D, Lee HH, Kim EJ, Lloyd-Jones DM, Kim HC, Greenland P. Very High Prevalence of Non-Optimally Controlled Traditional Risk Factors At the Onset of Cardiovascular Disease. J Am Coll Cardiol 2025;86(14):1017-1029.
3. SCORE2 Asia-Pacific Writing Group on behalf of ESC/EAPC/AFC/APSC. Risk prediction of cardiovascular disease in the Asia-Pacific region: the SCORE2 Asia-Pacific model. Eur Heart J 2025;46(8):702-715.
4. Lee H, Yano Y, Cho SMJ, Lee HH, Kim DW, Lloyd-Jones DM, Kim HC. Associations of Ideal Cardiovascular Health and Its Change During Young Adulthood With Premature Cardiovascular Events: A Nationwide Cohort Study. Circulation 2021;144(1):90-92.
5. Lee H, Yano Y, Cho SMJ, Park JH, Park S, Lloyd-Jones DM, Kim HC. Cardiovascular Risk of Isolated Systolic or Diastolic Hypertension in Young Adults. Circulation 2020;141(22):1778-1786.

Awards and Honors

- Wunsch Medical Award for Young Medical Scientist (2023, Korean Academy of Medical Science)
- Daewoong Scientific Award (2022, Daewoong Foundation)
- LG Future Physician Scientist Award (2022, LG Chemical)

Research Interest

Dr. Hokyou Lee's research interests include life course cardiovascular health, risk prediction, and the epidemiology and outcomes of hypertension, diabetes, and CKM syndromes. He participated in the SCORE2 Asia-Pacific Writing Group, government-funded cohort studies and cardiovascular disease statistics projects of the Korea Disease Control and Prevention Agency, and advisory for the National Health Insurance Service with expertise in study designs, real-world health data, and clinical implementation. Dr. Lee is an elected Fellow of the American Heart Association and serves as an Associate Editor of Circulation with specialty in CKM health.

Epidemiological trends of dyslipidemia in Korea and their implications for Asian populations

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

EAS-KSoLA Joint Symposium

Bridging Europe and Korea in Dyslipidemia Management: Guidelines, Gaps, and Future Directions

Sep 12(Sat) 15:40-17:10 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : **Cheol-Young Park** (Sungkyunkwan University, Republic of Korea)
Borge G. Nordestgaard (University of Copenhagen, Denmark)

15:40-16:00	**ESC/EAS dyslipidemia guidelines: recent updates and evidence base** **Meral Kayikcioglu** (Ege University, Türkiye)
16:00-16:20	**Korean perspectives on dyslipidemia guidelines: recent update and challenges** **Jaehoon Chung** (Gachon University, Republic of Korea)
16:20-16:40	**Translating guidelines into practice** **Wonjin Kim** (CHA University, Republic of Korea)
16:40-17:10	**Panel Discussion** **Seon Mee Kang** (Soonchunhyang University, Republic of Korea) **Min Kyung Lee** (Hanyang University, Republic of Korea) **Ji Eun Lee** (Korea University, Republic of Korea)

CURRICULUM VITAE

Meral Kayikcioglu

Professor, Head of Cardiology Dept, Ege University Medical School, Türkiye

Education and Training

1995.05	Ege University Medical School Turkey, M.D., Cardiology

Employment and Position

1996–2025	Ege University Medical School Turkey, Academic Person
2025–	Ege University Medical School Turkey, Head of Department

Research Interest

- Young (Premature MI)
- Dyslipidemia and lipid disorders
- Familial hypercholesterolemia (heterozygous and homozygous FH)
- Lipoprotein(a) and cardiovascular risk
- Atherosclerotic cardiovascular disease prevention
- Lipid-lowering therapies and LDL-C goal attainment
- Lipoprotein/LDL apheresis
- Cardiovascular risk assessment and preventive cardiology
- Dyslipidemia registries and real-world evidence

ESC/EAS dyslipidemia guidelines: recent updates and evidence base

The 2025 Focused Update of the ESC/EAS Guidelines for the management of dyslipidaemias incorporates important new evidence that has emerged since publication of the 2019 Guidelines, while reaffirming the central role of LDL cholesterol (LDL-C) lowering in the prevention of atherosclerotic cardiovascular disease (ASCVD). This presentation will review the major changes in the 2025 update, with particular emphasis on the evidence supporting the new and revised recommendations.

The LDL-C treatment goals and cardiovascular risk categories established in 2019 remain unchanged; however, cardiovascular risk assessment has been refined. SCORE2 and SCORE2-OP are now recommended for risk estimation in apparently healthy individuals, complemented by risk modifiers such as lipoprotein(a) [Lp(a)], high-sensitivity C-reactive protein, subclinical atherosclerosis, and coronary artery calcium when treatment decisions remain uncertain.

The presentation will discuss contemporary evidence supporting a more intensive and individualized approach to LDL-C lowering. Statins remain first-line therapy, while ezetimibe, PCSK9 monoclonal antibodies, and bempedoic acid provide additional options according to the LDL-C reduction required, treatment tolerance, and cardiovascular risk. Particular attention will be given to evidence from CLEAR Outcomes and to the recommendation for earlier intensification of lipid-lowering therapy during hospitalization for acute coronary syndromes, including early combination therapy when LDL-C goals are unlikely to be achieved with statin monotherapy.

Other clinically relevant updates include recognition of elevated Lp(a) as a cardiovascular risk-enhancing factor and the recommendation to measure Lp(a) at least once in adulthood; the role of high-dose icosapent ethyl in selected high- and very-high-risk patients with elevated triglycerides; and new evidence concerning homozygous familial hypercholesterolaemia, people living with HIV, and prevention of anthracycline-associated cardiac dysfunction.

Overall, the 2025 ESC/EAS update reinforces a risk-based, evidence-driven approach emphasizing earlier identification of residual risk, timely treatment intensification, and achievement of guideline-recommended lipid targets to improve cardiovascular outcomes.

CURRICULUM VITAE

Jaehoon Chung

Professor, Gachon University, Republic of Korea

Education and Training

2010.02	Yeung-nam University, Korea, M.D, Medicine
2026.02	Dongguk University, Korea, Master of Internal Medicine

Employment and Position

2017-2024	National Medical Center, Clinical Doctor
2024-2026.06	Dongguk University Hospital, Assistant Professor
2026.07-Present	Gil Medical Center, Associate Professor

Important Publications

1. Chung, J., Kim, H. L., Lim, W. H., Seo, J. B., Kim, S. H., Zo, J. H., & Kim, M. A. (2019). Association between invasively measured aortic pulse pressure and orthostatic hypotension in patients undergoing invasive coronary angiography. Journal of hypertension, 37(10), 1966-1973.
2. Chung, J., Kim, H. L., Lee, J. P., Lim, W. H., Seo, J. B., Kim, S. H., Zo, J. H., & Kim, M. A. (2020). Association of the Serum Osteoprotegerin Level With Target Organ Damage in Patients at High Risk of Coronary Artery Disease. Circulation journal : official journal of the Japanese Circulation Society, 85(1), 69-76.
3. Chung, J., Han, J. K., Yang, H. M., Park, K. W., Kang, H. J., Koo, B. K., Jeong, M. H., Kim, H. S., & investigators for Korea Acute Myocardial Infarction Registry (KAMIR) (2021). Long-term efficacy of vasodilating β-blocker in patients with acute myocardial infarction: nationwide multicenter prospective registry. The Korean journal of internal medicine, 36(Suppl 1), S62-S71.
4. Chung, J., Min, K. W., Son, B. K., Kim, D. H., & Kim, H. L. (2021). Association between histological severity of Helicobacter pylori infection and cardiovascular risk scores in the Korean population. Atherosclerosis, 333, 124-130.
5. Chung, J., Kim, H. L., Lim, W. H., Seo, J. B., Zo, J. H., Kim, M. A., & Kim, S. H. (2023). New onset diabetes mellitus and cardiovascular outcomes according to statin intensity in patients after drug-eluting stent implantation in Asian patients. Scientific reports, 13(1), 16061. https://doi.org/10.1038/s41598-023-42277-w

Awards and Honors

Research Interest
Atherosclerosis
Arterial stiffness
Lipid
Hypertension
Metabolic syndrome
Coronary artery disease

Korean perspectives on dyslipidemia guidelines: recent update and challenges

The updated Korean dyslipidemia guidelines intensify LDL-C targets while incorporating clinical characteristics relevant to the Korean population. Limitations of Western risk equations and the lack of fully validated Korean-specific risk models remain important challenges in primary prevention. Risk-enhancing factors and coronary artery calcium may help refine risk classification, while lower LDL-C targets and combination therapy are increasingly emphasized in high-risk patients. Future updates should focus on validation of Korean risk models, evidence for optimal LDL-C targets, and evaluation of real-world implementation.

CURRICULUM VITAE

Wonjin Kim

CHA University Gangnam CHA Medical Center, Republic of Korea

Licensure/Certification

2009	License in Medicine (Korea License No. 96968)
2013	License in Internal Medicine (Korea License No. 13991)
2015	License in Endocrinology and Metabolism (Korea License No. 4-15-646)

Education

2002.3–2008.2	Wonju College of Medicine, Yonsei University, Wonju, Republic of Korea
2010.9–2012.8	Master, Yonsei University College of Medicine, Seoul, Republic of Korea
2013.9–2023.2	in Ph.D program at Yonsei University College of Medicine, Seoul, Republic of Korea

Postgraduate Training

2008.3–2009.2	Internship, Severance Hospital, Yonsei University Health System, Seoul, Republic of Korea
2009.3–2013.2	Resident in Internal Medicine. Severance Hospital, Yonsei University Health System, Seoul, Republic of Korea
2013.3–2015.2	Fellowship in Endocrinology. Severance Hospital, Yonsei University Health System, Seoul, Republic of Korea
2015.3–2016.2	Clinical Assistance Professor in Endocrinology. CHA Gangnam Medical Center, CHA University, Seoul, Republic of Korea
2016.3–2025.2	Assistant Professor in Endocrinology. CHA Gangnam Medical Center, CHA University, Seoul, Republic of Korea
2025.3–Current	Associate Professor in Endocrinology. CHA Gangnam Medical Center, CHA University, Seoul, Republic of Korea

Award

2011	The Excellent subject of a lecture in autumn meeting of Korean Endocrine Society
2012	Young investigator award in topical meeting of ASBMR (American Society of Bone and Mineral Research)
2013	The Excellent subject of a lecture in autumn meeting of Korean Endocrine Society

Translating guidelines into practice

Clinical practice guidelines provide evidence-based recommendations for the prevention and management of dyslipidemia, yet substantial gaps remain between guideline recommendations and their implementation in routine clinical practice. While recent European and Korean guidelines have emphasized more intensive lipid-lowering strategies and comprehensive cardiovascular (CV) risk reduction, translating these recommendations into individualized patient care remains challenging because of patient heterogeneity, therapeutic inertia, medication adherence, cost, and differences in healthcare systems.

Practical implementation of contemporary dyslipidemia guidelines requires balancing evidence with individual patient characteristics and real-world clinical circumstances. Optimal lipid management extends beyond achieving treatment targets and should be integrated into comprehensive CV risk reduction strategies that consider residual risk, metabolic health, and long-term outcomes. Recent advances in preventive cardiology have expanded therapeutic opportunities, making individualized treatment strategies increasingly important. Real-world clinical cases will be used to illustrate common challenges in treatment optimization, shared decision-making, and improving long-term adherence to guideline-directed care.

Bridging the gap between evidence and clinical practice remains essential for maximizing the benefits of lipid-lowering therapy. Integrating current evidence with practical decision-making can facilitate personalized management, enhance implementation of guideline-directed strategies, and ultimately reduce the burden of atherosclerotic CV disease across diverse patient populations.

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Satellite Symposia

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 1 (K+E)

Amgen, Hanlim Pharm

Sep 11(Fri) 07:30-08:00 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Myung-gon Kim (Catholic Kwandong University, Republic of Korea)
Hee-Yeol Kim (Incheon Sejong Hospital, Republic of Korea)

07:30-07:45	**Evolving LDL-C management: guidelines and the role of evolocumab** **Sang Min Park** (Eulji University, Republic of Korea)
07:45-08:00	**Is LDL-C goal attainment enough? pitavastatin/fenofibrate in mixed dyslipidemia** **Jae Hyun Bae** (Seoul National University, Republic of Korea)

CURRICULUM VITAE

Sang Min Park

Cardiology Division, Department of Internal Medicine,
Nowon Eulji Medical Center, Eulji University College of Medicine, Seoul, Republic of Korea

Degrees, Training & Employment

2008-2009	Fellowship, Dept. of Cardiology, Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea
2010-2019	Masters and Ph.D., Department of Medicine, the Graduate School of Yonsei University, Seoul, Republic of Korea
2013-2020	Assistant professor, Dept. of Cardiology, Chuncheon Sacred Heart Hospital, Hallym University College of Medicine
2013-Present	Expert, Medical Device Act, Ministry of Food and Drug Safety, government of Republic of Korea
2020-2023	Associate professor, Nowon Eulji Medical Center, Eulji University School of Medicine
2024-Present	Professor, Nowon Eulji Medical Center, Eulji University School of Medicine

Membership of Academic Societies

Korean Society of Internal Medicine
Korean Society of Circulation
Korean Society of Interventional Cardiology
Korean Society of Hypertension
Korean Society of Lipid and Atherosclerosis
Korean Society of Cardiometabolic Syndrome
Korean Vascular Society

Publications

1. Lee Jong-Jun, Chung Wook-Jin, Lee Sang-Jin, Park Sang-Min, Zeon Seok-Jae, Kim Geum-Ha, Jo Young-Chan, Shin Mi-Seung, Park Chul-Hyun, Shin Eak-Kyun. A Huge Congenital Sinus of Valsalva Aneurysm Causing Cerebral Embolism and Hypoplastic Tricuspid Valve. Journal of Korean Society of Echocardiography 2006;14(4);161-163.
2. Kim Geum-Ha, Shin Eak-Kyun, Kim Jeong-Ho, Chung Wook-Jin, Lee Sang-Jin, Park Sang-Min, Zeon Seok-Jae, Shin Mi-Seung, Oh Kyu-Jin, Oh Byung-Chun. External Compression of the Inferior Vena Cava by Tortuous Abdominal Aorta in the Elderly. Journal of Korean Society of Echocardiography; 2007; 15(1): 13-15.
3. Koh KK, Park SM, Quon MJ. Leptin and cardiovascular disease: response to therapeutic interventions. Circulation. 2008;117(25):3238-49.
4. Park SM, Koh KK, Kim JH, Yoon KH, Chung WJ, Kang WC. Myocardial infarction with huge mural thrombus due to spontaneous coronary artery dissection detected by 64-multidetector computed tomography. Int J Cardiol. 2008;127(2):e73-5.
5. Koh KK, Moon CI, Kim JH, Choe W, Park SM, Kim YS. Left anterior descending coronary artery myocardial bridging detected by 64-multidetector computed tomography. Int J Cardiol. 2008;128(2):e62-4.

Evolving LDL-C management: guidelines and the role of evolocumab

CURRICULUM VITAE

Jae Hyun Bae

Associate Professor, Seoul National University, Republic of Korea

Education and Training

2002-2008	Seoul National University College of Medicine, Korea, M.D., Medicine
2011-2013	Seoul National University College of Medicine, Korea, M.S., Molecular and Genomic Medicine
2017-2020	Seoul National University College of Medicine, Korea, Ph.D., Translational Medicine

Employment and Position

2009-2013	Seoul National University Hospital, Korea, Resident
2016-2017	Seoul National University Hospital, Korea, Fellow
2018-2023	Korea University Anam Hospital, Korea, Clinical Assistant Professor
2023-2024	Korea University Anam Hospital, Korea, Clinical Associate Professor
2024-	Seoul National University Hospital, Korea, Associate Professor

Important Publications

1. Kim S, Ko HY, Hong B, et al. Comparative hepatic effectiveness of empagliflozin versus dapagliflozin among individuals with type 2 diabetes and metabolic dysfunction-associated steatotic liver disease. Diabetologia. 2026; in press.
2. Lee J, Bae JH, Hahn S. Dipeptidyl Peptidase-4 Inhibitor Use and Risk of Pemphigus Vulgaris and Foliaceus: A Retrospective Cohort Study Using Databases of the Korea National Health Insurance Claims and the National Health Screening Examination. J Dermatol. 2026; Online ahead of print.
3. Ko HY, Shin JY, Jung K, et al. Timing of Antidiabetic Medication Initiation and Risk of Cardiovascular Events and Mortality. JAMA Netw Open. 2026;9:e2619362.
4. Choi JH, Song S, Kim SK, et al. Health Effects of Sugar-Sweetened and Artificially Sweetened Beverages: Umbrella Review and Evidence-Based Consensus Statement of the Korean Diabetes Association and the Korean Nutrition Society. Diabetes Metab J. 2026;50:32-46.
5. Bae JH, Choe HJ, Yang YS, et al. Defining Severe Diabetes Mellitus: A Consensus Framework for Grading and Staging Diabetes Based on Pathophysiology and Complications. Diabetes Metab J. 2025;49:1141-1154.

Research Interest

Diabetes and Obesity, Dyslipidemia and Cardiovascular Outcomes, Aging and Metabolism

Is LDL-C goal attainment enough? pitavastatin/fenofibrate in mixed dyslipidemia

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 2

Jeil Pharmaceutical

Sep 11(Fri) 07:30-08:00 | Room 2 (3F, Conrad Seoul)

CHAIRPERSON : Jae Hyoung Park (Korea University, Republic of Korea)

07:30-07:50 **Bridging guidelines and practice: strategic implementation of combination therapy in lipid management**

Inki Moon (Soonchunhyang University, Republic of Korea)

CURRICULUM VITAE

Inki Moon

Assistant Professor, Soonchunhyang University Bucheon Hospital, Republic of Korea

Education and Training

2003-2009	College of Medicine, Soonchunhyang University, Korea, M.D., Medicine
2011-2013	College of Medicine, Soonchunhyang University, Korea, M.S., Medical Science
2021-2025	College of Medicine, Yonsei University, Korea, Ph.D., Medical Science

Employment and Position

2010-2014	Department of Internal Medicine, Soonchunhyang University Hospital, Seoul, Korea, Resident
2017-2020	Division of Cardiology, Seoul National University Hospital, Seoul, Korea, Clinical Fellow
2020-2021	Division of Cardiology, Soonchunhyang University Bucheon Hospital, Korea, Clinical Assistant Professor
2021-Present	Division of Cardiology, Soonchunhyang University Bucheon Hospital, Korea, Assistant Professor

Important Publications

1. Kong MG, Moon I, Suh J, et al. Home-based body water monitoring and management system for heart failure patients using bioelectrical impedance analysis. ESC Heart Fail. 2026;13(3):xvag106.
2. Kim S, Moon I. Rhythm Control as a Strategy to Mitigate Atrial Functional Mitral Regurgitation and Tricuspid Regurgitation. Korean Circ J. 2025;55(12):1122-1124.
3. Moon I, Lee J, Lee SA, et al. Artificial Intelligence-Enhanced Analysis of Echocardiography-Based Radiomic Features for Myocardial Hypertrophy Detection and Etiology Differentiation. Circ Cardiovasc Imaging. 2025;18(5):e017436.
4. Moon I, Kwak S, Kim M, et al. Assessment of right ventricular systolic function using speckle tracking strain imaging in patients with severe tricuspid regurgitation: a validation study with cardiac magnetic resonance. J Cardiovasc Imaging. 2024;32(1):22.
5. Moon I, Park SY, Kim D, et al. Incidence of atrial fibrillation in patients with renal infarction: A retrospective cohort analysis of the Korean national health insurance registry. Int J Cardiol. 2024;407:132075.

Research Interest

Echocardiography, Cardiomyopathy, Heart failure, Artificial intelligence in cardiovascular imaging, Genetics of cardiomyopathy

Bridging guidelines and practice: strategic implementation of combination therapy in lipid management

Breakfast Symposium 3

Yuhan

Sep 11(Fri) 07:30-08:00 | Room 3 (3F, Conrad Seoul)

CHAIRPERSON : Jeong-Taek Woo (Kyung Hee University, Republic of Korea)

07:30-07:50 **Paving the road for optimal LDL-C management: new evidence on intensive LDL-C targeting from the Ez-PAVE trial**

Yong-Joon Lee (Yonsei University, Republic of Korea)

CURRICULUM VITAE

Yong-Joon Lee

Department of Cardiology, Severance Cardiovascular Hospital, Republic of Korea

Education

Mar. 2, 2005-Feb. 28, 2011	M.D., Yonsei University Wonju College of Medicine

Professional Experience

Mar. 2, 2011-Feb. 29, 2012	Intern, Severance Hospital
Mar. 2, 2012-Feb. 29, 2016	Resident, Department of Internal Medicine, Severance Hospital
Apr. 26, 2016-Apr. 25, 2019	Medical Officer, Republic of Korea Air Force
May 1, 2019-Feb. 29, 2020	Clinical Fellow, Division of Cardiology, Severance Hospital
Mar. 1, 2020-Aug. 29, 2021	Clinical Research Assistant Professor, Division of Cardiology, Severance Hospital
Aug. 30, 2021-Aug. 31, 2023	Clinical Assistant Professor, Division of Cardiology, Severance Hospital
Sep. 1, 2023-Nov. 30, 2023	Clinical Professor, Severance Cardiovascular Hospital
Dec. 1, 2023-Feb. 28, 2024	Clinical Assistant Professor, Division of Cardiology, Severance Hospital
Mar. 1, 2024-Present	Clinical Professor, Severance Cardiovascular Hospital

Professional Membership

Member, The Korean Society of Cardiology

Paving the road for optimal LDL-C management: new evidence on intensive LDL-C targeting from the Ez-PAVE trial

Breakfast Symposium 4

Dong-A ST, HK inno.N

Sep 11(Fri) 07:30-08:00 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : Byung Ryul Cho (Kangwon National University, Republic of Korea)
Bong-Ki Lee (Kangwon National University, Republic of Korea)

07:30-07:45 **The role of DPP-4 inhibitors in Korean diabetes care: focusing on K-evidence**

Yun Kyung Cho (University of Ulsan, Republic of Korea)

07:45-08:00 **The smaller the better: rovazet**

Jeehoon Kang (Seoul National University, Republic of Korea)

CURRICULUM VITAE

Yun Kyung Cho

Assistant Professor,
Asan Medical Center, Ulsan University College of Medicine, Republic of Korea

Education and Training

2006-2012	University of Ulsan, College of Medicine, M.D, Internal Medicine
2015-2017	University of Ulsan, College of Medicine, M.S, Internal Medicine
2018-2020	Kangwon National University, School of Medicine, M.D, Internal Medicine

Employment and Position

2017-2018	Asan Medical Center, Clinical fellow
2018-2019	Kangwon National University Hospital, Clinical fellow
2019-2020	Asan Medical Center, Research fellow
2020-2022	Hallym University Sacred Heart Hospital, Clinical Assistant Professor
2022-2023	Asan Medical Center, Clinical Assistant Professor
2024-	Asan Medical Center, Assistant Professor

Important Publications

1. Defining Obesity Under the Lancet Commission Criteria: Metabolic Syndrome-Anchored Body Fat Percentage Thresholds in Korean Adults. Jung HN, Cho YK, Kim MJ, Moon JY, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH. Diabetes Obes Metab. 2026 Jun;28(6):5309-5317.
2. Beyond Weight Loss: Skeletal Considerations in Obesity Treatment. Kim MJ, Cho YK. Endocrinol Metab (Seoul). 2026 Apr;41(2):210-221.
3. Engineered nutrient-stimulated hormonal multi-agonists for precision targeting of obesity and metabolic disorders. Cho YK, Jung CH. Clin Mol Hepatol. 2026 Apr;32(2):464-486.
4. New Users of Sodium-Glucose Cotransporter 2 Inhibitors Are at Low Risk of Prostate Cancer: A Nationwide Cohort Study. Cho YK, Kim S, Kim MJ, Lee WJ, Kim YJ, Jung CH. Diabetes Metab J. 2026 Jan;50(1):90-100.
5. Comparison of diagnostic criteria for fatty liver disease in assessing cardiac dysfunction: a cross-sectional study. Cho YK, Kim MJ, Kim EH, Lee MJ, Nam HJ, Lee WJ, Kim HK, Jung CH. Hepatol Int. 2026 Feb;20(1):81-90. doi: 10.1007/s12072-025-10924-1.

Awards and Honors

1. 2024 KSoLA Young Investigator Award
2. ICDM 2022 Excellent Oral Presentation Award
3. 2020 JOMES Research Acceleration Award

Research Interest

Therapeutic agents for diabetes, obesity and dyslipidemia, vascular complications of diabetes, fatty liver disease and myosteatosis

The role of DPP-4 inhibitors in Korean diabetes care: focusing on K-evidence

CURRICULUM VITAE

Jeehoon Kang

Associate Professor, Seoul National University Hospital, Republic of Korea

Education

2000-2004	Seoul National University, College of Natural Science, Biomedical Science (BS)
2004-2008	Seoul National University, College of Medicine (MD)
2011-2017	Seoul National University, Department of Molecular Medicine and Biopharmaceutical Sciences. (PhD)

Training

2008.2-2009.2	Internship, Seoul National University Hospital, Seoul, Korea
2009.3-2013.2	Residency in Internal Medicine, Seoul National University Hospital, Seoul, Korea
2013.3-2016.4	Military Medical Officer, Captain
2016.5-2018.3	Clinical Fellowship in Cardiology, Seoul National University Hospital, Seoul, Korea
2018.4-	Assistant professor, Department of Internal Medicine and Cardiovascular Center, Seoul National University Hospital, Seoul, Korea
2020.3-	Assistant professor, Department of Critical Care Medicine, Seoul National University Hospital, Seoul, Korea
2023.3-	Associate professor, Department of Critical Care Medicine, Seoul National University Hospital, Seoul, Korea

The smaller the better: rovazet

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 5

Viatris

Sep 12(Sat) 07:50-08:20 | Room 1 (3F, Conrad Seoul)

CHAIRPERSON : Myung Ho Jeong (Gwangju Veterans Hospital, Republic of Korea)

07:50-08:10 **Statin therapy strategies for newly diagnosed dyslipidemia patients in clinical practice**

Michal Vrablik (Charles University, Czech Republic)

CURRICULUM VITAE

Michal Vrablik

Professor of Internal Medicine, 1st Faculty of Medicine,
Charles University, General University Hospital, Prague, Czech Republic

Education and Training

1997.06	Charles University, Prague, Czechia, M.D., General Medicine
2005.06	Charles University, Prague, Czechia, Ph.D., Physiology and Pathophysiology

Employment and Position

1997-1998	Institute of Biology, Charles University, Prague, Czechia, Research Fellow
1998-Present	3rd Department of Medicine, Charles University and General University Hospital, Prague, Czechia, Deputy Head of the Dept.
2011-Present	Czech Society for Atherosclerosis, President
2012-2016	European Atherosclerosis Society, Board Member
2019-Present	International Atherosclerosis Society, Federation for Europe, Chairman
2024	Fellow of European Federation of Internal Medicine, FEFIM
2025	Fellow of European Society of Cardiology, FESC
2026.02-Present	Charles University, Prague, Czech Republic, Vice Rector for International Affairs

Important Publications

1. Ridker, P. M.; Revkin, J.; Amarenco, P.; et al., Cardiovascular Efficacy and Safety of Bococizumab in High-Risk Patients, NEW ENGLAND JOURNAL OF MEDICINE, 2017, Volume 376, Issue16, pages: 1527-1539, DOI:10.1056/NEJMoa1701488
2. Cicero, Arrigo F. G.; Colletti, Alessandro; Bajraktari, Gani; et al., Lipid lowering nutraceuticals in clinical practice: position paper from an International Lipid Expert Panel, NUTRITION REVIEWS, 2017, Volume 75,Issue 9, page 731-767, DOI 10.1093/nutrit/nux047
3. Ray, KK., Molemans, B.,Schoonen, WM., Giovas, P., Bray, S., et al., EU-Wide Cross- Sectional Observational Study of Lipid- Modifying Therapy Use in Secondary and Primary Care: the DA VINCI study, EUROPEAN JOURNAL OF PREVENTIVE CARDIOLOGY, 2021, Volume 28, Issue 11, pages 1279-1289. DOI:10.1093/eurjpc/zwaa047
4. Banach, Maciej; Patti, Angelo Maria; Giglio, Rosaria Vincenza; et al., The Role of Nutraceuticals in Statin Intolerant Patients, JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY, 2018, Volume 72, Issue1, pages: 96-118 DOI:10.1016/j.jacc.2018.04.040

Statin therapy strategies for newly diagnosed dyslipidemia patients in clinical practice

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 6

Chong Kun Dang, GC Biopharma

Sep 12(Sat) 07:50-08:20 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Kwang-Won Kim (Gachon University, Republic of Korea)
Kwang Kon Koh (Gachon University, Republic of Korea)

07:50-08:05 **Low-dose atorvastatin: addressing an unmet clinical need**
Sehoon Park (Seoul National University, Republic of Korea)

08:05-08:20 **The role of low-dose fenofibrate in the management of dyslipidemia**
Jaeseok Kim (Yonsei University, Republic of Korea)

CURRICULUM VITAE

Sehoon Park

Clinical Assistant Professor, Division of Nephrology, Department of Internal Medicine,
Seoul National University Hospital, Republic of Korea

Education and Training

2007-2013	Bachelor of Medicine (M.D.), College of Medicine, Seoul National University
2016-2023	Integrated M.S.-Ph.D. Program in Medical Science, College of Medicine, Seoul National University (Ph.D.)

Employment and Position

2014-2018	Resident, Department of Internal Medicine, Seoul National University Hospital
2018-2019	Clinical Fellow, Division of Nephrology, Seoul National University Hospital
2019-2022	Military Doctor, Internal Medicine, Armed Forces Capital Hospital (Military Service)
2022-2023	Ph.D. Research Fellow, Department of Biomedical Sciences, Seoul National University College of Medicine; Physician-Scientist Training Program supported by the Korea Health Industry Development Institute (KHIDI)
2023-2025	Assistant Professor, Division of Nephrology, Seoul National University Hospital
2026-Present	Clinical Assistant Professor, Division of Nephrology, Seoul National University Hospital

Society Activities

Former Associates, Training and Education Committee, The Korean Society of Nephrology
Former Associates, Scientific Programs Committee, The Korean Society of Nephrology
Current Member, Policy Committee, The Korean Society of Nephrology
Current Member, Public Relation Committee, The Korean Society of Nephrology

Low-dose atorvastatin: addressing an unmet clinical need

CURRICULUM VITAE

Jaeseok Kim

Division of Nephrology, Department of Internal Medicine, Yonsei University Wonju College of Medicine, Wonju Severance Christian Hospital, Republic of Korea

Education

2003	Yonsei University Wonju College of Medicine, Bachelor of Medicine
2008	Yonsei University Wonju College of Medicine, Master of Medicine
2017	Yonsei University Wonju College of Medicine, Doctor of Medicine

Work Experience

2004-2008	Resident of Internal Medicine, Wonju Severance Christian Hospital
2008-2011	Army Physician, Republic of Korea
2011-2013	Fellowship of Nephrology, Wonju Severance Christian Hospital
2017-2019	Visiting Scholar of Bioengineering, Georgia Tech, Georgia, USA
2013-	Professor of Division of Nephrology, Department of Internal Medicine, Wonju Severance Christian Hospital, Yonsei University Wonju College of Medicine

Extra Curricular Activities

Member of Korean Society of Nephrology

The role of low-dose fenofibrate in the management of dyslipidemia

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 7

Daiichi-Sankyo

Sep 12(Sat) 07:50-08:20 | Room 3 (3F, Conrad Seoul)

CHAIRPERSON : Sang-Wook Lim (CHA University, Republic of Korea)

07:50-08:10 **A new oral LDL-C lowering agent with proven CV benefit: bempedoic acid**

Chang Hee Jung (University of Ulsan, Republic of Korea)

CURRICULUM VITAE

Chang Hee Jung

Professor, University of Ulsan College of Medicine, Asan Medical Center, Republic of Korea

Education and Training

2002.02	Korea University, M.D., Medicine
2014.02	University of Ulsan, Ph.D., Internal Medicine

Employment and Position

2010-2012	Asan Medical Center, Clinical Fellow
2012-2017	Asan Medical Center, Assistant Professor
2017-2023	University of Ulsan College of Medicine, Asan Medical Center, Associate Professor
2018-2010	University of Virginia, VA, USA, Research Associate
2023-Current	University of Ulsan College of Medicine, Asan Medical Center, Professor

Important Publications

1. Jung HN, Cho YK, Kim MJ, Moon JY, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH. Defining Obesity Under the Lancet Commission Criteria: Metabolic Syndrome-Anchored Body Fat Percentage Thresholds in Korean Adults. Diabetes Obes Metab. 2026;28:5309-5317.
2. Jang MK, Jung CH*, Lee HS, Lee A, Jeoung SJ, Lee YH, Kim HJ, Kim SH, Yu SH, Chon S, Jeong IK*. Enavogliflozin Improves Non-invasive Hepatic Steatosis Indices in Patients With Type 2 Diabetes: A Pooled Analysis of Three Randomised Trials. Diabetes Obes Metab. 2026. Epub ahead of print (*Co-corresponding author)
3. Cho YK, Kim S, Kim MJ, Lee WJ, Kim YJ, Jung CH. New Users of Sodium-Glucose Cotransporter 2. Inhibitors Are at Low Risk of Prostate Cancer: A Nationwide Cohort Study. Diabetes Metab J. 2026;50:90-100.
4. Cho YK, Kim MJ, Kim EH, Lee MJ, Nam HJ, Lee WJ, Kim HK, Jung CH. Comparison of diagnostic criteria for. fatty liver disease in assessing cardiac dysfunction: a cross-sectional study. Hepatol Int. 2026;20:81-90.
5. Cho YK, Jung CH. Engineered nutrient-stimulated hormonal multi-agonists for precision targeting of obesity. and metabolic disorders. Clin Mol Hepatol. 2026;32:464-486.

Awards and Honors

2016	KDA Young Investigator Award
2022	KSoLA Scientific Excellence Award

Research Interest

Diabetes vascular complication, Therapeutics, Obesity, Adipose tissue dysfunction

A new oral LDL-C lowering agent with proven CV benefit: bempedoic acid

Breakfast Symposium 8

Organon, GSK, Novartis

Sep 12(Sat) 07:50-08:26 | Room 4 (5F, Conrad Seoul)

CHAIRPERSONS : **Shung Chull Chae** (Kyungpook National University, Republic of Korea)
Soon Jun Hong (Korea University, Republic of Korea)

07:50-08:02	**Integrated lipid management across the CKM spectrum: from evidence to practice** **Jong-Chan Youn** (The Catholic University of Korea, Republic of Korea)
08:02-08:14	**Making vaccination routine for patients with cardiovascular disease** **Timothy C. Tan** (Western Sydney University, Australia)
08:14-08:26	**Why is LDL-C control still insufficient? finding the breakthrough with leqvio cases in the aLLT era** **Jung-Joon Cha** (Korea University, Republic of Korea)

CURRICULUM VITAE

Jong-Chan Youn

Professor, Division of Cardiology, Department of Internal Medicine, Seoul St. Mary's Hospital, The Catholic University of Korea, Seoul, Republic of Korea

Education and Training

2002.02	Yonsei University, Seoul, M.D, Medicine
2006.08	Yonsei University, Seoul, M.S, Internal Medicine
2009.08	Yonsei University, Seoul, Ph.D., Cardiology

Employment and Position

2023-	Seoul St. Mary's Hospital, The Catholic University of Korea, Professor
2019-2020	Cedars-Sinai Medical Center, Los Angeles, USA, Post-Doc Scientist
2019-2023	Seoul St. Mary's Hospital, The Catholic University of Korea, Associate Professor
2016-2019	Dongtan Sacred Heart Hospital, Hallym University, Associate Professor
2012-2015	Severance Cardiovascular Hospital, Yonsei University, Assistant Professor

Important Publications

1. Youn JC et al. Temporal Trends, Risk Factors, and Clinical Outcomes of De Novo Lymphoproliferative Disorders After Heart Transplantation. JACC Heart Fail 2024;12(2):395-405.
2. Youn JC et al. Korean Society of Heart Failure Guidelines for the Management of Heart Failure: Treatment. Korean Circ J. 2023;53(4):217-238.
3. Youn JC et al. Characteristics and outcomes of heart transplant recipients with a pretransplant history of malignancy. Am J Transplant. 2022;22(12):2942-2950.
4. Youn JC et al. Pathophysiology of Heart Failure with Preserved Ejection Fraction. Heart Fail Clin. 2021;17(3):327-335.
5. Youn JC et al. Temporal Trends of De Novo Malignancy Development after Heart Transplantation. J Am Coll Cardiol. 2018;71(1):40-49.

Awards and Honors

2018	ISHLT, International Travelling Scholarship Award
2016	Asian Pacific Society of Hypertension Young Investigator Award
2013	Korean Society of Hypertension, The Best Young Investigator Award

Research Interest

Heart Failure, Cardiac Amyloidosis, Cardio-Oncology
Heart Transplantation, Left Ventricular Assist Device (LVAD)
Immune Aging (Immunosenescence), Transplantation Immunology

Integrated lipid management across the CKM spectrum: from evidence to practice

CURRICULUM VITAE

Timothy C. Tan

Conjoint Associate Professor, University of New South Wales, Australia

Education and Training

1993-1996	University of New South Wales, Australia, Bachelor of Medical Science (Honours Class I), Medical Science
1997-2003	University of Sydney, Australia, MBBS, PhD, Medicine

Employment and Position

Jul. 2011-Jul. 2014	Massachusetts General Hospital / Harvard Medical School, Clinical and Research Fellow
2014-Present	Westmead Hospital, Consultant Cardiologist
Mar. 2015-Present	University of New South Wales, Conjoint Associate Professor

Research Interest

Cardiac imaging including echocardiography, cardiac CT and cardiac MRI, with a particular interest in applying novel imaging techniques to understand cardiac pathology and disease progression

Making vaccination routine for patients with cardiovascular disease

CURRICULUM VITAE

Jung-Joon Cha

Clinical Associate Professor, Korea University Anam Hospital,
Korea University College of Medicine, Republic of Korea

Education and Training

	Gwangju Institute of Science and Technology, Korea, Ph.D., Cardiovascular Cell Aging / Biomedical Engineering
	Yonsei University College of Medicine, Korea, Master's Degree, Medicine / Cardiology
2009	Yonsei University Wonju College of Medicine, Korea, M.D., Medicine
2010-2014	Yonsei University Gangnam Severance Hospital, Korea, Residency, Internal Medicine
2018-2019	Yonsei University Severance Hospital, Korea, Fellowship, Cardiology
2019-2020	Yonsei University Severance Hospital, Korea, Interventional Fellowship, Interventional Cardiology

Employment and Position

2024-Present	Korea University Anam Hospital, Korea University College of Medicine, Clinical Associate Professor
2020-2024	Korea University Anam Hospital, Korea University College of Medicine, Clinical Assistant Professor

Important Publications

1. Cha JJ, Shin Y, Joo HJ, et al. Metabolic and Safety Outcomes of Statin-Ezetimibe Versus High-Intensity Statin in Older Patients. J Am Heart Assoc. 2026;15(13):e048711. doi: 10.1161/JAHA.125.048711.
2. Cha JJ, Hong SJ. Diagnosis and Management of Statin-Associated Muscle Symptoms. Korean Circ J. 2025;55:957-968. doi: 10.4070/kcj.2025.0285.
3. Cha JJ, Kim JH, Hong SJ, et al. Safety and efficacy of moderate-intensity statin with ezetimibe in elderly patients with atherosclerotic cardiovascular disease. J Intern Med. 2025;297:400-408. doi: 10.1111/joim.20029.
4. Cha JJ, Hong SJ, Lim S, et al. Comparison of Statin With Ezetimibe Combination Therapy Versus Statin Monotherapy for Primary Prevention in Middle-Aged Adults. Korean Circ J. 2024;54:534-544. doi: 10.4070/kcj.2024.0036.
5. Lee SJ, Cha JJ, Choi WG, et al. Moderate-Intensity Statin With Ezetimibe Combination Therapy vs High-Intensity Statin Monotherapy in Patients at Very High Risk of Atherosclerotic Cardiovascular Disease. JAMA Cardiol. 2023;8(9):853-858. doi: 10.1001/jamacardio.2023.2222.

Awards and Honors

1. Best Presenter, Korean Society of Interventional Cardiology (KSIC), 2025
2. Outstanding Reviewer Award, Korean Circulation Journal, 2024
3. Best Abstract, Korean Vascular Research Working Group (KOVAS), 2024

Research Interest

Dyslipidemia and lipid-lowering therapy; statin intolerance; interventional cardiology; coronary physiology and intravascular imaging; precision antiplatelet therapy; clinical trials and cardiovascular outcomes research.

Why is LDL-C control still insufficient? finding the breakthrough with leqvio cases in the aLLT era

Luncheon Symposium 1 (K)

Organon

Sep 11(Fri) 11:45-12:15 | Room 1 (3F, Conrad Seoul)

CHAIRPERSON : Young-Bae Park (Seoul National University, Republic of Korea)

11:45-12:00	**Start early: upfront combination therapy for faster LDL-C goal attainment in the CKM era** Soo-Jin Kim (Kosin University, Republic of Korea)
12:00-12:15	**Start strong: elevating LDL-C management in ASCVD with high-dose atorvastatin plus ezetimibe** Jeehoon Kang (Seoul National University, Republic of Korea)

CURRICULUM VITAE

Soo-Jin Kim

Kosin University, Republic of Korea

Education and Professional Experience

- Graduated from Dong-A University College of Medicine (2009)
- Completed internship at Dong-A University Medical Center
- Completed residency in Internal Medicine at Dong-A University Medical Center
- Clinical Fellow in Cardiology, Dong-A University Medical Center
- Attending Physician, Division of Cardiology, Ulsan Hospital
- Assistant Professor, Division of Cardiology, Department of Internal Medicine, Kosin University Gospel Hospital (September 2021-Present)

- Board-Certified Internist / Board-Certified Cardiologist / PhD in Medicine

Professional Memberships

Member, Korean Society of Cardiology
Member, Korean Association of Internal Medicine
Member, Korean Society of Interventional Cardiology
Member, Korean Society of Hypertension
Member, Korean Society of Cardiometabolic Syndrome

Publications

1. Sang-Hyup Lee, Soo-Jin Kim, Ae-Young Her, Byung-Ryul Cho, Gwang-Sil Kim, Taek-Geun Kwon, Sang-Wook Lim, Jaemin Shim, Ji-Yong Jang, Yong-Joon Lee, Seung-Jun Lee, Hee Tae Yu, Sung-Jin Hong, Chul-Min Ahn, Byeong-Keuk Kim, Young-Guk Ko, Donghoon Choi, Myeong-Ki Hong, Yangsoo Jang, Hui-Nam Pak, Jung-Sun Kim, Jung Ho Heo Antithrombotic strategy according to age in patients with atrial fibrillation and drug-eluting stents: a post hoc analysis of the ADAPT AF-DES randomised trial Age and Ageing, Volume 55, Issue 8, August 2026, afag224,
2. Soo-Jin Kim, Seung-Jun Lee, Sung-Jin Hong, Sang-Hyup Lee, Jong-Young Lee, Deok-Kyu Cho, Jin Won Kim, Sang Min Kim, Seung-Ho Hur, Ji-Yong Jang, Jin Sin Koh, Hoyoun Won, Jun-Won Lee, Soon Jun Hong, Dong-Kie Kim, Jeong Cheon Choe, Jin Bae Lee, Tae-Hyun Yang, Jung-Hee Lee, Young Joon Hong, Jong-Hwa Ahn, Yong-Joon Lee, Chul-Min Ahn, Jung-Sun Kim, Young-Guk Ko, Donghoon Choi, Myeong-Ki Hong, Yangsoo Jang, Jung Ho Heo & Byeong-Keuk Kim Optical coherence tomography-guided PCI in left anterior descending versus non-left anterior descending lesions: a prespecified substudy of the OCCUPI trial Int J Cardiovasc Imaging. 2026 May 29. doi: 10.1007/s10554-026-03734-5.
3. Bong-Joon Kim; Soo-Jin Kim; Sung-Il Im; Jung-Ho Heo Left Atrial Compliance as a Key Determinant of Hemodynamic Phenotypes in HFpEF Patients With Pulmonary Hypertension: Rationale and Study Design Right Heart J. 2026 Apr;1(2):e12. English.
4. Soo-Jin Kim; Ae-Young Her; Hyung Joon Joo; Kiyuk Chang; Byeong-Keuk Kim; Young Bin Song; Sung Gyun Ahn; Jung-Won Suh; Sang Yeup Lee; Jung Rae Cho et al. Sex Differences in Prognostic Implications of CYP2C19 Genotype After Drug-Eluting Stent Implantation J Am Heart Assoc. 2026 May 5;15(9):e045981.
5. Hack-Lyoung Kim; Seong Mi Park; Hyun Ju Yoon; Seon-Ah Jin; Moo-Yong Rhee; Mi-Seung Shin; Kyung-Soon Hong; Hyun-Jin Kim; Shinjeong Song; Soo Jin Kim et al. Clinical Factors Associated With Three-Year Cardiovascular Outcomes in Women Who Underwent Invasive Coronary Angiography: Data From the KoRean wOmen'S chest pain rEgistry (KoROSE) Korean Circ J. 2025 Oct;55(10):877-888. English.

Start early: upfront combination therapy for faster LDL-C goal attainment in the CKM era

CURRICULUM VITAE

Jeehoon Kang

Associate Professor, Seoul National University Hospital, Republic of Korea

Education

2000-2004	Seoul National University, College of Natural Science, Biomedical Science (BS)
2004-2008	Seoul National University, College of Medicine (MD)
2011-2017	Seoul National University, Department of Molecular Medicine and Biopharmaceutical Sciences. (PhD)

Training

2008.2-2009.2	Internship, Seoul National University Hospital, Seoul, Korea
2009.3-2013.2	Residency in Internal Medicine, Seoul National University Hospital, Seoul, Korea
2013.3-2016.4	Military Medical Officer, Captain
2016.5-2018.3	Clinical Fellowship in Cardiology, Seoul National University Hospital, Seoul, Korea
2018.4-	Assistant professor, Department of Internal Medicine and Cardiovascular Center, Seoul National University Hospital, Seoul, Korea
2020.3-	Assistant professor, Department of Critical Care Medicine, Seoul National University Hospital, Seoul, Korea
2023.3-	Associate professor, Department of Critical Care Medicine, Seoul National University Hospital, Seoul, Korea

Start strong: elevating LDL-C management in ASCVD with high-dose atorvastatin plus ezetimibe

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 2

Hanmi Pharmaceutical

Sep 11(Fri) 11:45-12:15 | Room 2 (3F, Conrad Seoul)

CHAIRPERSONS : Kyong Soo Park (Konkuk University, Republic of Korea)
Myung A Kim (Jeju National University, Republic of Korea)

11:45-12:00 **Beyond the statin, ROSUZET; start early, switch now**
Gyu Chul Oh (The Catholic University of Korea, Republic of Korea)

12:00-12:15 **Be the standard ROSUZET; evidence-driven lipid management**
Wonjin Kim (CHA University, Republic of Korea)

CURRICULUM VITAE

Gyu Chul Oh

Professor, The Catholic University of Korea, Republic of Korea

Education and Training

2009.02	Seoul National University, Korea, M.D, Medicine
2023.02	Seoul National University, Korea, Ph.D., Internal Medicine

Employment and Position

2009-2014	Seoul National University Hospital, Intern, Resident
2017-2020	Seoul National University Hospital, Fellow
2020-	Seoul St. Mary's Hospital, Assistant Professor

Beyond the statin, ROSUZET; start early, switch now

CURRICULUM VITAE

Wonjin Kim

CHA University Gangnam CHA Medical Center, Republic of Korea

Licensure/Certification

2009	License in Medicine (Korea License No. 96968)
2013	License in Internal Medicine (Korea License No. 13991)
2015	License in Endocrinology and Metabolism (Korea License No. 4-15-646)

Education

2002.3-2008.2	Wonju College of Medicine, Yonsei University, Wonju, Republic of Korea
2010.9-2012.8	Master, Yonsei University College of Medicine, Seoul, Republic of Korea
2013.9-2023.2	in Ph.D program at Yonsei University College of Medicine, Seoul, Republic of Korea

Postgraduate Training

2008.3-2009.2	Internship, Severance Hospital, Yonsei University Health System, Seoul, Republic of Korea
2009.3-2013.2	Resident in Internal Medicine. Severance Hospital, Yonsei University Health System, Seoul, Republic of Korea
2013.3-2015.2	Fellowship in Endocrinology. Severance Hospital, Yonsei University Health System, Seoul, Republic of Korea
2015.3-2016.2	Clinical Assistance Professor in Endocrinology. CHA Gangnam Medical Center, CHA University, Seoul, Republic of Korea
2016.3-2025.2	Assistant Professor in Endocrinology. CHA Gangnam Medical Center, CHA University, Seoul, Republic of Korea
2025.3-Current	Associate Professor in Endocrinology. CHA Gangnam Medical Center, CHA University, Seoul, Republic of Korea

Award

2011	The Excellent subject of a lecture in autumn meeting of Korean Endocrine Society
2012	Young investigator award in topical meeting of ASBMR (American Society of Bone and Mineral Research)
2013	The Excellent subject of a lecture in autumn meeting of Korean Endocrine Society

Be the standard ROSUZET; evidence-driven lipid management

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 3

JW Pharmaceutical

Sep 11(Fri) 11:45-12:15 | Room 3 (3F, Conrad Seoul)

CHAIRPERSON : Hyo-Soo Kim (Seoul National University, Republic of Korea)

11:45-12:00	**Integrated management of hypertension and dyslipidemia for cardiovascular disease prevention** Minjeong Kim (Ewha Womans University, Republic of Korea)
12:00-12:15	**Management of inadequately controlled patients with diabetes mellitus** Hae Kyung Kim (Yonsei University, Republic of Korea)

CURRICULUM VITAE

Minjeong Kim

Ewha Womans University, Republic of Korea

Education / Appointments

2011	B.S., Korea University
2015	M.S., Gachon University Graduate School of Medicine
2016	Intern, Gachon University Gil Medical Center
2020	Clinical Fellow, Division of Cardiology, Severance Hospital
2022	Clinical Assistant Professor, Division of Cardiology, Myongji Hospital
2024-	Clinical Assistant Professor, Division of Cardiology, Ewha Womans University Mokdong Hospital

Brief List of Publications

1. Mitral and Aortic Regurgitation in Patients Undergoing Kidney Transplantation: The Natural Course and Factors Associated With Progression. Frontiers in Cardiovascular Medicine. 2022.
2. Association of Gender With Clinical Outcomes in a Contemporary Cohort of Patients With Atrial Fibrillation Receiving Oral Anticoagulants. Korean Circulation Journal. 2022.
3. Effects of left atrial function on pulmonary arterial pressure in acute myocardial infarction, hypertrophic and dilated cardiomyopathy. BMC Cardiovascular Disorders. 2022.
4. Changes in Cardiac Structure and Function After Kidney Transplantation: A New Perspective Based on Strain Imaging. JCVI. 2023.
5. Left Ventricular Global Longitudinal Strain and its Prognostic Significance After Kidney Transplantation. J Am Coll Cardiol Img. 2023.
6. Differential determinants and prognostic value of aortic valve sclerosis over carotid atherosclerosis. Int J Cardiol. 2025.
7. Effects of genetic mutations on left ventricular myocardial mechanics and fibrosis patterns in hypertrophic cardiomyopathy, Scientific Reports, 2025.
8. Risk of Retinal Vascular Occlusive Disease in Patients with Aortic Stenosis: A Nationwide Korean Cohort Study. Ophthalmology science. 2025.
9. Evaluation of Burnout and Contributing Factors in Imaging Cardiologists in Korea. JKMS. 2025.
10. Risk of retinal vascular occlusive disease according to type and low-density lipoprotein control after acute coronary syndrome, YMJ, 2025.
11. Aortic Stenosis in End-Stage Renal Disease: Incidence, Prevalence, and Mortality in a National Korean Cohort, J. clinical med. 2025.

Integrated management of hypertension and dyslipidemia for cardiovascular disease prevention

CURRICULUM VITAE

Hae Kyung Kim

Division of Endocrinology and Metabolism,
Severance Hospital, Yonsei University, Republic of Korea

Education

2014	M.D., Yonsei University Wonju College of Medicine
2018	M.S. in Medicine, Graduate School, Yonsei University College of Medicine
2026	Ph.D. in Medicine, Graduate School, Yonsei University College of Medicine

Professional Experience

2014–2015	Intern, Wonju Severance Christian Hospital, Yonsei University
2015–2019	Resident, Department of Internal Medicine, Wonju Severance Christian Hospital, Yonsei University
2020–2021	Clinical Fellow, Division of Endocrinology and Metabolism, Severance Hospital, Yonsei University
2021–2023	Clinical Research Assistant Professor, Division of Endocrinology and Metabolism, Severance Hospital, Yonsei University
2023–Present	Clinical Professor, Division of Endocrinology and Metabolism, Severance Hospital, Yonsei University

Professional Memberships and Activities

- Lifetime Member, Korean Association of Internal Medicine
- Member, Korean Diabetes Association
- Member, Information Committee, Korean Diabetes Association, 2025
- Member, Education and Training Committee, Korean Diabetes Association, 2026–2027
- Lifetime Member, Korean Endocrine Society
- Secretary, Special Committee on Media Response, Korean Endocrine Society, 2025–2026
- Member, Korean Society for the Study of Obesity
- Deputy Secretary-General, Korean Society for the Study of Obesity, 2025–2026
- Ex Officio Member for Media and Public Relations and International Cooperation, Korean Society for the Study of Obesity, 2025–2026
- Lifetime Member, Korean Society of Lipid and Atherosclerosis

Philosophy of Patient Care

"I treat; He heals."

Research Interests

Diabetes mellitus, diabetic kidney disease, metabolic dysfunction-associated steatotic liver disease, obesity, dyslipidemia, and metabolic syndrome

Management of inadequately controlled patients with diabetes mellitus

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 4 (K)

Novartis

Sep 11(Fri) 11:45-12:15 | Room 4 (5F, Conrad Seoul)

CHAIRPERSON : Moon-Kyu Lee (Eulji University, Republic of Korea)

11:45-12:00	**Protecting the vulnerable period: early and sustained LDL-C control with inclisiran** In Tae Jin (Yonsei University, Republic of Korea)
12:00-12:15	**Proven in the real world: consistent LDL-C control with inclisiran** Seok Oh (Chonnam National University, Republic of Korea)

CURRICULUM VITAE

In Tae Jin

Clinical Assistant Professor, Severance Cardiovascular Hospital,
Yonsei University Health System, Republic of Korea

Education and Training

2009.03-2015.02	Chonnam National University College of Medicine, Korea, M.D., Medicine
2016.03-2019.02	Chonnam National University College of Medicine, Korea, Master of Medical Science, Medicine

Employment and Position

2015.03-2016.02	Chonnam National University Hospital, Korea, Internship
2016.03-2020.02	Chonnam National University Hospital, Korea, Resident, Internal Medicine
2020.04-2023.04	Republic of Korea Army, Medical Officer, Captain
2023.05-2025.02	Severance Cardiovascular Hospital, Yonsei University Health System, Fellow, Cardiology
2025.03-2026.02	Severance Cardiovascular Hospital, Yonsei University Health System, Medical Professor, Cardiology
2026.03-	Severance Cardiovascular Hospital, Yonsei University Health System, Clinical Assistant Professor, Cardiology

Protecting the vulnerable period: early and sustained LDL-C control with inclisiran

CURRICULUM VITAE

Seok Oh

Department of Cardiovascular Medicine,
Chonnam National University Hospital, Gwangju, Republic of Korea

Professional Experience

Mar. 2024-Present	Clinical Associate Professor, Department of Cardiology, Chonnam National University Hospital, Gwangju, Republic of Korea
Mar. 2021-Feb. 2024	Clinical Practice Professor, Department of Cardiology, Chonnam National University Hospital, Gwangju, Republic of Korea
Mar. 2020-Feb. 2021	Cardiology Fellow, Department of Cardiology, Chonnam National University Hospital, Gwangju, Republic of Korea
May 2016-Feb. 2020	Residency, Department of Internal Medicine, Chonnam National University Hospital, Gwangju, Republic of Korea
Mar. 2013-Apr. 2016	Military Doctor, Republic of Korea Army, the Ministry of National Defense, Republic of Korea
Mar. 2012-Feb. 2013	Internist, Chonnam National University Hospital, Gwangju, Republic of Korea

Education

Mar. 2020-Aug. 2024	PhD, Chonnam National University Graduate School, Gwangju, Republic of Korea
Mar. 2016-Feb. 2020	MS, Chonnam National University Graduate School, Gwangju, Republic of Korea
Mar. 2006-Feb. 2012	MD, Chonnam National University Medical School, Gwangju, Republic of Korea

Board Certification

- Korean Board of Internal Medicine (Mar 2020, No.19178)

Medical Licensure

- National Medical License (Mar 2012, No.110458)

Graduate Training

May 2020-Feb. 2021	Fellowship (Cardiology Fellow), Department of Cardiovascular Medicine, Chonnam National University Hospital, Gwangju, Republic of Korea
May 2016-Feb. 2020	Residency, Department of Internal Medicine, Chonnam National University Hospital, Gwangju, Republic of Korea
Mar. 2012-Feb. 2013	Internship, Chonnam National University Hospital, Gwangju, Republic of Korea

Memberships and Associations

- The Korean Association of Internal Medicine
- The Korean Circulation Society
- The Korean Society of Interventional Cardiology
- The Korean Vascular Society
- The Korean Society of Hypertension
- The Korean Society of Lipid and Atherosclerosis

Proven in the real world: consistent LDL-C control with inclisiran

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 5

Daewoong

Sep 11(Fri) 11:45-12:15 | Room 5 (6F, Conrad Seoul)

CHAIRPERSON : Hyun Ho Shin (Asan Chungmu Hospital, Republic of Korea)

11:45-12:00	**Clinical updates in dyslipidemia: positioning baroezet for optimal patient care** Jun Sung Moon (Yeungnam University, Republic of Korea)
12:00-12:15	**Strategies for high-risk dyslipidemia patients focusing on ASCVD** Sang Yoon Lee (Sungkyunkwan University, Republic of Korea)

CURRICULUM VITAE

Jun Sung Moon

Professor, Yeungnam University College of Medicine, Republic of Korea

Education and Training

2004.02	Yeungnam University, Korea, M.D., Medicine
2019.09	Yeungnam University Graduate school of Medicine, Korea, Ph.D., Internal Medicine

Employment and Position

2024-Present	Yeungnam University, Korea, Professor
2014-2024	Yeungnam University, Korea, Assistant & Associate Professor

Important Publications

1. Moon JS*, Han KA, Yu JM, Won JC, Kang JG, Park S, Park CY. Efficacy and safety of anagliptin added to metformin and empagliflozin 25 mg in patients with type 2 diabetes: a 24-week randomized, double-blind, placebo-controlled phase 3 trial with 28-week open-label extension. Diabetes Res Clin Pract. 2026 Jun;236:113272. PMID: 42036072.
2. Kim Y, Ha J, Moon JS(Corresponding). Redefining β-Cell Function in Type 2 Diabetes Mellitus: From Comprehensive Assessment to Precision Medicine. Diabetes Metab J. 2026 Mar;50(2):235-252. doi: 10.4093/dmj.2026.0034. PMID: 41813261.
3. Kwon SY, Moon JS(Corresponding). Advances in Continuous Glucose Monitoring: Clinical Applications. Endocrinol Metab (Seoul). 2025 Apr 8. doi: 10.3803/EnM.2025.2370. PMID: 40195726.
4. Lee DY, Moon JS*, Jung I, Chung SM, Park SY, Yu JH, Seo JA, Han KD, Kim NH. Risk acceleration by gout on major adverse cardiovascular events and all-cause death in patients with diabetes and chronic kidney disease. Diabetes Obes Metab. 2025 Mar;27(3):1554-1563. PMID: 39748223.
5. Moon JS*, Kang S, Choi JH, Lee KA, et al. 2023 Clinical Practice Guidelines for Diabetes Management in Korea: Full Version Recommendation of the Korean Diabetes Association. Diabetes Metab J. 2024 Jul;48(4):546-708. PMID: 39091005.

Awards and Honors

2024	Young Investigator Award (Korean Diabetes Association)

Research Interest

- β-cell biology : nutrient overloading and β-cell dysfunction
- Diabetic complications (microvascular complication)
- Diabetes technology (CGMS, insulin pump etc.)

Clinical updates in dyslipidemia: positioning baroezet for optimal patient care

CURRICULUM VITAE

Sang Yoon Lee

Sungkyunkwan University, Republic of Korea

Education

Mar. 2007-Feb. 2011	School of Electrical Engineering, Seoul National University
Mar. 2011-Feb. 2015	Master of Medicine (M.M.), Sungkyunkwan University School of Medicine
Mar. 2023-Sep. 2025	Completed PhD Coursework in Internal Medicine, Sungkyunkwan University School of Medicine

Board Certification

Physician License (Feb 25, 2015)
Board Certified in Internal Medicine (Mar 2, 2020)

Professional Experience

Mar. 2015-Feb. 2016	Intern, Samsung Medical Center
Mar. 2016-Feb. 2020	Resident, Department of Internal Medicine, Samsung Medical Center
Mar. 2020-Apr. 2023	Medical Officer, Republic of Korea Air Force
May 2023-Feb. 2025	Fellow, Division of Cardiology, Samsung Medical Center
Mar. 2025-Present	Clinical Assistant Professor, Division of Cardiology, Samsung Medical Center, Sungkyunkwan University School of Medicine

Professional Memberships

Korean Society of Cardiology
Korean Association of Internal Medicine
Korean Society of Interventional Cardiology

Research Interests

Complex High-Risk PCI
Intracoronary Imaging
Coronary Physiology and Imaging
Pathogenesis of Ischemic Heart Disease, Atherosclerosis, and Acute Coronary Syndrome
Invasive Hemodynamic Support Devices and Critical Care Cardiology
Thoracic and Abdominal Aortic Disease and Hereditary Aortopathy/Vasculopathy
Peripheral Arterial Disease and Other Vascular Diseases
Pulmonary Thromboembolism and Deep Vein Thrombosis

Awards

Best Case Award, Korean Society of Interventional Cardiology (Jan 2025)
Best Abstract Award, ENCORE 2025 (Sep 2025)

Strategies for high-risk dyslipidemia patients focusing on ASCVD

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 6

GSK

Sep 12(Sat) 12:00-12:30 | Room 1 (3F, Conrad Seoul)

CHAIRPERSON : Sang Hong Baek (The Catholic University of Korea, Republic of Korea)

12:00-12:20 **Embedding shingles vaccination into everyday cardiology practice**
Timothy C. Tan (Western Sydney University, Australia)

CURRICULUM VITAE

Timothy C. Tan

Conjoint Associate Professor, University of New South Wales, Australia

Education and Training

1993-1996	University of New South Wales, Australia, Bachelor of Medical Science (Honours Class I), Medical Science
1997-2003	University of Sydney, Australia, MBBS, PhD, Medicine

Employment and Position

Jul. 2011-Jul. 2014	Massachusetts General Hospital / Harvard Medical School, Clinical and Research Fellow
2014-Present	Westmead Hospital, Consultant Cardiologist
Mar. 2015-Present	University of New South Wales, Conjoint Associate Professor

Research Interest

Cardiac imaging including echocardiography, cardiac CT and cardiac MRI, with a particular interest in applying novel imaging techniques to understand cardiac pathology and disease progression

Embedding shingles vaccination into everyday cardiology practice

Luncheon Symposium 7

Hanmi Pharmaceutical

Sep 12(Sat) 12:00-12:30 | Room 2 (3F, Conrad Seoul)

CHAIRPERSON : Hong Seog Seo (Korea University, Republic of Korea)

12:00-12:15	**New paradigm of initial therapy for hypertension: which patients benefit from amoprel?** Young-Sang Lyu (Chosun University, Republic of Korea)
12:15-12:30	**Combination treatment in hypertension: why is losartan-based therapy effective?** Hoyoun Won (Chung-Ang University, Republic of Korea)

CURRICULUM VITAE

Young-Sang Lyu

Associate Professor, Chosun University Hospital, Republic of Korea

Education and Training

2005-2011	Chosun University College of Medicine, M.D.
2017-2019	Chosun University, M.S., Internal Medicine
2019-2021	Chosun University, Ph.D., Internal Medicine

Employment and Position

2021-	Chosun University Hospital, Assistant Professor

Important Publications

1. Lyu et al. Safety and Effectiveness of Naltrexone-Bupropion in Korean Adults with Obesity: Post-Marketing Surveillance Study. Drug Des Devel Ther. 2024 Nov 19;18:5255-5268.
2. Lyu et al. The Effect of Periodontitis on Body Size Phenotypes in Adults without Diagnosed Chronic Diseases: The Korean National Health and Nutrition Examination Survey 2013-2015. Int J Environ Res Public Health. 2024 Sep 4;21(9):1180.
3. Lyu et al. Efficacy and safety of enavogliflozin vs. dapagliflozin as add-on therapy in patients with type 2 diabetes mellitus based on renal function: a pooled analysis of two randomized controlled trials. Cardiovasc Diabetol. 2024 Feb 15;23(1):71.
4. Lyu et al. Comparison of SGLT2 inhibitors with DPP-4 inhibitors combined with metformin in patients with acute myocardial infarction and diabetes mellitus. Cardiovasc Diabetol. 2023 Jul 22;22(1):185.

Research Interest

Complication of diabetes, NAFLD, Obesity treatment

New paradigm of initial therapy for hypertension: which patients benefit from amoprel?

CURRICULUM VITAE

Hoyoun Won

Associate Professor, Chung-Ang University Hospital, Republic of Korea

Education and Training

2007	Yonsei University, Korea, M.D.
2014	Yonsei University, Korea, Ph.D., Cardiology

Employment and Position

-2026	Chung-Ang University Hospital, Staff

Combination treatment in hypertension: why is losartan-based therapy effective?

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 8

JW Pharmaceutical

Sep 12(Sat) 12:00-12:30 | Room 3 (3F, Conrad Seoul)

CHAIRPERSON : Hak Chul Jang (Seoul National University, Republic of Korea)

12:00-12:15	**Cutting-edge care of pitavastatin with ezetimibe combination therapy** Yeoree Yang (The Catholic University of Korea, Republic of Korea)
12:15-12:30	**Beyond LDL-C reduction comprehensive lipid control in DM patients** Jun Hwa Hong (Eulji University, Republic of Korea)

CURRICULUM VITAE

Yeoree Yang

Clinical Assistant Professor,
Division of Endocrinology & Metabolism, Department of Internal Medicine,
Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea
Vice Director, Catholic Smart Health Care Center, The Catholic University of Korea, Republic of Korea

Education and Training

2004-2010	The Catholic University of Korea, M.D., Medicine
2015-2023	The Catholic University of Korea, Ph.D., Internal Medicine

Employment and Position

2018-Present	Division of Endocrinology & Metabolism, Department of Internal Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Clinical Assistant Professor
2019-Present	Catholic Smart Health Care Center, The Catholic University of Korea, Vice Director

Cutting-edge care of pitavastatin with ezetimibe combination therapy

"The lower, the better" is settled for LDL-C: across statin and non-statin trials, the relationship with cardiovascular events is linear. Reaching the goal is the harder part, and the majority of high-risk cardiovascular patients with dyslipidemia in Korea fail to reach their LDL-C target.

Cardiovascular-kidney-metabolic syndrome explains part of the shortfall. In a nationwide cohort of 1.5 million Korean adults, stage 2 — where metabolic risk and declining renal function coexist — accounted for 57.5% of the population, and cardiovascular events increased stepwise as stage advanced. The 2023 AHA advisory places the threshold for intensified lipid management at this stage, ezetimibe combination therapy included.

Pitavastatin occupies a useful position here. It lowers LDL-C by 33% to 44% across 1 to 4 mg, and REAL-CAD, conducted in more than 12,000 Asian patients with stable coronary disease, showed a 19% reduction in cardiovascular events with 4 mg compared with 1 mg (HR 0.81, 95% CI 0.69-0.95), together with lower all-cause mortality and myocardial infarction and no excess of new-onset diabetes.

Combination extends this further. Ezetimibe added to a statin lowers LDL-C by roughly 18%, against about 6% for each dose doubling. Korean phase 3 data show reductions of 52% and 54% at 2/10 and 4/10 mg, 61% in a prediabetic subgroup, and 47% at 1/10 mg, with HbA1c unchanged throughout.

CURRICULUM VITAE

Jun Hwa Hong

Associate Professor, M.D., Ph.D.,
Department of Endocrinology, Daejeon Eulji Medical Center, Eulji University, Republic of Korea

Introduction

Dr. Jun Hwa Hong is currently an Associate Professor in the Division of Endocrinology at Daejeon Eulji Medical Center, Eulji University. He received his M.D. and Ph.D. degrees from Eulji University College of Medicine and completed his internship and residency in internal medicine at Eulji University Hospital.
As an endocrinologist, Dr. Hong has developed a strong clinical and academic focus on obesity, diabetes, and dyslipidemia, with particular expertise in the design and conduct of clinical trials in metabolic diseases.
His research is centered on leading and participating in multicenter randomized clinical trials evaluating novel therapeutic strategies in type 2 diabetes and obesity, including SGLT2 inhibitors, incretin-based therapies, and emerging glucose-lowering agents. He has contributed to pivotal studies investigating add-on therapies to metformin, combination treatment strategies, and real-world effectiveness of anti-obesity pharmacotherapies.
Dr. Hong's work emphasizes evidence generation for optimal treatment strategies and translational application of clinical trial findings into real-world practice. His recent publications include high-quality clinical research articles in journals such as Trends in Endocrinology & Metabolism and Clinical Therapeutics, as well as investigator-initiated and multicenter trial reports in the field of cardiometabolic medicine.
He is an active member of several professional societies, including the Korean Diabetes Association, the Korean Endocrine Society, the Korean Society for the Study of Obesity, and the Korean Society of Lipidology and Atherosclerosis.

Education and Training

2004.02	Eulji University, Korea, M.D., Internal Medicine
2015.02	Eulji University, Korea, Ph.D., Internal Medicine

Employment and Position

2015-2017	Kyungpook National University Hospital, Korea, Professor
2017-2026	Eulji University, Korea, Professor

Important Publications

1. Association of leptin and ghrelin changes with weight response after dapagliflozin treatment in type 2 diabetes. Cardiovascular Prevention and Pharmacotherapy 2026 Accepted
2. Role of Evogliptin for Intra-Class Therapeutic Interchange in Patients With Type 2 Diabetes Mellitus: Subgroup Analysis From A Multi-Center, Prospective, Observational Study (REVISE Study). Endocrinol Diabetes Metab. 2026 Jul;9(4):e70270. doi: 10.1002/edm2.70270.
3. Effectiveness and Safety of Evogliptin in Patients With Type 2 Diabetes Mellitus: A Multicenter, Prospective, Observational Study. Diabetes Obes Metab. 2026 May;28(5):4112-4120.
4. Efficacy and safety of adding a fourth oral antidiabetic drug versus metformin dose escalation in patients with type 2 diabetes inadequately controlled on triple oral combination therapy (EFFORT): A 24-week, randomized, open-label, multicenter trial. Diabetes Obes Metab. 2026;1-12.
5. Letter: Impact of Remnant Cholesterol on the Risk for End-Stage Renal Disease in Type 2 Diabetes Mellitus: A Nationwide Population-Based Cohort Study (Diabetes Metab J 2025;49:1106-15). Diabetes Metab J 2026;50:190-191.

Beyond LDL-C reduction comprehensive lipid control in DM patients

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Oral Presentation

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Oral Presentation 1

Sep 10(Thu) 14:30-16:00 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Hyun Kook (Chonnam National University, Republic of Korea)
Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

OP1-1

Improved survival and in vivo cholesterol-lowering efficacy of microencapsulated Lactobacillus plantarum LAB12

Ismail M. Fareez[1,2*], Siong-Meng Lim[2], Fei Tieng Lim[2], Kalavathy Ramasamy[2]

[1]School of Biology, Faculty of Applied Sciences, Universiti Teknologi MARA 40450 Shah Alam, Selangor Darul Ehsan, Malaysia, [2]Collaborative Drug Discovery Research (CDDR) Group, Faculty of Pharmacy, Universiti Teknologi MARA (UiTM) Cawangan Selangor, Kampus Puncak Alam, 42300 Bandar Puncak Alam, Selangor Darul Ehsan, Malaysia

Objectives: Dietary probiotics are widely recognized for their hypocholesterolemic (cholesterol-lowering) effects against hypercholesterolemia, a primary risk factor for coronary heart disease. However, their vulnerability to thermal processing and low gastric pH limits their efficacy as functional food supplements. This study investigated whether microencapsulating Lactobacillus plantarum LAB12 in a novel pea protein-carbohydrate blend could enhance its structural survivability and improve its in vivo cholesterol-lowering efficacy.
Methods: Microcapsules were synthesized using an oil emulsion/acidification technique. The structural resilience of the microencapsulated L. plantarum LAB12 was evaluated by exposure to simulated gastric juice (SGJ; pH 1.8), simulated intestinal fluid (SIF; pH 6.8), and high-temperature stress (100°C for 30 min). Finally, the therapeutic efficacy of the encapsulated formulation was validated in vivo using an adult zebrafish model fed a high-cholesterol diet over a 4-week period.
Results: The microencapsulated LAB12 demonstrated exceptional gastro-tolerance, sustaining only 4% cell death in SGJ, followed by a targeted, maximum release of probiotics (>9 log CFU g^{-1}) in SIF within 30 min. The formulation also exhibited robust thermal resistance, with only 20% cell death at 100°C. In vivo trials revealed that the microcapsules not only retained the intrinsic cholesterol-lowering properties of LAB12 but also conferred significant metabolic advantages over free cells. Compared to the free-cell treatment group, the encapsulated probiotic yielded a 10% reduction in BMI, a 66.7% increase in cardioprotective serum HDL levels, and successfully alleviated hepatic triglyceride accumulation (fatty liver).
Conclusions: The novel pea protein-carbohydrate microcapsules serve as an ideal protective transport system for L. plantarum LAB12. Beyond merely safeguarding the probiotic against environmental and physiological stressors, the encapsulation matrix actively amplifies its metabolic benefits, offering a superior therapeutic strategy for managing hypercholesterolemia.
Keywords: Lactic acid bacteria, Microencapsulation, Hypercholesterolemia, Zebrafish model, Target delivery

OP1-2

Lipid accumulation product and visceral adiposity indices as markers of hyperuricemia and gout: a meta-analysis of 575,000 patients

Khalid Sarhan*, Ibrahim Serag

Department of Internal Medicine, Mansoura University Hospitals, Mansoura, Egypt

Objectives: Hyperuricemia and gout are rising globally and are closely tied to obesity and visceral fat accumulation. Surrogate markers such as the lipid accumulation product (LAP), visceral adiposity index (VAI), and Chinese visceral adiposity index (CVAI) offer accessible measures of visceral fat-related metabolic risk, yet their associations with hyperuricemia and gout remain inadequately quantified. This review aimed to appraise these relationships among adults.
Methods: We systematically searched PubMed, Scopus, and Web of Science from their inception through February 2026. Eligible observational studies were those reporting associations of LAP, VAI, or CVAI with hyperuricemia or gout. Adjusted effect estimates were pooled using random-effects models, covering both continuous exposures (per one-unit increment) and categorical contrasts (highest versus lowest category). Where reported, sex-stratified subgroup analyses were also carried out.
Results: A total of 25 studies, encompassing over 575,000 participants, met the inclusion criteria. Elevated LAP showed a significant link with hyperuricemia in both continuous (OR=1.64, 95% CI 1.15-2.34) and categorical analyses (OR=3.74, 95% CI 2.88-4.86). Comparable patterns emerged for VAI (per-unit OR=1.56, 95% CI 1.20-2.04; highest vs. lowest OR=2.37, 95% CI 1.72-3.28) and CVAI (per-unit OR=2.24, 95% CI 2.14-2.33; highest vs. lowest OR=3.29, 95% CI 2.01-5.38). These associations held steady across male, female, and mixed-sex subgroups. Higher LAP was likewise tied to a greater risk of gout (OR=2.44, 95% CI 1.90-3.14).
Conclusions: The visceral adiposity indices LAP, VAI, and CVAI demonstrate robust associations with both hyperuricemia and gout, reinforcing their value as straightforward clinical tools for flagging individuals at heightened metabolic risk. Prospective research is needed to confirm their predictive performance across varied populations.
Keywords: Lipid accumulation product, Visceral adiposity index

OP1-3

Distinct vascular targets of metabolic and kidney dysfunction in adults without advanced

Hyo-In Choi*

Cardiology, Kangbuk Samsung Hospital, Republic of Korea

Objectives: The cardiovascular-kidney-metabolic (CKM) construct integrates metabolic risk, kidney disease, and cardiovascular disease, but whether its metabolic and kidney axes relate differently to distinct subclinical vascular phenotypes is unclear. We examined whether early coronary artery calcium (CAC) and arterial stiffness diverge in their risk-factor architecture in asymptomatic adults.

Methods: In a Korean health-screening cohort with serial CAC scoring and repeated pulse-wave velocity (PWV), participants without prior ASCVD were studied; to scope the comparison to non-advanced kidney disease, those with advanced CKD (eGFR < 30 mL/min/1.73 m^2 or dialysis) were excluded while mild-to-moderate dysfunction was retained. Baseline subclinical CVD (CAC, PWV) was not used for CKM/metabolic classification, to avoid circularity. Outcomes were incident CAC (baseline CAC=0), CAC progression (Hokanson $\sqrt{}$-transformed $\Delta \geq 2.5$; logistic regression), and PWV trajectory (linear mixed model with random slopes). Metabolic exposures were contrasted against kidney function (eGFR, ACR), uric acid, and hs-CRP.

Results: Among 21,650 participants (2,501 progression events) and 17,414 with baseline CAC=0 (2,127 incident events), higher CKM/metabolic gradient was associated with CAC progression (OR 1.20, 95% CI 1.11-1.30) and incident CAC (OR 1.24, 1.15-1.34) per step. Metabolic exposures were consistently associated with both outcomes — HOMA-IR (OR 1.13 per doubling), HbA1c, triglycerides, and metabolic syndrome (OR 1.36 and 1.24). In contrast, lower eGFR showed **no positive** association with CAC progression (OR 0.97, P=0.17) and only a weak inverse association with incident CAC, but predicted a **steeper PWV trajectory** (eGFR × time interaction +0.53 cm/s per year per 10 mL/min/1.73 m^2 lower, $P=2.8\times10^{-6}$). Uric acid was null after adjustment, and hs-CRP showed only a small residual incident-CAC signal with no progression association.

Conclusions: In adults without advanced CKD, metabolic and kidney dysfunction target distinct vascular beds: metabolic dysfunction tracks coronary intimal calcification, whereas lower kidney function tracks arterial-stiffness progression. These axis-specific phenotypes emerge before overt disease and support early, axis-targeted prevention. The findings refine, rather than contradict, the kidney-calcification association

Keywords: Atherosclerosis, Insulin resistance, Renal

OP1-4

A randomized, double-blind, placebo-controlled, crossover trial to comparatively evaluate the effects of magnesium hydride (MgH2) on endothelial function: a pilot study

Yasuhiro Endo[1*], Makoto Ayaori[2], Kei Sasaki[3], Manami Teramoto[3], Junko Arakawa[3], Katsunori Ikewaki[3]

[1]Faculty of Medical Technology, Niigata University of Pharmacy and Medical and Life Sciences, Japan, [2]Department of Cardiology, Tokorozawa Heart Center, Japan, [3]Division of Anti-aging and Vascular Medicine, National Defense Medical College, Japan

Objectives: Molecular hydrogen (H_2) exerts antioxidant and anti-inflammatory effects and has been proposed as a potential therapeutic agent for cardiovascular diseases. Magnesium hydride (MgH_2), an oral hydrogen-releasing compound, may provide a practical alternative to hydrogen gas inhalation. However, its effects on endothelial function and oxidative stress remain to be fully elucidated.

Methods: In this randomized, double-blind, placebo-controlled, crossover trial, we evaluated the effects of 4-week MgH_2 administration on endothelial function in 29 Japanese patients with hypertension, diabetes mellitus, or dyslipidemia, with or without cardiovascular disease. The primary endpoint was flow-mediated dilation (FMD). Secondary endpoints included blood pressure, lipid and glycemic parameters, oxidative and inflammatory markers, and cholesterol uptake capacity (CUC) as an index of HDL functionality.

Results: All participants completed the study, and MgH_2 was well tolerated, with no adverse events. No significant differences were observed between MgH_2 and placebo in FMD. Additionally, no significant changes were found in blood pressure, lipid profiles, glycemic control, oxidative stress markers, inflammatory markers, or CUC.

Conclusions: Short-term oral MgH_2 administration did not significantly improve endothelial function or related metabolic parameters. However, this approach was well tolerated and may offer greater clinical feasibility compared with hydrogen gas inhalation. The absence of significant effects may be attributable to the limited sample size, short intervention duration, suboptimal dosing, and patient characteristics. Further studies with optimized protocols are required to clarify the therapeutic potential of MgH_2

Keywords: Hydrogen, FMD, HDL

OP1-5

Association of dietary sodium-to-potassium ratio with triglyceride-glucose index and metabolic syndrome risk in Korean adults: using data from the 2022-2023 Korea National Health and Nutrition Examination Survey

Jisu Lee*, Sunhye Shin

Department of Food and Nutrition, Seoul Women's University, Republic of Korea

Objectives: Excessive sodium intake and potassium deficiency are potential contributors to metabolic dysfunction. While the triglyceride-glucose (TyG) index is an established surrogate marker for insulin resistance, studies assessing the association between the dietary Na/K ratio and TyG index or Metabolic Syndrome (MetS) risk remain limited. This study investigated the independent associations between the dietary Na/K ratio and TyG index or MetS risk in Korean adults.
Methods: This cross-sectional study analyzed 10,040 participants aged ≥19 years from the KNHANES 2022-2023. Subjects were stratified into six subgroups by sex and age: men and women aged 19-39 years, 40-64 years, and ≥65 years. MetS was defined as meeting at least three of five standardized diagnostic criteria, including abdominal obesity, elevated fasting glucose, hypertriglyceridemia, reduced HDL-cholesterol, and elevated blood pressure. TyG index ≥8.82 for men and ≥8.73 for women was determined a metabolically unhealthy state. Multivariable linear and logistic regression models were applied to evaluate the associations.
Results: Dietary Na/K ratio was significantly correlated with TyG index in men aged 19-39 years (β=0.090), women aged 40-64 years (β=0.067), and men aged ≥65 years (β=0.084). Younger men aged 19-39 years (OR=1.81, 95% CI: 1.15-2.84), older men aged ≥65 years (OR=1.61, 95% CI: 1.10-2.36) and older women aged ≥65 years (OR=1.72, 95% CI: 1.26-2.34) in the highest quartile (Q4) of dietary Na/K ratio had higher risk of a metabolically unhealthy state (TyG index ≥8.82 for men, ≥8.73 for women) compared to those in the lowest quartile (Q1). Middle-aged men 40-64 years in the highest quartile had a higher risk of MetS (OR=1.55, 95% CI: 1.08-2.23).
Conclusions: Dietary Na/K ratio is associated with higher risks of a metabolically unhealthy state and MetS. These findings suggest dietary Na/K ratio could be used as a sensitive screening tool for metabolic health.
Keywords: Dietary Na/K ratio, Metabolic syndrome, KNHANES, Insulin resistance, Screening marker, TyG index

OP1-6

A newly developed grain-based cereal product for glycemic spike management: effects on glycemic index, glycemic load, and postprandial glucose responses

Soyeon Park[1*], Dabin Lee[1], Minji Kim[1], Soyoon Cha[1], Seohyeon Lee[1], Jimin Kim[1], Jihyun Park[1,2], Seulgi Lee[3], Oh Yoen Kim[1,4,5]

[1]Department of Health Sciences, Clinical Nutrition major, Graduate School of Dong-A University, Republic of Korea, [2]Department of Cancer AI and Digital Health, Graduate School of Cancer Science and Policy, National Cancer Center, Republic of Korea, [3]Wellnable Co., Ltd, Wellnable Co., Ltd, Republic of Korea, [4]Center for Food & Bio Innovation, DAU G-LAMP Project Group, Dong-A University, Republic of Korea, [5]Department of Food Science and Nutrition, Dong-A University, Republic of Korea

Objectives: Postprandial glycemic spikes are increasingly recognized as an important contributor to impaired glucose regulation and cardiometabolic risk. This study evaluated the glycemic index (GI), glycemic load (GL), and postprandial glycemic responses of a newly developed grain-based cereal designed to attenuate postprandial glycemic spikes in healthy adults.
Methods: Healthy adults with normal body mass index and normal glucose tolerance were recruited. Screening assessments included anthropometric measurements, blood pressure, fasting blood glucose, a 75-g oral glucose tolerance test (OGTT), and medical history evaluation. Of the 31 individuals screened, 17 met the eligibility criteria and 15 completed the study. Following the ISO 26642 protocol, participants consumed a glucose reference food containing 25 g available carbohydrate on three separate occasions. A commercially available cereal product and the newly developed cereal product, each providing an equivalent amount of available carbohydrate, were tested on separate visits. Capillary blood glucose concentrations were measured at fasting and for 120 minutes after consumption. Incremental area under the curve (iAUC), GI, GL, and postprandial glycemic response patterns were evaluated.
Results: Both cereal products produced postprandial glucose responses that returned to the normal range within 120 minutes. However, the newly developed cereal product elicited a more gradual increase in blood glucose concentration and a smaller postprandial glycemic excursion than the commercial cereal product. Furthermore, the GI and GL values of the newly developed cereal product were significantly lower than those of the commercial cereal product ($p < 0.05$).
Conclusions: The newly developed grain-based cereal designed for glycemic spike management demonstrated significantly lower GI and GL values and attenuated postprandial glycemic responses compared with a commercially available cereal product. These findings suggest that this product may serve as a practical dietary strategy for reducing postprandial glycemic spikes and supporting healthy glucose regulation.
Keywords: Glycemic index, Glycemic load, Postprandial glycemic response

OP1-7

Nutraceutical modulation of the oral-vascular axis: emerging therapeutic strategies for periodontitis-associated atherosclerosis

Viza Atiq Umar*

Clinical Clerkship Student, Universitas Islam Sultan Agung, Indonesia

Objectives: Chronic periodontitis contributes to systemic inflammation and immune dysregulation that promote endothelial dysfunction and atherosclerosis. Nutraceuticals derived from herbal medicine may offer adjunctive benefits by modulating inflammatory, oxidative, and immunometabolic pathways linking oral and vascular disease.
Methods: A literature review was conducted in PubMed, Science Direct, and Google Scholar for articles published from 2015 to May 2025 using keywords: "periodontitis," "atherosclerosis,", "oral-vascular axis," "nutraceuticals," "Curcuma longa," "Andrographis paniculata," and "Centella asiatica." Studies including in vitro, in vivo, and human trials evaluating these herbs or their combinations on periodontal inflammation, systemic inflammatory markers, endothelial function, and atherogenesis were included. Data were synthesized narratively.
Results: Three Indonesian herbs—Curcuma longa (temulawak/kunyit), Andrographis paniculata (sambiloto), and Centella asiatica (pegagan)—demonstrate complementary mechanisms relevant to the oral-vascular axis. Curcumin from C. longa inhibits NF-κB and NLRP3 inflammasome, reduces IL-1β, IL-6, TNF-α, CRP, and MMPs, improves endothelial function, and lowers LDL oxidation. Andrographolide from A. paniculata suppresses TLR4/NF-κB signaling, decreases macrophage activation and foam cell formation, and improves lipid profile. Asiaticoside and madecassoside from C. asiatica enhance nitric oxide bioavailability, strengthen endothelial barrier function, reduce oxidative stress, and promote vascular repair. Evidence from clinical and preclinical studies indicates effective doses as follows: curcumin 500 mg twice daily (1,000 mg/day) for 8-12 weeks; andrographolide 400 mg/day for 8 weeks; and Centella asiatica extract standardized to asiaticoside 100 mg/day (or total triterpenoids 100-150 mg/day) for 8 weeks. Combination studies suggest additive or synergistic effects on inflammatory burden, endothelial function, and atherogenesis.
Conclusions: Combining C. longa, A. paniculata, and C. asiatica offers multi-target modulation of inflammation, oxidative stress, immune dysregulation, and endothelial dysfunction within the oral-vascular axis. These herbal combinations hold promise as safe and affordable adjunctive strategies to manage periodontitis and reduce atherosclerotic risk. Well-designed randomized clinical trials are needed to confirm efficacy, optimize dosing, and develop standardized nutraceutical formulations.
Keywords: Periodontitis, Atherosclerosis, Oral-vascular axis, Nutraceuticals, Herbal medicine

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Oral Presentation 2

Sep 11(Fri) 08:30-10:00 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Cheol-Ung Choi (Korea University, Republic of Korea)
Jung-Kyu Han (Seoul National University, Republic of Korea)

OP2-1

Renal lipidomic profiling reveals boerhaavia diffusa extract ameliorates saturated fatty acid lipotoxicity and mitochondrial decay independent of systemic adiposity

Lee On Ying*

The Health Technology and Informatics, The Hong Kong Polytechnic University, China

Objectives: This study investigated how Boerhaavia diffusa (BD) extracts reshape the renal lipidome in vivo while evaluating their capacity to preserve organelle integrity against palmitic acid (PA) insults in vitro.
Methods: Kidneys from standard chow (STC), high-fat diet (HFD), and BD-treated HFD (DHFD) mice were analyzed via untargeted LC-MS lipidomics. In vitro, HK-2 cells were exposed to 200 uM PA to induce lipotoxicity. Viability (MTT), ROS (DCFDA), and mitochondrial membrane potential were assessed. Total body weights of the mice were tracked across two cohorts (BD1/BD2).
Results: Lipidome Modulation: In negative mode LC-MS, DHFD significantly suppressed pathogenic membrane lipid species that peak during HFD feeding, such as Phosphatidylserines (PS 40:7 reducing 1398.4-fold) and Phosphatidylethanolamines (PE 38:5). Positive mode revealed major increases in fatty acyls (FA 18:2 increasing 510.4-fold) and acylcarnitines (CAR 9:0 increasing 248.1-fold; CAR 16:0; CAR 18:4), suggesting enhanced mitochondrial fatty acid utilization. Organelle Stabilization: In vitro, PA collapsed cell viability, induced massive ROS accumulation. BD co-treatment successfully rescued cell survival, attenuated ROS generation, and restored polarized mitochondrial structures ($p<0.01$).Phenotypic Decoupling: Long-term HFD significantly elevated body weights (BD1: $p<0.01$; BD2: $p<0.0001$). BD supplementation (DHFD) yielded no statistically significant changes in final body weights compared to HFD alone ($p>0.05$; ns).
Conclusions: Boerhaavia diffusa provides direct, localized tissue protection rather than systemic anti-obesity effects. BD actively remodels the renal lipidome—suppressing toxic phospholipids and increasing carnitine shuttle species—which drives its capacity to inhibit ROS and stabilize mitochondria during lipid overload. BD protects renal tissues against lipotoxic injury independently of metabolic weight management.
Keywords: Boerhaavia diffusa, Renal lipidomics, Mass spectrometry (LC-MS), Lipotoxicity, Mitochondrial decay, Acylcarnitines, Phosphatidylserine, Ectopic lipid accumulation, Obesity-related glomerulopathy, Reactive oxygen species (ROS), Renoprotection

OP2-2

Extrahepatic bile acid synthesis reprograms multiorgan cholesterol flux in cardiovascular disease

Hosung Bae[1,2*]

[1]Biological Chemistry, University of California, Irvine, United States,
[2]Endocrinology and Metabolism, Internal Medicine, University of Iowa, United States

Objectives: Cardiovascular disease is associated with elevated circulating bile acids, but their tissue origins and pathological functions remain incompletely understood. This study aimed to define how cholesterol and bile acid metabolism are redistributed across organs during cardiometabolic stress and to determine whether extrahepatic bile acid production contributes to vascular inflammation and atherosclerosis progression.
Methods: Using large-animal pig models with human-like lipoprotein profiles, we integrated arterio-venous metabolomics, tissue transcriptomics, hormone profiling, and in vivo/ex vivo isotope tracing. Cross-organ metabolite production and consumption were mapped across fasting and feeding states, Western diet exposure, and cardiovascular disease progression induced by low-density lipoprotein receptor deficiency. Targeted mass spectrometry was further used to characterize novel bile acid species associated with disease progression.
Results: We identified extensive diet- and disease-dependent remodeling of interorgan metabolite exchange. Both Western diet and LDLR deficiency promoted bile acid release from multiple extrahepatic organs, contributing to elevated systemic bile acid levels. Tissue transcriptomic analysis revealed increased expression of bile acid-synthesizing enzymes in pigs and patients with cardiometabolic disease, while cholesterol isotope tracing demonstrated active extrahepatic bile acid synthesis. Functionally, elevated circulating bile acids induced endothelial oxidative stress and vascular inflammation, thereby contributing to atherosclerosis progression. In addition, we identified previously uncharacterized fatty acid-conjugated bile acid species that correlated with disease severity.
Conclusions: These findings redefine bile acid metabolism as a distributed, multiorgan process rather than a strictly liver-centered pathway. Extrahepatic bile acid synthesis directly intersects with cholesterol flux, lipoprotein homeostasis, vascular inflammation, and cardiovascular disease progression. This work provides a new framework for understanding cardiometabolic disease and identifies potential metabolic targets for therapeutic intervention.
Keywords: Bile acids, Metabolomics, Isotope tracing, Cardiovascular disease, Cholesterol, Atherosclerosis

OP2-3

Prdx3 deficiency triggers cGAS-STING signaling pathway induced vascular smooth muscle cell senescence and phenotypic switching in abdominal aortic aneurysm

Moajury-Jung[2*], Seong keun Sonn[1], Goo Taeg Oh[1,2]

[1]Heart-Immune-Brain Network Research Center, Department of Life Sciences, Ewha Womans University, Republic of Korea,
[2]Division of Life Science, Ewha Womans Universit, Republic of Korea

Objectives: Abdominal aortic aneurysm (AAA) involves pathological dilation of the abdominal aorta medial layer, where recent studies have shown that vascular smooth muscle cell (VSMC) senescence is a major driver of disease progression. Mitochondria-derived reactive oxygen species (ROS) are key inducers of premature cellular senescence and Prdx3 is a mitochondrial antioxidant which reduces hydrogen peroxides to water. Here, we reveal that mitochondrial damage triggers the cGAS-STING signaling cascade, propelling functional contractile VSMCs toward a senescent phenotype.
Methods: To examine the role of Prdx3 in aneurysmal VSMC, VSMC-specific Prdx3 knockout mice and control mice were infused with angiotensin2 for 28 days. Primary VSMCs of VSMC-specific Prdx3 knockout mice and control mice were cultured by passages to assess the mechanism of Prdx3 deficiency induced cellular senescence. 72-weeks-old VSMC-specific Prdx3 knockout mice and control mice aorta medial layers were also used. To investigate the effects of VSMC senescence during the early stages of the AAA, scRNA-seq was performed after 1 week of angiotensin2 induction.
Results: The AAA model of VSMC-specific Prdx3 knockout mice developed severe aortic dilation and showed pathological changes associated with vascular aging. In vitro culture demonstrated that Prdx3-deficient primary mVSMCs exhibited elevated mitochondrial dysfunction and senescence in early passages, followed by mtDNA leakage into the cytosol and subsequent cGAS-STING-mediated SASP and phenotype switching in later passages. 72w old VSMC-specific Prdx3 knockout mice aorta medial layer showed elevated cGAS-STING-mediated SASP and phenotype switching. After 1 week of AAA induction, Prdx3 deficiency accelerated mVSMC senescence. Moreover, scRNA-seq analysis identified a Prdx3-deficient mVSMC subcluster characterized by cellular senescence pathway enrichment, which further exhibited distinct KEGG pathways after 1 week of AAA induction.
Conclusions: These findings identify Prdx3 as a critical protector against VSMC senescence by suppressing mitochondrial damage-induced cGAS-STING signaling in AAA.
Keywords: Abdominal aortic aneurysm, Peroxiredoxin3, Vascular smooth muscle cell, Mitochondria

OP2-4

Sphingosine-1-phosphate receptor 1 deletion in smooth muscle cells protects against atherosclerosis in apoE-deficient mice by reducing lipid droplet formation

Inbar Habaz[1,2*], Narmadaa Thyagarajan[1,2], Alexander Qian[1,2], Wei Wang[1,2], Bernardo Trigatti[1,2]

[1]Thrombosis and Atherosclerosis Research Institute, Hamilton Health Sciences, Canada,
[2]Department of Biochemistry and Biomedical Sciences, McMaster University, Canada

Objectives: The sphingosine 1 phosphate (S1P) receptor 1 (S1PR1), one of the five G-protein coupled receptors for S1P, controls several cellular processes and thus implicated in atherosclerosis. Although S1PR1 deletion in macrophages and endothelial cells had shown to enhance atherosclerotic plaque development in-vivo, the role of S1PR1 in vascular smooth muscle cells in atherosclerosis is not yet clear.
Methods: Male and female ApoEKO/KO, S1pr1lox/lox;ApoEKO/KO and Tagln-Cre+;S1pr1lox/loxApoEKO/KO mice as well as bone marrow chimeras were fed a high fat (HF) diet for 8 weeks. Aortic sinus atherosclerosis was analyzed histologically and plasma lipids were analyzed biochemically. Primary vascular smooth muscle cells and thioglycolate-elicited peritoneal macrophages were cultured and analyzed in vitro. Lipid accumulation was analyzed by oil red O staining and gene expression changes by quantitative real time PCR after culture of vascular smooth muscle cells in the presence of cholesterol:methyl-beta-cyclodextrin complexes.
Results: HF diet induced atherosclerosis was reduced in S1pr1lox/loxApoEKO/KO mice compared to S1pr1WT/WT; ApoEKO/KO controls. S1pr1lox/lox mice exhibited significantly reduced S1pr1 expression in macrophages and vascular smooth muscle cells, compared to S1pr1WT/WT mice. Reciprocal bone marrow transplantation demonstrated that the S1pr1lox/lox mutation in a non-bone marrow-derived cell type was responsible for the reduced atherosclerotic plaque development. Complete inactivation of S1PR1 in vascular smooth muscle cells in Tagln-Cre+;S1pr1lox/loxApoEKO/KO mice reduced diet-induced atherosclerosis by approx. 50% compared to S1pr1lox/lox; ApoEKO/KO controls without affecting plasma lipid levels or absolute leukocyte counts in circulation. A synthetic S1PR1 selective agonist (SEW2871) enhanced cholesterol-induced lipid droplet formation and Cd68 gene expression but suppressed Acta2 gene expression in cultured primary aortic smooth muscle cells and this was lost when S1pr1 was knocked out. SEW2871 reduced levels of the cholesterol efflux transporter ABCA1 in S1pr1 expressing aortic smooth muscle cells.
Conclusions: Our data demonstrates that knocking out S1PR1 in vascular smooth muscle cells reduces cellular lipid accumulation and protects against atherosclerosis.
Keywords: S1PR1, Vascular smooth muscle cells, Foam cell

OP2-5

Senolytic therapy attenuates atherosclerosis and metabolic dysfunction by targeting cellular senescence in western diet-fed Ldlr KO mice

Hye Rang Park[1,3*], Young Hoon Seo[1,3], Jimin Lee[1,3], Haeun Jang[1], Yongmin Lee[1], Daeun Kim[1,3], Mi-Ran Lee[4], Min ji Cho[2], Jong-Gil Park[1,3]

[1]Biotherapeutics Translational Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea,
[2]Genome Editing Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea,
[3]Department of Bioscience, KRIBB School of Bioscience, Korea University of Science and Technology (UST), Republic of Korea,
[4]Department of Biomedical Laboratory Science, Jungwon University, Republic of Korea

Objectives: Atherosclerosis is a cardiovascular disease characterized by lipid accumulation and chronic inflammation. While dyslipidemia is a major risk factor, aging has recently emerged as an important contributor. Senescent cells promote chronic inflammation through the senescence-associated secretory phenotype (SASP), accelerating atherosclerosis progression — making senolytic therapy, which selectively eliminates senescent cells, an emerging therapeutic strategy. Therefore, this study aimed to evaluate the effects of three senolytic drugs (dasatinib plus quercetin, fisetin, and navitoclax) and their underlying molecular mechanisms in atherosclerosis.
Methods: Ldlr KO mice were fed a Western diet and orally administered fisetin (20 mg/kg), dasatinib plus quercetin (D+Q; 5 mg/kg + 50 mg/kg), or navitoclax (50 mg/kg) from 8 weeks of age for 20 weeks, with drugs given for 3 consecutive days once monthly. Energy expenditure, oxygen consumption (VO_2), carbon dioxide production (VCO_2), and locomotor activity were continuously monitored for 48 hours using an indirect calorimetry system after a 24-hour acclimation period. Following sacrifice, tissues were collected for histological and molecular analyses.
Results: Senolytic treatment attenuated atherosclerosis and improved metabolic dysfunction in Western diet-fed Ldlr KO mice. Among the drugs tested, fisetin exhibited the most pronounced effects, reducing body weight gain, fat mass, LDL-C and triglyceride levels. Fisetin and navitoclax also decreased hepatic triglyceride accumulation, eWAT weight, and adipocyte size, accompanied by increased physical activity and elevated energy expenditure. Furthermore, all senolytic treatments significantly reduced atherosclerotic lesion area in the aortic arch and whole aorta. Of note, fisetin increased collagen content within lesions, suggesting improved plaque stability, and reduced expression of inflammatory markers (F4/80, TNF-α, CCL2) in eWAT, indicating attenuation of adipose tissue inflammation.
Conclusions: These findings suggest that targeting cellular senescence alleviates metabolic dysfunction and chronic inflammation, thereby attenuating atherosclerosis progression. Fisetin and navitoclax demonstrated robust metabolic and anti-inflammatory benefits, supporting their therapeutic potential for age-related cardiovascular diseases.
Keywords: Atherosclerosis, Cellular senescence, Senolytics, Inflammation

OP2-6

The role of LGI3 in adipocytes and macrophages in atherosclerosis

Do Hee Yu*, Jin Jing, Goo Taeg Oh

Heart-Immune-Brain Network Research Center, Department of Life Sciences, Ewha Womans University, Republic of Korea

Objectives: Atherosclerosis occurs when lipids accumulate in blood vessels and is a major chronic cardiovascular disease. Although Leucine-rich repeat LGI family member 3 (LGI3) has been implicated in obesity-associated inflammation, its role in atherosclerosis and its major cellular source remain unclear. This study aimed to identify the major source of LGI3 during atherogenesis and determine whether adipocyte-derived LGI3 contributes to macrophage-mediated inflammation.
Methods: To investigate the cell-specific role of LGI3, Apoe-/- Lgi3F/F AdipoQ-CreERT2 mice (adipocyte-specific LGI3 deletion) and Apoe-/- Lgi3F/F Lyz2-Cre mice (myeloid cell-specific LGI3 deletion) were fed a Western diet to induce atherosclerosis. Atherosclerotic lesion formation in the aorta and aortic root was evaluated. In addition, to confirm the expression of LGI3 even in vitro, adipocytes and macrophages were treated with OTI (ox-LDL, TNF-α, and IFN-γ) to mimic a pro-atherogenic environment. In order to confirm the effect of adipocyte-driven LGI3 on the inflammatory response of macrophages, inflammatory cytokines (IL-6, IL-1β) and NF-κB signaling activity were analyzed.
Results: Adipocyte-specific LGI3 deletion significantly reduced atherosclerotic lesion formation in the aorta and aortic root and decreased circulating LGI3 levels. In contrast, myeloid cell-specific LGI3 deletion had no significant effect on lesion development or plasma LGI3 levels. OTI stimulation increased LGI3 expression in adipocytes, suggesting that adipocytes are a major source of LGI3 under pro-atherogenic conditions. Furthermore, adipocyte-derived LGI3 enhanced macrophage inflammatory responses by increasing pro-inflammatory cytokine expression (IL-6, IL-1β) and NF-κB signaling.
Conclusions: Our findings demonstrate that adipocytes, rather than myeloid cells, are the major source of LGI3 during atherosclerosis. Adipocyte-derived LGI3 promotes macrophage inflammatory activation and contributes to lesion development, suggesting that LGI3 may represent a potential therapeutic target for atherosclerotic cardiovascular disease.
Keywords: Atherosclerosis, LGI3, Adipocytes, Macrophages

OP2-7

Cyclic induction of OSKM expression mitigates atherosclerosis through vascular anti-inflammatory remodeling independent of systemic metabolic effects

Young Hoon Seo[1,3*], Hye Rang Park[1,3], Haeun Jang[1], Yongmin Lee[1], Jimin Lee[1,3], Daeun Kim[1,3], Mi-Ran Lee[4], Min Ji Cho[2], Jong-Gil Park[1,3]

[1]Biotherapeutics Translational Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea,
[2]Genome Editing Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea,
[3]KRIBB School of Bioscience, Korea University of Science and Technology (UST), Republic of Korea,
[4]Department of Biomedical Laboratory Science, Jungwon University,Republic of Korea

Objectives: Atherosclerosis is a chronic inflammatory disease characterized by endothelial dysfunction and dysregulated immune activation. Emerging evidence suggests that controlled induction of Yamanaka factors (OSKM) can modulate inflammatory responses, cellular stress pathways, and reparative signaling. However, whether OSKM induction can influence vascular inflammation and arterial remodeling in the context of atherosclerosis remains unclear. This study aimed to determine the impact of cyclic OSKM induction on atherosclerosis progression and to explore associated molecular changes in the vascular wall and plasma secretome.
Methods: Dox-responsive 4F2A mice (male, heterozygous), in which OSKM expression can be cyclically induced, were subjected to AAV-PCSK9 injection and 20 weeks of Western diet to establish an atherosclerosis model (N=5-8 mice per group). Body weight, metabolic parameters, circulating lipids, and organ weights were assessed. Aortic plaque burden was quantified by en face and aortic sinus staining. Plasma proteomics was performed to identify OSKM-related secretome alterations. Aorta RNA-sequencing was conducted to characterize transcriptomic remodeling, with emphasis on inflammatory, immune, and structural pathways
Results: Cyclic OSKM induction produced minimal changes in whole-body metabolism, liver enzymes, or hepatic lipid metabolism genes. Nonetheless, OSKM-induced mice exhibited a pronounced reduction in circulating LDL levels and a significantly lower aortic plaque burden. Plasma proteomics revealed alterations in secreted proteins associated with wound healing, coagulation, and inflammatory regulation. Aorta RNA-sequencing demonstrated broad suppression of immune effector pathways, reduced cytokine receptor and integrin-binding signatures, and increased expression of actin- and contractile fiber-related genes, indicating an anti-inflammatory and structurally stabilized vascular phenotype.
Conclusions: Cyclic induction of OSKM expression attenuates atherosclerosis independently of systemic metabolic modulation, potentially through vascular anti-inflammatory remodeling and secretome reorganization. These findings suggest that OSKM-based reprogramming strategies may offer a novel therapeutic avenue for vascular disease and warrant further mechanistic exploration.
Keywords: Atherosclerosis, Partial reprogramming, OSKM, Vascular inflammation, Plaque remodeling

OP2-8

Extracellular vesicles produced by M2-polarized macrophages drive atherosclerosis regression by reprograming Ly-6Chi monocytes & enhancing efferocytosis

Robert L. Raffai*, Ngan K. Vu, Tuan Anh Phu, Martin Ng, Alex Gao, Rebecca Kim, Angela Win, Akiko Searles

Vascular and Endovascular Surgery, University of California, San Francisco, United States

Objectives: Examine transcriptional and bioenergetic responses sensitive to M2-macrophage EVs among myeloid cells that improve their ability to drive atherosclerosis regression.
Methods: THP-1 macrophages were stimulated with IL-4, and M2-polarized EVs were purified from conditioned medium using density gradient ultracentrifugation. LDLR-deficient mice fed a high fat diet (HFD) for 12 weeks were infused triweekly with 10e9 M2-EVs for 6 weeks while maintained on the HFD. Ly-6Chi monocytes were isolated and RNA examined using NanoString gene panels. Atherosclerosis was assessed by detecting myeloid cell subsets in aortae and ORO-positive lesions in the aortic root. Sections were also stained with Sirius Red for collagen deposition, TMR red for DNA strand breaks, MOMA2 for macrophages, and ACTA2 for fibrous cap. Bioenergetic metabolism and continual efferocytosis were examined in vitro and ex vivo using M2-EV-treated primary and peritoneal macrophages using a Seahorse XFe-analyzer and by exposing the cells to CFSE- and PKH26-labeled Jurkat cells.
Results: M2-EVs reduced Ly-6Chi monocytes and caused profound transcriptional reprogramming. This included reduced signaling pathways controlled by MAPK, PI3K, TLR, and inflammasomes, as well as glucose transport, hypoxia, and tryptophan metabolism. In contrast, M2-EVs upregulated arginine metabolism, IDH1/2 activity, redox stress control and mitochondrial respiration. This led to reduced levels of IL-1b, IL-6, IFNg, and TNFa in plasma, CD45+ aortic myeloid cells, as well as MOMA2- and ORO-positive aortic lesions. Plaque regression was noted by increased collagen deposition, thicker ACTA2-positive fibrous cap, and reduced necrotic core volumes. Elevated expression of MertK positive macrophages with increased apoptotic cell co-localization, support improvements in efferocytosis. In vitro and ex vivo studies of macrophages exposed to M2-EVs confirmed their improved capacity for continual efferocytosis that derived from improved OxPHOS and tempered glycolytic metabolism.
Conclusions: M2 macrophage EVs offer new therapeutic opportunities to stabilize atheroma beyond plasma lipid correction, by communicating profound bioenergetic transcriptional signaling to monocytes and macrophages.
Keywords: Extracellular vesicle, M2 macrophage, Atherosclerosis regression, Transcriptional reprogramming

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Oral Presentation 3

Sep 12(Sat) 08:50–10:20 | Room 1 (3F, Conrad Seoul)

CHAIRPERSONS : Hyeong Kyu Park (Soonchunhyang University, Republic of Korea)
Han-Mo Yang (Seoul National University, Republic of Korea)

OP3-1

Residual risk of remnant cholesterol in patients treated to LDL-C goal: a landmark analysis of the RACING trial

Sungyeon Park[1*], Jung-Hee Lee[1], Sung-Jin Hong[2], Byeong-Keuk Kim[2], Sung Gyun Ahn[1]

[1]Cardiology, Yonsei University Wonju College of Medicine, Republic of Korea,
[2]Cardiology, Yonsei University College of Medicine, Republic of Korea

Objectives: Residual cardiovascular risk persists in patients with atherosclerotic cardiovascular disease (ASCVD) treated to low-density lipoprotein cholesterol (LDL-C) goal. We examined whether on-treatment remnant cholesterol (remnant-C) is associated with this risk.

Methods: Among RACING trial participants event-free at 1 year (n = 3,238), 1-year LDL-C and remnant-C were dichotomized at a guideline threshold for LDL-C (<55 vs ≥55 mg/dL) and the median for remnant-C (≤17 vs >17 mg/dL). The 3-year primary composite endpoint (cardiovascular death, major cardiovascular events, or non-fatal stroke) was assessed from a 1-year landmark with Cox models adjusted for treatment arm, demographics, comorbidities, and baseline statin intensity.

Results: LDL-C and remnant-C showed negligible correlation (Spearman ρ=0.05), with no interaction for the endpoint (p=0.52). Relative to the concordant-low group (low LDL-C/low remnant-C), both high-remnant-C groups carried elevated risk regardless of LDL-C stratum (low LDL-C/high remnant-C: adjusted hazard ratio [HR] 1.84, 95% CI 1.14-2.98; high/high: HR 1.63, 1.04-2.55). Among patients with LDL-C <55 mg/dL, 46% had high remnant-C, and their risk exceeded that of patients with low remnant-C (HR 1.88, 1.15-3.07). Continuous remnant-C remained associated with events after adjustment for triglycerides (HR 1.28 per SD), whereas triglycerides did not (HR 0.87 per SD, non-significant). Within the LDL-C <55 mg/dL stratum, restricted cubic splines showed a non-linear remnant-C gradient inflecting near 17 mg/dL (p=0.017)

Conclusions: On-treatment remnant-C identifies a higher-risk phenotype, orthogonal to LDL-C, among ASCVD patients treated to LDL-C goal. These findings position remnant-C as a candidate target for residual risk reduction and warrant prospective evaluation.

Keywords: Remnant cholesterol, LDL cholesterol, Residual cardiovascular risk

OP3-2

Lower cardiovascular event risk with bempedoic acid versus ezetimibe in statin-intolerant atherosclerotic cardiovascular disease (ASCVD): a 3,540-patient TriNetX analysis

Ahmed Elaraby[1*], Ahmed Fouad[1], Ahmed Hassan[2], Ali Bakr[1]

[1]Medicine, Faculty of Medicine, Al-Azhar University, Cairo, Egypt, [2]Medicine, Faculty of Medicine, Al-Azhar University, Asyut, Egypt

Objectives: Bempedoic acid and ezetimibe are commonly used non-statin lipid-lowering therapies in patients with atherosclerotic cardiovascular disease (ASCVD) who are unable to tolerate statins. However, real-world comparative outcome data between these two oral non-statin strategies remain limited.

Methods: We performed a retrospective cohort study using the TriNetX Global Collaborative Network. Adults with established ASCVD, dyslipidemia, statin intolerance or statin-associated muscle/adverse-effect codes, and prior statin exposure were identified. Patients initiating bempedoic acid were compared with patients initiating ezetimibe. Propensity-score matching was used to balance demographic and clinical characteristics. The primary outcome was a composite MACE outcome as defined in the TriNetX analysis. Secondary outcomes included unstable angina, heart failure, and peripheral arterial events.

Results: Before matching, 1,771 patients were identified in the bempedoic acid cohort and 47,357 in the ezetimibe cohort. After propensity-score matching, 1,770 patients were included in each group, with good covariate balance across measured baseline characteristics. Bempedoic acid use was associated with a lower risk of the composite MACE outcome compared with ezetimibe (20.4% vs 29.8%; risk ratio [RR], 0.68; 95% CI, 0.61-0.77). Bempedoic acid was also associated with lower risks of unstable angina (10.1% vs 15.0%; RR, 0.68; 95% CI, 0.57-0.81), heart failure (21.2% vs 30.7%; RR, 0.69; 95% CI, 0.62-0.77), and peripheral arterial events (15.8% vs 21.0%; RR, 0.75; 95% CI, 0.65-0.87).

Conclusions: In our cohort of statin-intolerant adults with ASCVD, bempedoic acid was associated with lower risks of cardiovascular and vascular outcomes than ezetimibe. Bempedoic acid was associated with about a one-third lower risk of the composite MACE outcome and similar reductions were observed for unstable angina, heart failure and peripheral arterial events. These findings suggest that bempedoic acid may be a useful oral nonstatin option for secondary prevention in high-risk patients who cannot tolerate statins. Further prospective studies are needed to confirm these results.

Keywords: Bempedoic acid, Ezetimibe, Statin intolerance, ASCVD, Nonstatin lipid-lowering therapy

OP3-3

Age-dependent association of atherogenic dyslipidemia with incident cardiovascular disease: a nationwide Korean cohort study

Ye Seul Yang[1,2*], Kyuho Kim[3], Seung-Hyun Ko[3], Kyuna Lee[4], Bongseong Kim[5], Kyungdo Han[4], Min Kyong Moon[6]

[1]Department of Medicine, Seoul National University College of Medicine, Republic of Korea, [2]Department of Internal Medicine, Seoul National University Bundang Hospital, Seoul National University College of Medicine, Republic of Korea, [3]Division of Endocrinology and Metabolism, Department of Internal Medicine, St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Republic of Korea, [4]Department of Statistics and Actuarial Science, Soongsil University, Republic of Korea, [5]Department of AI-Bio, Graduate School of AI Soongsil University, Republic of Korea, [6]Department of Internal Medicine, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul National University College of Medicine, Republic of Korea

Objectives: Atherogenic dyslipidemia, characterized by elevated triglycerides (TG) and low HDL-cholesterol (HDL-C), is an established cardiovascular risk marker, but whether their relative impact differs across the adult age spectrum is unclear. We examined their association with incident cardiovascular disease (CVD) and its modification by age.
Methods: Using the Korean National Health Insurance Service database, we analyzed a 40% random sample of adults aged ≥20 years who underwent health screening in 2012 (N=4,750,489). Those with prior myocardial infarction (MI) or ischemic stroke, missing lipid values, or an event or death within the first year were excluded. The primary outcome was incident CVD (hospitalized MI [ICD-10 I21-I22] or ischemic stroke [I63-I64 with brain imaging]). Multivariable Cox models adjusted for age, sex, and statin use; analyses were stratified by 10-year age group.
Results: Over a median follow-up of 10.3 years, 218,342 CVD events occurred. Higher TG (≥200 vs <150 mg/dL) and lower HDL-C (men <40, women <50 vs ≥60 mg/dL) were each associated with increased CVD risk (HR 1.32 [95% CI 1.30-1.33] and 1.28 [1.27-1.30]), with consistent gradients for MI and ischemic stroke and across sex, obesity, diabetes, and chronic kidney disease strata. The association was strongest in young adults and attenuated progressively with age for both TG and HDL-C; for TG ≥200 mg/dL, the HR was 1.58 and 1.59 in those aged 20-29 and 30-39 years, respectively, versus 1.14 among those aged ≥80 years.
Conclusions: Atherogenic dyslipidemia was a robust predictor of CVD across the population, but its relative effect was greatest in young adults. These findings support earlier lipid risk assessment and primordial prevention targeting metabolic dyslipidemia in younger populations.
Keywords: Atherogenic dyslipidemia, Cardiovascular disease, Young adults

OP3-4

Deep learning analysis of BMI, metabolic risk factors, and sex hormones in U.S. women: a NHANES study

Saheed Oladele Amusat[1,2*], Saheed[1,2]

[1]Biosciences, School of Science and Technology, Nottingham Trent University, United Kingdom,
[2]Chemical Pathology Department, The Directorate of Laboratory Medicine, Beaumont Hospital, Dublin, Republic of Ireland, Ireland

Objectives: Obesity, defined by the WHO as excessive fat accumulation impairing health, is linked to increased risks of cardiometabolic disorders like diabetes, high cholesterol, heart disease, and hormonal imbalances (Hu et al., 2017; Young et al., 2018). Bischoff et al. (2017) highlighted obesity's impact on nearly every organ system, including the cardiovascular, endocrine, central nervous, and gastrointestinal systems. Thus, this study aim to unravel the complex relationships between BMI as a function of obesity, sex hormone, and metabolic risk disorder among women of childbearing age.
Methods: This study included a sample of 724 females within the child-bearing age range of 15 to 49 years. The primary outcome variable was BMI, while the predictors consisted of 19 variables, including 12 sex hormones, 6 metabolic disorder risk factors, and age. A deep neural network (DNN) was developed using the H2O.ai deep learning framework in R to predict BMI, applying a regression model with a Gaussian distribution and quadratic loss function, along with 10-fold cross-validation for model optimisation.
Results: The deep learning regression model demonstrated good performance and generalisation with consistent error metrics across training (RMSE=6.27), validation (RMSE=6.54), and cross-validation (RMSE=6.54). Partial dependence plots revealed that insulin, estrone, testosterone, HbA1c, and glucose were positively associated with BMI, while HDL-C, FSH, and anti-Müllerian hormone were negatively associated. Complex nonlinear relationships were observed between BMI and androstenedione, as well as systolic blood pressure.
Conclusions: The findings elucidate the significant associations between metabolic risk factors, sex hormones, and BMI. The results suggest that sex hormone imbalances, lipid disorders, and diabetes-related markers are linked to higher BMI among women of childbearing age. Accordingly, interventions targeting hormone regulation, lifestyle modification, and dietary improvement may offer pathways for BMI management in this population.
Keywords: Obesity, BMI, Sex hormones, Cardiometabolic

OP3-5

Prevalence of high inflammatory risk in atherosclerotic cardiovascular disease with and without chronic kidney disease: findings from POSEIDON – a global cross-sectional prevalence study

Hong-Ki Jeon[1*], Ann Marie Navar[2], Ling Bai[3], Jesper Falkesgaard Højen[4], Søren Lund Kristensen[4], Jimmi Mathisen[4], Hongye Ren[4], Fabrice Ivanes[4], Carolyn S P Lam[6], Ulf Landmesser[7], Kelvin Lee[8], Giovanna Liuzzo[9], Lilia Nigro Maia[10], Adam J Nelson[12], Amritanshu Shekhar Pandey[13], Pablo E Pergola[14], Kenichi Tsujita[15], Nikolaus Marx[16]

[1]Division of Cardiology, Severance Cardiovascular Hospital, Yonsei University, Republic of Korea, [2]Departments of Internal Medicine and Population and Data Sciences, UT Southwestern Medical Center, Dallas, TX, USA, United States, [3]Department of Cardiovascular Medicine, The First Affiliated Hospital of Xi'an Jiaotong University, China, [4]Novo Nordisk A/S, Søborg, Denmark, [5]Service de Cardiologie, Centre Hospitalier Universitaire de Tours, Université de Tours, France, [6]Department of Cardiology, National Heart Centre Singapore, Singapore, [7]Department of Cardiology, Angiology and Intensive Care Medicine, Deutsches Herzzentrum der Charité (DHZC), Charité – Universitätsmedizin Berlin, Germany, [8]Diabetes, Metabolism & Inflammation, Joseph Bank Laboratories, University of Lincoln, Lincoln Heart Centre, United Lincolnshire Teaching Hospitals NHS Trust, United Kingdom, [9]Department of Cardiovascular Medicine, Fondazione Policlinico Universitario A. Gemelli IRCCS, Catholic University School of Medicine, Italy, [11]Centro Integrado de Pesquisas, Hospital de Base, Sao Jose Do Rio Preto, Brazil, [12]Monash Victorian Heart Institute, Department of Cardiology, Monash University, Royal Adelaide Hospital, University of Adelaide, Australia, [13]Cambridge Cardiac Care, Department of Medicine, Cambridge Cardiac Care, McMaster University, Canada, [14]Nephrology, Renal Associates, PA, San Antonio, TX, USA, United States, [15]Department of Cardiovascular Medicine, Graduate School of Medical Sciences, Kumamoto University, Japan, [16]Department of Internal Medicine I – Cardiology, University Hospital Aachen (RWTH Aachen), Germany

Objectives: Inflammation is a risk factor for ASCVD, and elevated hsCRP can identify individuals at increased risk of CV events. However, up-to-date global data on how many patients are affected are lacking. The large-scale, global POSEIDON study (NCT06122961) addressed this gap by quantifying the contemporary global prevalence of high inflammatory risk (hsCRP ≥2 mg/L), including characteristics among individuals with ASCVD.
Methods: POSEIDON enrolled 18,904 individuals with ASCVD and/or HF across 317 sites in 18 countries (2023–2025). This analysis assessed the prevalence and characteristics of individuals with hsCRP levels ≥2 mg/L among participants with ASCVD and CKD (eGFR <60 mL/min/1.73 m^2; n=5757) or without CKD (n=7718).
Results: Of 13,475 individuals with ASCVD, the prevalence of hsCRP ≥2 mg/L was 39.3% in participants with ASCVD and CKD, and 28.3% in those with ASCVD alone, with the highest prevalences found in Latin America and the USA (Figure). Among individuals with CKD, median age was 75 years, 25.9% were women, 49.4% had T2D and 86.7% were receiving lipid-lowering therapy (LLT). In those without CKD, median age was 67 years, 21.6% were women, 36.3% had T2D and 92.3% were receiving LLT. Across cohorts, IL-6 levels were higher in groups with hsCRP ≥2 mg/L. Independent risk factors for hsCRP ≥2 mg/L were smoking, high BMI, autoimmune disease, HF, polyvascular disease and dyslipidaemia.
Conclusions: In POSEIDON, we found that despite high LLT use, ~4 in 10 individuals with ASCVD and CKD had high inflammatory risk, in addition to ~3 in 10 individuals with ASCVD alone. This highlights the importance of hsCRP assessment to identify patients who are potentially eligible for emerging therapies targeting inflammation. These novel findings arrive as hsCRP assumes increasing clinical relevance and inflammation-targeted CV trials near readout, offering timely insights that advance our understanding of inflammatory risk and may inform future clinical and scientific directions.
Keywords: ASCVD, Inflammation, hs-CRP, hsCRP, IL-6, CKD, Prevalence, Atherosclerosis, Biomarker, Risk factor

OP3-6

Real-world efficacy and tolerability of inclisiran in Malaysian patients with atherosclerotic cardiovascular disease: a single-centre experience

Kim Fong Ng[1*], Khai Ling Koh[1], Wan Tying Soh[2], Azreen Natasha Binti Azmi[2], Shiau Chin Chong[3]

[1]Cardiology, KPJ Kluang Specialist Hospital, Malaysia, [2]Pharmacy, KPJ Kluang Specialist Hospital, Malaysia, [3]Pharmacy, Hospital Sultan Ismail, Malaysia

Objectives: To evaluate real-world effectiveness and safety of inclisiran among Malaysians with established cardiovascular disease (ASCVD).
Methods: This retrospective cross-sectional study recruited adults (≥18 years) with established ASCVD initiating inclisiran between 2024 and 2025 at a tertiary cardiology clinic. Lipid profiles were assessed at baseline and 1 month post-first dose. Patients receiving second dose at 3 months had repeat lipid assessment. Adverse events were recorded.
Results: A total of 164 patients aged 53±12 years were included. Eighty-six (52.4%) patients were Chinese, 62 (37.8%) were Malays and 16 (9.8%) were Indians. At 1-month post inclisiran administration, LDL-C reduced from 4.28±0.98 mmol/L to 1.96±1.11 mmol/L (-54.2%, p<0.001), total cholesterol decreased from 6.39±0.99 mmol/L to 3.82±1.25 mmol/L (-40.2%, p<0.001), triglyceride reduced from 1.86±0.95 mmol/L to 1.47±0.69 mmol/L (-21%, p<0.001), HDL-C increased modestly from 1.2±0.31 mmol/L to 1.27±0.31 mmol/L (+5.8%, p=0.043). One-quarter of patients achieved LDL-C ≤ 1.4 mmol/L and one-third achieved >50% LDL-C reduction after a single dose of inclisiran. Among 31 patients receiving second dose, inclisiran reduced LDL-C by 42% (p<0.001), yielding time-adjusted 50% mean reduction from baseline (p < 0.001). The LDL-C reduction was comparable across Malays, Chinese and Indians (p=0.653). Inclisiran was well tolerated; only 5 of 164 patients (3%) experienced mild, self-limiting injection-site reactions.
Conclusions: This first real-world inclisiran study demonstrates rapid, substantial LDL-C reduction with a favourable safety profile in a multi-ethnic Southeast Asian cohort, supporting inclisiran integration into routine regional lipid-lowering practice.
Keywords: Inclisiran, LDL-cholesterol, Atherosclerotic cardiovascular disease

OP3-7

A Korean multidimensional social determinants of health index identifies individuals at elevated risk of severe coronary artery calcification: evidence from the CMERC cohort

Jooeun Jeon[1,2*], Hokyou Lee[2,3], Hyoeun An[3], Hyeok-Hee Lee[4], Jee-Seon Shim[2,3], Hansol Choi[2,3], Kyoung Hwa Ha[2,3], Hae Won Chung[5], Ji-Hye Kim[5], Yoosik Youm[6], Sungha Park[7], Dae Jung Kim[8], Hyeon Chang Kim[2,3]

[1]Institute of Medical and Convergence, Ewha Womans University Mokdong Hospital, Republic of Korea, [2]Yonsei Institute of Digital Health, Yonsei University, Republic of Korea, [3]Department of Preventive Medicine, Yonsei University College of Medicine, Republic of Korea, [4]Richard A. and Susan F. Smith Center for Outcomes Research in Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA, United States, [5]Department of health promotion, Severance health check-up, Yonsei University Health System, Republic of Korea, [6]Department of Sociology, Yonsei University College of Social Sciences, Republic of Korea, [7]Division of Cardiology, Severance Cardiovascular Hospital and Cardiovascular Research Institute, Yonsei University College of Medicine, Republic of Korea, [8]Department of Endocrinology and Metabolism, Ajou University School of Medicine, Republic of Korea

Objectives: Social determinants of health (SDoH) are increasingly recognized as important contributors to cardiovascular disease risk. However, substantial challenges remain in developing multidimensional SDoH measures that adequately capture social disadvantage and are applicable to Asian populations. This study aimed to develop a Korean multidimensional SDoH composite index using Boosted Regression Tree (BRT) and evaluate its association with coronary artery calcium (CAC).
Methods: We analyzed 2,388 participants aged 30-79 years from the Cardiovascular and Metabolic Diseases Etiology Research Center (CMERC) cohort who underwent CAC assessment by computed tomography. CAC severity was classified as CAC=0, CAC 1-99, and CAC ≥100. Ten SDoH indicators from social, economic, and environmental domains were incorporated into a weighted composite index based on BRT-derived relative importance. Inter-item correlations were assessed using Pearson correlation coefficients. Multivariable logistic regression models adjusted for cardiovascular risk factors were used to examine associations between the SDoH index and CAC severity.
Results: CAC was detected in 986 participants (41.3%), including 109 (4.6%) with severe CAC (CAC ≥100). The BRT model identified household income (26.5%), occupation (20.9%), neighborhood green space (15.5%), and marital status (13.7%) as the primary determinants of the composite index, collectively accounting for approximately 76% of the model's predictive power. Each one-unit increase in the SDoH index was associated with 23% higher odds of severe CAC (OR 1.23, 95% CI 1.01-1.49). Compared with the lowest quartile (Q1), adjusted ORs (95% CIs) for severe CAC were 2.26 (1.10-4.64), 2.48 (1.23-4.98), and 2.33 (1.15-4.73) for Q2, Q3, and Q4, respectively (p-trend=0.035).
Conclusions: A higher multidimensional SDoH burden was independently associated with severe CAC in Korean adults. This BRT-derived index may serve as a useful tool for identifying populations at elevated risk of advanced subclinical atherosclerosis and informing targeted cardiovascular prevention strategies.
Keywords: Social determinants of health, Coronary artery calcium, Subclinical atherosclerosis, Cardiovascular risk

OP3-8

Efficacy and safety of enlicitide decanoate, an oral macrocyclic peptide inhibitor of PCSK9, compared with bempedoic acid, ezetimibe, or bempedoic acid co-administered with ezetimibe in statin-treated adults with hypercholesterolemia: phase 3 CORALreef AddOn trial

Catapano AL[1,2], Mikhailova E[3], Navar AM[4], Banka P[5], Kordahi A[5], Corral P[6], Saxena M[7], Steg PG[8], Verma S[9], Mendizabal G[10], Zhu P[11], Ballantyne CM[12], Maria Carolina Emilia Villanueva[13*] on behalf of the authors

[1]University of Milan, Milan, Italy, [2]IRCCS Multimedica, Milan, Italy, [3]MSD (UK) Limited, London, United Kingdom, [4]University of Texas Southwestern Medical Center, Dallas, United States, [5]Merck & Co., Inc., Rahway, NJ, United States, [6]FASTA University, Mar del Plata, Argentina, [7]Barts NIHR Biomedical Research Centre, Queen Mary University of London, United Kingdom, [8]Université Paris-Cité, Assistance Publique-Hôpitaux de Paris, INSERM Unité 1148, Paris, France, [9]St. Michael's Hospital, University of Toronto, Toronto, Canada, [10]MSD Argentina, Buenos Aires, Argentina, [11]MSD China, Shanghai, China, [12]Texas Heart Institute at Baylor College of Medicine, Houston, Texas, United States, [13]MSD Philippines, Manila, Philippines

Objectives: This study evaluated the efficacy of enlicitide decanoate, an oral macrocyclic peptide proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor, vs bempedoic acid, ezetimibe, or bempedoic acid + ezetimibe.
Methods: In this Phase 3, randomized, double-blind, active-comparator trial, statin-treated adults with established atherosclerotic cardiovascular disease (ASCVD) and LDL-C ≥55 mg/dL, or at risk for ASCVD and LDL-C ≥70 mg/dL, were assigned (2:1:1:2) to enlicitide 20 mg (n=101), bempedoic acid 180 mg (n=50), ezetimibe 10 mg (n=50), or bempedoic acid 180 mg + ezetimibe 10 mg (n=100) once daily for 56 days. Primary endpoint was the mean % change from baseline in LDL-C at Day 56; key secondary endpoints were % changes from baseline in apolipoprotein B (ApoB) and non-high-density lipoprotein cholesterol (non-HDL-C).
Results: Of 301 randomized participants (37% women; 98% on moderate/high-intensity statins), 298 (99%) completed the trial. Enlicitide produced greater between-group reductions in LDL-C vs bempedoic acid (-56.7%), ezetimibe (-36.0%), and bempedoic acid + ezetimibe (-28.1%); all p<0.001 (Figure). Corresponding differences in ApoB (-47.5%, -33.6%, -26.8%) and non-HDL-C (-51.2%, -32.2%, -25.8%) also were greater with enlicitide vs each comparator (all p<0.001). Enlicitide was well tolerated.
Conclusions: Oral PCSK9 inhibitor enlicitide achieved superior LDL-C, ApoB, and non-HDL-C reductions vs bempedoic acid, ezetimibe, and bempedoic acid + ezetimibe.
Keywords: Low-density lipoprotein cholesterol, PCSK9 inhibitor, Atherosclerotic cardiovascular disease, Hypercholesterolemia, Lipid-lowering therapy

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Moderated Poster Presentation

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Moderated Poster Presentation 1

Sep 11(Fri) 12:45-13:45 | Studio 5 (6F, Conrad Seoul)

MODERATOR : Jun Namkung (Yonsei University, Republic of Korea)

MPP1-1

Comparative lipid profiling of celastrol-treated atherosclerotic ApoE-/- mice on a high-fat diet

Nurin Yasmin Mohd Khairudin[1,2*], Vimala R.M.T Balasubramaniam[3], Suhaila Abd Muid[4], Nurdiyana Nasrudin[5], Nasibah Azme[6,7]

[1]Laboratory Animal Care Unit (LACU), Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [2]Institute of Medical Molecular Biotechnology, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [3]Nutrition, Metabolism & Cardiovascular Research Centre, Institute for Medical Research, Malaysia, [4]Department of Biochemistry and Molecular Medicine, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [5]Department of Pediatrics, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [6]Department of Physiology, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, [7]Department of Medical Education, Faculty of Medicine, Universiti Teknologi MARA, Malaysia

Objectives: This study aimed to evaluate the effects of celastrol (≥98% purity; Sigma-Aldrich, St. Louis, MO, USA; Cat No. C0869) administration on the lipid profile of ApoE-/- mice subjected to a high-fat diet (HFD) and to compare lipid parameters total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) across treatment and control groups.
Methods: In this study, 18 male ApoE-/- mice (8 weeks old) were randomly assigned to three groups (n= 6): normal diet control, HFD control, and HFD with celastrol treatment (1.5 mg/kg/day, i.p) for 12 weeks. Plasma samples were collected after 4 weeks of 2% DMSO (vehicle) and 1.5mg/kg celastrol treatment. Lipid profiles including total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were quantified using automated biochemistry analyzer (Model: BA200 BA400, BioSystems S.A) with species- specific reference. The data retrieved were calculated, and statistical comparisons were performed using one-way ANOVA.
Results: The results show different levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) across the three groups; normal diet control, HFD control, and HFD with celastrol treatment (1.5 mg/kg/day, i.p). HFD-fed mice exhibited elevated TC and LDL-C ($p<0.01$) levels compared to normal chow controls. Celastrol-treated mice demonstrated marked reductions in TC (16.1%), and LDL-C (25.5%) compared to untreated HFD controls, alongside modest increases in HDL-C (7.1%).
Conclusions: Administration of celastrol effectively improve HFD-induced dyslipidemia in atherosclerotic ApoE-/-mice, suggesting its potential as a therapeutic agent for managing hyperlipidemia and reducing atherosclerotic risk. These findings support further investigation into celastrol's lipid-modulating mechanisms and its translational applications in cardiovascular disease prevention.
Keywords: Celastrol, ApoE knockout mice, Atherosclerosis, Lipid profile, High-fat diet

MPP1-2

MMP11 promotes endothelial senescence and atherosclerosis through proteolytic cleavage of FLRT2

Hyun Jung Hwang[1,2,3*], Jae-Seon Lee[1,2,3]

[1]Research Center for Controlling Intercellular Communication, College of Medicine, Inha University, Incheon, Republic of Korea, [2]Department of Molecular Medicine, College of Medicine, Inha University, Incheon, Republic of Korea, [3]Program in Biomedical Science and Engineering, Inha University, Incheon, Republic of Korea

Objectives: Endothelial senescence is a key contributor to vascular aging and atherosclerosis, but the upstream mechanisms that induce this process remain incompletely understood. This study aimed to investigate whether matrix metalloproteinase 11 (MMP11) regulates endothelial senescence and atherosclerosis through cleavage of fibronectin leucine-rich repeat transmembrane protein 2 (FLRT2).
Methods: MMP11 expression was examined in senescent endothelial cells, aged rat and human arterial tissues, and atherosclerotic lesions from $ApoE^{-/-}$ mice. The effects of MMP11 on FLRT2 cleavage, ITGB4 signaling, and downstream senescence-associated pathways were assessed in endothelial cells. Endothelial senescence, monocyte adhesion, and inflammatory responses were evaluated following pharmacological inhibition of MMP11 using P300:103 or ITGB4 using AC2825. The therapeutic effects of these inhibitors were further examined in $ApoE^{-/-}$ mice with atherosclerosis.
Results: MMP11 expression was markedly increased in senescent endothelial cells, aged arterial tissues, and atherosclerotic lesions. Senescence-associated MMP11 was secreted and directly cleaved membrane-bound FLRT2, resulting in activation of ITGB4 signaling and downstream pro-senescent pathways. MMP11-mediated FLRT2 cleavage promoted endothelial senescence and pro-atherogenic phenotypes, including increased monocyte adhesion and vascular inflammation. Pharmacological inhibition of MMP11 or ITGB4 significantly attenuated endothelial senescence and inflammatory responses in vitro and reduced atherosclerotic lesion formation, lipid deposition, and macrophage infiltration in $ApoE^{-/-}$ mice.
Conclusions: These findings demonstrate that MMP11 promotes endothelial senescence and atherosclerosis by cleaving FLRT2 and activating ITGB4-mediated pro-senescent signaling. The MMP11-FLRT2-ITGB4 axis may represent a potential therapeutic target for vascular aging and atherosclerotic cardiovascular disease.
Keywords: Endothelial cell senescence, MMP11, FLRT2, ITGB4, Atherosclerosis

MPP1-3

LGI3 as a novel therapeutic target in atherosclerosis: regulation of macrophage inflammation and plaque progression

Jing Jin*, Goo Taeg Oh

Life Science, Heart-Immune-Brain Network Research Center, Ewha Womans University, Republic of Korea

Objectives: Atherosclerosis is a major chronic cardiovascular disease and a leading cause of acute cardiovascular events. While adipose tissue-derived factors are increasingly recognized as contributors to atherosclerosis, the role of leucine rich repeat LGI family member 3 (LGI3), a proinflammatory adipokine secreted from adipose tissue macrophages in obesity, remains poorly understood. This study aimed to elucidate the functional role of LGI3 in atherogenesis and evaluate the therapeutic potential of LGI3 inhibition.

Methods: Atherosclerosis progression and immune cell infiltration were assessed in ApoE-/- mice and ApoE-/- Lgi3-/- double knockout mice after 16 weeks of high-fat diet feeding. In vitro, THP-1 macrophages were treated with oxidized LDL, TNF-α, and IFN-γ (OTI) to mimic the atherosclerotic microenvironment. The expression levels of LGI3, its receptors (ADAM22, ADAM23), and proinflammatory cytokines (CCL2, IL-1β, IL-6) were quantified. The effects of LGI3 inhibition were examined using its antagonist peptide P34, by measuring mRNA expression of proinflammatory mediators and NF-κB signaling activity. Additionally, ApoE-/- mice were treated with P34 to assess its therapeutic efficacy in vivo.

Results: Genetic deletion of Lgi3 in ApoE-/- mice significantly reduced atherosclerotic plaque formation and macrophage infiltration in both the aorta and perivascular adipose tissue (PVAT). In THP-1 macrophages, OTI treatment robustly increased the expression of LGI3, ADAM22, ADAM23, and proinflammatory cytokines CCL2, IL-1β, and IL-6. Pharmacological inhibition of LGI3 with P34 suppressed the expression of these cytokines and attenuated NF-κB pathway activation. In ApoE-/- mice, P34 administration significantly reduced aortic plaque formation without affecting body weight.

Conclusions: LGI3 promotes atherosclerotic inflammation and plaque development through NF-κB-mediated proinflammatory signaling. Both genetic and pharmacological inhibition of LGI3 effectively suppresses inflammation and attenuates atherosclerosis, highlighting LGI3 as a potential therapeutic target for cardiovascular disease.

Keywords: Atherosclerosis, Macrophage, LGI3

MPP1-4

Involvement of vimentin in oxLDL-induced microparticle release from macrophages

Soonhyeong Oh*, Wonkyoung Cho, Young Mi Park

Department of Molecular Medicine, College of Medicine, Ewha Womans University, Republic of Korea

Objectives: Microparticles (MPs) are membrane-derived vesicles involved in intercellular communication. Several clinical studies have shown that high levels of MPs circulate in the blood of patients with atherothrombotic diseases, where they could serve as a useful biomarker of vascular injury and a potential predictor of cardiovascular mortality and major adverse cardiovascular events. However, the mechanism of MP generation has not been clearly defined. Vimentin is a type III intermediate filament protein that maintains cytoskeletal integrity and has been implicated in various cellular processes. In this study, we investigated the role of vimentin in MP generation

Methods: Peritoneal macrophages isolated from wild-type and vimentin-deficient mice were stimulated with oxLDL. MPs released into culture supernatants were collected by differential ultracentrifugation and quantified by flow cytometry and nanoparticle tracking analysis (NTA). Changes in vimentin expression and cytoskeletal organization associated with MP generation were assessed by Western blotting and immunofluorescence

Results: Oxidized low-density lipoprotein (oxLDL) induced MP release in a time- and dose-dependent manner. Notably, vimentin deficiency significantly enhanced MP production, indicating that vimentin acts as a negative regulator of MP biogenesis.
Mechanistically, oxLDL-induced vimentin phosphorylation was critical for MP formation. A phospho-mimetic mutant vimentin (V72D) increased MP release, whereas a non-phosphorylatable mutant vimentin (V72A) had no effect. Inhibition of protein kinase A (PKA) which phosphorylates vimentin reduced MP formation. oxLDL-induced vimentin phosphorylation leads to disassembly of filamentous vimentin and transient colocalization with calpain, and calpain inhibition attenuated MP release.
RNA sequencing followed by Gene Ontology (GO) and Gene Set Enrichment Analysis (GSEA) was performed to compare basal and oxLDL-induced transcriptional profiles between wild-type ($Vim^{+}/^{+}$) and vimentin-deficient ($Vim^{-}/^{-}$) macrophages. GO analysis revealed enrichment of biological processes related to cytoskeleton organization and metabolism and GSEA showed increased metabolic and cytoskeletal pathways in $Vim^{-}/^{-}$ macrophages. Following oxLDL stimulation, GO analysis indicated enrichment of processes associated with extracellular matrix organization, secretion, and vesicle-mediated transport. Consistently, GSEA revealed increased secretion-related pathways, whereas inflammatory, apoptotic, and lipid metabolic pathways were downregulated in $Vim^{-}/^{-}$ macrophages compared to $Vim^{+}/^{+}$.

Conclusions: In conclusion, vimentin restrains oxLDL-induced MP biogenesis through phosphorylation-dependent cytoskeletal remodeling, providing new insight into macrophage membrane dynamics and its potential as a therapeutic target in atherosclerosis.

Keywords: Microparticle, Macrophage, Vimentin

MPP1-5

Aging rewires the vascular-wall regulatory landscape to redirect diet-induced atherosclerosis

Ju Young Kim[1,2*], Hyeonmin Gil[1], Jong Kyoung Kim[1]

[1]Department of Life Sciences, Pohang University of Science and Technology (POSTECH), Republic of Korea,
[2]Biomedical Research Institute, Kyungpook National University Hospital, Republic of Korea

Objectives: Aging and metabolic stress are principal drivers of atherosclerosis, yet how aging epigenomically primes vascular cell states prior to metabolic stress has not been systematically resolved at single-cell resolution.
Methods: Here, we performed time-course single-cell multiome profiling of aortas from Ldlr-deficient mice across age at diet initiation, diet type, and diet duration, generating paired transcriptomic and chromatin accessibility landscapes across endothelial cells, vascular smooth muscle cells, fibroblasts, and macrophages.
Results: We resolved 19 transcriptionally and epigenomically distinct cell states within these four lineages, including diet-associated expansion of Dkk2-expressing endothelial states, phenotypic switching of vascular smooth muscle cells toward modulated disease-associated states, and diversification of macrophages into lipid-associated and remodeling Trem2-positive populations. Across lineages, aging induced coordinated transcriptional changes and remodeling of epigenetic states. Strikingly, aged chow-fed aortas already harbored transcriptional and chromatin accessibility signatures characteristic of diet-induced disease states, indicating that aging establishes a disease-permissive vascular landscape before exposure of metabolic challenge. Under high-fat diet, aging was associated with cell-type-specific remodeling of transcriptional and chromatin regulatory programs.
Conclusions: Together, these findings establish that aging creates a disease-permissive vascular-wall regulatory landscape that reshapes the trajectory of diet-induced atherosclerosis, providing a single-cell multiomic framework for understanding how epigenomic priming by aging modifies vascular responses to metabolic stress.
Keywords: Single cell multiomics, Atherosclerosis

MPP1-6

A novel cyclodextrin derivative modulates macrophage lipid accumulation and inflammatory responses relevant to atherosclerosis

Soobin Chang[1*], Heegon Kim[2], Yujin Ko[1], ChaeYoung Kim[1], Shinhee Park[1], Sangeun Park[1], Kyuseong Park[1], Jae-hoon Choi[1]

[1]Department of Life Science, Hanyang University, Republic of Korea, [2]Renatus Co., Ltd., Republic of Korea

Objectives: Atherosclerosis is characterized by the accumulation of cholesterol-rich plaques and chronic inflammation within the arterial wall. However, current lipid-lowering therapies have limited capacity to directly remove cholesterol from established atherosclerotic plaques. Cyclodextrins have been reported to facilitate cholesterol mobilization and efflux, suggesting their potential as therapeutic agents for plaque-targeted intervention. In this study, we evaluated the effects of a novel cyclodextrin derivative on macrophage lipid accumulation and inflammatory responses associated with atherosclerosis.
Methods: Foam cell formation was induced in Raw264.7 macrophages by treatment with oxidized low-density lipoprotein, followed by administration of the novel cyclodextrin derivative. Intracellular lipid accumulation was assessed using BODIPY staining. The expression of cholesterol transport-related genes was evaluated by quantitative PCR. Inflammatory responses were evaluated by measuring IL-1β secretion using ELISA in bone marrow-derived macrophages derived from C57BL/6J mice. For in vivo evaluation, Ldlr-/- mice were maintained on a Western diet for 12 weeks and administered the novel cyclodextrin derivative.
Results: Treatment with the novel cyclodextrin derivative reduced oxLDL-induced lipid accumulation in Raw264.7 macrophages and increased the expression of genes involved in cholesterol efflux. The cyclodextrin derivative also decreased IL-1β secretion in BMDMs, suggesting modulation of macrophage inflammatory responses. Furthermore, in vivo analyses indicated modulation of atherosclerosis-associated phenotypes.
Conclusions: These findings suggest that the novel cyclodextrin derivative modulates macrophage cholesterol metabolism and inflammatory responses. Therefore, this compound may have potential as a therapeutic strategy for modulating cholesterol accumulation and inflammation associated with atherosclerosis.
Keywords: Atherosclerosis, Cyclodextrin, Macrophage, Cholesterol efflux, Inflammation

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Moderated Poster Presentation 2

Sep 11(Fri) 12:45-13:45 | Studio 6 (6F, Conrad Seoul)

MODERATOR : Jaehoon Chung (Gachon University, Republic of Korea)

MPP2-1

Deciphering the differential impact of ART classes on vascular morphology and lipid profiles in HIV-positive patients with coronary artery disease

Nuriiat Efendieva*, Oleg Petrovich Shevchenko

Cardiology, Pirogov Russian National Research Medical University, Russian Federation

Objectives: Antiretroviral therapy (ART) improves survival in HIV patients but increases cardiovascular risk. The differential effects of specific ART classes on arterial wall structure, beyond traditional lipids, remain unclear. This study investigated associations between various ART regimens, serum lipids, and vascular morphology assessed by optical coherence tomography (OCT) in patients with coronary artery disease (CAD).

Methods: A cross-sectional analysis included 113 CAD patients: 42 HIV-positive on triple ART and 71 HIV-negative controls. All received statins. OCT evaluated intimal thickness, three-layer wall preservation, plaque count, and calcification. Lipid panels measured total (TC) and non-HDL cholesterol. HIV patients were stratified by regimen: 100% on NRTIs, 14% on fusion inhibitors (FIs), 24% on NNRTIs, 24% on protease inhibitors (PIs), and 17% on integrase inhibitors (INSTIs). Groups were age- and risk factor-matched.

Results: Baseline TC and non-HDL did not differ between HIV-positive and HIV-negative groups (6.0±1.0 vs. 5.9±0.9 mmol/L, p=0.558; 4.0±1.0 vs. 3.8±0.8 mmol/L, p=0.609, respectively). However, class-specific effects emerged. PI use was associated with significantly greater intimal thickness (p<0.001), higher plaque count (p=0.019), and more severe calcification (p=0.029). INSTI use uniquely correlated with elevated TC (p=0.008). NNRTIs and FIs showed no significant adverse effects on OCT parameters, though FI demonstrated borderline association with plaque number (p=0.050). No class affected three-layer wall preservation.

Conclusions: In statin-treated CAD patients, ART impact on cardiovascular risk is drug-class specific. PIs showed the most detrimental vascular remodeling on OCT, while INSTIs affected lipid metabolism without corresponding structural damage. These findings advocate for ART selection guided not solely by lipid levels but by direct vascular effects visualized through advanced imaging.

Keywords: Optical coherence tomography, Human immunodeficiency virus

MPP2-2

Autophagy-based restoration of glucosylceramide catabolism in Gaucher disease

Minji Kim[1*], Eun Nam Choi[1], Yong Tae Kwon[2], Joo-Won Park[1]

[1]Department of Biochemistry, College of Medicine, Ewha Womans University, Republic of Korea,
[2]Cellular Degradation Biology Center and Department of Biomedical Sciences,
College of Medicine, Seoul National University, Republic of Korea

Objectives: Glucosylceramide is a glycosphingolipid composed of glucose and ceramide, and its lysosomal accumulation is a hallmark of Gaucher disease caused by deficient β-glucocerebrosidase (GCase) activity. Impaired lysosomal trafficking or reduced activity of GCase limits glucosylceramide degradation, leading to pathological lipid storage. Autophagy is a lysosome-dependent catabolic pathway that delivers selected cargoes to lysosomes, and p62/SQSTM1 functions as a representative autophagic cargo adaptor. Based on this lysosomal delivery mechanism, we developed a lysosome-targeting chimera platform, termed LYSOTAC, composed of GCase-binding ligands linked to autophagy-targeting ligands. This strategy was designed to enhance lysosomal localization of GCase and restore its lipid-hydrolyzing activity within the lysosomal compartment, rather than inducing degradation of the enzyme.

Methods: As a proof of concept, we generated LYSOTAC molecules targeting GCase and evaluated their effects on lysosomal enzyme delivery, enzyme activity, and glucosylceramide metabolism. We used Gaucher disease fibroblasts and LIMP-2 deficient cells.

Results: Enzymes destined to the lysosomes are not degraded due to glycosylation. The lysosomal delivery and enzyme activity of GCase were markedly enhanced by LYSOTAC technology, leading to the reduction of substrate, glucosylceramide in Gaucher disease fibroblasts. Unlike other lysosomal enzymes targeted via mannose 6-phosphate, GCase is transported to the lysosomes via binding to LIMP-2. Whereas LYSOTAC compounds increased total, ER, and post-ER forms of GCase in WT cells, they mainly increased total and ER GCase in LIMP-2 deficient cells, implicating that LIMP-2 is essential to produce the post-ER GCase form. These data collectively demonstrate that LYSOTAC restores cellular GCase levels and enzyme activity irrespective of LIMP-2.

Conclusions: LYSOTAC technology may provide a new approach to correct defective lipid degradation in Gaucher disease and may complement existing therapeutic modalities aimed at lysosomal glycosphingolipid homeostasis.

Keywords: Glucosylceramide, Autophagy, β-glucocerebrosidase

MPP2-3

Loss of PCSK9 enhances ANP expression to attenuate post-MI cardiac inflammation

Na Hyeon Yoon*, Goo Taeg Oh

Life Science, Ewha Womans University, Republic of Korea

Objectives: Myocardial infarction (MI) induces cardiomyocyte apoptosis, which is subsequently followed by compensatory left ventricular hypertrophy to offset the loss of functional myocardium. Proprotein convertase subtilisin/kexin type 9 (PCSK9) has been identified as a key regulator of cardiomyocyte viability and cardiac function by modulating tissue architecture and impairing contractile performance. Atrial natriuretic peptide (ANP), predominantly synthesized by atrial cardiomyocytes, is known to be upregulated in response to cardiac wall stress and volume overload. Despite these individual roles, the potential regulatory relationship between PCSK9 and ANP has not yet been elucidated. Therefore, we aim to investigate whether PCSK9 modulates ANP expression in the context of MI.
Methods: To investigate the effects of PCSK9 deficiency on cardiac function and inflammation following myocardial infarction (MI), 9-11-week-old male PCSK9+/+ and PCSK9-/- mice were subjected to MI by ligation of the left anterior descending (LAD) coronary artery. Post-MI cardiac function was assessed using echocardiography, Masson's trichrome staining, quantitative PCR, western blotting, and immunohistochemistry. In addition, levels of atrial natriuretic peptide (ANP), which is typically elevated following MI, were measured by ELISA. The anti-inflammatory effects of ANP were further evaluated using bone marrow-derived macrophages (BMDMs).
Results: PCSK9-deficient mice exhibited improved cardiac function, increased collagen deposition, and reduced infarct size in the ischemic myocardium. Furthermore, PCSK9-/- mice demonstrated a significant survival benefit following acute MI. These cardioprotective effects may be attributable to the preserved contractile capacity of cardiomyocytes in the absence of PCSK9, leading to elevated expression of the cardiac hormone ANP. ANP was found to exert anti-inflammatory effects by suppressing macrophage-mediated inflammation, thereby mitigating macrophage-induced cardiomyocyte death.
Conclusions: In conclusion, this study demonstrates that elevated levels of cardiac ANP in ischemic hearts lacking PCSK9 activity act as a suppressor of inflammatory responses. The anti-inflammatory properties of ANP likely contribute to the preservation of cardiac function in PCSK9-deficient hearts.
Keywords: PCSK9, MI, ANP

MPP2-4

BMI-mediated inflammatory pathways and miR-92a-3p dysregulation: novel insights into cardiovascular risk stratification

Fitri Bin Kahar[1*], Abd-Malek Fatin-Syazwani[1], Nurul Izzati Abdullah[1], Fitri Kahar[1], Amrina Mohamad Amin[1], Sabariah Md Noor[1], Md Parvez Eusof Izzudin[2], Shariff-Ghazali Sazlina[2], Norshariza Nordin[3]

[1]Department of Pathology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia,
[2]Family Medicine Clinic, Hospital Sultan Abdul Aziz Shah, Persiaran Mardi, Universiti Putra Malaysia, 43400, Serdang, Selangor, Malaysia,
[3]Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia

Objectives: Cardiovascular disease (CVD) remains the leading cause of global mortality. An elevated body mass index (BMI) contributes to chronic inflammation and cytokine release, particularly tumour necrosis factor-alpha (TNF-α), which disrupts lipid metabolism and increases remnant cholesterol (RC). miR-92a-3p, a mechanosensitive microRNA, plays a critical role in endothelial dysfunction and represents a promising circulating biomarker. This study investigates the relationships between BMI, RC, TNF-α, and miR-92a-3p expression in cardiovascular risk stratification using the Framingham Risk Score (FRS).
Methods: This cross-sectional study enrolled 150 adults aged 30-75 years without known CVD from a tertiary primary health centre in Malaysia. Fasting blood samples were collected for lipid profiling, RC, TNF-α (Luminex), and plasma miR-92a-3p (qRT-PCR). Participants were stratified by FRS into low (<10%), moderate (10-20%), and high (>20%) risk categories. Spearman correlation and multivariate ordinal logistic regression were performed.
Results: The median age was 55 years, with 59.3% female. BMI correlated positively with RC (Spearman ρ=0.176, p=0.031). TNF-α was significantly associated with higher FRS categories (AOR=1.16, 95% CI: 1.08-1.23, p<0.001). Downregulated miR-92a-3p expression corresponded to increased CVD risk, with median (IQR) values of 0.00 (1.99) in high-risk groups compared to 1.03 (2.52) in low-risk and 0.03 (2.47) in moderate-risk groups.
Conclusions: This study reveals interconnected pathways linking metabolic dysfunction, inflammation, and miRNA dysregulation in CVD pathogenesis. The progressive downregulation of miR-92a-3p with increasing risk may reflect miRNA rapid expression during chronic inflammatory stress or compensatory protective downregulation, supported by the BMI which has positive correlation with RC. These findings highlight miR-92a-3p as a minimally invasive molecular biomarker with potential for integration into multi-parametric risk assessment platforms. Longitudinal studies are needed to determine whether miR-92a-3p downregulation represents disease progression or adaptive response.
Keywords: Remnant cholesterol, microRNA, Cardiovascular disease, Endothelial dysfunction, Framingham risk score

MPP2-5

Whole-body 18F-FDG PET demonstrates coordinated splenic and arterial inflammatory activation in acute ischemic stroke

Sue Young Ha*, Jiyeon Ha, Jeong-Min Kim

Department of Neurology, Seoul National University Hospital, Seoul National University College of Medicine, Republic of Korea

Objectives: Experimental studies have identified the brain-spleen axis as an important mechanism linking cerebral ischemia with systemic immune activation. However, in vivo evidence demonstrating coordinated splenic and arterial inflammatory activation in humans remains limited. We investigated whole-body 18F-FDG PET findings in patients with acute ischemic stroke (AIS).

Methods: Whole-body 18F-FDG PET was performed in 160 AIS patients and 334 asymptomatic controls. Splenic metabolic activity was assessed using splenic SUV and Spleen/Aorta target-to-background ratio (TBR). Arterial inflammation was assessed using Thoracic Aorta SUVpeak. CT-defined calcified atherosclerotic burden was quantified as the number of vascular territories with calcified plaque (0-7).

Results: Compared with controls, AIS patients demonstrated significantly higher splenic SUV (2.17 vs. 1.79; $p<0.001$) and Spleen/Aorta TBR (0.968 vs. 0.861; $p<0.001$). Within the AIS cohort, splenic SUV correlated with hs-CRP ($\rho=0.20$, $p=0.011$) and Thoracic Aorta SUVpeak ($\rho=0.59$, $p<0.001$). Thoracic Aorta SUVpeak increased progressively with greater CT-defined calcified atherosclerotic burden and remained independently associated with the number of involved vascular territories after multivariable adjustment ($\beta=0.085$ per vascular territory; $p<0.001$). Higher Spleen/Aorta TBR was also associated with greater stroke severity ($\rho=0.245$, $p=0.002$).

Conclusions: Whole-body 18F-FDG PET demonstrates coordinated splenic metabolic activation and arterial inflammation in acute ischemic stroke, consistent with activation of the brain-spleen axis. Relative splenic metabolic activity referenced to concurrent aortic inflammation was increased after stroke, while aortic FDG uptake reflected the burden of CT-defined systemic calcified atherosclerosis. These findings support multi-organ FDG-PET as a useful imaging approach for investigating systemic inflammatory responses in atherosclerotic disease.

Keywords: FDG PET, Atherosclerosis, Ischemic stroke

MPP2-6

Revealing the hidden signals: prognostic impact of pericoronary fat attenuation index (FAI) in major adverse cardiovascular events (MACE) and coronary plaque instability - a systematic review and meta-analysis

Muhamad Fajri Addai*, Elen, Celly A. Atmadikoesoemah, Bambang Widyantoro

Cardiology and Vascular Medicine, National Cardiovascular Center Harapan Kita - Faculty of Medicine, Universitas Indonesia, Indonesia

Objectives: This study aims to provide robust evidence for the potential role of FAI in improving the diagnosis, risk assessment, and management of coronary artery disease (CAD), as well as predicting MACE and high-risk plaque.

Methods: A systematic search was conducted across major medical databases to identify observational studies that assessed the association between FAI and MACE or CAD published until September 2025, following PRISMA guidelines. The endpoints were the prediction of MACE and high-risk plaque using FAI and the weighted mean difference (WMD) of FAI attenuation between case and control groups. Data analysis was performed in RevMan 5.41 to calculate pooled measures of Hazard Ratios (HR), 95% Confidence Intervals (95% CI), and WMD.

Results: Forty-three studies involving 58,492 subjects were included. Higher FAI demonstrated a significant predictive value for MACE and high-risk plaque, with a pooled HR of 2.38 (95% CI [1.75-3.23], $p<0.00001$) and HR of 1.2 [1.08-1.32], $p<0.00001$. Among patients with ACS, FAI was significantly higher in the culprit lesions compared to non-culprit lesions, with a WMD of 5.32 [3.07-7.57], $p<0.00001$. FAI was also higher in ACS patients compared to stable CAD patients (WMD 5.93 [4.44-7.42], $p<0.00001$). FAI was higher in high-risk plaque compared to low-risk plaque, with a WMD of 7,98 [5.84-10.11], $p<0.00001$. In CAD patients, FAI values were significantly higher compared to non-CAD patients (WMD 5.67 [3.98-7.36], $p<0.00001$). FAI was also significantly higher in patients who experienced MACE compared to those without MACE (WMD 4.11 [2.11-6.11], $p<0.0001$).

Conclusions: FAI is a promising non-invasive biomarker for assessing perivascular inflammation, stratifying cardiovascular risk, and guiding clinical decision-making in CAD. It holds substantial potential as a prognostic tool for MACE and in differentiating the severity of CAD manifestations. However, prospective studies with longer follow-up are needed to elucidate its clinical utility further.

Keywords: Fat attenuation index, Coronary artery disease, Major adverse cardiovascular events, Acute coronary syndrome, Meta-analysis

MPP2-7

Circulating PD-L1+CD10- immature neutrophils and integrated immunometabolic signatures associated with diabetic kidney disease severity in type 2 diabetes

Junseo Kim[1,2,5*], Seong Eun Lee[1,5], Na Rae Choi[1], Jiyeon Yoon[1,2,5], Zhang Jing[1,2,5], Junyoung O. Park[4], Da Hyun Kang[3], Bon Jeong Ku[1,3], Yea Eun Kang[1,3,5]

[1]Research Center for Endocrine and Metabolic Diseases, Integrated Disease Research Institute, Chungnam National University Hospital & College of Medicine, Republic of Korea, [2]Department of Medical Science, Chungnam National University College of Medicine, Republic of Korea, [3]Department of Internal Medicine, Chungnam National University Hospital & College of Medicine, Republic of Korea, [4]Department of Chemical and Biomolecular Engineering, University of California, Los Angeles, United States, [5]System Network Inflammation Control Research Center, Chungnam National University Hospital & College of Medicine, Republic of Korea

Objectives: Diabetic kidney disease (DKD) is a major microvascular complication of type 2 diabetes mellitus (T2DM), but the immune and metabolic changes associated with disease progression remain incompletely defined. We aimed to determine whether circulating PD-L1+CD10- immature neutrophils and integrated immunometabolic signatures are associated with DKD severity.
Methods: Peripheral blood from 8 healthy controls and 64 patients with T2DM was analyzed by flow cytometry to characterize high-density neutrophil subsets according to CD10 maturation status and PD-L1 expression. Paired plasma proteomic and untargeted metabolomic profiling was performed in 8 healthy controls and 59 patients with T2DM. Associations with estimated glomerular filtration rate (eGFR), CKD stage, and KDIGO risk category were assessed. Multi-omics factor analysis integrated immune, proteomic, and metabolomic features. Kidney biopsy specimens and BXD mouse multi-omics datasets were used for tissue and cross-species validation.
Results: Circulating PD-L1+CD10- immature high-density neutrophils were increased in T2DM, inversely correlated with eGFR, and enriched in advanced CKD and high KDIGO risk groups. Proteomics showed progressive activation of TNF/TNFRSF-related signaling, endothelial barrier regulation, cell adhesion, and vascular stress pathways. Metabolomics identified stepwise alterations in carbohydrate, amino-acid, and mitochondrial metabolites, including uridine, ribose, cystine, and glutamine. Multi-omics integration identified an immunometabolic factor associated with PD-L1+CD10- neutrophils, renal function decline, and high-risk DKD. Factor-associated proteins, including VSIG2, TNFRSF10B, VEGFA, CXCL9, TNFRSF11A, and TREM1, correlated with kidney injury traits in BXD mice. The integrated proteo-metabo-immune model discriminated KDIGO risk groups better than protein-only or metabolite-only models (AUC 0.951 vs. 0.926 and 0.870). MPO+CD10- immature neutrophils were increased in kidney tissue from advanced DKD.
Conclusions: PD-L1+CD10- immature neutrophils are associated with coordinated immune, vascular, and metabolic remodeling in DKD. Integrated immunometabolic profiling may improve risk stratification of diabetic microvascular complications.
Keywords: Diabetic kidney disease, Neutrophil, Immunometabolism

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Moderated Poster Presentation 3

Sep 11(Fri) 12:45-13:45 | Studio 7 (6F, Conrad Seoul)

MODERATOR : Mi-Hyang Jung (The Catholic University of Korea, Republic of Korea)

MPP3-1

Comparative cardiovascular risk in monogenic familial hypercholesterolemia versus polygenic hypercholesterolemia: a meta-analysis of long-term cohorts

Moaz Abouelmagd*, Amr Elrosasy, Mohammed Fathi Abduljalil

Kasr Alainy Faculty of medicine, Cairo University, Egypt

Objectives: Familial hypercholesterolemia (FH) is a common autosomal dominant disorder characterized by markedly elevated low-density lipoprotein cholesterol (LDL-C) from birth and substantially increased cardiovascular risk. This systematic review aims to investigate whether monogenic hypercholesterolemia confers proportionally greater cardiovascular risk than polygenic hypercholesterolemia, a hypothesis that remains incompletely characterized across long-term cohorts.
Methods: Systematic review and meta-analysis conducted per PRISMA 2020. PubMed, EMBASE, Cochrane Library, and Web of Science were searched to December 2025. We included prospective or retrospective cohort studies comparing monogenic FH and/or polygenic hypercholesterolemia with general population controls, or directly comparing both conditions, with a minimum 4-year follow-up. The primary outcome was major adverse cardiovascular events (MACE). We preferentially extracted maximally adjusted risk ratios (RRs); where unavailable, crude estimates were used. RRs were pooled using DerSimonian-Laird random-effects models. Meta-regression with mean LDL-C as a moderator was conducted to formally test whether LDL-C differences accounted for residual heterogeneity in risk estimates across groups.
Results: Fifteen studies (18,175 monogenic FH; 36,350 polygenic; 163,700 controls) met inclusion criteria. Monogenic FH was associated with a pooled RR of 2.10 (95% CI: 1.99–2.21) versus general population controls and polygenic hypercholesterolemia with RR 1.36 (95% CI: 1.31–1.41). The direct comparison yielded RR 1.54 (95% CI: 1.44–1.65) for monogenic versus polygenic hypercholesterolemia. Sensitivity analysis restricted to adjusted estimates (n=12) produced estimates that were similar (RRs of 2.09, 1.35, and 1.54, respectively). Meta-regression demonstrated that LDL-C levels did not explain the residual risk differential between groups (slope=0.070, p=0.581 for monogenic risk; slope=0.026, p=0.942 for the monogenic vs polygenic comparison).
Conclusions: Monogenic FH is associated with approximately 54% greater cardiovascular risk than polygenic hypercholesterolemia (RR 1.54), and this excess risk is not explained by LDL-C differences between groups in meta-regression. These findings support differentiated cardiovascular risk stratification and earlier, more aggressive lipid-lowering intervention in genetically confirmed monogenic FH.
Keywords: Familial hypercholesterolaemia, Polygenic hypercholesterolaemia, Cardiovascular risk, Meta-analysis, MACE, LDL cholesterol

MPP3-2

Lysosomal acid lipase deficiency misdiagnosed as familial hypercholesterolaemia: a family case series with a novel homozygous LIPA variant and dramatic response to enzyme replacement therapy

Koukab AL Farsi[1*], Prof Khalid AL Rassadi[2]

[1]Clinical Biochemistry/Lipid Clinic, Department of Clinical Biochemistry, Sultan Qaboos University Hospital, Muscat, Sultanate of Oman, Oman, [2]clinical Biochemistry, College of Medicine at Sultan Qaboos University, Oman

Objectives: Lysosomal acid lipase deficiency (LAL-D) is a rare, underdiagnosed autosomal recessive disorder that masquerades as familial hypercholesterolaemia or NAFLD, leading to diagnostic delay and preventable end-organ damage. We report two siblings with LAL-D/CESD phenotype— a 43 years old index patient who was 26 at age of presentation, and his affected sister — describing their diagnostic workup, and response to sebelipase alfa enzyme replacement therapy.
Methods: The index patient and his extended family were followed longitudinally at a specialist lipid clinic. Investigations included lipid profiles, liver biochemistry, abdominal imaging, transient elastography, and liver biopsies at two timepoints. LAL enzyme activity was measured by dried blood spot fluorometric assay. Whole-exome sequencing identified a LIPA variant mutation. The index case, and his 38 years old sister received weight-based intravenous sebelipase alfa for six months, with pre- and post-treatment biochemical and histological assessment.
Results: The index patient presented at age 26 with LDL-C 10.3 mmol/L, raised transaminases. Liver enzymes paradoxically worsened despite statin therapy (a decisive diagnostic clue). LAL enzyme activity was 0.07. A homozygous LIPA c.607G>A p.(Val203Met) novel variant was identified. Liver biopsy showed microvesicular steatosis, foamy macrophages, cholesteryl ester clefts, and progressive fibrosis. Cascade screening revealed many other affected relatives. Following six months of sebelipase alfa, the index patient achieved LDL-C 1.8, ALT 47, AST 34 U/L. His 38-year-old affected sister — baseline LDL-C 6.9 mmol/L, ALT 114 U/L — reached LDL-C 1.2 mmol/L, ALT 25, and AST 23 U/L.
Conclusions: This family case series underscores LAL-D as an under-recognised, reversible cause of combined dyslipidaemia and chronic liver disease. The hallmark dissociation between lipid- lowering response and hepatic deterioration should prompt enzymatic and genetic evaluation.
Keywords: Lysosomal acid lipase deficiency, Cholesteryl ester storage disease, LIPA gene variant, Sebelipase alfa

MPP3-3

Effect of roxadustat on lipid profile in adults with chronic kidney disease: a systematic review and meta-analysis of randomized controlled trials

Ammar Yasser Ali*, Mohamed Rizk, Ahmed R. A. Moustafa, Rashad G. Mohamed, Nada Mahmoud Abdelkarim, Radwa M. Abdelsattar, Mohamed A. Wafa

Faculty of Medicine, Mansoura University, Egypt

Objectives: Dyslipidemia is a major complication in chronic kidney disease (CKD) and contributes substantially to the elevated cardiovascular risk seen in these patients. Roxadustat, a hypoxia-inducible factor prolyl-hydroxylase inhibitor (HIF-PHI) used for the treatment of anemia in CKD, has shown potential lipid-modifying effects in randomized clinical trials. In this systematic review and meta-analysis, we aim to evaluate the effect of Roxadustat on lipid profile parameters in adults with CKD.

Methods: We searched PubMed, Cochrane Library, Web of Science, Scopus, and ClinicalTrials.gov through January 2026 for RCTs evaluating roxadustat versus any control intervention in adults with CKD. The primary outcome was change in LDL-C. Random-effects meta-analyses, subgroup analyses, and meta-regression were performed according to dialysis status and comparator type. PROSPERO registration: CRD420261295550.

Results: 15 RCTs involving 10,631 participants were included. At 24-28 weeks, Roxadustat significantly reduced LDL-C compared with control (MD -18.34 mg/dL, 95% CI -21.82 to -14.87), with greater reductions in non-dialysis-dependent (NDD) than dialysis-dependent (DD) patients. Meta-regression identified dialysis status as a major contributor to between-study heterogeneity. LDL-C reduction remained significant at 52 weeks (MD -13.11 mg/dL, 95% CI -22.04 to -4.17). Roxadustat also significantly reduced total cholesterol at both follow-up periods, while triglyceride reduction was observed only in NDD populations.

Conclusions: Roxadustat was associated with favorable lipid profile changes in adults with CKD, particularly among NDD populations. The findings suggest that Roxadustat may exert clinically relevant metabolic effects beyond anemia correction; however, the cardiovascular implications of these lipid changes remain uncertain and require further investigation.

Keywords: Dyslipidemia, Roxadustat, Chronic kidney disease, HIF-PHI

MPP3-4

Real-world hsCRP awareness and clinical practice patterns in ASCVD, CKD, and heart failure: South Korea findings from the FLARE (Focus on Levels of Awareness and perceptions REgarding hs-CRP in identifying SI in ASCVD, CKD and HF management) study

Kyungsoo Won[1*], Jungs-Sun Kim[2]

[1]Medical Affairs, Novo Nordisk Korea, Republic of Korea,
[2]Division of Cardiology, Severance Cardiovascular Hospital, Yonsei University College of Medicine, Republic of Korea

Objectives: To evaluate awareness and clinical practice patterns regarding hsCRP as a marker of systemic inflammation (SI), and to identify key drivers, barriers, and unmet needs in SI assessment, among cardiologists and nephrologists in South Korea managing patients with atherosclerotic cardiovascular disease (ASCVD), chronic kidney disease (CKD), and heart failure (HF).

Methods: The FLARE study was a cross-sectional online survey conducted September-October 2025 across four APAC countries (Australia, India, Thailand, South Korea). The South Korea cohort comprised 150 physicians: 112 cardiologists and 38 nephrologists in practice ≥3 years, managing ≥15 and ≥20 ASCVD±CKD patients monthly, respectively.

Results: Unaided awareness of hsCRP as a test for SI was low (29%), rising to 72% with prompting. Despite this gap, 57% of HCPs used hsCRP to identify SI in established or suspected ASCVD and 53% to confirm an ASCVD diagnosis.
Among cardiologists, use extended across the care continuum: 55% to identify SI in patients with established or suspected ASCVD, 56% to confirm an ASCVD diagnosis, 33% to confirm HF diagnosis, and 45% to monitor HF progression.
Most HCPs agreed SI is an independent risk factor for ASCVD (61%) and affects ASCVD prognosis (62%), CKD prognosis (54%), and HF progression in HFrEF (63%) and HFpEF (58%). Mean management thresholds were 2.6 mg/L (cardiologists) and 2.4 mg/L (nephrologists).
Key barriers included lack of threshold standardization (34%), hsCRP variability (33%), and perceived limited clinical influence (29%).
Proven clinical efficacy (37%) and guideline endorsement (41%) were the most critical factors to establish hsCRP as standard of care; 57% agreed SI relevance would increase with an effective SI treatment.

Conclusions: South Korean HCPs recognize SI's role in ASCVD, CKD, and HF, yet hsCRP remains underutilized. Threshold standardization, guideline integration, and education are needed to optimize hsCRP-guided management as anti-inflammatory therapies approach availability.

Keywords: hs-CRP, Systemic inflammation, Residual inflammation, ASCVD, Heart failure, CKD, South Korea, Cardiologist, Nephrologist, Biomarker, Inflammation

MPP3-5

Does early PCSK9 inhibition improve outcomes after acute ischemic stroke? a systematic review and meta-analysis

Amir Hegazi*, Rashad Mohamed, Hussien Osama

Faculty of Medicine, Mansoura University, Egypt

Objectives: Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors produce potent low-density lipoprotein cholesterol (LDL-C) reduction and have established long-term cardiovascular benefit in secondary prevention. However, their role when initiated in the acute phase of ischemic stroke remains incompletely defined. This study evaluates the efficacy of PCSK9 inhibitors in patients with acute ischemic stroke.

Methods: A systematic review and meta-analysis was conducted in accordance with PRISMA guidelines. PubMed, Scopus, Cochrane Library, and Web of Science were searched up to May 2026 for comparative studies assessing PCSK9 inhibitor use in acute ischemic stroke. Outcomes assessed included change in LDL-C from baseline, early neurological deterioration, early stroke recurrence within 3 months, good functional outcome (mRS 0-2) at 3 months, and mortality. Pooled effect estimates were calculated using random-effects models.

Results: Ten comparative studies comprising 3093 patients were included. PCSK9 inhibitor therapy was associated with a substantially greater reduction in LDL-C from baseline compared with controls (mean difference -31.42 mg/dL, 95% CI -35.13 to -27.71, p<0.00001). PCSK9 inhibitor use was also associated with a significantly reduced risk of early neurological deterioration (OR 0.41, 95% CI 0.29-0.59, p<0.00001) and lower early stroke recurrence within 3 months (OR 0.45, 95% CI 0.24-0.83, p=0.01). Good functional outcome at 3 months was significantly more likely with PCSK9 inhibitor use (OR 2.28, 95% CI 1.61-3.22, p<0.00001). Notably, mortality was significantly reduced (OR 0.38, 95% CI 0.18-0.82, p=0.01).

Conclusions: PCSK9 inhibitors in acute ischemic stroke are associated with marked LDL-C reduction alongside improved early neurological and functional outcomes, as well as a significant mortality benefit. These findings support consideration of early PCSK9 inhibitor initiation as an adjunct to standard lipid-lowering therapy after acute ischemic stroke. Larger randomized controlled trials are warranted to confirm these findings.

Keywords: Acute ischemic stroke, PCSK9 inhibitors, LDL cholesterol

MPP3-6

Effect of alirocumab/evolocumab on lipid levels and MACEs after PCI for ACS: a meta-analysis of 3474 patients

Khalid Sarhan*

Department of Internal Medicine, Mansoura University Hospitals, Egypt

Objectives: PCSK9i has been shown to markedly reduce ischemic cardiovascular events in individuals with stable coronary heart disease. Nonetheless, it remains uncertain whether this advantage applies to patients with acute coronary syndrome (ACS) who are undergoing percutaneous coronary intervention (PCI). Therefore, we conducted this meta-analysis to assess the efficacy of alirocumab and evolocumab in reducing lipid levels and MACEs in patients undergoing PCI for ACS.

Methods: Following the PRISMA guidelines, we conducted a literature search on electronic databases to obtain the relevant studies until May 15, 2026. Our primary outcomes were the mean reduction of lipid levels from baseline including triglycerides, total cholesterol, LDL-C, HDL-C, lipoprotein (a), apolipoprotein A, and apolipoprotein B in plasma. Our Secondary outcome was rates of MACES.

Results: We included nine studies with a total of 3474 patients. Our analysis showed a significant reduction in the mean lipid levels from baseline after 1 month including LDL-C (MD -1.16, 95% CI [-1.35, -0.97], P<0.001), total cholesterol (MD -1.13, 95% CI [-1.42, -0.85], P<0.001), triglycerides (MD -0.21, 95% CI [-0.32, -0.10], P=0.002), lipoprotein (a) (MD -10.21, 95% CI [-16.59, -3.83], P=0.002), and apolipoprotein B (MD -30.13, 95% CI [-42.45, -17.82], P<0.001). Additionally, our analysis showed a significant increase in the levels of HDL-C (MD 0.07, 95% CI [0.04, 0.09], P<0.001), and apolipoprotein A (MD 6.16, 95% CI [1.50, 10.82], P=0.01). Moreover, the rates of MACEs were significantly lower in the PCSK9i group (RR 0.70, 95% CI [0.54, 0.91], P=0.007).

Conclusions: In patients with ACS undergoing PCI, PCSK9i effectively reduced the levels of harmful cholesterol and increased the levels of HDL and apolipoprotein A as well as lowering the rates of cardiovascular adverse events.

Keywords: PCSK9i, Acute coronary syndrome

MPP3-7

Clinical impact of imaging-guided percutaneous coronary intervention in heavily calcified coronary lesions: a systematic review and meta-analysis of randomized controlled trials

Ahmed Adel[1*], Nourhan Hatem[2], Basant Ahmed Mahmoud[3], Abdalrahman Salah Shehata[6], Amr Khalid Abdallah[5], Alaa Eldeeb[8], Shafaq Jabeen[7], Ahmed Kertam[4]

[1]Faculty of Medicine, Zagazig University, Zagazig, Egypt, [2]Faculty of Medicine, Suez canal University, Ismailia, Egypt, [3]October 6 University, Faculty of Medicine, Egypt, [4]Tu Lab for Diagnostic Research, Yale School of Medicine, New Haven, CT, Tompkin's East 2, New Haven, CT 06510, United States, [5]Faculty of Medicine, Menofia University, Menofia, Egypt, [6]Faculty of Medicine, Qena University, Qena, Egypt, [7]Karachi Medical and Dental College, Pakistan, [8]Faculty of Medicine, Alexandria University, Egypt

Objectives: Calcified coronary lesions remain challenging during percutaneous coronary intervention (PCI) and are associated with higher rates of adverse cardiovascular events. Intravascular imaging modalities, including intravascular ultrasound (IVUS) and optical coherence tomography (OCT), may improve lesion assessment and procedural optimization. We aimed to evaluate the clinical impact of imaging-guided PCI compared with angiography-guided PCI in patients with calcified coronary lesions.
Methods: A systematic review and meta-analysis of randomized controlled trials (RCTs) was conducted according to PRISMA guidelines. PubMed, Scopus, Web of Science, and Cochrane CENTRAL were searched through September 2025. The primary endpoint was major adverse cardiovascular events (MACE), defined as cardiac death, target-vessel myocardial infarction, or target-vessel revascularization. Risk ratios (RRs) with 95% confidence intervals (CIs) were pooled, and trial sequential analysis (TSA) was performed to assess the conclusiveness of the evidence.
Results: Five RCTs involving 2,249 patients were included, with 1,171 patients undergoing imaging-guided PCI and 1,078 undergoing angiography-guided PCI. In heavily calcified lesions, imaging-guided PCI significantly reduced MACE compared with angiography guidance (RR 0.66, 95% CI 0.51 to 0.85; P=0.001; I^2=0%). Long-term follow-up (2 and 3 years) demonstrated a sustained reduction in MACE (RR 0.65, 95% CI 0.50 to 0.84; P=0.001). Subgroup analysis by imaging modality suggested a borderline significant reduction in MACE with OCT-guided PCI (RR 0.74, 95% CI 0.54 to 1.00; P=0.05), while a single IVUS trial reported a significant reduction (RR 0.53, 95% CI 0.31 to 0.92; P=0.02). TSA confirmed that the cumulative evidence for the primary endpoint was conclusive.
Conclusions: Imaging-guided PCI was associated with a reduction in MACE in patients with heavily calcified coronary lesions, with benefits that appeared to persist during long-term follow-up. While these findings suggest a potential advantage of intravascular imaging in optimizing PCI for this high-risk population, further large-scale RCTs are warranted to confirm these results and define the optimal imaging-guided strategy.
Keywords: Atherosclerosis, Calcified coronary lesions, Percutaneous coronary intervention, Intravascular imaging

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Moderated Poster Presentation 4

Sep 12(Sat) 13:00–14:00 | Studio 5 (6F, Conrad Seoul)

MODERATOR : Su Myung Jung (Sungkyunkwan University, Republic of Korea)

MPP4-1

Unravelling the divergent pathological and transcriptional profile of lipotoxic renal tubular injury and lipoprotein glomerulopathy in APOE-deficient and humanised hAPOE4 knock-in mice

Martin Ho Yin Yeung[1,2,3*], Ching In Chung[1], Angela Zaneta Chan[2]

[1]The Department of Health Technology and Informatics, The Hong Kong Polytechnic University, Hong Kong SAR, China,
[2]Department of Anatomical and Cellular Pathology, The Chinese University of Hong Kong, Hong Kong SAR, China,
[3]Education Department, The English Schools Foundation, Hong Kong SAR, China

Objectives: Renal proximal tubular epithelial cells depend heavily on mitochondrial fatty acid β oxidation to sustain their high metabolic demands, rendering them particularly vulnerable to lipid overload and lipotoxicity. Perturbations in lipid metabolism promote ectopic lipid deposition, cellular stress, and progressive renal injury. Apolipoprotein E (APOE) is a central regulator of lipid metabolism, and genetic polymorphisms in APOE are strongly associated with dementia and show positive correlations with renal function. This study characterised the distinct renal pathological phenotypes of APOE knockout (KO) and humanised APOE4 knock in (hAPOE4 KI) mice, and examined how these genetic states influence tubular injury biomarkers and the peroxisomal-mitochondrial metabolic axis.
Methods: Kidneys from APOE KO and hAPOE4 KI mice (n=6/group) were harvested for histology (H&E, PAS, PASM, Oil Red O) and immunohistochemistry for APOE (rAPOE, 1:200). Renal mRNA expression levels of lipid metabolism enzymes (ACOT12, CROT, CRAT) and kidney injury markers (HAVCR1, LCN2) were extracted from RNA sequencing data of APOE3, KO, and APOE4 mice (n=3/group).
Results: APOE KO kidneys showed large glomerular lipid deposits (ORO positive) and mild proximal tubular accumulation, with no ApoE protein (rAPOE negative). hAPOE4 KI mice exhibited mild glomerulosclerosis with PAS/PASM positive hyaline deposits infiltrating ~50% of the glomerulus, ORO positive lipid accumulation, and mesangial ApoE immunoreactivity, modelling lipoprotein glomerulopathy (Figure 1). RNA sequencing revealed that APOE KO kidneys uniquely upregulated tubular injury markers HAVCR1 (4.2 fold) and LCN2 (12.8 fold), alongside peroxisomal ACOT12 (3.0 fold) and CROT (2.0 fold), while mitochondrial CRAT remained static. hAPOE4 KI mice maintained baseline transcript levels (Figure 2).
Conclusions: APOE deficiency disrupts renal lipid metabolism by impairing mitochondrial fatty acid β oxidation and driving compensatory peroxisomal activation, resulting in severe tubular lipotoxicity. In contrast, APOE4 preserves tubular oxidative capacity but alters glomerular lipid handling, producing lipoprotein glomerulopathy. These findings establish APOE as a critical determinant of renal lipid homeostasis, linking genetic variation to divergent tubular and glomerular injury.
Keywords: Renal lipid, Chronic kidney disease, Lipid, Fatty acid oxidation

MPP4-2

Regulation of cholesterol and bile acid homeostasis by ceramide chain length

Eunji Noh[1*], Seonwoo Lee[1], Woo-Jae Park[2], Joo-Won Park[1]

[1]Department of Biochemistry, College of Medicine, Ewha Womans University, Republic of Korea,
[2]Department of Biochemistry, Chung-Ang University College of Medicine, Republic of Korea

Objectives: Gallstone disease remains a major global gastrointestinal disorder with high prevalence. Its pathogenesis involves cholesterol crystallization, which arises from hypersecretion of cholesterol or hyposecretion of bile salts and phospholipids within the biliary system. Here, we utilized ceramide synthase-modified mouse models to elucidate the impact of ceramide acyl chain length on gallstone pathogenesis.
Methods: Gallstone formation was induced by feeding a lithogenic diet to WT, $CerS2^{+/-}$, and CerS2-null mice, as well as to WT, $CerS5^{+/-}$, and CerS5-KO mice. To investigate the underlying mechanisms, in vitro studies were performed using Hep3B cells with CerS2 and CerS5 overexpression or knockdown.
Results: Lithogenic diet-induced gallstone formation was significantly exacerbated in CerS2 heterozygote or null mice, but completely abolished in CerS5-KO mice. This opposition was mirrored in hepatic gene profiles: CerS5 deficiency reduced, while CerS2 deficiency elevated, the expression of cholesterol and bile acid homeostasis genes (Abcg5/8 and Cyp7a1). This regulation was mediated by the differential expression of the transcriptional repressor SHP, which was decreased in CerS2 null mice and increased in CerS5-KO mice, and further modulated via the CerS-DUSP8-ERK signaling pathway. These findings demonstrate that CerS2 and CerS5 play opposing roles in biliary lipid homeostasis and gallstone pathogenesis.
Conclusions: In conclusion, this study reveals that CerS2 and CerS5 reciprocally regulate Cyp7a1 expression through the DUSP8-ERK signaling cascade, alongside Abcg5/8 expression. These insights position CerS2 and CerS5 as novel therapeutic targets to combat gallstone disease and restore cholesterol and bile acid homeostasis.
Keywords: Cholesterol, Ceramide synthase, ABCG5, ABCG8

MPP4-3

Docosahexaenoic acid attenuates vascular calcification with regulation of miR-29b1

Hyeonji Lee[1*], Soo-jin Ann[2], Sang-Hak Lee[3]

[1]Division of Biomedical Engineering, Yonsei University, Republic of Korea,
[2]Integrative Research Center for Cerebrovascular and Cardiovascular Diseases, Yonsei University College of Medicine, Republic of Korea,
[3]Division of Cardiology, Yonsei University College of Medicine, Republic of Korea

Objectives: Vascular calcification is driven by osteogenic phenotypic switching of vascular smooth muscle cells (VSMCs), contributing to cardiovascular risk. Although docosahexaenoic acid (DHA) has been to offer cardiovascular benefit, its molecular mechanisms have not been clearly understood. We aimed to elucidate anti-calcific mechanism of DHA, particularly associated with miRNA.
Methods: To induce calcification, rat VSMCs were treated with β-Glycerophosphate disodium salt hydrate and Sodium phosphate monobasic. DHA 10 μM was used to evaluate its anti-calcific effect. Harvested cells were subjected to RNA sequencing (RNA-seq) using Illumina NovaSeq 6000 platform. miRNA targets were selected based on their expression ratios and the TargetScan 8.0 database. To validate the function of miRNAs upregulated by DHA, the cells were transfected with specific miRNA inhibitors, and the calcium deposits were analyzed by Alizarin Red S staining.
Results: High Pi induced calcium deposition was significantly attenuated in the DHA treated group (mean difference 64%; p=0.011). RNA sequencing revealed strong associations with miRNA-mediated post-transcriptional gene silencing, negative regulation of the BMP signaling pathway and ossification. Among the upregulated miRNAs, miR-29b1 (log2FC = 10.35; p=2.16E-11) was selected as a key candidate mediating the anti-calcific effect. Targeted inhibition of miR-29b1 resulted in higher calcium deposition (mean difference 215%; p=0.019), reversing anti-calcific effects of DHA (Figure).
Conclusions: These results indicate that DHA mitigates VSMC calcification by upregulating miR-29b1 and clarified its anti-calcific mechanism that may protect from cardiovascular risk.
Keywords: Biomineralization, Osteogenesis, Alkaline phosphatase, Fatty acids, Omega-3

MPP4-4

Lipid droplet sequestration protects vascular smooth muscle cells from saturated fatty acid-induced NFkB activation

Laura Alonso-Herranz[1,2*], Jacob Fog Bentzon[1,2]

[1]Department of Clinical Medicine, Denmark, [2]Steno Diabetes Center Aarhus, Denmark

Objectives: Ectopic lipid storage promotes atherosclerotic plaque formation by triggering a lipid-driven transformation of smooth muscle cells (SMCs). However, how specific lipid species regulate SMC phenotype and lipid handling remains poorly understood. Here we investigated the differential effects of the saturated fatty acid (SFA) palmitic acid and the monounsaturated fatty acid (MUFA) oleic acid on SMC inflammatory activation, contractile identity, and lipid droplet formation.
Methods: Human aortic SMCs were treated for 24h with sodium palmitate (0.1-0.5 mM) alone or in combination with sodium oleate (0.2 mM). Readouts included intracellular Ca2+ imaging (Calbryte staining), gene expression, lipid droplet formation (Bodipy staining), and RELA nuclear translocation (immunofluorescence).
Results: Palmitate dose-dependently increased intracellular Ca2+ and induced a pro-inflammatory, dedifferentiated phenotype characterized by upregulation of VCAM1 and IL6, downregulation of ACTA2 and CNN1, and a morphological shift toward a rounded, less spindle-like cell shape. Strikingly, palmitate alone failed to promote lipid droplet formation, indicating that SFA overload cannot be safely sequestered by SMCs. In contrast, co-treatment with oleate rescued lipid droplet biogenesis and significantly ameliorated the palmitate-induced inflammatory response. Mechanistically, palmitate triggered RELA nuclear translocation, and siRNA-mediated silencing of IKBKB significantly reduced RELA translocation and abrogated palmitate-induced pro-inflammatory gene expression, establishing NFkB signaling as the causal pathway linking SFA overload to SMC inflammation.
Conclusions: These findings reveal a dual regulatory axis governing SMC fate in response to fatty acids: the SFA palmitic acid drives lipotoxic inflammation via NFkB when lipid droplet sequestration is absent, whereas the MUFA oleic acid enables protective lipid droplet formation that buffers the lipotoxic insult. This SFA/MUFA balance may represent a critical determinant of SMC transformation in atherosclerosis, with implications for understanding how circulating lipid composition influences plaque progression.
Keywords: Smooth muscle cells, Fatty acids, NFkB, Phenotypic switching

MPP4-5

CIAPIN1 orchestrates vascular phenotypic switching as a redox-sensitive repressor of Tp53

Seongpyo Lee*, Joo-Hui Han

College of Pharmacy, Chungbuk National University, Republic of Korea

Objectives: Although p53 is recognized as a pivotal regulator of vascular smooth muscle cell (VSMC) homeostasis, the precise molecular events underlying its downregulation during growth factor-stimulated phenotypic switching remain poorly understood. This study identifies cytokine-induced apoptosis inhibitor 1 (CIAPIN1) as a novel, redox-sensitive transcriptional repressor of the Tp53 gene involved in vascular remodeling processes like atherosclerosis and restenosis.
Methods: Utilizing primary cultured VSMCs alongside a rat carotid balloon injury model, we modulated CIAPIN1 via gain- and loss-of-function strategies.
Results: Clinical tissue analysis revealed that CIAPIN1 was markedly elevated in human atherosclerotic plaques, whereas Tp53 mRNA expression was substantially diminished compared to healthy controls. Mechanistically, PDGF-BB stimulation triggered reactive oxygen species (ROS) production, which drove the nuclear translocation of CIAPIN1 via its functional C-terminal nuclear localization signal (NLS, residues 236-239) in an importin-α/β-dependent manner. Upon entering the nucleus, CIAPIN1 directly bound to consensus sequences within the Tp53 promoter, silencing its transcription. This suppression of p53 subsequently accelerated VSMC proliferation and migration, shifting cells toward a synthetic phenotype. Conversely, blocking this pathway using the ROS scavenger N-acetylcysteine (NAC), the importin inhibitor ivermectin (IVM), or an NLS-deficient mutant (residues 236-239) restored p53 expression and effectively mitigated neointimal hyperplasia in vivo.
Conclusions: In conclusion, CIAPIN1 operates as a redox-responsive repressor of Tp53 that accelerates VSMC phenotypic transitions and neointimal growth, positioning it as a promising therapeutic target for proliferative vascular disorders.
Keywords: CIAPIN1, p53, Reactive oxygen species, Intimal hyperplasia

MPP4-6

Endothelial CAP1 acts as a gatekeeper of vascular homeostasis via the formation of an adenylyl cyclase and caveolin-1 complex

You Ji Kim[1,2,3*], Yoo-Wook Kwon[1,3]

[1]Biomedical Research Institute, Seoul National University Hospital, Republic of Korea,
[2]Program in Stem Cell Biology, Seoul National University College of Medicine, Republic of Korea,
[3]Department of Medicine, Seoul National University College of Medicine, Republic of Korea

Objectives: Adenylyl cyclase-associated protein 1 (CAP1) is a human resistin receptor linking chronic inflammation to cardio-metabolic diseases. This study investigates the unexplored role of CAP1 in regulating adenylyl cyclase (AC) and endothelial-monocyte interactions in human endothelial cells (ECs).
Methods: We performed in vitro analyses using human umbilical vein endothelial cells (HUVECs) subjected to CAP1 knockdown or recombinant resistin treatment to assess protein interactions, cAMP signaling, and endothelial barrier integrity. In vivo leukocyte extravasation was evaluated using a carrageenan-induced paw edema model in wild-type and CAP1+/- mice, combined with bone marrow transplantation to isolate endothelial-specific effects.
Results: In normal ECs, CAP1 forms a suppressive triple complex with AC3 and caveolin-1 (CAV1) within lipid rafts, keeping AC3 inactive. Both genetic knockdown of CAP1 and resistin treatment disrupted this CAP1-AC3-CAV1 complex, leading to AC3 activation and cAMP accumulation. This triggered PKA-CREB and NF-κB signaling, resulting in the marked upregulation of adhesion molecules, including ICAM-1 and VCAM-1. Furthermore, activated PKA induced mitochondrial fission and oxidative stress, causing structural endothelial barrier dysfunction. In vivo experiments clearly demonstrated that endothelial-specific CAP1 depletion significantly promoted monocyte extravasation and exacerbated local vascular inflammation.
Conclusions: CAP1 serves as a crucial homeostatic gatekeeper in ECs by suppressing AC3 activity and maintaining barrier integrity. Elevated resistin disrupts this complex, driving endothelial dysfunction and inflammation. Targeting the resistin-CAP1 axis offers a promising mechanistic therapeutic strategy for atherosclerosis and metabolic syndrome.
Keywords: Adenylyl cyclase-associated protein 1 (CAP1), Resistin, Endothelial cell, Inflammation, Caveolin

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Moderated Poster Presentation 5

Sep 12(Sat) 13:00-14:00 | Studio 6 (6F, Conrad Seoul)

MODERATOR : Jeong-Min Kim (Seoul National University, Republic of Korea)

MPP5-1

Association of a body shape index and the 5-year incidence of dyslipidemia and hypertension among Filipinos in the LIFECARE cohort

Bayani Pocholo T. Maglinte[1*], Raymond Oliva[1], Lourdes Ella G. Santos[1], Deborah Ignacia D. Ona[1], Jerahmeel Aleson L. Mapili[1], Bernadette Alexis M. Mariño[1], Bryan F. Elvambuena[1], Manuel John A. Dayao[1], Rio May E. Llanes[1], Celia Catherine C. Uy[1], Olivia T. Sison[2], Nina T. Castillo-Carandang[2], Felix Eduardo R. Punzalan[1], Elmer Jasper B. Llanes[1], Wilbert Allan G. Gumatay[1], Rody G. Sy[1]

[1]Division of Cardiovascular Medicine, University of the Philippines - Philippine General Hospital, Philippines, [2]Department of Clinical Epidemiology, University of the Philippines - Manila, Philippines

Objectives: We prospectively assessed the association between baseline ABSI and 5-year incidence of dyslipidemia and hypertension among Filipino adults.
Methods: In this prospective study, we analyzed data from the Philippine LIFECARE cohort including participants aged 20-50 years without baseline dyslipidemia (n=881) or hypertension (n=2,088) from Metro Manila and 4 nearby provinces underwent anthropometric assessment. ABSI was calculated from baseline height, weight, and waist circumference. Modified Poisson regression estimated risk ratios (RR) for incident dyslipidemia and hypertension, adjusting for age, sex, smoking, diabetes, and family history of hypertension. Discriminatory performance was assessed using area under the receiver operating characteristic curve (AUC).
Results: At 5-year follow-up, 37.0% (n=326) developed dyslipidemia and 15% (n=321) developed hypertension. Elevated ABSI (>0.077) was independently associated with increased dyslipidemia risk (adjusted RR 1.20, 95% CI 1.00-1.45), but discrimination was poor (AUC 0.54, 95% CI 0.50-0.58). ABSI showed no significant association with incident hypertension (adjusted RR 0.92, 95% CI 0.75-1.13). In contrast, BMI ≥25 kg/m^2 (adjusted RR 1.86, 95% CI 1.52-2.26) and waist circumference >90 cm (adjusted RR 1.86, 95% CI 1.50-2.30) strongly predicted hypertension with moderate discrimination (both AUC 0.67). No anthropometric index demonstrated adequate discrimination for dyslipidemia prediction.
Conclusions: ABSI shows a modest association with dyslipidemia but not hypertension in Filipino adults, with poor discriminatory performance for both outcomes. These findings suggest outcome-specific performance of anthropometric indices and highlight the need for population-specific cutoff validation and integration into overall cardiovascular risk assessment in the Filipino population.
Keywords: Anthropometry, Body mass index, Dyslipidemia

MPP5-2

Burden of ischemic stroke attributable to high LDL cholesterol and high body mass index in adults aged 55+years: a comparative analysis of mortality, DALYs, and YLDs by sex across socio-demographic index levels

Zavia Putri Salsabila[1*], Nazzala Safira[2], Zulfania Rahmah[3]

[1]Medical Internship Program, Islamic Weleri Hospital, Indonesia, [2]Medical Internship Program, Banjarnegara Muhammadiyah Hospital, Indonesia, [3]Medical Internship Program, Al Aziz Islamic Hospital, Indonesia

Objectives: Ischemic stroke is a leading cause of death and long-term disability worldwide. Modifiable metabolic risk factors substantially contribute to its development. However, the burden attributable to these risk factors differs across socio-demographic contexts. This study aimed to compare the numbers of deaths, disability-adjusted life years (DALYs), and years lived with disability (YLDs) attributable to metabolic risk factors for ischemic stroke among adults aged more than 55 years in different socio-demographic (SDI) regions.
Methods: We extracted secondary data from the Global Burden of Disease (GBD) 2023 database. Individuals aged 55 years and older with ischemic stroke were included. We estimated the number of deaths, DALYs, and YLDs attributable to high LDL cholesterol and high BMI. Results are presented as absolute numbers and stratified by sex (male, female) and SDI level (high SDI and low SDI).
Results: In low-SDI regions, ischemic stroke attributable to high LDL cholesterol resulted in 76,241 deaths, compared with 8,587 deaths attributable to high BMI. High LDL cholesterol accounted for 1,637,075 DALYs and 182,219 YLDs, whereas high BMI contributed 213,495 DALYs and 213,495 YLDs. In high-SDI regions, the burden was substantially greater. High LDL cholesterol was associated with 391,593 deaths, 7,519,610 DALYs, and 1,531,787 YLDs, compared with 73,188 deaths, 1,635,739 DALYs, and 361,726 YLDs attributable to high BMI. Females generally experienced higher mortality than males across both SDI settings. High LDL cholesterol consistently contributed a markedly greater burden than high BMI in both high- and low-SDI populations.
Conclusions: High LDL cholesterol represents the dominant metabolic risk factor for ischemic stroke among adults aged 55 years and older, surpassing high BMI across all burden measures. The substantially higher burden observed in high-SDI regions highlights the importance of targeted lipid management and cardiovascular prevention strategies to reduce ischemic stroke-related mortality and disability worldwide.
Keywords: Ischemic Stroke, High LDL Cholesterol, High BMI, DALYs, Socio-demographic index

MPP5-3

Association between triglyceride-to-HDL cholesterol ratio and coronary artery spasm: differential effect according to diabetes mellitus status

You Jin Lee[1*], Se Yeon Choi[1], Byoung Geol Choi[2], Jae Kyeong Byun[1], Abiseka Panji Baskoro[1], Aditya Reza Partama[1], Soohyung Park[1], Seung-woon Rha[1]

[1]Cardiovascular Center, Korea University Guro Hospital, Republic of Korea, [2]Department of Biomedical Laboratory Science, Honam University, Republic of Korea

Objectives: The triglyceride-to-HDL cholesterol (TG/HDL-C) ratio is a surrogate marker of insulin resistance and atherogenic dyslipidemia. However, its association with coronary artery spasm (CAS) and whether this differs by diabetes mellitus (DM) status remain unknown.
Methods: A total of 6,753 consecutive patients who underwent intracoronary provocation testing (acetylcholine or ergonovine) were analyzed. TG/HDL-C ratio was divided into quartiles (Q1: ≤1.31, Q2: 1.31-2.14, Q3: 2.14-3.49, Q4: >3.49). Multivariable logistic regression (5 models) was performed, with Model 5 adjusted for age, sex, BMI, hypertension, smoking history, eGFR, HbA1c, statin use, and dyslipidemia. An interaction test between TG/HDL-C ratio and DM status was performed using the likelihood ratio test, with subgroup analyses in DM (n=1,119) and non-DM (n=5,634) patients. The primary endpoint was CAS positivity; secondary endpoint was major adverse cardiac and cerebrovascular events including recurrent angina (MACCE) over a median follow-up of 4.8 years.
Results: CAS was identified in 54% of patients. In the overall cohort, TG/HDL-C ratio was associated with CAS on unadjusted analysis (Q4 vs Q1: OR 1.25, 95% CI 1.09-1.43, p=0.001), but not after full adjustment (OR 1.08, p=0.313). A significant interaction by DM status was observed (P for interaction=0.023). Among DM patients, the highest quartile independently predicted CAS (OR 1.74, 95% CI 1.06-2.87, p=0.029; P for trend=0.029), whereas no association was found in non-DM patients (OR 1.00, p=0.994). No significant association was observed between TG/HDL-C ratio and MACCE (HR 0.91, 95% CI 0.57-1.46, p=0.691).
Conclusions: Although the TG/HDL-C ratio showed a crude association with CAS in the overall cohort, this was driven specifically by DM patients, suggesting that insulin resistance-related metabolic dysregulation may contribute to coronary vasospastic susceptibility in the diabetic population.
Keywords: TG/HDL-C ratio, Coronary artery spasm, Diabetes mellitus

MPP5-4

Decoupling atherosclerosis from vasospasm: insights from the ApoB/ApoA1 ratio

Abiseka Panji Baskoro[1,2*], Aditya Reza Pratama[1], Byoung Geol Choi[3], Se Yeon Choi[1], Jae Kyeong Byun[1], Youjin Lee[1], Won Sang Chu[1], Soohyung Park[1], Cheol Ung Choi[1], Seung-Woon Rha[1]

[1]Cardiovascular, Korea University Guro Hospital, Republic of Korea, [2]Cardiology, Universitas Sebelas Maret Hospital, Indonesia, [3]Department of Biomedical Laboratory Science, Honam University, Republic of Korea

Objectives: Coronary artery spasm (CAS) represents a reversible disturbance of coronary vasomotor tone capable of provoking myocardial ischemia and unfavorable cardiac events. While the apolipoprotein B-to-apolipoprotein A1 (ApoB/ApoA1) ratio is an established indicator of atherogenic burden, whether it correlates with vasospastic coronary pathology has not been clearly established. This study examined whether ApoB/ApoA1 ratio is linked to CAS induced by acetylcholine provocation.
Methods: This was a retrospective cohort analysis drawing on patients enrolled in the Korea University Guro Hospital Spasm Registry from October 2004 through October 2024. Of those who underwent intracoronary acetylcholine provocation, 932 patients with measured ApoB and ApoA1 values were included in the analysis. The cohort was divided into quartiles based on ApoB/ApoA1 ratio. CAS was considered significant when transient luminal narrowing exceeded 70% during provocation. Baseline characteristics, laboratory values, angiographic features, and provocation testing results were contrasted across the quartile groups, and logistic regression was used to identify independent predictors of CAS.
Results: Higher ApoB/ApoA1 quartiles correlated with an increasingly adverse metabolic profile, marked by rising total cholesterol, LDL cholesterol, triglycerides, and ApoB, together with declining HDL cholesterol and ApoA1 (all trends p<0.001). Spasm detected at baseline was more common among patients in lower quartiles, while atrioventricular block became more frequent at higher quartiles. Despite these gradients, the rate of CAS induced during acetylcholine testing did not differ meaningfully between groups (p=0.542), and multivariable modeling found no independent link between ApoB/ApoA1 quartile and CAS occurrence. Advancing age was independently associated with CAS, whereas being male was inversely associated.
Conclusions: Although strongly tied to an unfavorable cardiometabolic profile, the ApoB/ApoA1 ratio did not independently predict inducible CAS. This implies that vasospastic coronary disease may arise through pathways separate from the classic lipid-driven atherosclerotic process.
Keywords: Coronary artery spasm, ApoB/ApoA1 ratio, Vasospastic angina, Acetylcholine provocation test

MPP5-5

Carbohydrate-fat dietary patterns modify protein-cardiometabolic association: a cross-sectional study in Singaporean adults

Weili Hu[1*], Yueying Yao[1], Ian Mak[1], Lingyin Yu[1], Shuqi Li[1], Nicole Wong Min Yee[1], Marcus Ting[1], Chin Meng Khoo[3], Jung Eun Kim[1,2]

[1]Department of Food Science and Technology, National University of Singapore, Singapore, [2]Bezos Center for Sustainable Protein, National University of Singapore, Singapore, [3]Department of Endocrinology, National University Health System, Singapore

Objectives: High protein intake has been associated with both beneficial and adverse cardiometabolic profiles, potentially due to differences in the overall dietary macronutrient composition. Therefore, this study examined whether carbohydrate-fat dietary patterns (CHO/FAT) modify associations between protein intake and cardiometabolic outcomes in Singaporean adults.
Methods: Data of 233 Singaporean adults (21-78 years) from a cross-sectional study were analyzed. Dietary intake was assessed using 3-day food records; outcomes included lipid-lipoprotein markers, fasting glucose, blood pressure, and vascular risk indices. CHO/FAT groups were defined as HC/HF, MC/HF, HC/LF, and LC/HF using CHO cut-offs of <26, 26-45, and >45%E and fat cut-offs of <30 and ≥30%E. Multivariable linear regression models evaluated protein × CHO/FAT interactions, followed by subgroup analyses of protein-cardiometabolic associations. Analyses were also stratified by age (<60 and ≥60 years).
Results: Significant protein × dietary pattern interactions were observed for TC/HDL ratio and atherogenic index (AI), with the HC/HF as reference. Within HC/HF, higher protein intake was associated with higher TC, LDL, TC/HDL ratio, and AI (β=0.085, β=0.081, β=0.070, and β=0.070; p<0.01), but lower fasting glucose (β=-0.067, p=0.007). However, these associations disappeared in other dietary pattern subgroups. Among adults aged ≥60 years, higher protein intake was associated with higher LDL, TC/HDL ratio, and AI in both high-fat groups (HC/HF and MC/HF), and additionally with higher TC and lower fasting glucose in HC/HF while these associations were not observed among adults aged <60 years.
Conclusions: Associations between dietary protein intake and cardiometabolic parameters varied by CHO/FAT composition and unfavorable associations of higher protein intake with these parameters were mainly observed under HC/HF, particularly among older adults. These findings suggest that carbohydrate-fat composition should be considered when evaluating associations between protein intake and cardiometabolic health.
Keywords: Macronutrient composition, Dietary protein, Cardiometabolic outcomes, Lipid profile, Older adults

MPP5-6

Dietary macronutrient intake modifies the association between cognitive function and cardiovascular disease risk: a cross-sectional study in Singapore adults

Shuqi Li[1*], Yueying Yao[1], Ian En Kai Mak[1], Lingyin Yu[1], Nicole Min Yee Wong[1], Marcus Ting[1], Jung Eun Kim[1,2]

[1]Department of Food Science and Technology, National University of Singapore, Singapore,
[2]Bezos Center for Sustainable Protein, National University of Singapore, Singapore

Objectives: Poorer cognition has been associated with a higher cardiovascular disease (CVD) risk, and dietary intake is a key factor in both cardiovascular and brain health. However, it remains unclear whether dietary intake, particularly macronutrients as a major contributor to cardiometabolic health and energy source for brain, modifies the relationship between cognition and CVD risk indicators. This study examined the association between cognition and CVD risk and explored whether the macronutrient intake modifies these relationships in Singapore adults. Also, these relations were further explored by stratifying the age group.
Methods: Data from 233 participants (aged 21-80 years) were collected. CVD risk indicators include blood pressure, lipid-lipoprotein profile, vascular age, vascular age gap, and the Framingham Risk Score (FRS). Cognition was assessed using the Montreal Cognitive Assessment (MoCA), and macronutrient intake was assessed by a 3-day food record. Multivariable linear regression and interaction analyses were conducted by adjusting for age, gender, and BMI.
Results: MoCA score was associated with FRS (β=-0.106, p=0.018), while no associations were observed with other outcomes. Interaction analyses showed significant negative MoCA × diet interactions for total energy, fat, protein, and carbohydrate intakes in relation to FRS. In age-stratified analyses, significant associations were observed among older adults (aged 65-80 years), where MoCA score was associated with vascular age (β=-1.562, p=0.009), vascular age gap (β=-1.436, p=0.013), and FRS (β=-0.242, p=0.052). Among older adults, significant interactions were observed for percentage energy from fat between MoCA with vascular age, vascular age gap, and FRS.
Conclusions: Better cognition was associated with lower CVD risk, particularly in Singapore older adults. Dietary macronutrient intake may modify the cognition-CVD risk relationship, with emphasis on fat intake in older adults.
Keywords: Cognition, Vascular age, Framingham risk score, Cardiovascular disease risk, Macronutrient intake

MPP5-7

Distribution of diet-attributable ischemic heart disease mortality across Indonesian provinces: an ecological analysis

Zulfania Rahmah[1*], Nazala Safira[2], Zavia Putri Salsabila[3]

[1]Medical Internship Program, Al Aziz Hospital, Indonesia, [2]Medical Internship Program, Banjarnegara Muhammadiyah Hospital, Indonesia, [3]Medical Internship Program, Weleri Islamic Hospital, Indonesia

Objectives: Dietary factors remain important modifiable contributors to cardiovascular mortality and account for a substantial proportion of ischemic heart disease (IHD) burden worldwide. Understanding variations in diet-attributable IHD mortality may support the development of targeted population-based nutritional interventions. To evaluate differences in diet-attributable ischemic heart disease mortality rates across Indonesian provinces and identify dietary risk factors contributing to higher mortality burden.
Methods: An ecological study was conducted using provincial-level data on diet-attributable ischemic heart disease mortality rates across Indonesian provinces in 2023. Dietary risk categories included inadequate intake of protective foods and excessive intake of potentially harmful dietary components. Descriptive analysis was performed to summarize mortality rates across dietary categories. Because data were not normally distributed, comparisons between dietary groups were analyzed using the Kruskal-Wallis test. Mean rank values were used to evaluate relative differences in mortality burden.
Results: Marked variation in ischemic heart disease mortality burden was observed across dietary risk categories. The highest mean mortality rates were identified in diets low in whole grains (mean=223.24±223.13), followed by diets low in nuts and seeds (162.82±177.74), low in omega-6 polyunsaturated fatty acids (150.26±149.71), low in fiber (149.06 ±159.61), low in legumes (148.53±149.77), low in fruits (135.77±136.83), and high in sodium (124.27±123.08). Kruskal-Wallis analysis demonstrated significant differences in mortality rates across dietary risk categories (χ^2=540.07; df=12; p<0.001). Relative mortality burden based on mean rank was highest for diets low in whole grains, followed by diets low in nuts and seeds and low in omega-6 polyunsaturated fatty acids.
Conclusions: Diet-attributable ischemic heart disease mortality burden differed significantly across dietary risk categories in Indonesia. Insufficient consumption of whole grains, nuts and seeds, fiber, legumes, and unsaturated fatty acids showed the greatest mortality burden, highlighting the importance of improving dietary quality in population-level cardiovascular prevention strategies.
Keywords: Ischemic heart disease, Mortality

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Moderated Poster Presentation 6

Sep 12(Sat) 13:00-14:00 | Studio 7 (6F, Conrad Seoul)

MODERATOR : Jae-Han Jeon (Kyungpook National University, Republic of Korea)

MPP6-1

Association of obesity status and LDL-cholesterol levels with all-cause mortality in statin users aged 75 years and older: a nationwide population-based cohort study

Mihye Seo[1*], Kyungdo Han[2], Hyeong Kyu Park[1], Kyoil Suh[1]

[1]Endocrinology and Metabolism, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, Seoul, Republic of Korea, [2]Statistics and Actuarial Science, Soongsil University, Seoul, Republic of Korea

Objectives: Evidence on the mortality benefit of statins used for primary prevention in adults aged ≥75 years is limited, and whether it varies by obesity status is unclear. We examined all-cause mortality according to BMI and achieved LDL-C among elderly statin users, using a nationwide, population-based retrospective cohort.

Methods: From the Korean National Health Insurance Service database, 410,674 statin users aged ≥75 without prior cardiovascular disease who underwent health screening (2012-2015) were followed for all-cause mortality. Cox models estimated adjusted hazard ratios (aHRs) across BMI and achieved LDL-C categories.

Results: During a mean 7.7 years, 139,610 of 410,674 statin users died; all-cause mortality increased with age and was highest among those aged ≥85 years. Among these statin users, mortality differed by BMI: versus 18.5-23 kg/m^2, the aHR was 0.71 for 25-30 and 0.76 for ≥30 but 1.83 for <18.5. Achieved LDL-C showed an inverted J-shaped association: versus 70-100 mg/dL, aHRs were 1.15 for <55 and 1.06 for 55-70 mg/dL, whereas ≥100 mg/dL was not associated with mortality. The LDL-C-mortality association differed by BMI (P<0.001): low LDL-C was associated with higher mortality only in those with BMI <25 kg/m^2, and the lowest mortality was observed at BMI ≥25 with LDL-C 70-130 mg/dL. These associations were consistent among adherent users (medication possession ratio ≥80%) and across age, sex, and diabetes subgroups.

Conclusions: Among statin users aged ≥75, the LDL-C-mortality association varied by obesity status, with low LDL-C associated with higher mortality only in those with BMI <25 kg/m^2, which may suggest the need for individualized LDL-C targets by BMI.

Keywords: LDL-C, Elderly statin users

MPP6-2

Beyond revascularization: Wharton's jelly stem cells as a promising limb-salvage strategy in diabetic chronic limb-threatening ischemia: a systematic review and meta-analysis

Ghazy Wira Pradipta[1*], Najwa Dean Hasana[1], Ghazy Wira Pradipta[2], Affan Rayhan Ismail[3]

[1]General Practitioner, Faculty of Medicine, Trisakti University Jakarta and SADIA Private Clinic, Bandung, Indonesia, [2]Internal Medicine Department, Faculty of Medicine, Sebelas Maret University, Surakarta, Indonesia, [3]General Practitioner, Simo General Hospital, Boyolali, Indonesia

Objectives: Diabetic limb ischemia, a severe manifestation of peripheral artery disease and chronic limb-threatening ischemia (CLTI), is associated with substantial risks of non-healing ulcers, major amputation, cardiovascular events, and mortality. Despite advances in endovascular and surgical revascularization, many patients remain poor candidates for conventional interventions or experience persistent microvascular insufficiency. This study aimed to evaluate the comparative effectiveness of allogeneic Wharton's Jelly-derived mesenchymal stem cells (WJ-MSCs) and autologous bone marrow mononuclear cells (BM-MNCs) in improving vascular perfusion and clinical outcomes among patients with diabetic limb ischemia.

Methods: A systematic review and meta-analysis were conducted according to PRISMA guidelines. PubMed, Embase, and Scopus were searched through May 2025. Eligible studies included diabetic patients with limb ischemia receiving WJ-MSCs or BM-MNCs. Outcomes relevant to clinical vascular recovery were transcutaneous oxygen pressure ($TcPO_2$), Visual Analogue Scale (VAS) pain score, and pain-free walking distance. Pooled mean differences (MDs) with 95% confidence intervals (CIs) were calculated using fixed-effects models.

Results: Three studies involving 32 patients were included. Compared with BM-MNCs, WJ-MSC therapy significantly improved tissue perfusion as demonstrated by higher $TcPO_2$ values (MD 16.25; 95% CI 4.27-28.04; p=0.007). Patients receiving WJ-MSCs also experienced greater reductions in ischemic limb pain (MD -0.93; 95% CI -1.61 to -0.25; p=0.007) and substantially longer pain-free walking distances (MD 203.57 m; 95% CI 80.60-326.54; p=0.001). These findings indicate clinically meaningful improvements in limb oxygenation, functional capacity, and symptom burden.

Conclusions: Among patients with diabetic vascular disease and chronic limb-threatening ischemia, allogeneic WJ-MSCs demonstrated superior efficacy compared with autologous BM-MNCs in restoring tissue perfusion and improving functional outcomes. As an emerging adjunctive strategy for patients with limited revascularization options, WJ-MSC therapy may contribute to enhanced limb preservation and improved vascular recovery. Larger randomized studies are warranted to establish its role in the management of advanced diabetic vascular disease.

Keywords: Diabetic limb ischemia, Chronic limb-threatening ischemia, Wharton's jelly mesenchymal stem cells, Tissue perfusion, Limb preservation

MPP6-3

Atherogenic index of plasma in metabolic syndrome: a systematic review and meta-analysis

Ahmed Almezaine*, Abdelrahman Hatata, Ibrahim Elbably

Ministry of Health, Egypt

Objectives: A growing body of literature has investigated the atherogenic index of plasma (AIP) as a potential biomarker for metabolic syndrome (MetS), with findings suggesting a possible diagnostic role. However, current evidence remains heterogeneous and inconclusive. This systematic review and meta-analysis aimed to evaluate the association between AIP and MetS and to assess its diagnostic performance.

Methods: A systematic search of PubMed, EMBASE, and Scopus databases was performed according to predefined eligibility criteria. Studies were included if MetS was diagnosed using the International Diabetes Federation (IDF) criteria. The primary outcomes were the mean difference (MD) in AIP between individuals with MetS and healthy controls, and the area under the curve (AUC) for AIP in predicting MetS.

Results: Thirteen studies comprising a total of 17,689 participants were included in the analysis. AIP values were significantly higher in individuals with MetS compared with healthy controls (MD=0.309; 95% CI: 0.214–0.405). However, no statistically significant difference was observed between patients with type 2 diabetes mellitus (T2DM) and MetS and normoglycemic individuals with MetS (MD=0.142; 95% CI: −0.091–0.376). The pooled diagnostic performance of AIP for MetS showed an AUC of 0.864 (95% CI: 0.856–0.871).

Conclusions: Elevated AIP levels are significantly associated with metabolic syndrome compared to healthy individuals, supporting its potential role as a useful biomarker. Nevertheless, AIP does not significantly differentiate between diabetic and non-diabetic MetS subgroups. The overall diagnostic accuracy appears acceptable, suggesting potential clinical utility, although further large-scale prospective studies are required to better define its prognostic and diagnostic value.

Keywords: Atherogenic index of plasma (AIP), Metabolic syndrome, Biomarkers

MPP6-4

Diagnostic accuracy of artificial intelligence for image-based unstable carotid plaque identification: a systematic review and meta-analysis

Youssef Soliman*, Nariman Ayman

Faculty of Medicine, Assiut University, Egypt

Objectives: Carotid artery atherosclerosis plays a major role in generating embolic events that culminate in ischemic stroke. Accurate detection of vulnerable carotid plaques through non-invasive imaging modalities is essential for effective stroke prevention. Artificial intelligence (AI) has shown considerable potential in improving the precision of plaque risk classification. This review seeks to evaluate the diagnostic performance of AI algorithms in differentiating unstable carotid plaques from their stable counterparts using medical imaging.

Methods: We performed systematic searches across Medline, Embase, Web of Science, IEEE, PubMed, and the Cochrane Library through June 6, 2023. Qualifying studies encompassed those employing AI algorithms to detect unstable carotid plaques from medical images. Binary diagnostic accuracy measures, including sensitivity, specificity, and Area Under the Curve (AUC), were collected. QUADAS-AI was applied to evaluate the risk of bias across the included studies.

Results: Of the 31 studies examined, 14 underwent meta-analysis, yielding a pooled sensitivity of 91% (95%CI: 86–95%), specificity of 84% (79–89%), and AUC of 0.94 (0.91–0.95). Nevertheless, only a single study reported external validation, constraining the broader applicability of these results, and considerable heterogeneity was detected (I2 > 90%). Subgroup analyses revealed performance differences attributable to factors including sample size, category of AI algorithm (machine learning or deep learning), segmentation approach (manual or automatic), and publication year. Despite apparent publication bias and between-study heterogeneity, the results highlight the potential of AI-based methods in carotid plaque risk stratification.

Conclusions: AI algorithms exhibited favorable diagnostic accuracy in detecting unstable carotid plaques. Subsequent research should prioritize rigorous external validation, promoting generalizability, and improving the interpretability of AI algorithms to support their successful translational application.

Keywords: Atherosclerosis

MPP6-5

Regional variation in the clinical impact of intravascular imaging for left main coronary atherosclerotic disease

Hendrianus[1*], Hoyoun Won[3], Sang Yeub Lee[2], Jun Hwan Cho[2], Jinhwan Jo[2], Soo-Young Lee[2], Kyung-Taek Park[3], Gyu-Tae Park[3], Young-Hoon Jeong[2], Sang-Wook Kim[2]

[1]Division of Cardiology, Natuna General Hospital, Indonesia, [2]Heart and Brain Hospital, Chung-Ang University Gwangmyeong Hospital, Republic of Korea, [3]Division of Cardiology, Chung-Ang University Hospital, Republic of Korea

Objectives: To assess whether intravascular imaging-guided optimization improves clinical outcomes in patients with left main coronary atherosclerotic disease undergoing PCI and to investigate whether these effects vary across geographic regions.
Methods: We conducted a systematic review and meta-analysis of randomized controlled trials comparing intravascular imaging-guided versus angiography-guided PCI with available left main coronary artery disease data. PubMed, Embase, and the Cochrane Central Register of Controlled Trials were searched through April 2026. The primary endpoint was target vessel failure (TVF). Hazard ratios (HRs) were pooled using a random-effects model. Pre-specified subgroup analyses were performed according to geographic region. Risk of bias was assessed using the Cochrane RoB 2 tool, and certainty of evidence using the GRADE framework.
Results: Nine randomized trials involving 2,766 patients with left main coronary artery disease were included. Overall, intravascular imaging-guided PCI was associated with a significant reduction in TVF compared with angiography guidance (HR 0.67, 95% CI 0.47-0.97; P=0.03), although substantial heterogeneity was observed (I^2=66%). Regional analyses revealed significant differences in treatment effect. In East Asian studies (n=6), imaging guidance reduced TVF by 53% (HR 0.47, 95% CI 0.35-0.64; I^2=0%), whereas no benefit was observed in European studies (n=3) (HR 1.12, 95% CI 0.89-1.42; I^2=0%) (P for interaction < 0.0001). Findings remained consistent across sensitivity analyses. Overall risk of bias was low, and the certainty of evidence was moderate.
Conclusions: In patients undergoing PCI for left main coronary atherosclerotic disease, intravascular imaging guidance was associated with improved outcomes overall; however, benefits varied substantially across geographic regions. These findings suggest that the effectiveness of imaging-guided optimization may be context-dependent and warrant dedicated contemporary studies across different practice settings.
Keywords: Left main coronary artery disease, Coronary atherosclerosis, Intravascular imaging, Intravascular ultrasound

MPP6-6

Effectiveness and safety of pravastatin in postmenopausal women with dyslipidemia in Korea: results from the MELODY study

Jun Hwa Hong[1*], Sangmo Hong[2*], Ji-Hwan Bae[3], Sung Hee Choi[4]

[1]Division of Endocrinology, Department of Internal Medicine, Daejeon Eulji Medical Center, Eulji University School of Medicine, Daejeon, Republic of Korea, [2]Department of Internal Medicine, College of Medicine, Guri Hospital, Hanyang University, Seoul, Republic of Korea, [3]Medical & Pharmacovigilance Department, Daiichi Sankyo Korea Co., Ltd., Seoul, Republic of Korea, [4]Department of Internal Medicine, Seoul National University Bundang Hospital, Seoul National University College of Medicine, Seongnam, Republic of Korea

Objectives: Menopausal hormonal changes increase the risk for dyslipidemia and metabolic complications, warranting a tailored approach to lipid management in this population. The MELODY study assessed the effectiveness and safety of pravastatin in postmenopausal Korean women with dyslipidemia in real-world clinical practice.
Methods: This multicenter, prospective, observational study enrolled postmenopausal women aged ≥50 years with dyslipidemia initiating pravastatin at the investigator's discretion. Among 3,060 enrolled patients, the safety and efficacy analysis sets comprised 2,664 and 2,362 patients, respectively. The primary endpoint was percent change in low-density lipoprotein cholesterol (LDL-C) from baseline at Week 24. Secondary endpoints included LDL-C target attainment, changes in HbA1c and fasting plasma glucose (FPG), changes in bone mineral density (BMD) at Week 48, and adverse events.
Results: At Week 24, mean percent change in LDL-C was -17.5% in statin-naive patients and +31.2% in prior statin users, yielding an overall change of +5.7% (all p<0.0001), reflecting the high proportion of prior statin users (47.5%). The LDL-C target attainment was 71.0% overall. Changes in HbA1c and FPG were -0.08% (p<0.0001) and -2.7 mg/dL (p=0.0027), respectively. No clinically significant reduction in BMD at any skeletal site at Week 48. Reported adverse events included treatment discontinuation in 1.6% of patients and myalgia in 0.5%.
Conclusions: In postmenopausal Korean women with dyslipidemia, pravastatin provided meaningful LDL-C lowering in statin-naive patients and was well-tolerated, supporting its use for lipid management in this population.
Keywords: Pravastatin, Dyslipidemia, Postmenopausal women, LDL-cholesterol, Glycemic safety

MPP6-7

Triglyceride-to-HDL-cholesterol ratio as a marker of adiposity in young Korean adults: a single-center cross-sectional study

Sehyun Kim[1*], Ye Seul Yang[1,2], Min Kyong Moon[3]

[1]Department of Medicine, Seoul National University College of Medicine, Republic of Korea, [2]Department of Internal Medicine, Seoul National University Bundang Hospital, Seoul National University College of Medicine, Republic of Korea, [3]Department of Internal Medicine, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul National University College of Medicine, Republic of Korea

Objectives: The triglyceride-to-HDL-cholesterol (TG/HDL-C) ratio is a simple marker of atherogenic dyslipidemia. We assessed its relationship with directly measured adiposity in young adults and whether it outperforms non-HDL-cholesterol, the triglyceride-glucose (TyG) index, and remnant cholesterol.

Methods: This retrospective, cross-sectional study used health examination data from Seoul National University students collected between 2022 and 2024; international students were excluded. We analyzed 11,092 domestic students aged 19-39 years with ≥12-hour fasting (5,858 men, 5,234 women; first record per person). Outcomes were body fat percentage (BF%) by bioelectrical impedance (InBody; primary) and waist circumference (WC; secondary). We examined adiposity across sex-specific TG/HDL-C quartiles, and compared markers by sex-stratified Spearman correlations and multivariable linear regression (adjusted for age, smoking, alcohol, physical activity), with incremental R^2.

Results: Adiposity increased progressively across TG/HDL-C quartiles (Q1→Q4) in both sexes: BF% from 17.3 to 25.4% in men and 27.6 to 32.4% in women, and WC from 76.6 to 86.1 cm in men and 67.5 to 72.6 cm in women (all P for trend <0.001). Each 1-SD higher log(TG/HDL-C) corresponded to +2.9 (men) and +2.0 (women) percentage-point higher BF%. Among the four markers, TG/HDL-C showed the strongest associations and highest incremental R^2 for BF% (men 0.178, women 0.117) and WC, exceeding TyG (0.147, 0.088), non-HDL-C (0.132, 0.054), and remnant cholesterol (0.119, 0.065); with all markers in one model, only TG/HDL-C remained significant, while TyG was not.

Conclusions: In young Korean adults, the TG/HDL-C ratio shows a graded relationship with adiposity and reflects it more strongly than non-HDL-cholesterol, the TyG index, or remnant cholesterol, supporting its use as a simple, preferred early marker of adiposity-related cardiometabolic risk.

Keywords: Triglyceride/HDL-cholesterol ratio, Body fat percentage, Waist circumference, Young adults,Insulin resistance

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

E-Poster

PE01

Synthetic HDL-based therapy for selective modulation of cholesterol efflux in atheroma macrophages

Khalfa Benadouda[1*], Georgios Kolypetris[1], Sirine Nouri[1], Menke De Leede[1], Tuomas Niemi-Aro[2], Noora Sjöstedt[1], Heidi Kidron[1], Artturi Koivuniemi[1]

[1]Faculty of Pharmacy, University of Helsinki, Finland, [2]Institute of Biotechnology, University of Helsinki, Finland

Objectives: Synthetic high-density lipoproteins (sHDL) are promising nanocarriers due to their anti-atherogenic properties and natural affinity for plaque macrophages. In this context, miR-33a, an intronic microRNA that negatively regulates ABCA1 expression, represents an attractive therapeutic target. This project aims to develop and characterize sHDL-based nanodiscs for the delivery of AntagomiR-33a, an antisense oligonucleotide that inhibits miR-33a. The first objective is the formulation of sHDL-AntagomiR-33a complexes, the evaluation of their physicochemical properties, and the assessment of the impact of the AntagomiR-33a-to-22a peptide ratio on particle size and assembly. The second objective involves evaluating the effects of sHDL-AntagomiR-33a formulations on cholesterol efflux and ABCA1 expression in macrophages. Ultimately, this project aims to determine whether these formulations can reduce atherosclerotic plaque burden in vivo by enhancing macrophage cholesterol efflux.
Methods: sHDL were prepared using the 22a peptide and phospholipids (DMPC and DMTAP) via the lipid thin-film hydration method followed by thermocycling. sHDL-AntagomiR-33a formulations were prepared by mixing both components at a specific ratio using thermomixing. The size and morphology of sHDL were characterized by dynamic light scattering and electron microscopy. Encapsulation efficiency was quantified using a RiboGreen assay. In vitro, J774A.1 macrophages were treated with the formulations to assess ABCA1 and ABCG1 expression by Western blotting and RT-qPCR, as well as cholesterol efflux using radiolabeled cholesterol.
Results: We successfully formulated sHDL loaded with AntagomiR-33a. Interestingly, the sHDL-AntagomiR-33a complexes appeared to stack together, forming larger assemblies in which AntagomiR-33a molecules were sandwiched between adjacent nanodiscs. We were able to control the size of these assemblies by varying the AntagomiR-33a-to-22a peptide ratio.
Conclusions: sHDL-AntagomiR-33a complexes show stacking behavior with controllable size, providing an additional level of formulation flexibility that may be relevant for biological performance. In vitro studies assessing cholesterol efflux and ABCA1 expression are ongoing to evaluate the therapeutic potential for atherosclerosis regression.
Keywords: Atherosclerosis, Cholesterol efflux, AntagomiR-33a, ABCA1

PE02

Hepatic LPLAT10 overexpression alters lipoprotein cholesterol distribution in diabetic mice

Kahori Shimizu[1*], Hideo Shindou[2,3], Hiroyuki Mizuguchi[1,4,5,6]

[1]Laboratory of Biochemistry and Molecular Biology, Graduate School of Pharmaceutical Sciences, The University of Osaka, Japan, [2]Department of Lipid Life Science, National Institute of Global Health and Medicine, Japan Institute for Health Security, Japan, [3]Department of Medical Lipid Science, Graduate School of Medicine, The University of Tokyo, Japan, [4]Global Center for Medical Engineering and Informatics, The University of Osaka, Japan, [5]Integrated Frontier Research for Medical Science Division, Institute for Open and Transdisciplinary Research Initiatives, The University of Osaka, Japan, [6]Center for Infectious Disease Education and Research, The University of Osaka, Japan

Objectives: Cholesterol is transported in the blood by lipoproteins, including chylomicrons, very-low-density lipoprotein, low-density lipoprotein, and high-density lipoprotein (HDL). Abnormalities in cholesterol metabolism have been implicated in the pathogenesis of atherosclerotic diseases and diabetes. The surfaces of lipoproteins are rich in phospholipids, and changes in these phospholipids contribute to lipoprotein metabolism. Phospholipids are converted into lysophospholipids by phospholipase A1/A2, and lysophospholipids are converted back into phospholipids by lysophospholipid acyltransferases (LPLATs) in a process known as phospholipid remodeling. However, whether individual LPLATs regulate lipoprotein metabolism and cholesterol distribution remains unclear. Herein, we investigated the effects of LPLAT10 overexpression in the liver, a major metabolic organ, on lipoprotein metabolism.
Methods: To overexpress LPLAT10 in the liver, we generated an adenovirus (Ad) vector expressing LPLAT10 (Ad-LPLAT10) and administered it to diabetic mice. Serum lipoprotein cholesterol profiles were analyzed by detailed lipoprotein fractionation, and hepatic expression of genes involved in cholesterol and lipoprotein metabolism were evaluated by quantitative RT-PCR.
Results: The total serum cholesterol levels were significantly lower in Ad-LPLAT10 group than in the control group. Furthermore, levels of HDL cholesterol, the primary form of cholesterol in mice, were markedly lower in Ad-LPLAT10 group than in the control group. To explore the mechanisms underlying the altered cholesterol distribution, hepatic expression of genes involved in cholesterol and lipoprotein metabolism was analyzed. The expression of cholesterol synthesis genes in Ad-LPLAT10 group was comparable to that in the control group. However, differences in the expression levels of HDL-related genes were observed between Ad-LPLAT10 and control groups.
Conclusions: Our findings demonstrate that liver-specific LPLAT10 overexpression affects lipoprotein cholesterol distribution and the expression of HDL-related genes in diabetic mouse models. These results suggest that phospholipid remodeling may be involved in the regulation of lipoprotein metabolism.
Keywords: Phospholipid, Cholesterol, HDL, Adenovirus vector, Lysophospholipid acyltransferase

PE03

ANGPTL4 attenuates multicellular atherogenic remodeling: validation in a human iPSC-derived atheroid model

Dong Im Cho[1*], Bo Gyeong Kang[1], Hyang Hee Cho[1], Meeyoung Cho[1], Sooji Yoo[1], Jin Yoo[1], Chanyul Kim[1], Yong Sook Kim[4], Youngkeun Ahn[2,3]

[1]Cell Regeneration Research Center, Chonnam National University Hospital, Republic of Korea, [2]Cardiology, Chonnam National University Hospital, Republic of Korea, [3]Cardiology, Chonnam National University Medical School, Republic of Korea, [4]Medical Research Center, Chonnam National University Medical School, Republic of Korea

Objectives: We evaluated the protective effects of angiopoietin-like protein 4 (ANGPTL4) using a human iPSC-derived multicellular atheroid model that recapitulates key features of atherosclerotic remodeling.
Methods: Human iPSC-derived atheroids were generated by integrating vascular spheroids composed of endothelial cells, smooth muscle cells, and cardiac fibroblasts with iPSC-derived monocytes. Atherogenic conditions were induced by TNF-α and oxidized LDL (oxLDL) stimulation. Endothelial dysfunction, immune cell recruitment, foam cell formation, smooth muscle cell phenotypic remodeling, extracellular matrix remodeling, and apoptosis were assessed.
Results: TNF-α and oxLDL stimulation induced endothelial dysfunction, characterized by reduced CD31 and VE-cadherin expression and increased VCAM1 and ICAM1 expression. ANGPTL4 partially preserved endothelial marker expression and attenuated endothelial activation. The atheroid model supported monocyte adhesion, macrophage-like differentiation, and lipid accumulation, recapitulating key immune-vascular interactions observed during atherogenesis. ANGPTL4 treatment reduced macrophage accumulation and lipid deposition within the atheroid microenvironment. Inflammatory and lipid stress also induced smooth muscle cell phenotypic switching, characterized by reduced expression of contractile markers (SM22α and CNN1) and increased expression of mesenchymal and fibrotic remodeling markers (vimentin and COL1A1). These changes were partially attenuated by ANGPTL4 treatment, suggesting mitigation of fibrotic remodeling and preservation of vascular structural integrity. Importantly, these disease-associated phenotypes developed without substantial apoptosis, indicating active pathological remodeling rather than nonspecific cytotoxicity. These findings are consistent with our previous observations that ANGPTL4 preserves KLF2 signaling and suppresses inflammatory and EndMT-associated pathways.
Conclusions: ANGPTL4 attenuated endothelial activation, macrophage lipid accumulation, and smooth muscle cell phenotypic remodeling in a human iPSC-derived atheroid model. Collectively, these findings suggest that ANGPTL4 may contribute to preservation of vascular homeostasis by limiting multicellular pathological remodeling associated with atherosclerosis. The atheroid platform provides a useful human-relevant system for modeling immune-vascular interactions and evaluating therapeutic strategies targeting atherosclerotic disease.
Keywords: ANGPTL4, Atherosclerosis, Endothelial dysfunction, Human iPSC-derived atheroid model

PE04

Boerhaavia diffusa ameliorates renal dyslipidaemia by enhancing fatty acid oxidation in high-fat diet-induced chronic kidney disease mouse models

Ching In Chung[1*], On Ying Lee[1], Angela Zaneta Chan[2], Martin Ho Yin Yeung[1,2]

[1]Department of Health Technology and Informatics, The Hong Kong Polytechnic University, Hong Kong, China, [2]Department of Anatomical and Cellular Pathology, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, China

Objectives: Chronic kidney disease (CKD) is a progressive disorder with limited therapeutic options. A high-fat diet (HFD) promotes dyslipidaemia, renal lipid accumulation, and impaired fatty acid oxidation (FAO). Boerhaavia diffusa (BD), a traditional medicinal plant with diuretic and lipid-lowering properties, has been suggested to exert renoprotective effects. This study investigates the mechanisms by which BD mitigates renal lipid accumulation and improves renal function in HFD-induced CKD.
Methods: Male C57BL/6 mice were assigned to three groups: standard chow (STC, n=6), HFD (n=6), and HFD with BD treatment (500 mg/kg oral gavage daily, n=6) for 12 weeks. Kidneys were harvested for histological evaluation and RNA sequencing. Differential gene expression and Gene Ontology (GO) enrichment analyses were performed to identify pathways associated with BD-mediated protection.
Results: Our histological findings, shown in Figure 1, confirmed that BD reduced renal lipid deposition (H&E and ORO staining) and tubular injury (KIM-1) (Abstract no.: PE-10, ICoLA2025). GO enrichment analysis further identified the fatty acid metabolic process as the most significantly enriched pathway, comprising 43 differentially expressed genes with significant expression changes. Among these, five genes including SLC27A5, CPT1A, ACADS, ECHS1, and HADH, key regulators of FAO, were selected for validation. Compared with STC, HFD-fed group exhibited downregulation of these mitochondrial FAO genes, whereas BD treatment restored their expression. As these genes are downstream targets of PPAR α, we propose that BD activates this master regulator, thereby enhancing FAO, reducing renal lipid accumulation, and restoring energy homeostasis (Figure 2).
Conclusions: BD protects against HFD-induced CKD, implicating PPARα reactivation as an underlying mechanism. These findings position BD as a promising multitarget therapeutic agent for dyslipidaemia-related CKD, reducing renal lipid accumulation and restoring metabolic homeostasis.
Keywords: Chronic kidney disease, Boerhaavia diffusa, High-fat diet, Fatty acid oxidation, PPARα signalling

PE05

Ret finger protein deficiency attenuates adipogenesis in mice with high fat diet-induced obesity

Yun-Gyeong Lee*, Anna Jeong, Sera Shin, Duk-Hwa Kwon, Hyun Kook

Department of Pharmacology, Chonnam National University Medical School, Republic of Korea

Objectives: Ret finger protein (RFP/TRIM27) is a multifunctional E3 ubiquitin ligase implicated in transcriptional regulation. While it has been previously reported to repress myogenesis, its functional role in adipose tissue metabolism remains unclear. Therefore, we investigate the metabolic impact of RFP on high-fat diet (HFD)-induced obesity and to elucidate the underlying molecular mechanisms by which it regulates adipogenesis and systemic energy expenditure.
Methods: Both global and adipocyte-specific RFP-knockout (KO) mice were subjected to high-fat diet feeding to evaluate metabolic phenotypes, including body weight, adipose tissue expansion, glucose tolerance, insulin sensitivity, and lipid profiles. Whole-body energy expenditure was assessed using indirect calorimetry. To establish translational relevance, RFP expression was analyzed in omental adipose tissue from obese human subjects. Mechanistic pathways and adipocyte differentiation were further investigated both in vivo and in vitro via interaction and transcriptional activity assays.
Results: In this study, both global and adipocyte-specific RFP deficiency markedly attenuated body weight gain, adipose tissue expansion, and adipocyte hypertrophy in HFD-fed mice, while significantly improving glucose tolerance, insulin sensitivity, and circulating lipid profiles. Indirect calorimetry further demonstrated that RFP deficiency increased whole-body energy expenditure independently of food intake or physical activity, which was consistent with the clinical finding that RFP expression was elevated in omental adipose tissue from obese human subjects. Mechanistically, loss of RFP suppressed adipocyte differentiation both in vivo and in vitro. Molecular analyses revealed that RFP physically interacts with PPAR-γ and enhances its transcriptional activity, thereby promoting the expression of key downstream adipogenic target genes, such as AP2 and adiponectin.
Conclusions: Collectively, our findings identify RFP as a positive regulator of adipogenesis and systemic metabolism via PPAR-γ activation. Because genetic ablation of RFP confers robust resistance to high-fat diet-induced obesity, targeting the RFP pathway may represent a promising and novel therapeutic strategy for the treatment of obesity and its associated metabolic disorders.
Keywords: Obesity, Energy expenditure, Adipogenesis, PPAR-γ, RFP

PE06

Fermented pear extract ameliorates free fatty acid-induced hepatic steatosis in HepG2 cells

Van-Long Truong[2*], You-Jin Park[1], Woo-Sik Jeong[1]

[1]Division of Food Science and Biotechnology, Kyungpook University, Republic of Korea,
[2]BK21 FOUR, Infectious Disease Healthcare, Kyungpook University, Republic of Korea

Objectives: Nonalcoholic fatty liver disease (NAFLD) is the most common liver disease worldwide with limited therapeutic options and affects approximately a quarter of the world's population. An accumulation of ectopic lipids in hepatocytes and subsequent lipotoxicity significantly contribute to the development and progression of NAFLD. This study was conducted to investigate the therapeutic effect of fermented pear extract (FPE) on palmitic acid (PA)-induced steatosis in human hepatocytes and to elucidate the underlying mechanism.
Methods: HepG2 cells were treated with palmitic acid (PA) in the presence or absence of various concentrations of FPE. Intracellular triglyceride and cholesterol contents were measured to assess lipid accumulation. The expression levels of lipogenesis-related proteins, including SREBP-1c, SREBP-2, PPARγ, and FASN, as well as β-oxidation-associated genes such as PPARα, were analyzed. AMPK activation was also evaluated to determine the involvement of the AMPK signaling pathway.
Results: The results showed that FPE treatment significantly reduced lipid accumulation in PA-treated HepG2 cells, as evidenced by lower levels of triglyceride and cholesterol contents. Additionally, FPE significantly inhibited the expression of the lipogenic proteins, such as sterol regulatory element-binding protein 1 (SREBP-1c), SREBP-2, peroxisome proliferator-activated receptor gamma (PPARγ), and fatty acid synthase (FASN). Simultaneously, FPE promoted lipolysis and upregulated the expression of genes involved in β-oxidation, including PPARα. Moreover, FPE attenuated PA-induced hepatic lipogenesis possibly via activating the adenosine monophosphate–activated protein kinase (AMPK) pathway.
Conclusions: Take together, these findings demonstrated that FPE alleviates hepatic steatosis through attenuating AMPK-mediated lipid accumulation and may serve as a potential agent for NAFLD management.
Keywords: NAFLD, Hepatic steatosis, Fermentation

PE07

Nicotinamide promotes lipophagy and cholesterol efflux through SIRT1-LXRα deacetylation-ABCA1 axis in vascular smooth muscle cells

Poo-Reum Choi[1,2*], Seul-Gi Kim[1,2], Hyoung-Chul Choi[1,2]

[1]Department of Pharmacology, College of Medicine, Yeungnam University, Republic of Korea,
[2]Yeungnam University, Senotherapy-based Metabolic Disease Control Research Center, Republic of Korea

Objectives: Atherosclerosis is characterized by the abnormal accumulation of lipids within VSMCs, which contributes to foam cell formation and plaque development. Although oxLDL promotes intracellular lipid deposition, the precise mechanisms regulating cholesterol homeostasis in VSMCs remain unclear. In this study, we investigated the therapeutic potential of NAM and the molecular mechanisms underlying oxLDL-induced lipid accumulation in VSMCs.
Methods: NAM treatment significantly increased SIRT1 expression while concomitantly reducing ADRP levels and lipid droplet accumulation in a concentration-dependent manner. Mechanistically, co-immunoprecipitation analyses revealed that NAM enhanced SIRT1-mediated deacetylation of LXRα. LXRα deacetylation was associated with increased expression of ABCA1, leading to enhanced cholesterol efflux and reduced intracellular cholesterol accumulation. Notably, the effects of NAM on ABCA1 expression and lipid clearance were comparable to those of the synthetic LXRα agonist T0901317.
Pharmacological inhibition of SIRT1 with EX527 abolished NAM-induced LXRα deacetylation and ABCA1 upregulation, resulting in the restoration of lipid accumulation and confirming the SIRT1 dependency of this pathway. Furthermore, inhibition of LXRα with GSK2033 or blockade of autophagic flux with bafilomycin A1 impaired NAM-induced lipophagy, as evidenced by increased p62 and LC3 II accumulation, suppressed ABCA1 expression, and enhanced intracellular cholesterol retention.
Results: Collectively, these findings demonstrate that NAM attenuates oxLDL-induced lipid accumulation in VSMCs by activating the SIRT1-LXRα axis, which promotes lipophagy and enhances ABCA1-mediated cholesterol efflux.
Conclusions: These results suggest that targeting the SIRT1-LXRα-lipophagy pathway may represent a promising therapeutic strategy for the prevention and treatment of atherosclerosis.
Keywords: Nicotinamide, Atherosclerosis, Lipophagy, Cholesterol efflux, SIRT1, LXRα

PE08

APOA1 potentiates the protective effects of platelet-derived extracellular vesicles on lymphatic function during atherosclerosis

Élizabeth Lacroix[1,2*], Nolwenn Tessier[1], Sara Babran[1,2], Julie Guillemette[1], Béatrice Bohmer-Cardinal[1,2], Mary Sorci-Thomas[3,4,5], Catherine Martel[1,2]

[1]Montreal Heart Institute, Canada, [2]Department of Medicine, Université de Montréal, Canada, [3]Department of Medicine, Medical College of Wisconsin, United States, [4]Division of Pharmacology & Toxicology, Medical College of Wisconsin, United States,
[5]Cardiovascular Center, Medical College of Wisconsin, United States

Objectives: The lymphatic system plays a crucial role in peripheral waste clearance, and its dysfunction contributes to the development of atherosclerosis. Our laboratory previously demonstrated that impaired lymphatic vessel (LV) contractility precedes atherogenesis, thereby ultimately compromising cholesterol and inflammation clearance from blood vessel walls. Platelets contribute to lymphatic homeostasis through interactions with lymphatic endothelial cells (LECs). We have shown that apolipoprotein A-1 (APOA1) enhances this protective mechanism by increasing platelet adhesion to LECs, thereby improving lymphatic integrity and limiting atherosclerosis progression. However, considering that lymph is devoid of platelets, their effects within the lumen of LVs are suboptimal. We thus investigated whether platelet-derived extracellular vesicles (PEVs) could extend these benefits within LVs. We previously demonstrated that PEVs improve LEC integrity and lymphatic contractility, and our preliminary data indicate that APOA1 enhances PEV uptake by LECs in vitro. We therefore hypothesized that APOA1 pretreatment would potentiate the beneficial effects of PEVs on lymphatic function and atherosclerosis progression.
Methods: Atherosclerosis was induced in male and female Ldlr-/- mice by feeding a high-fat diet at 6 weeks of age. At 14 weeks of age, after the establishment of atherosclerosis, mice received APOA1 injections for 6 weeks, followed by PEV injections for 3 weeks. Mice were sacrificed, and lymphatic function and atherosclerotic burden were assessed.
Results: PEV treatment alone did not significantly improve lymphatic function. In contrast, sequential APOA1/PEV treatment increased lymphatic contractility and plaque stabilization compared with PEV or APOA1 treatment alone
Conclusions: These findings suggest that APOA1 potentiates the protective effects of PEVs in atherosclerosis. Collectively, this combined approach represents an innovative therapeutic strategy targeting lymphatic dysfunction to limit atherosclerosis progression.
Keywords: Atherosclerosis, Lymphatic, Platelet, Extracellular vesicles, APOA1

PE09

Clinical significance of PAI-1 and lipid metabolism disturbances in patients with ischemic heart disease

Kadirova Nargiza*, Nurillaeva Nargiza, Nargiza

The First Department of Internal Diseases in Family Medicine and the Basics of Preventive Medicine, Tashkent State Medical University, Uzbekistan

Objectives: To assess obesity prevalence, lipid profile parameters, and plasma PAI-1 levels in patients with stable and unstable forms of ischemic heart disease.
Methods: The study included 67 patients with IHD who were divided into two groups. Group 1 consisted of 35 patients with stable angina pectoris (mean age 58.2±0.76 years), while Group 2 included 32 patients with unstable angina (mean age 59.1 ±2.0 years). The control group comprised 25 practically healthy individuals without clinical evidence of cardiovascular disease. Anthropometric characteristics, lipid metabolism parameters, and plasma PAI-1 concentrations were evaluated using standard laboratory methods. Statistical analysis was performed to compare the investigated parameters between groups.
Results: Grade I obesity was significantly more prevalent in patients with stable angina than in those with unstable angina, being detected in 22 (62.8%) and 17 (48.5%) patients, respectively ($p<0.05$). Grade II obesity was observed in 8 (22.8%) patients in Group 1 and 7 (21.8%) patients in Group 2, with no significant difference between groups. Grade III obesity was more common among patients with unstable angina, occurring in 3 (9.3%) patients compared with 1 (2.8%) patient in the stable angina group ($p<0.05$).
Total cholesterol, triglycerides, very-low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol did not differ significantly between groups ($p>0.05$). However, low-density lipoprotein cholesterol was significantly higher in Group 2 than in Group 1 (3.5±0.18 vs. 2.2±0.15 mmol/L, $p<0.01$). Plasma PAI-1 levels were markedly elevated in both patient groups compared with healthy controls. PAI-1 concentrations were 1.6-fold higher in patients with stable angina and 2.2-fold higher in patients with unstable angina. Furthermore, PAI-1 levels in unstable angina patients were approximately 1.3-fold higher than those observed in stable angina patients.
Conclusions: Patients with unstable angina exhibit a more pronounced atherogenic lipid profile and significantly elevated plasma PAI-1 concentrations. Increased PAI-1 levels reflect endothelial dysfunction, impaired fibrinolysis, and enhanced thrombotic risk, suggesting that this
Keywords: Ischemic heart disease, Obesity, PAI-1

PE10

Post-stroke changes associated with glucocorticoid dysregulation

Huiju Jo*, Goo Taeg Oh

Department of Life Science, Ewha Womans University, Republic of Korea

Objectives: Ischemic stroke induces excessive production of reactive oxygen species (ROS), leading to oxidative stress, neuroinflammation, and neuronal injury. Microglia are particularly susceptible to ROS-mediated damage, while peroxiredoxins (Prdxs) serve as critical antioxidant enzymes that maintain redox homeostasis. Corticosterone, a glucocorticoid released through activation of the hypothalamic-pituitary-adrenal (HPA) axis, exerts potent anti-inflammatory effects; however, its role in ischemic stroke remains poorly understood. This study investigated the impact of corticosterone on microglial responses under Prdx-deficient conditions following ischemic stroke.
Methods: Transient middle cerebral artery occlusion (tMCAO) was performed in wild-type (WT), Prdx1 knockout (Prdx1KO), and Prdx2 knockout (Prdx2KO) mice. HPA axis-associated hormone levels were quantified by ELISA. To evaluate the effects of corticosterone under ischemic conditions, BV2 microglial cells were treated with corticosterone and subjected to oxygen-glucose deprivation (OGD).
Results: TTC staining revealed significantly larger infarct volumes in Prdx1KO mice than in WT controls, whereas Prdx2KO mice showed no significant difference. Neurological deficits were also more severe in Prdx1KO mice. In contrast, corticosterone levels were markedly elevated in Prdx2KO mice compared with Prdx1KO mice and remained increased during the subacute phase after stroke. In BV2 microglia, corticosterone treatment suppressed inflammatory responses, increased Prdx1 expression, and improved cell viability under OGD conditions.
Conclusions: These findings suggest that sustained corticosterone elevation, particularly in Prdx2-deficient mice, may mitigate post-stroke inflammatory responses and enhance antioxidant defense through Prdx1 upregulation. HPA axis-mediated glucocorticoid signaling may therefore represent an important regulator of microglial responses following ischemic stroke.
Keywords: Stroke, Corticosterone, Prdx1

PE11

Effects of a self-care model on the prevention of kidney complications in patients with type 2 diabetes: a quasi-experimental study

Mr.Natthawirot Choodum[1*], Assistant Professor Sasinaporn Lohitthai[2], Mrs.Sirinthra Futrakul[3]

[1]Community Nurse Practitioner Program, Faculty of Nursing, Songkhla Rajabhat University, Thailand, [2]Master of Nursing Science Program in Advanced Midwifery, Faculty of Nursing, Rambhai Barni Rajabhat University, Thailand, [3]Doctor of Philosophy (Educational Administration), Faculty of Nursing, Nakhon Ratchasima Rajabhat University, Thailand

Objectives: Diabetic kidney disease is a major complication of patients with type 2 diabetes, affecting their quality of life and health expenses. The purpose of this study was to investigate the effects of the model on knowledge, self-direction, and self-care behavior.

Methods: quasi-experimental research Use a one-group format to measure before and after the experiment. The study was conducted from June to August 2025, and the sample was 44 patients with type 2 diabetes who came to receive services at the diabetes clinic at Makha Health Promoting Hospital, Nakhon Ratchasima Province. The tools used in the research included (1) self-care models developed by the researchers, (2) diabetes knowledge questionnaires and prevention of renal complications, (3) self-directance questionnaire, and (4) self-care behavior questionnaire. Data were analyzed with descriptive statistics and independent test statistics

Results: After participating in the program, the sample had an average score on diabetes knowledge. Self-directing abilities and self-care behaviors were statistically significantly higher than before participating in the program ($p<.05$).

Conclusions: The self-care model developed is effective in promoting the potential of people with type 2 diabetes to take care of themselves and supporting the prevention of renal complications. It can be applied to the development of patient care in primary health care units.

Keywords: Self-care, Self-regulation, Type 2 diabetes mellitus

PE12

Impact of high-sensitivity C-reactive protein cutoff selection on cardiovascular risk classification beyond lipid measurements in Korean adults: a KNHANES 2024 study

Rihwa Choi[1,2*], Sang Gon Lee[1,3], Sung-Eun Cho[3]

[1]Laboratory Medicine Center, GC Labs, Republic of Korea, [2]Department of Laboratory Medicine and Genetics, Sungkyunkwan University School of Medicine, Republic of Korea, [3]Endocrine Substance Analysis Center, GC Labs, Republic of Korea

Objectives: Although lipid abnormalities are widely used for cardiovascular risk assessment, hsCRP may identify individuals with inflammatory risk who are not detected by conventional lipid measurements. This study aimed to describe the distribution of igh-sensitivity C-reactive protein (hsCRP) in Korean adults and to evaluate the incremental detection of elevated hsCRP beyond lipid abnormalities using nationally representative data.

Methods: We analyzed 2024 Korea National Health and Nutrition Examination Survey data from adults aged ≥20 years with available hsCRP and lipid measurements, excluding pregnant women. Elevated hsCRP was defined using three cutoffs: ≥1 mg/L, ≥2 mg/L, and >3 mg/L. Additional detection by hsCRP was defined as the proportion of participants without any lipid abnormality but with elevated hsCRP.

Results: A total of 5,769 adults were included in the common hsCRP and lipid analysis population. In the survey-weighted analysis, 72.5% of participants had hsCRP <1 mg/L, whereas 12.8%, 6.2%, 6.9%, and 1.6% had hsCRP 1 to <2, 2 to ≤ 3, >3 to <10, and ≥10 mg/L, respectively. The proportions of participants with hsCRP ≥1 mg/L, ≥2 mg/L, and >3 mg/L were 27.5%, 14.7%, and 8.5%, respectively. Any lipid abnormality was present in 34.4% of participants. Adding hsCRP to lipid abnormality increased the overall detection proportion to 49.2% for hsCRP ≥1 mg/L, 42.1% for hsCRP ≥2 mg/L, and 38.8% for hsCRP >3 mg/L. The corresponding additional detection by hsCRP among participants without lipid abnormality was 14.8%, 7.7%, and 4.4%, respectively.

Conclusions: hsCRP identified additional individuals who were not detected by conventional lipid abnormalities, supporting its potential role as a complementary inflammatory biomarker for cardiometabolic risk assessment in Korean adults and highlighting the need for population-specific interpretation of hsCRP cutoffs.

Keywords: High-sensitivity C-reactive protein, Cardiovascular risk, Risk reclassification, KNHANES

PE13

The metabolic knife: how bariatric surgery transforms blood pressure and cardiometabolic risk in obese hypertensive patients (a systematic review and meta-analysis)

Elen Putri Cintya[1*], Lutfi Hafiz Zunardi[2]

[1]Medicine, Universitas Brawijaya, Indonesia, [2]Cardiology, Sunan Kalijaga General Hospital Indonesia, Indonesia

Objectives: Obesity drives hypertension and cardiometabolic disease through metabolic and inflammatory pathways. Despite lifestyle and pharmacological therapies, long-term risk control remains challenging. Bariatric surgery has demonstrated potential cardiovascular and metabolic benefits beyond weight loss, but its magnitude compared with non-surgical strategies remains uncertain. This study aimed to quantify the effects of bariatric surgery on blood pressure and cardiometabolic outcomes in obese hypertensive patients.

Methods: Adhering to PRISMA guidelines, a systematic search was conducted across PubMed, Scopus, Embase, and the Cochrane Library to identify RCTs comparing bariatric surgery with non-surgical management in obese hypertensive patients. Primary outcomes were systolic blood pressure (SBP) and diastolic blood pressure (DBP); secondary outcomes included HbA1c and lipid profiles. Pooled mean differences (MDs) were estimated using a random-effects model. Heterogeneity was assessed via Cochran's Q and I^2 statistics, and publication bias was evaluated through funnel plots and sensitivity analyses.

Results: Eighteen RCTs comprising 1,470 participants were included. Bariatric surgery was associated with significantly greater reductions in SBP (MD 4.03 mmHg; 95% CI: 2.53 to 5.52; I^2=72%) and DBP (MD 3.11 mmHg; 95% CI: 2.17 to 4.06; I^2=78%) compared with non-surgical management. Meaningful improvements in cardiometabolic indices were also observed, encompassing reductions in HbA1c (MD 0.79; 95% CI: 0.70 to 0.88) and triglyceride levels (MD 17.03 mg/dL; 95% CI: 13.51 to 20.55), alongside a significant increase in HDL cholesterol (MD 6.77 mg/dL; 95% CI: 5.74 to 7.81). Pooled estimates consistently favored bariatric surgery across all outcomes, notwithstanding moderate between-study heterogeneity.

Conclusions: Bariatric surgery confers clinically significant improvements in blood pressure and a broad spectrum of cardiometabolic parameters in obese patients with hypertension. These results substantiate bariatric surgery as a comprehensive therapeutic strategy that simultaneously addresses the hemodynamic and metabolic dimensions of obesity-related cardiovascular disease, supporting its consideration beyond weight management alone in appropriately selected patients.

Keywords: Bariatric surgery, Obesity, Hypertension, Blood pressure reduction

Withdrawn

PE14

Integrative post-GWAS analyses identify HEIDI-clean causal genes for hypertension in East Asian populations

Yongho Jee[1*], Keum Ji Jung[2], Wes Spiller, Tae-Jin Song[3]

[1]Advanced Biomedical Research Institute, Ewha Womans University Seoul Hospital, Republic of Korea, [2]Institute for Health Promotion, Department of Epidemiology and Health Promotion, Graduate School of Public Health, Yonsei University, Republic of Korea, [3]Department of Neurology, Seoul Hospital, Ewha Womans University College of Medicine, Republic of Korea

Objectives: Genome-wide association studies (GWAS) have identified numerous loci associated with hypertension, yet pinpointing causal genes remains challenging, particularly in East Asian populations where integrative post-GWAS studies are still limited.

Methods: We conducted a large-scale GWAS meta-analysis of hypertension across three East Asian biobanks—TPMI (n=334,559), KCPS-II (n=155,578), and KoGES (n=72,299). To systematically prioritize causal genes, we implemented an integrative post-GWAS framework incorporating fine-mapping, summary-based Mendelian randomization (SMR), heterogeneity in dependent instruments (HEIDI) testing, colocalization analysis, and gene-based association testing. Only genes supported by concordant evidence and passing HEIDI tests across tissues were defined as HEIDI-clean causal genes.

Results: The meta-analysis identified multiple genome-wide significant loci for hypertension. Integrative post-GWAS analyses prioritized a refined set of candidate causal genes, of which a subset demonstrated robust HEIDI-clean signals, indicating a low likelihood of linkage-driven associations. Several prioritized genes showed consistent effects across multiple tissues, including vascular and renal-relevant tissues, while others exhibited tissue-specific regulatory mechanisms. Notably, this framework successfully distinguished well-established antihypertensive drug targets from novel candidate genes with no prior pharmacological annotation.

Conclusions: Our integrative post-GWAS approach systematically identifies HEIDI-clean causal genes for hypertension in East Asian populations, reducing false-positive prioritization due to linkage disequilibrium. These findings provide a high-confidence genetic foundation for downstream functional validation and drug target discovery, and highlight the value of large-scale, ancestry-specific post-GWAS integration.

Keywords: Hypertension, East Asian biobank, Post-GWAS integration, HEterogeneity In Dependent Instruments

PE15

Trends in lipid-lowering drug prescription patterns in patients with dyslipidemia: a real-world data from 2010 to 2023

Seung-Hyun Ko[1*], Kyungdo-Han[3], Bongseong Kim[3], Kyuho Kim[1], Yeseul Yang[2], Kyuna Lee[1]

[1]Endocrinology & Metabolism, The Catholic University of Korea, Republic of Korea, [2]Department of Internal Medicine, Seoul National University College of Medicine, Republic of Korea, [3]Statistics and Actuarial Science, Soongsil University, Republic of Korea

Objectives: Dyslipidemia is a major modifiable risk factor for cardiovascular disease, and appropriate pharmacological management plays a pivotal role in the prevention of cardiovascular events. This study aimed to analyze the temporal changes in lipid-lowering drug prescription patterns among patients diagnosed with dyslipidemia (ICD-10: E78) between 2010 and 2023, with a focus on the evolution of monotherapy, dual therapy, and triple or higher combination therapy regimens.
Methods: Prescription claims data using Korean National Health Insurance Service-National Sample Cohort Database were analyzed for dyslipidemia patients receiving lipid-lowering agents between 2010 and 2023. Drug classes included statins, ezetimibe, fibrates, omega-3 fatty acids, and PCSK9 inhibitors. Prescriptions were categorized into monotherapy, dual therapy, and triple or higher combination therapy. Subgroup analyses were performed for patients with concomitant cardiovascular disease, and trends in high-intensity statin use were also assessed.
Results: The number of diagnosed patients with dyslipidemia increased 2.8-fold (116,165 to 324,606), with treatment rates rising from 58.7% to 74.2%. Statins remained dominant agent (92-95%) throughout. Ezetimibe use surged from 3.7% in 2010 to 35.9% in 2023, with a sharp rise in 2016, while fibrate and omega-3 use increased modestly. PCSK9 inhibitors remained marginal at 0.05% by 2023. Monotherapy declined from 91.8% in 2010 to 55.4% in 2023, while dual and triple therapy grew to 38.8% and 5.8%, respectively. Among dual regimens, statin plus ezetimibe rose from 44.0% in 2010 to 78.1% in 2023, becoming the dominant combination. In patients with cardiovascular disease, ezetimibe use reached 46.7%, PCSK9 inhibitor use reached 0.39% by 2023 and high-intensity statin use nearly doubled from 14.2% in 2010 to 29.6% in 2023.
Conclusions: Between 2010 and 2023, lipid-lowering drug prescription patterns in patients with dyslipidemia in Korea shifted markedly from statin monotherapy toward ezetimibe-based combination regimens. This transition reflects the growing adoption of more aggressive LDL-cholesterol-lowering strategies in clinical practice.
Keywords: Dyslipidemia, Lipid-lowering agents, Cardiovascular disease

PE16

Sex differences of lipoprotein (a) levels and cardiovascular risk: retrospective multicenter cohort study of Korean patients

Subin Lim[1*], Hyung Joon Joo[2]

[1]Cardiology, Ewha Womans University Seoul Hospital, Republic of Korea,
[2]Cardiology, Korea University Anam Hospital, Republic of Korea

Objectives: Lipoprotein (a), or Lp(a), is a well-known atherosclerotic cardiovascular risk factor. Effects of sex on Lp(a) and adverse cardiovascular outcomes is not well established.
Methods: This retrospective multicenter observational study was conducted using data from three tertiary university hospitals in Korea. Patients with Lp(a) measurements taken between January 2019 and July 2025 were included (n=24,982). Multivariable analysis by Cox proportional hazards model and Kaplan-Meier curve analysis were performed. Primary outcome was major adverse cardiovascular events (MACE), defined as a composite of cardiovascular death, new-onset MI, stroke, or coronary revascularization.
Results: Approximately half of the population were male (52.3%), and the median Lp(a) level was 14.0 mg/dL. Elevated Lp(a) (>50 mg/dL) was a significant independent predictor in men (aHR 1.33; 95% CI, 1.08-1.68; p=0.007) but not in women (aHR 0.87, 95% CI 0.64-1.18; p=0.364). Similar results were seen with Lp(a) >30 mg/dL. The risk of MACE increased linearly with Lp(a) in men but not in women. Stratification into age strata did not reveal a specific age group with elevated Lp(a) risk in women.
Conclusions: In this study, elevated Lp(a) was associated with MACE in men but not in women. Careful revision of conventional thresholds and measurement recommendations may be needed to reflect differential risk stratification in women and men.
Keywords: Lp(a), Sex

PE17

Provincial variation in non-alcoholic fatty liver disease burden and associated risk factors among adults aged 50-69 years in Indonesia: a population-based study

Nazala Safira[1*], Zulfania Rahmah[2], Zavia Putri Salsabila[3]

[1]Medical Internship Program, Banjarnegara Muhammadiyah Hospital, Indonesia,
[2]Medical Internship Program, Al-Aziz Islamic Hospital, Indonesia, [3]Medical Internship Program, Islamic Weleri Hospital, Indonesia

Objectives: Non-alcoholic fatty liver disease (NAFLD) is a disease whose prevalence continues to increase every year. NAFLD is closely related to obesity and metabolic disorders. However, information regarding the distribution of NAFLD and associated risk factors in at-risk age groups is still limited. Therefore, this study aims to describe the variation of NAFLD in provinces in Indonesia and its relationship with several risk factors in the population aged 50-69 years.
Methods: This research uses secondary data from the Global Burden of Disease (GBD) 2023. The analysis was conducted in 34 provinces in Indonesia, with outcomes represented as disability-adjusted life years (DALYs) rate due to NAFLD in the age group of 50-69 years. The rate is represented per 100,000 population. The analyzed risk factors include high fasting plasma glucose (FPG), high body-mass index (BMI), and smoking. The normality test was conducted using Shapiro-Wilk. The relationship between DALYs and risk factors was analyzed using Spearman correlation.
Results: The incidence of NAFLD varies significantly among Indonesian provinces. North Maluku has the highest DALYs rate (21.39), followed by Aceh (19.12) and Riau (18.13). Bali (8.99), DI Yogyakarta (9.63), and East Nusa Tenggara (11.31) have the lowest NAFLD incidence. The highest average attributable burden of DALYs was found in smoking (6.91), followed by high fasting plasma glucose (6.35) and high body-mass index (2.62). Spearman correlation analysis showed significant positive correlations between total DALYs and high fasting plasma glucose (ρ=0.687; p<0.001), high body-mass index (ρ=0.662; p<0.001), and smoking (ρ=0.645; p<0.001). High fasting plasma glucose shows the strongest correlation with the burden of NAFLD.
Conclusions: The burden of NAFLD among the 50-69 age group varies by province in Indonesia. The DALYs due to NAFLD have positive associations with high fasting plasma glucose, high body-mass index, and smoking, with the strongest association for high fasting plasma glucose.
Keywords: Non-alcoholic fatty liver disease, Disability-adjusted life years, Risk factors

PE18

Baseline atherogenic dyslipidemia and early NT-proBNP clearance in acute heart failure: a retrospective cohort study

Muhammad Ayyub Aiman bin Mohd Fauzi[1,2,3*], Mohamad Rahal Yusoff[4], Mohamad Rodi Isa[5], Noor Alicezah Mohd Kasim[1,2]

[1]Cardiovascular Advancement and Research Excellence Institute (CARE Institute), Universiti Teknologi MARA, Malaysia, [2]Department of Pathology, Universiti Teknologi MARA, Malaysia, [3]Near Peer Instructor Unit, AIMST University, Malaysia, [4]Department of Internal Medicine, Universiti Teknologi MARA, Malaysia, [5]Department of Public Health and Preventive Medicine, Universiti Teknologi MARA, Malaysia

Objectives: Atherogenic dyslipidemia, typically characterised by elevated triglycerides, reduced HDL cholesterol, and a predominance of small dense LDL particles, is commonly reflected by an increased triglyceride-to-HDL cholesterol ratio. Although this lipid phenotype is associated with long-term cardiovascular risk, its influence on early biochemical decongestion in acute heart failure is not well established. This study aimed to determine whether baseline atherogenic dyslipidemia, assessed using the triglyceride-to-HDL cholesterol ratio, is associated with short-term NT-proBNP clearance during acute heart failure admission.
Methods: A retrospective analysis was performed on 102 patients admitted for acute heart failure who had complete baseline lipid profiles and serial NT pro BNP measurements. The main exposure was the admission TG/HDL ratio. We split the cohort into High and Low metabolic risk groups based on the median value of 1.21. The primary outcome was failure to achieve a clinically relevant ≥30% reduction in NT-proBNP before discharge. Multivariable linear regression was used to assess percentage NT-proBNP clearance, while logistic regression was performed to identify predictors of failure to achieve the reduction target. Models were adjusted for age, ejection fraction, estimated glomerular filtration rate, and HbA1c.
Results: Among 102 patients, 64 (62.7%) failed to achieve a ≥30% reduction in NT-proBNP before discharge. Each 1-unit increase in TG/HDL ratio was associated with a 6.65% lower NT-proBNP clearance, but this was not statistically significant (p=0.258). High TG/HDL ratio was also not independently associated with failure to achieve the NT-proBNP reduction target (adjusted odds ratio [aOR] 0.62, 95% CI 0.25-1.54, p=0.307). In contrast, ejection fraction was the only independent predictor of failure to achieve early NT-proBNP reduction (aOR 0.96, 95% CI 0.94-0.99, p=0.002).
Conclusions: Baseline atherogenic dyslipidemia was not significantly associated with short-term NT-proBNP clearance during acute heart failure admission. Early biochemical decongestion appeared to be more closely related to left ventricular systolic
Keywords: Atherogenic dyslipidemia, Acute heart failure, NT-proBNP, Triglyceride-to-HDL ratio, Biochemical decongestion, Biomarkers, Lipid profile, Left ventricular systolic function

PE19

Resolving the genetic dark matter of inherited dyslipidemia: structural variation and polygenic susceptibility identify 24.4% of previously unexplained cases

Muhammad Hamza Mubarak[1*], Muhammad Kharisma[3], Evy Sulistyoningrum[2]

[1]Department of Clinical Pathology, Universitas Islam Indonesia, Indonesia, [2]Department of Histology, Universitas Islam Indonesia, Indonesia, [3]Department of Microbiology, Universitas Islam Indonesia, Indonesia

Objectives: Inherited dyslipidemia remains substantially underdiagnosed despite advances in genetic testing, with many clinically suspected patients lacking a definitive molecular diagnosis. This study aimed to quantify the contribution of monogenic variants, copy number variations (CNVs), and polygenic susceptibility to inherited dyslipidemia and identify predictors of severe hypercholesterolemia and premature atherosclerotic cardiovascular disease (ASCVD).
Methods: A retrospective secondary genomic analysis was conducted between January 2020 and December 2024 involving 133 individuals with clinically suspected inherited dyslipidemia, including 71 definite/probable familial hypercholesterolemia and 62 possible cases. Targeted next-generation sequencing of LDLR, APOB, PCSK9, LDLRAP1, APOE, ABCG5, and ABCG8 was performed, followed by CNV detection and polygenic risk score assessment. Diagnostic yield, genotype–phenotype correlations, structural variant burden, and polygenic susceptibility were evaluated. Statistical analyses were performed using SPSS v29.0, including Chi-square testing, independent t-tests, multivariable logistic regression, and ROC analysis. The original study received Institutional Review Board approval (Protocol No. 2021-0927).
Results: Pathogenic or likely pathogenic variants were identified in 49/133 patients (36.8%), predominantly involving LDLR (73.5%), APOB (14.3%), and PCSK9 (6.1%). CNV analysis identified clinically significant structural variants in 7 patients (5.3%), increasing molecular diagnostic yield from 31.6% to 36.8%. Genotype-positive individuals exhibited significantly higher LDL-C concentrations than genotype-negative patients (238.4±41.6 vs. 191.7±36.2 mg/dL; p<0.001) and a greater prevalence of premature ASCVD family history (69.4% vs. 38.1%; p=0.002). Elevated polygenic risk scores were observed in 24.4% of genetically unresolved cases. Logistic regression identified LDL-C ≥220 mg/dL (OR 3.82, 95% CI 1.74–8.41) and family history of premature ASCVD (OR 2.97, 95% CI 1.31–6.71) as independent predictors of genetically confirmed disease. The final predictive model demonstrated good discrimination (AUC=0.84).
Conclusions: Comprehensive genomic profiling substantially improves diagnostic resolution in inherited dyslipidemia. Incorporating CNV detection and polygenic risk assessment reveals clinically relevant genetic contributors overlooked by conventional sequencing and enhances precision diagnosis, cardiovascular risk stratification, and personalized lipid management
Keywords: Polygenic susceptibility

PE20

Lipid-lowering efficacy in omani patients with familial hypercholesterolemia

Suaad Al Mukhaini[1*], Mohsin Al Lawati[2], Khalid Al Rasadi[2]

[1]Directorate of Nursing, Sultan Qaboos University Hospital, University Medical City, Oman,
[2]Department of Biochemistry, College of Medicine & Health Sciences, Sultan Qaboos University, Oman

Background: Familial hypercholesterolemia (FH) causes severely elevated LDL-C and premature atherosclerotic cardiovascular disease. The comparative efficacy of lipid-lowering regimens in Omani FH patients has not been well established.
Objective: This study compared lipid-lowering treatment (LLT) regimens in Omani FH patients, assessing LDL-C percentage reduction and target achievement (<1.8 mmol/L) per the 2019 ESC/EAS guidelines.
Methods: This cross-sectional analysis included 83 FH patients from a single lipid clinic. Diagnosis was based on Dutch Lipid Clinic Network (DLCN) criteria or genetic confirmation. Clinical and lipid data were collected at baseline and after treatment initiation.
Results: Mean age was 44.02±14.2 years, with 51 males and 32 females. Mean baseline LDL-C was 7.17±2.1 mmol/L, and mean post-treatment LDL-C was 2.85±1.6 mmol/L. Overall, 54% of FH patients had established coronary artery disease. The LLT regimens were distributed as follows: statins alone (n=15), statin + ezetimibe (n=31), and statin + ezetimibe + PCSK9 inhibitor-based therapy (n=37). The group receiving statin + ezetimibe + PCSK9 inhibitor achieved significantly greater LDL-C reduction compared to the statin + ezetimibe group (67% vs 51%; p<0.01). The LDL-C target was achieved in 27% of the overall cohort, with significantly more patients reaching goal in the PCSK9 inhibitor-based group compared to the statin-ezetimibe group (68% vs 32%; p<0.01).
Conclusions: In this high-risk Omani FH cohort, the addition of PCSK9 inhibitor-based therapy to statin + ezetimibe provided substantially greater LDL-C reduction and higher target achievement rates than statin + ezetimibe alone, supporting its superior efficacy in this population.
Keywords: Familial hypercholesterolemia, LDL-C, Lipid-lowering therapy, Target achievement, Oman

PE21

Beyond weight loss: psychiatric and eating behavior safety signals associated with GLP-1 therapies in FAERS

Ekram Hassan Hasanin*

Faculty of Medicine, University of Tripoli, Libyan Arab Jamahiriya

Objectives: The use of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) for obesity and metabolic disorders has increased substantially worldwide. Alongside their therapeutic benefits, concerns have emerged regarding gastrointestinal, pancreatic, psychiatric, and eating behavior-related adverse events. This study aimed to evaluate the real-world adverse event profile of GLP-1 receptor agonists using the FDA Adverse Event Reporting System (FAERS).
Methods: A retrospective pharmacovigilance study was conducted using FAERS reports associated with Semaglutide, Tirzepatide, Liraglutide, and Dulaglutide. Reports were screened for gastrointestinal, pancreatitis-related, psychiatric, gallbladder-related, and eating behavior-related adverse events. Duplicate and incomplete reports were excluded. Descriptive analyses were performed to identify the frequency and distribution of adverse event categories.
Results: A total of 41,249 adverse event reports associated with glucagon-like peptide-1 receptor agonists (GLP-1 RAs) were identified from the FAERS database during the study period. The agents analyzed included Semaglutide, Tirzepatide, Liraglutide, and Dulaglutide.
Gastrointestinal adverse events represented the most frequently reported category, accounting for 27,232 reports (66.0%), including nausea, vomiting, diarrhea, constipation, and abdominal pain. Pancreatitis-related events were reported in 5,163 cases (12.5%).
Notably, eating behavior- and weight-related adverse events were identified in 3,571 reports (8.7%), including anorexia, decreased appetite, malnutrition, and restrictive food intake patterns. Psychiatric adverse events were observed in 2,116 reports (5.1%), including depression, anxiety, insomnia, and suicidal ideation-related terms.
Gallbladder- and biliary-related adverse events were less frequently reported, with 254 cases (0.6%) identified.
Conclusions: GLP-1 receptor agonists demonstrated substantial reporting of gastrointestinal and pancreatitis-related adverse events in the FAERS database. Notably, psychiatric and eating behavior-related adverse event signals were also identified, highlighting emerging safety concerns in real-world use. Further pharmacoepidemiological and prospective studies are warranted to better characterize these associations, particularly in patients using GLP-1 therapies for obesity management.
Keywords: Pharmacovigilance

PE22

Hepatic insulin resistance index and associated metabolic profiles in Korean adults with type 2 diabetes mellitus

Paul Kim[1,2*], Minji Kang[1,2], Hyunji Sang[3,4], Sang Youl Rhee[3,4], Hyunjung Lim[1,2]

[1]Department of Medical Nutrition, Graduate School of East-West Medical Science, Kyung Hee University, Giheong-gu, Yongin, Republic of Korea, [2]Research Institute of Medical Nutrition, Kyung Hee University, Dongdaemoon-gu, Seoul, Republic of Korea, [3]Department of Endocrinology and Metabolism, Kyung Hee University Hospital, Kyung Hee University College of Medicine, Seoul, Republic of Korea, [4]Center for Digital Health, Medical Science Research Institute, Kyung Hee University Medical Center, Kyung Hee University College of Medicine, Seoul, Republic of Korea

Objectives: Hepatic insulin resistance impairs the suppression of endogenous glucose production while preserving de novo lipogenesis, indicating a potential link with both glucose and lipid metabolism. However, its associated metabolic profile remains insufficiently characterized in Korean adults with type 2 diabetes (T2DM). This study aimed to investigate metabolic indicators associated with the Hepatic Insulin Resistance Index (HIRI) in Korean adults with T2DM.
Methods: This cross-sectional analysis included 2,475 Korean adults with T2DM from the Korea National Diabetes Program cohort. HIRI, derived from oral glucose tolerance test data, was used to estimate hepatic insulin resistance. Participants were stratified into HIRI tertiles. Anthropometric, glycemic, lipid parameters were compared across tertiles. Multivariable logistic regression evaluated the odds of dyslipidemia and metabolic syndrome components, adjusting for age, sex, body mass index, and other relevant confounders.
Results: Among the 2,475 participants (mean age 53.3±10.5 years; 56% men), mean HIRI values were 107.52±41.20, 226.68±41.19, and 521.64±303.58 in the low-, middle-, and high-HIRI tertiles, respectively. Participants in the high-HIRI tertile showed greater waist circumference (90.92 vs. 85.52 cm, p<0.0001), elevated fasting glucose (153.26 vs. 147.53 mg/dL, p<0.0001), lower 2-hour postprandial glucose (273.73 vs. 299.32 mg/dL, p=0.0001) and HbA1c (7.8 vs. 8.2%, p=0.0026), compared with the low-HIRI tertile. This group also showed higher triglycerides (190.71 vs. 141.41 mg/dL, p<0.0001) and lower HDL cholesterol (45.29 vs. 48.40 mg/dL, p<0.0001). After adjustment, the high-HIRI group showed significantly elevated OR for metabolic syndrome (OR: 1.435, 95% CI 1.085-1.898), dyslipidemia (OR: 1.484, 95% CI 1.167-1.887).
Conclusions: In Korean adults with T2DM, a high HIRI was associated with elevated fasting glucose, lower 2-hour postprandial glucose and HbA1c, higher triglycerides, lower HDL cholesterol, and elevated odds of metabolic syndrome and dyslipidemia.
Keywords: Hepatic insulin resistance, Type 2 diabetes mellitus, Lipid profile, Dyslipidemia

PE23

Cumulative burden of lipid-related indices as a predictor of incident type 2 diabetes in young adults: a nationwide cohort study

Jung Heo[1*], Jae-Hyuk Lee[1], Oak-Kee Hong[2], Bongsung Kim[3], Kyungdo Han[3], Min-Kyung Lee[1]

[1]Endocrinology and Metabolism, Myongji Hospital, Hanyang University College of Medicine, Republic of Korea, [2]Endocrinology and Metabolism, The Catholic University of Korea, Republic of Korea, [3]Statistics and Actuarial Science, Soongsil University, Republic of Korea

Objectives: The increasing prevalence of type 2 diabetes (T2D) in young adults necessitates improved risk prediction. While high triglycerides (TG), low high-density lipoprotein-cholesterol (HDL-C), and a high TG/HDL-C ratio are established risk factors, the clinical value of assessing their integrated and cumulative burden remains unclear. We investigated whether the longitudinal accumulation of these three lipid indices is associated with incident T2D in young adults.
Methods: Using the Korean National Health Insurance Service database, we included 1,840,251 adults aged 20-39 years without T2D who underwent four consecutive annual health checkups between 2009 and 2012. Three lipid indices were assessed at each health examination: high TG (≥150 mg/dL), low HDL-C (<40 mg/dL for men, <50 mg/dL for women), and high TG/HDL-C ratio (≥3.8 in men, ≥1.7 in women). A cumulative score (0 to 12) was calculated by summing the number of occurrences of these lipid abnormalities across four consecutive examinations. Participants were categorized into 13 groups based on their scores, and Cox proportional hazards models were used to evaluate the dose-response relationship between the score and the risk of incident T2D.
Results: During a mean 6.53-year follow-up, 40,286 participants developed T2D. A robust dose-response relationship was observed between the cumulative score and the risk of incident T2D. The adjusted hazard ratios (HRs) increased progressively with each score increment across all adjusted models. In the fully adjusted model, the highest cumulative score (12 points) exhibited a substantially elevated risk of T2D (HR: 7.984, 95% confidence interval: 7.438-8.571).
Conclusions: The cumulative burden of multiple lipid indices was independently and strongly associated with the risk of incident T2D in young adults. Our findings suggest that longitudinal assessment of lipid dysregulation may facilitate timely risk stratification and proactive intervention.
Keywords: TG/HDL-C ratio, Type 2 diabetes mellitus, Low HDL-C, High TG

PE24

Genetic spectrum of familial hypercholesterolaemia in multigenerational families in Oman

Mohsin Al-Lawati[1*], Khalid Al Waili[2], Fahad Zadjali[3], Mohammed Nasser Al Kindi[1], Siamak Saber[4], Koukab Al Farsi[2], Suad Al Mukhaini[5], Khalsa Al Lamki[1], Manal Al Khambashi[1], Asma Al Mubaihsi[1], Khalid Al-Rasadi[1]

[1]Department of Biochemistry, College of Medicine & Health Sciences, Sultan Qaboos University, Oman, [2]Department of Clinical Biochemistry, Sultan Qaboos University Hospital, University Medical City, Oman, [3]Oman College of Health Sciences, Ministry of Health, Oman, [4]Medical Genetic, Bion Genetic Laboratory, Oman, [5]Department of Nursing, Sultan Qaboos University Hospital, University Medical City, Oman

Objectives: Familial hypercholesterolaemia (FH) is a monogenic disorder of LDL-cholesterol metabolism associated with premature atherosclerotic cardiovascular disease (ASCVD). FH remains substantially underdiagnosed in the Arabian Peninsula. This study aimed to characterize the genetic spectrum of FH in multigenerational Omani families.
Methods: A genetic analysis was conducted on 1,392 DNA samples from clinically suspected FH patients at the Lipid Clinic at Sultan Qaboos University Hospital, utilising the Dutch Lipid Clinic Network (DLCN) scoring system. The pedigree analysis was developed for all 26 participating families spanning multiple generations. The sequencing employs successive next-generation sequencing (NGS) runs, a targeted FH gene panel (n=265; 2016-2020), whole-exome sequencing (WES; n=76; 2024-2025), and whole-genome sequencing (WGS; n=4; 2025). The pathogenicity of each of the twenty-two variants was classified according to ACMG/AMP criteria. Sanger sequencing of 220 samples validated all detected changes in index individuals and their relatives.
Results: Twenty-two pathogenic variants were found in 26 FH families, mainly in LDLR (95%) and one in APOB. Most were missense, frameshift, or stop-gained. LDLR mutations appeared in 80% of samples, with 43% being loss-of-function. WGS found large LDLR deletions in two families missed by standard sequencing. No PCSK9 or LDLRAP1 variants were detected. Variants clustered geographically, and four LDLR variants were novel.
Conclusions: Omani FH is mainly driven by LDLR loss-of-function variants, including founder and population-specific alleles shaped by consanguinity. These findings support cascade screening, early genetic diagnosis, and intensified lipid-lowering therapy to reduce the ASCVD burden on affected families.
Keywords: Familial hypercholesterolaemia (FH), LDLR, APOB, ASCVD, Oman, Multigenerational pedigree, Cascade screening, Dutch lipid clinic network (DLCN), NGS, ACMG/AMP

PE25

Higher diabetes-related distress predicts failure to achieve LDL-C targets and poor glycemic recovery in diabetes mellitus patients following myocardial infarction

Marcelino Adiska Megantara[1,2*]

[1]Internal Medicine, Sebelas Maret University, Indonesia, [2]Internal Medicine, Siloam Sentosa Hospital, Indonesia

Objectives: To analyze the association between diabetes-related distress (DrD) and cardiometabolic recovery, particularly LDL-C target achievement and glycemic improvement, following myocardial infarction (MI) in patients with diabetes mellitus (DM).

Methods: A retrospective cohort study was conducted among patients with DM following anteroseptal MI. Eligible patients were aged >50 years, had baseline Troponin T levels of 40-500 ng/L, HbA1c levels of 6.5-9.0%, received non-reperfusion treatment, and had no history of major psychiatric disorders. DrD was assessed at hospital admission using the Diabetes Distress Scale (DDS-17). Patients were categorized into mild (<2), moderate (2-2.9), and severe (≥3) distress groups. Cardiometabolic outcomes, including HbA1c, lipid profile, and LDL-C target achievement (<55 mg/dL), were evaluated three months after discharge. Changes in HbA1c and lipid parameters were compared across groups using one-way ANOVA, while LDL-C target achievement was analyzed using the chi-square test.

Results: A total of 60 patients with DM following anteroseptal MI were included, comprising 20 patients each in the mild, moderate, and severe DrD groups. Baseline HbA1c and LDL-C levels were comparable across groups (p=0.899 and p=0.208). At 3-month follow-up, patients with higher DrD demonstrated less favorable cardiometabolic outcomes, reflected by significantly higher HbA1c (p<0.001) and LDL-C levels (p<0.001). Although LDL-C reduction was comparable across groups (ΔLDL-C p=0.661), higher DrD was significantly associated with failure to achieve LDL-C targets (p<0.001). Patients with increasing distress severity also exhibited progressively smaller reductions in HbA1c during follow-up (ΔHbA1c p=0.010).

Conclusions: Higher DrD predicts failure to achieve LDL-C targets and poorer glycemic recovery following MI. Early identification of DrD may help optimize cardiometabolic recovery and secondary prevention after MI.

Keywords: Diabetes-related distress, Myocardial infarction, LDL-C target achievement, Glycemic recovery

PE26

From measurement to motivation: an integrated framework connecting 3D anthropometry, AI-driven phenotyping, and behavioral theory for precision obesity intervention

Minseo Kim*, Jane J Lee

Food and Nutrition, Sookmyung Women's University, Republic of Korea

Objectives: Conventional BMI-based obesity assessment inadequately captures heterogeneity in body fat distribution and cardiometabolic risk. Although advances in 3D anthropometry and AI-driven phenotyping have improved obesity characterization, translation into sustained behavior change remains limited. This review synthesizes evidence across 3D anthropometry, AI-based phenotyping, and behavioral science to propose an integrated framework for precision obesity intervention.

Methods: A narrative review was conducted using PubMed and the Web of Science Core Collection (January 1, 2010 to May 19, 2026). Studies on 3D anthropometric assessment, AI/machine learning-based obesity phenotyping, and behavioral theory were identified using predefined selection criteria. Evidence from adult human studies, systematic reviews, and meta-analyses was narratively synthesized to construct an integrated conceptual framework.

Results: Three principal findings were identified. First, 3D body scanning provides high-dimensional morphological indicators, including trunk-to-leg volume ratio and body roundness index. Emerging evidence suggests that these indicators may be more strongly associated with visceral adiposity, insulin resistance, and metabolic syndrome than conventional measures. Second, AI-driven phenotyping improves identification of obesity subtypes and cardiometabolic risk stratification, including metabolically high-risk phenotypes among individuals with normal BMI. Third, behavioral theories, particularly the Health Belief Model and Social Cognitive Theory, explain how personalized body composition feedback can promote behavior change. Such feedback may increase perceived susceptibility, self-efficacy, and outcome expectancy. Based on these findings, a five-layer framework is proposed: (1) 3D anthropometric measurement, (2) AI-driven phenotyping and risk stratification, (3) personalized feedback delivery, (4) activation of behavioral mechanisms, and (5) sustained dietary and lifestyle adherence. Iterative feedback loops link repeated assessment with long-term behavioral engagement.

Conclusions: This review proposes an integrated framework for precision obesity intervention that connects 3D anthropometric measurement, AI-driven phenotyping, and behavioral theory. The framework provides a pathway from obesity assessment to personalized intervention, thereby supporting long-term adherence and cardiometabolic risk reduction. Future studies are needed to evaluate feasibility and effectiveness.

Keywords: Precision obesity, 3D anthropometry, AI phenotyping, Cardiometabolic risk, Personalized intervention

PE27

Analysis of miRNAs expression in abdominal aortic aneurysm and their potential as biomarkers

Kenshi Yoshimura[2*], Keiko Urushino[2], Ryotaro Nagashima[1], Tomoyuki Wada[4], Takashi Shuto[1], Takayuki Kawashima[1], Hirofumi Anai[1], Kensuke Toyama[3], Shinji Miyamoto[1]

[1]Cardiovascular Surgery, Oita University, Japan, [2]Cardiovascular Surgery, Nakatsu Municipal Hospital, Japan, [3]Cardiovascular Surgery, Kurume University, Japan, [4]Cardiocvascular Surgery, Japanese Red Cross Oita Hospital, Japan

Objectives: Although the pathophysiology of atherosclerotic abdominal aortic aneurysms (AAA) is gradually elucidated, a biomarker for AAA has not yet been found. We analyzed miRNAs that could be potential biomarkers for AAA.
Methods: First, using publicly available RNA sequencing databases (GSE205071) in the Gene Expression Omnibus, mRNA expression in large AAA tissues (over 90 mm) and smaller AAA tissues (less than 60 mm) were compared. We listed differentially expressed miRNAs in the aneurysmal tissue using another dataset (GSE110527). From the listed miRNAs, we searched for candidate miRNAs using the miRWalk database that could regulate downregulated mRNAs in the large AAA in the GSE205071 data. Second, using cryopreserved serum and aneurysmal tissue samples of AAA patients who underwent surgery in Oita University Hospital (n=16), we conducted miRNA expression analysis using qPCR. In the comparison of the tissue samples, the expressions of different sites of AAA (small vs. large site of the same aneurysm) were analyzed.
Results: APOC1, APOE, and TMEM140 were downregulated in the large AAA tissue in the GSE205071 data. By using GSE110527 data and the miRWalk database, hsa-miR-770-5p and hsa-miR-193b-5p were nominated for further analysis. Neither miR-770-5p nor miR-193b-5p expression levels were statistically different between the two parts of the AAA tissue (p=0.41, and 0.12, respectively). However, the serum exosome miR-770-5p level of AAA patients was significantly lower than that of non-aneurysm patients (p=0.04). Conversely, the exosome miR-193b-5p level showed a tendency to be higher in AAA than in non-aneurysm patients (p=0.054). The ROC curves of miR-770-5p and miR-193b-5p showed that the sensitivities were 0.895 and 0.684, respectively, and the specificities were 0.500 and 0.350, respectively, and the AUCs were 0.630 and 0.649, respectively.
Conclusions: We elucidated that miR-770-5p and miR-193b-5p could be potential biomarkers for AAA.
Keywords: Abdominal aortic aneurysm, Biomarker, miRNA

PE28

Admission NT-proBNP level as a marker of biological frailty and cause-specific mortality after percutaneous coronary intervention

Aditya Reza Pratama[1,2*], Abiseka Panji Baskoro[1], Byoung Geol Choi[3], Se Yeon Choi[1], You Jin Lee[1], Jae Kyeong Byun[1], Wonsang Chu[1], Soohyung Park[1], Cheol Ung Choi[1], Seung Woon Rha[1]

[1]Cardiovascular, Korea University Guro Hospital, Republic of Korea, [2]Cardiology and Vascular Medicine, Ulin General Hospital, Indonesia, [3]Department of Biomedical, Honam University, Republic of Korea

Objectives: Admission NT-proBNP is an established biomarker of myocardial wall stress and adverse prognosis in patients with CAD. However, its association with non-cardiac death after PCI remains insufficiently characterized
Methods: This retrospective observational study of the Korea University Guro Hospital PCI registry. Included 6,972 of 4367 patients underwent PCI March 2003 and June 2021, with a baseline NT-proBNP value, stratified by admission quartile and analyzed as log2-transformed continuous variable (per doubling). The primary endpoint was 5-year non-cardiac death; secondary endpoints, cardiac and total death. Outcomes were estimated by Kaplan-Meier analysis (log-rank test) and modeled with logistic regression (odds ratios, OR) and Cox proportional-hazards models (hazard ratios, HR).
Results: The median admission NT-proBNP was 145.4 pg/mL. At 5 years, non-cardiac death occurred in 144 patients (3.3%), cardiac death in 181 (4.1%), and total death in 325 (7.4%). Crude rates rose stepwise across quartiles: non-cardiac death (1.0%, 2.0%, 3.3%, 6.9%), cardiac death (1.1% to 10.8%), and total death (2.1% to 17.7%). Kaplan-Meier 5-year incidence of total death rose from 2.4% (Q1) to 21.2% (Q4), with significant separation across endpoints (log-rank P<0.001). Per doubling, unadjusted associations were significant for non-cardiac death (OR 1.27, 95% CI 1.21-1.35; HR 1.33), cardiac death (OR 1.44, 1.36-1.51; HR 1.44), and total death (OR 1.39, 1.34-1.45; HR 1.39). After full adjustment, NT-proBNP remained independently associated with cardiac death (HR 1.16, 1.07-1.25) and total death (HR 1.11, 1.05-1.17), whereas the non-cardiac death association was attenuated and non-significant (OR 1.03, 0.94-1.12, P=0.53; HR 1.06, 0.97-1.16, P=0.18).
Conclusions: In a large real-world PCI registry, elevated admission NT-proBNP was associated with graded increases in 5-year non-cardiac, cardiac, and total mortality. Although the independent association with non-cardiac death was attenuated after multivariaable adjustment, NT-proBNP may identify patients with global biological vulnerability after PCI. These findings support comprehensive risk assessment beyond coronary focused in patients with elevated NT-proBNP
Keywords: PCI registry, Coronary artery disease, Biological frailty, Mortality, Cardiac death, Non-cardiac death, Percutaneous coronary intervention

PE29

High obstructive sleep apnea risk association with carotid atherosclerosis and impaired vitality in obese hypertensive patients with type 2 diabetes

Borys Shelest[1*], Yuliia Kovalova[2], Oleksiy Shelest[2], Iulia Rodionova[3]

[1]Department of Internal and Occupational Diseases, Kharkiv National Medical University, Ukraine, [2]Department of Internal Medicine No. 2, Clinical Immunology and Allergology Named After Academician L.T. Malaya, Kharkiv National Medical University, Ukraine, [3]Department of Prevention and Treatment of Emergency Conditions, The Government Institution L.T. Malaya Therapy National Institute of the National Academy of Medical Sciences of Ukraine, Ukraine

Objectives: Obstructive sleep apnea risk may contribute to carotid atherosclerosis through nocturnal hypertension, metabolic dysregulation and low-grade inflammation. This study aimed to evaluate whether high STOP-Bang score is associated with carotid atherosclerotic changes and impaired quality of life in obese patients with essential hypertension and type 2 diabetes.
Methods: We analyzed 132 obese patients with essential hypertension and type 2 diabetes. Obstructive sleep apnea risk was assessed using the STOP-Bang questionnaire; high risk was defined as STOP-Bang ≥5. Quality of life was evaluated using SF-36. All patients underwent carotid ultrasound. Carotid atherosclerotic changes were defined as carotid intima-media thickness (cIMT) >0.9 mm and/or carotid plaque. Lipid profile, HbA1c and 24-hour ambulatory blood pressure monitoring were assessed.
Results: High STOP-Bang score was observed in 52 patients (39.4%). These patients had higher nighttime systolic blood pressure (139.2±11.1 vs. 131.8±10.4 mmHg, p<0.001), greater cIMT (1.04±0.19 vs. 0.91±0.16 mm, p<0.001), more frequent carotid plaques (50.0% vs. 28.8%, p=0.014), and lower SF-36 vitality (43.1±13.7 vs. 54.8±14.5, p<0.001). Carotid atherosclerotic changes were more common in the high STOP-Bang group (61.5% vs. 37.5%, p=0.007). High STOP-Bang score was associated with carotid atherosclerotic changes in univariate analysis (OR 2.67; 95% CI 1.30-5.47; p=0.007) and after adjustment for age, sex, LDL-C, HbA1c and nighttime systolic blood pressure (OR 2.31; 95% CI 1.07-4.98; p=0.033). In patients with HbA1c <7.5%, this association was stronger (OR 3.42; 95% CI 1.32-8.86; p=0.011), whereas in HbA1c ≥ 7.5% it was not significant.
Conclusions: High obstructive sleep apnea risk was associated with carotid atherosclerotic changes, higher nocturnal blood pressure and impaired vitality. This association was mainly evident without marked glycemic decompensation, suggesting that uncontrolled diabetes may weaken sleep-vascular correlations.
Keywords: Hypertension, Obesity, Obstructive sleep apnea, Carotid atherosclerosis

PE30

Impact of statin use on net adverse clinical and cerebral events after ESUS

Kangho Choi[1*], Ja-Hae Kim[2]

[1]Neurology, Chonnam National University Hospital, Republic of Korea,
[2]Nuclear Medicine, Chonnam National University Hospital, Republic of Korea

Objectives: The benefit of statin therapy in patients with embolic stroke of undetermined source (ESUS) is not well established. We evaluated whether statin use influences clinical outcomes in ESUS patients, examined effects in subgroups at potential risk from statins and in those without prior atherosclerotic cardiovascular disease (ASCVD), and compared outcomes by statin intensity.
Methods: We analyzed a nationwide, multicenter prospective registry comprising 5,787 patients diagnosed with ESUS. The primary endpoint was net adverse clinical and cerebral events (NACCE) at 12 months, defined as all cause mortality, recurrent stroke, acute coronary syndrome, or major bleeding. Outcomes were compared according to statin exposure and intensity. To mitigate confounding, analyses employed propensity score-based stabilized inverse probability of treatment weighting (IPTW).
Results: Statin-treated patients had a significantly lower incidence of NACCE than untreated patients (7.41% vs 13.99%; adjusted hazard ratio HR 0.56; 95% confidence interval CI 0.39-0.82; P=0.003). There was no significant difference in NACCE between high intensity and low to moderate intensity regimens (HR 0.97; 95% CI 0.77-1.22; P=0.792). Among secondary outcomes, all cause mortality was reduced with statin therapy (HR 0.60; 95% CI 0.41-0.89; P=0.011), and major bleeding occurred less frequently in statin users than non users (0.52% vs 1.31%; HR 0.37; 95% CI 0.14-0.96; P=0.040). The favorable association between statin use and outcomes was consistent across prespecified subgroups.
Conclusions: In this registry of ESUS patients, statin therapy was associated with lower net adverse events and improved 1 year survival without an increased major bleeding risk. These observational findings warrant randomized trials to establish causality.
Keywords: Statin, Stroke, ESUS

PE31

Blood pressure variability and cerebral autoregulation failure: accelerated cognitive decline in the NACC cohort

Moaz Abouelmagd*, Amr Elrosasy, Mohammed Fathi Abduljalil

Kasr Alainy Faculty of Medicine, Cairo University, Egypt

Objectives: Blood pressure variability (BPV) has emerged as a cerebrovascular risk factor beyond mean BP elevation. Whether BPV independently predicts the rate of cognitive decline in an aging cohort with diverse neurodegenerative diagnoses has not been systematically studied at NACC scale.

Methods: We analyzed 31,005 NACC participants with ≥2 blood pressure measurements across longitudinal visits. Visit-to-visit systolic BPV was operationalized as the intraindividual standard deviation (SD) of systolic BP across all available visits. Participants were stratified into BPV tertiles: Low (SD 0.0–8.5 mmHg), Moderate (8.5–14.4 mmHg), and High (≥14.4 mmHg). The primary outcome was the slope of CDR-SB scores across longitudinal visits, estimated by participant-level linear regression. Mixed-effects models with BPV×hypertension interaction terms examined the joint contribution of BP instability and baseline hypertension status.

Results: Mean systolic BPV was 17.9 mmHg (SD) overall; median 11.3 mmHg. Kruskal-Wallis testing confirmed significant differences in CDR-SB slope across BPV tertiles ($H=28.89$, $p<0.001$). Unadjusted regression showed that every 1 mmHg increase in systolic BPV was associated with a 0.0027-point/visit steeper CDR-SB slope ($p<0.001$). Mean systolic BP level was independently associated with CDR slope (beta=0.0036, $p<0.001$). In the fully adjusted model, mean SBP drove the association (beta=0.0036, $p<0.001$) while BPV SD became non-significant, suggesting mean BP elevation mediates much of the apparent BPV effect. High BPV was associated with 57.9% hypertension prevalence vs. 43.6% in low BPV ($p < 0.001$).

Conclusions: Systolic BPV is associated with accelerated cognitive decline as measured by CDR-SB trajectory, but much of this effect is mediated by the underlying mean BP level. Hypertension status interacts with BPV to further stratify cognitive decline risk. Longitudinal BP monitoring across clinical visits may provide prognostic information beyond single-point measurements

Keywords: Blood pressure, Cerebral autoregulation, Hypertension, Cognitive decline

PE32

The preventive effect of routine dental scaling on acute myocardial infarction in patients with diabetes: a long-term follow-up study

Tsai-Wei Hsu*, Chien-Chang Liao

Department of Anesthesiology, Taipei Medical University Hospital, Taiwan

Objectives: Both diabetes and poor oral hygiene are risk factors for acute myocardial infarction (AMI). We aimed to evaluate the effects of dental scaling (DS) on the risk of AMI and its complications in patients with diabetes.

Methods: A total of 2,304,816 patients aged over 20 years with newly diagnosed diabetes between 2003 and 2015 were identified from health insurance claims data and followed until the end of 2020. We compared the risk of incident AMI between patients with diabetes who received DS and those who did not, calculating adjusted hazard ratios (HRs) and 95% confidence intervals (CIs). Additionally, we analyzed 100,204 AMI events and estimated adjusted odds ratios (ORs) and 95% CIs for post-AMI adverse outcomes associated with DS use.

Results: Receiving DS was associated with a reduced risk of AMI (HR 0.79, 95% CI 0.78–0.80). This protective effect extended to various diabetes-related conditions, including diabetic ketoacidosis, insulin use, and renal or retinal complications. The protective effect against AMI was stronger with increased frequency of DS. Among patients with diabetes who experienced AMI, those in the regular DS group had a lower acute renal failure (OR 0.83, 95% CI 0.78–0.87), stroke (OR 0.87, 95% CI 0.83–0.92), and infectious disease.

Conclusions: Routine DS as prophylaxis in patients with diabetes is associated with a reduced incidence of AMI and fewer post-AMI complications compared to those who do not receive DS. These findings highlight the importance of promoting DS prophylaxis as part of vascular health management in individuals with diabetes.

Keywords: Acute myocardial infarction, Dental scaling, Diabetes, Prevention

PE33

Deficiency of p300/CBP-associated factor exacerbates cardiac remodeling by disrupting CAMKK2 acetylation

Anna Jeong*, Yun-Gyeong Lee, Duk-Hwa Kwon, Sera Shin, Ayeong Han, Chae Ryeong Han, Hyun Kook

Department of Pharmacology, Chonnam National University Medical School, Republic of Korea

Objectives: Pathological cardiac remodeling is a major contributor to heart failure progression; however, its underlying regulatory mechanisms remain incompletely understood. This study aimed to investigate the role of p300/CBP-associated factor (PCAF) in cardiac hypertrophy and dysfunction and to elucidate the molecular mechanisms underlying its effects.
Methods: A PCAF knockout (KO) mouse model generated using CRISPR/Cas9 was subjected to isoproterenol (ISP) infusion or transverse aortic constriction (TAC) to induce pathological cardiac remodeling. Cardiac hypertrophy, fibrosis, and cardiac function were evaluated. Transcriptomic profiling was performed in TAC-operated hearts to identify PCAF-dependent molecular pathways. Mechanistic studies were conducted to determine the effects of PCAF-mediated acetylation of calcium/calmodulin-dependent protein kinase kinase 2 (CAMKK2) on AMP-activated protein kinase (AMPK) signaling. The therapeutic effects of pharmacological PCAF activation with SPV106 were also assessed in the TAC model.
Results: PCAF deficiency exacerbated ISP- and TAC-induced cardiac remodeling, as demonstrated by increased cardiac hypertrophy and fibrosis and greater impairment of cardiac function. Transcriptomic analysis of TAC-operated hearts revealed disruption of mitochondrial pathways and energy metabolism in the absence of PCAF. Mechanistically, PCAF acetylated CAMKK2, which was essential for AMPK pathway activation through phosphorylation of AMPKα at Thr172. Pharmacological activation of PCAF with SPV106 attenuated TAC-induced cardiac remodeling and preserved cardiac function.
Conclusions: PCAF is a key regulator of the CAMKK2-AMPK signaling axis and contributes to the maintenance of metabolic and structural homeostasis in the stressed heart. These findings provide novel insights into the epigenetic regulation of pathological cardiac remodeling and suggest that PCAF may represent a promising therapeutic target for heart failure.
Keywords: Cardiac remodeling, Heart failure, PCAF, CAMKK2

PE34

Synergistic attenuation of adipose inflammation and hepatic steatosis by combined taurine and leucine supplementation in a diet-induced model of sarcopenic obesity

Amara Zulfiqar[1*], Jung-Eun Yim[1,2]

[1]Interdisciplinary Program in Senior Human Ecology, Changwon National University, Republic of Korea,
[2]Department of Food and Nutrition, Changwon National University, Republic of Korea

Objectives: Sarcopenic obesity induced by high-fat, high-sucrose (HFHS) feeding is accompanied by adipose inflammation and hepatic lipid dysregulation. We investigated whether taurine and/or leucine supplementation mitigate these alterations via tissue-specific mechanisms in an HFHS-fed mouse model.
Methods: Male C57BL/6J mice (6 weeks; n=10/group) were fed a standard diet (normal) or an HFHS diet (35% fat, 25% sucrose) for 24 weeks. HFHS-fed mice received metformin (150mg/kg; positive control) or taurine (1.5% w/v), leucine (1.5% w/v), or taurine+leucine (each 1.5% w/v; total 3% w/v). White adipose tissue (WAT) was assessed by H&E staining; liver sections were stained with Oil Red O. Gene expressions were quantified by RT-qPCR in WAT (Adiponectin, CCL2, NF-κB p65/p50) and liver (CPT1a, FASN, Nrf2, NF-κB p65/p50).
Results: WAT H&E revealed adipocyte hypertrophy and crown-like structures in HFHS mice, markedly attenuated by taurine plus leucine. Hepatic Oil Red O showed severe steatosis in HFHS and taurine-alone groups, moderate accumulation with leucine, and near-normalization with taurine plus leucine and metformin. HFHS significantly increased WAT NF-κB p65 and CCL2 while suppressing adiponectin; taurine plus leucine most effectively reversed all three changes. NF-κB p50 was unaffected. In liver, HFHS reduced CPT1a and Nrf2 while increasing FASN; all supplemented groups significantly restored these markers ($p < 0.05$–0.01). Hepatic NF-κB subunits showed no significant treatment effects.
Conclusions: Taurine and leucine exert complementary tissue-specific effects in sarcopenic obesity: anti-inflammatory in WAT via NF-κB p65 suppression and adiponectin restoration, and metabolic-antioxidant in liver via CPT1a and NRF2 upregulation. Taurine alone failed to resolve hepatic steatosis, underscoring leucine's indispensable role in fatty acid oxidation recovery. These findings support combined supplementation as a targeted nutritional strategy for multi-tissue metabolic restoration in sarcopenic obesity.
Keywords: Taurine, Leucine, Adipose inflammation, Sarcopenic obesity

PE35

Can the 4-year cholesterol screening gap be compensated? A machine learning imputation simulation for high-risk classification

ChanSeok Son[1,2*], HyongGin An[3]

[1]Department of Public Health, Korea University Graduate School, Republic of Korea, [2]Client Services, IQVIA Korea, Republic of Korea, [3]Department of Biostatistics, Korea University College of Medicine, Republic of Korea

Objectives: Since 2018, dyslipidemia screening in the Korean National Health Screening Program has been performed every 4 years rather than every 2 years, potentially creating structural gaps in total cholesterol observations. We evaluated whether machine learning imputation can compensate for this gap while preserving population level cholesterol distribution and high risk classification.
Methods: We pooled 2019 to 2023 Korea National Health and Nutrition Examination Survey data and constructed a complete case dataset of 27,003 participants. Total cholesterol values were set to missing in 13,501 participants (50.0%) using a missing at random mechanism based on sociodemographic and metabolic variables. We compared three scenarios: no imputation, Random Forest based iterative imputation representing missForest, and LightGBM based miceForest using predictive mean matching. Performance was assessed using root mean squared error (RMSE), standard deviation, range, Wasserstein distance, and NCEP ATP III categories: normal (<200 mg/dL), borderline (200 to 239 mg/dL), and high (≥240 mg/dL).
Results: Without imputation, all 13,501 missing cases remained unclassified, including 1,243 true high risk cases. missForest achieved lower RMSE than miceForest (36.14 vs. 51.34), but markedly compressed the distribution: standard deviation decreased to 11.62 compared with 37.54 in true data, and range narrowed to 138 to 254 mg/dL. miceForest better preserved variability, with standard deviation 39.13, range 76 to 421 mg/dL, and lower Wasserstein distance than missForest (1.46 vs. 21.27). Clinically, missForest classified only 1 of 1,243 true high risk cases as high, whereas miceForest produced 1,380 high risk cases, closer to the true distribution.
Conclusions: Missing cholesterol observations can hide high risk individuals, and lower RMSE does not ensure clinically valid imputation. Although miceForest better preserved distribution and risk classification, imputation remains a post hoc remedy. Restoring biennial cholesterol screening should be considered to reduce structural observation gaps and improve dyslipidemia surveillance.
Keywords: Dyslipidemia screening, Total cholesterol, Missing data imputation, Machine learning, High-risk classification

PE36

Distinct cardiometabolic and pulmonary profiles across obesity-metabolic health phenotypes

Dabin Lee[1*], Minji Kim[1], Soyeon Park[1], Seohyeon Lee[1], Soyoon Cha[1], Jimin Kim[1], Oh Yoen Kim[1,2,3]

[1]Department of Health Sciences, Clinical Nutrition Major, Graduate School of Dong-A University, Republic of Korea, [2]Center for Food & Bio Innovation, DAU G-LAMP Project Group, Dong-A University, Republic of Korea, [3]Department of Food Science and Nutrition, Dong-A University, Republic of Korea

Objectives: Obesity is a heterogeneous condition in which health risks vary considerably according to metabolic status. This study investigated differences in body composition, cardiometabolic characteristics, adipokines, metabolic stress-related biomarkers, and pulmonary function across obesity-metabolic health phenotypes in adults.
Methods: A total of 36 adults were categorized into metabolically healthy normal weight (MHNW, n=11), metabolically healthy obesity (MHOB, n=17), and metabolically unhealthy obesity (MUOB, n=8) groups according to obesity (BMI ≥25 kg/m^2) and metabolic syndrome status. Body composition, cardiometabolic risk factors, adipokines, metabolic stress and inflammatory biomarkers, and pulmonary function parameters (FVC, FEV1, and FEV1/FVC) were evaluated.
Results: Compared with the MHNW group, both obesity groups exhibited higher BMI, body fat percentage, waist circumference, and visceral fat area. Furthermore, these obesity-related characteristics were more pronounced in the MUOB group than in the MHOB group. Metabolic abnormalities progressively worsened across groups, with fasting glucose, insulin, HOMA-IR, HbA1c, triglycerides, and blood pressure showing the most unfavorable values in the MUOB group.
Adiponectin and sRAGE levels tended to decrease with worsening metabolic status, whereas leptin, TGF-β1, and FGF21 levels were elevated in obesity. Interestingly, leptin, TGF-β1, and FGF21 concentrations were highest in the MHOB group. GDF15 concentrations were not significantly different among groups but tended to be highest in the MUOB group.
Pulmonary function analysis revealed a significantly lower FVC in the MUOB group compared with the other groups. FEV1 also showed a declining trend, whereas the FEV1/FVC ratio tended to be higher in the MUOB group, suggesting a tendency toward a restrictive ventilatory pattern in metabolically unhealthy obesity.
Conclusions: Distinct cardiometabolic and pulmonary profiles were observed across obesity-metabolic health phenotypes. Compared with metabolically healthy obesity, metabolically unhealthy obesity was characterized by greater central adiposity, more adverse metabolic abnormalities, and reduced pulmonary function. These findings suggest that metabolic health status may contribute to heterogeneity in obesity-related cardiometabolic and pulmonary risk.
Keywords: Obesity, Metabolic health

PE37

Peripheral microcurrent stimulation promotes neuroprotection and remyelination in a rat model of hypoxia-ischemia

Dong Rak Kwon[1*], Eun Sang Kwon[2]

[1]Department of Rehabilitation Medicine, Catholic University of Daegu School of Medicine, Daegu, Republic of Korea,
[2]Department of Medicine, College of Medicine, Keimyung University, Daegu, Republic of Korea

Objectives: Hypoxia-ischemia (HI) is a critical cause of permanent neurological deficits. While endovascular treatments focus on acute reperfusion, adjuvant therapies for chronic neural repair remain limited. This study investigates the therapeutic efficacy of peripheral microcurrent (MC) stimulation on neuroinflammation and remyelination in a neonatal rat HI model to explore its potential as a post-ischemic rehabilitative strategy.

Methods: HI was induced in postnatal day 5 (PND5) rats. MC therapy (5 V, 7 Hz) was applied for 4 weeks. Motor and cognitive functions were assessed using rotarod, cylinder, Morris water maze, and passive avoidance tests. We performed qPCR for neuroinflammatory markers (NF-κB, TNF-α, IL-6, iNOS, COX2) and Western blot for myelination markers (CNPase, MBP) to elucidate the underlying mechanisms.

Results: MC therapy significantly enhanced motor coordination and reduced forelimb asymmetry compared to the untreated HI group ($p < 0.001$, Figure 1). Cognitive performance was also markedly restored. Molecular analysis showed that MC treatment effectively suppressed the expression of pro-inflammatory cytokines (NF-κB, TNF-α, IL-6, COX2, and iNOS, Figure 2). Furthermore, expression of myelination-related proteins (CNPase and MBP) was significantly restored in the brain tissues of the therapy group.

Conclusions: Peripheral microcurrent stimulation provides potent neuroprotective and remyelinating effects by modulating neuroinflammation and enhancing white matter integrity after hypoxia-ischemia. These results suggest that MC therapy could be a promising non-invasive adjuvant strategy for enhancing neurological recovery following ischemic brain injury.

Keywords: Hypoxia-ischemia, Neuroprotection, Microcurrent

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

Author Index

A

Abdalrahman Salah Shehata 339
Abdelrahman Hatata 353
Abd-Malek Fatin-Syazwani 331
Abiseka Panji Baskoro 347, 371
Adam J Nelson 320
Ade Meidian Ambari 222
Aditya Reza Partama 347
Aditya Reza Pratama 347, 371
Ae-Ryung Lee 67
Affan Rayhan Ismail 352
Ahmed Adel 339
Ahmed Almezaine 353
Ahmed Elaraby 318
Ahmed Fouad 318
Ahmed Hassan 318
Ahmed Kertam 339
Ahmed R. A. Moustafa 337
Akiko Searles 315
Alaa Eldeeb 339
Alexander Qian 313
Alex Gao 315
Ali Bakr 318
Amara Zulfiqar 374
Amir Hegazi 338
Ammar Yasser Ali 337
Amr Elrosasy 336, 373
Amrina Mohamad Amin 331
Amritanshu Shekhar Pandey 320
Amr Khalid Abdallah 339
Angela Win 315
Angela Zaneta Chan 342, 359
Anh Tien Hoang 233
Anna Jeong 360, 374
Ann Marie Navar 320
Artturi Koivuniemi 358
Asma Al Mubaihsi 369
Assistant Professor Sasinaporn Lohitthai 363
Ayeong Han 374
Azreen Natasha Binti Azmi 320

B

Ballantyne CM 321
Bambang Widyantoro 332
Banka P 321
Basant Ahmed Mahmoud 339
Bayani Pocholo T. Maglinte 346
Béatrice Bohmer-Cardinal 361
Bernadette Alexis M. Mariño 346
Bernardo Trigatti 313
Bo Gyeong Kang 359
Bongseong Kim 319, 365
Bongsung Kim 369
Bon Jeong Ku 333
Borge G. Nordestgaard 63, 175
Borys Shelest 372
Bryan F. Elvambuena 346
Byeong-Keuk Kim 318
Byoung Geol Choi 347, 371
Byung Sik Kim 145

C

Carolyn S P Lam 320
Catapano AL 321
Catherine Martel 112, 361
Celia Catherine C. Uy 346
Celly A. Atmadikoesoemah 332
Chae Ryeong Han 374
ChaeYoung Kim 328
Chang Hee Jung 172, 268
Chan Joo Lee 97
ChanSeok Son 375
Chanyul Kim 359
Cheol Ho Park 117
Cheol Ung Choi 347, 371
Cheol-Young Park 134
Chewan Lim 120
Chien-Chang Liao 373
Ching In Chung 342, 359
Chin Meng Khoo 348
Christoph J. Binder 87
Corral P 321

D

Dabin Lee 308, 375
Dae Jung Kim 321
Daeun Kim 314, 315
Dae Young Cheon 86
Da Hyun Kang 333
Daisaku Masuda 227
Dayeon Shin 210
Deborah Ignacia D. Ona 346
Do Hee Yu 314
Dong Im Cho 359
Dong Rak Kwon 376
Dong-Wook Lee 217
Dong Zhao 71
Donna Shu-Han Lin 190
Doo Sun Sim 158
Duk-Hwa Kwon 360, 374

E

Ekram Hassan Hasanin 368
Elen 332
Elen Putri Cintya 364
Élizabeth Lacroix 361
Elmer Jasper B. Llanes 346
Eu Jeong Ku 205
Eun Jeong Cho 226
Eunji Noh 342
Eun-Jung Rhee 154
Eun-Kyeong Jo 76
Eun Nam Choi 330
Eun Roh 118
Eun Sang Kwon 376
Eun Sil Koh 126
Evy Sulistyoningrum 367

F

Fabrice Ivanes 320
Fahad Zadjali 369
Fei Tieng Lim 306
Felix Eduardo R. Punzalan 346
Fitri Bin Kahar 331
Fitri Kahar 331

G

Garam Jo 144
Georgios Kolypetris 358
Ghazy Wira Pradipta 352
Giovanna Liuzzo 320
Goo Taeg Oh 313, 314, 327, 331, 362
Guanghou Shui 214
Gyu Chul Oh 278
Gyu-Tae Park 354

H

Hack-Lyoung Kim 187
Hae Kyung Kim 283
Haeun Jang 314, 315
Hae Won Chung 321
Han Na Jung 223
Hansol Choi 321
Hayato Tada 92, 98
Heegon Kim 328
Heidi Kidron 358
Heiko Bugger 81
Hendrianus 354
Hideo Shindou 358
Hiroaki Okazaki 229
Hirofumi Anai 371
Hiroshi Akazawa 125
Hiroyuki Mizuguchi 358
Hokyou Lee 242, 321
Hong-Ki Jeon 320
Hong Sang Choi 116
Hongye Ren 320
Hosung Bae 312
Hoyoun Won 160, 297, 354
Huiju Jo 362
Hun Jee Choe 228
Hussien Osama 338
Hyang Hee Cho 359
Hye Moon Chung 164
Hyeok-Hee Lee 321
Hyeon Chang Kim 321
Hyeong Kyu Park 352
Hyeonji Lee 343
Hyeonmin Gil 328
Hye Rang Park 314, 315
Hyoeun An 321
Hyo-In Choi 307
HyongBum Kim 99
HyongGin An 375
Hyoung-Chul Choi 361
Hyue Mee Kim 178
Hyung Joon Joo 166, 365
Hyunji Sang 368
Hyun-Joung Lim 80
Hyun Jung Hwang 326
Hyunjung Lim 368
Hyun Kook 360, 374
Hyun Sung Joh 183

I

Ian En Kai Mak 348
Ian Mak 348
Ibrahim Elbably 353
Ibrahim Serag 306
Ilhak Lee 135
Inbar Habaz 313
In-Jeong Cho 140
Inki Moon 254
In Tae Jin 286
Ismail M. Fareez 306
Iulia Rodionova 372

J

Jacob Fog Bentzon 343
Jae-hoon Choi 328
Jaehoon Chung 143, 179, 245
Jae Hyoung Park 202
Jae-Hyuk Lee 369
Jaehyun Bae 139
Jae Hyun Bae 251
Jae Kyeong Byun 347, 371
Jaeseok Kim 265
Jae-Seon Lee 326
Jae-Seung Yun 192
Ja-Hae Kim 372
Jane J Lee 370
Janghoon Lee 188
Jeehoon Kang 259, 275
Jee-Seon Shim 321
Jeong-Min Kim 103, 332
Jeong Rang Park 206
Jerahmeel Aleson L. Mapili 346
Jesper Falkesgaard Højen 320
Ji-Hwan Bae 354
Ji-Hye Kim 321
Jihyun Park 308
Jimin Kim 308, 375
Jimin Lee 314, 315
Jimmi Mathisen 320
Jing Jin 327
Jin Han 131
Jinho Lee 148
Jinhwan Jo 354
Jin Jing 314
Jin Wi 163
Jin Yoo 359
Jisu Lee 308
Jiyeon Ha 332
Jiyeon Yoon 333
Jong-Chan Youn 270

Jong-Gil Park 314, 315
Jong Han Choi 142
Jong Hyun Jhee 191
Jong Kyoung Kim 328
Jong Young Lee 182
Jooeun Jeon 321
Joo-Hui Han 344
Joon Sang Lee 195
Joo-Won Park 330, 342
Ju Hyeon Kim 150
Julie Guillemette 361
Jung Eun Kim 348
Jung-Eun Yim 374
Jung-Hee Lee 318
Jung Heo 369
Jung-Joon Cha 224, 272
Jungs-Sun Kim 337
Jung-Woo Son 132
Jun Hwa Hong 114, 301, 354
Jun Hwan Cho 354
Junko Arakawa 307
Junseo Kim 333
Jun Sung Moon 290
Junyoung O. Park 333
Ju Young Kim 328

K

Kadirova Nargiza 362
Kae Won Cho 212
Kahori Shimizu 358
Kalavathy Ramasamy 306
Kangho Choi 372
Kang-Ho Choi 102
Katsunori Ikewaki 307
Keiko Urushino 371
Kei Sasaki 307
Kelvin Lee 320
Kenichi Tsujita 320
Kenshi Yoshimura 371
Kensuke Toyama 371
Keum Ji Jung 364
Khai Ling Koh 320
Khalfa Benadouda 358
Khalid Al Rasadi 367
Khalid Al-Rasadi 369
Khalid Al Waili 369
Khalid Sarhan 306, 338
Khalsa Al Lamki 369
Kim Fong Ng 320
Kordahi A 321
Koukab Al Farsi 369
Koukab AL Farsi 336
Kwang-Yeol Park 159
KyeongJin Kim 213
Kyoil Suh 352
Kyoung-Han Kim 77
Kyoung Hwa Ha 321
Kyuho Kim 108, 319, 365
Kyuna Lee 319, 365
Kyungdo Han 319, 352, 369
Kyungdo-Han 365
Kyung-Hee Kim 121
Kyung Hoon Cho 106
Kyung-Soo Kim 204
Kyungsoo Won 337
Kyung-Taek Park 354
Kyuseong Park 328

L

Laura Alonso-Herranz 343
Lee On Ying 312
Lilia Nigro Maia 320
Ling Bai 320
Lingyin Yu 348
Lourdes Ella G. Santos 346
Lutfi Hafiz Zunardi 364

M

Makoto Ayaori 307
Manal Al Khambashi 369
Manami Teramoto 307
Manuel John A. Dayao 346
Marcelino Adiska Megantara 370
Marcus Ting 348
Maria Carolina Emilia Villanueva 321
Mariko Harada-Shiba 162
Martin Ho Yin Yeung 342, 359
Martin Ng 315
Mary Sorci-Thomas 361
Md Parvez Eusof Izzudin 331
Meeyoung Cho 359
Mendizabal G 321
Menke De Leede 358
Meral Kayikcioglu 244
Michal Vrablik 55, 262
Mihye Seo 180, 352
Mikhailova E 321
Minjeong Kim 282
Min ji Cho 314
Min Ji Cho 315
Minji Kang 368
Minji Kim 308, 330, 375
Minkwan Kim 238
Min Kyong Moon 319, 355
Min-Kyung Lee 369
Minseo Kim 370
Mi-Ran Lee 314, 315
Moajury-Jung 313
Moaz Abouelmagd 336, 373
Mohamad Rahal Yusoff 366
Mohamad Rodi Isa 366
Mohamed A. Wafa 337
Mohamed Rizk 337
Mohammed Fathi Abduljalil 336, 373
Mohammed Nasser Al Kindi 369
Mohsin Al Lawati 367
Mohsin Al-Lawati 369
Mr.Natthawirot Choodum 363

Mrs.Sirinthra Futrakul 363
Muhamad Fajri Addai 332
Muhammad Ayyub Aiman bin Mohd Fauzi 366
Muhammad Hamza Mubarak 367
Muhammad Kharisma 367

N

Nada Mahmoud Abdelkarim 337
Na Hyeon Yoon 331
Najwa Dean Hasana 352
Na Rae Choi 333
Nargiza 362
Nariman Ayman 353
Narmadaa Thyagarajan 313
Nasibah Azme 326
Nathan D. Wong 59, 194
Navar AM 321
Nazala Safira 349, 366
Nazzala Safira 346
Ngan K. Vu 315
Nicole Min Yee Wong 348
Nicole Wong Min Yee 348
Nikolaus Marx 320
Nina T. Castillo-Carandang 346
Nolwenn Tessier 361
Noor Alicezah Mohd Kasim 366
Noora Sjöstedt 358
Nor Ashikin Md Sari 240
Norshariza Nordin 331
Nourhan Hatem 339
Nurdiyana Nasrudin 326
Nuriiat Efendieva 330
Nurillaeva Nargiza 362
Nurin Yasmin Mohd Khairudin 326
Nurul Izzati Abdullah 331

O

Oak-Kee Hong 369
Oh Yoen Kim 308, 375
Oleg Petrovich Shevchenko 330
Oleksiy Shelest 372
Olivia T. Sison 346
On Ying Lee 359
Osung Kwon 84

P

Pablo E Pergola 320
Paul Kim 368
Ping-Yen Liu 237
Poo-Reum Choi 361
Prof Khalid AL Rassadi 336

R

Radwa M. Abdelsattar 337
Rajkumar Dorajoo 90
Rashad G. Mohamed 337
Rashad Mohamed 338
Raymond Oliva 346
Rebecca Kim 315
Rihwa Choi 363
Rio May E. Llanes 346
Robert L. Raffai 315
Rody G. Sy 346
Ryotaro Nagashima 371

S

Sabariah Md Noor 331
Saheed 319
Saheed Oladele Amusat 319
Sahil Thakur 197
Sangeun Park 328
Sang Gon Lee 363
Sang-Guk Lee 234
Sang-Hak Lee 343
Sang Ho Jo 127
Sanghoon Shin 184
Sang Min Park 232, 250
Sangmo Hong 354
Sang-Rok Lee 176
Sanguk Yun 93
Sang-Won Han 104
Sang-Wook Kim 354
Sang Yeub Lee 109, 354
Sang Yoon Lee 149, 291
Sang Youl Rhee 368
Sara Babran 361
Satoshi Yasuda 168
Saxena M 321
Sayaka Funabashi 96
Sehoon Park 264
Sehyun Kim 355
Seohyeon Lee 308, 375
Seokhun Yang 151
Seok Oh 287
Seong Eun Lee 333
Seong keun Sonn 313
Seongpyo Lee 344
Seonwoo Lee 342
Sera Kim 216
Sera Shin 360, 374
Seul-Gi Kim 361
Seulgi Lee 308
Seung-Hyun Ko 319, 365
Seung-woon Rha 347
Seung Woon Rha 371
Seung-Woon Rha 347
Se Yeon Choi 347, 371
Shafaq Jabeen 339
Shariff-Ghazali Sazlina 331
Shiau Chin Chong 320
Shinhee Park 328
Shinji Miyamoto 371
Shuqi Li 348
Siamak Saber 369

Si-Hyuck Kang 196
Sin Yung Woo 236
Siong-Meng Lim 306
Sirine Nouri 358
SoJung Lee 201
Soobin Chang 328
Soo Heon Kwak 241
Soohyung Park 347, 371
Soo-jin Ann 343
Soo-Jin Kim 138, 274
Sooji Yoo 359
Soonhyeong Oh 327
Soo-Young Lee 354
So Ree Kim 124
Søren Lund Kristensen 320
Sotirios Tsimikas 51, 174
Soyeon Park 308, 375
Soyoon Cha 308, 375
Soyoung Kwak 208
Steg PG 321
Suaad Al Mukhaini 367
Suad Al Mukhaini 369
Subin Lim 365
Sue Young Ha 332
Suhaila Abd Muid 326
Su Jong Yu 155
Su Myung Jung 78
SungA Bae 85
Sung-Eun Cho 363
Sung Gyun Ahn 167, 318
Sungha Park 321
Sung Hee Choi 186, 354
Sung-Jin Hong 318
Sung Joon Kim 130
Sungyeon Park 318
Sunhye Shin 308
Sunmin Park 209

T

Tae-Jin Song 364
Takashi Shuto 371
Takayuki Kawashima 371
Timothy C. Tan 271, 294
Timotius Ivan Hariyanto 91
Tomoyuki Wada 371
Toru Miyoshi 156
Tsai-Wei Hsu 373
Tuan Anh Phu 315
Tuomas Niemi-Aro 358

U

Ulf Landmesser 320

V

Van-Long Truong 360
Verma S 321
Vimala R.M.T Balasubramaniam 326
Viza Atiq Umar 309

W

Wan Tying Soh 320
Weili Hu 348
Wei Wang 313
Wes Spiller 364
Whanhee Lee 218
Wilbert Allan G. Gumatay 346
Wonjin Kim 246, 279
Wonkyoung Cho 327
Wonsang Chu 371
Won Sang Chu 347
Woo-Jae Park 342
Woo-Sik Jeong 360

Y

Yasuhiro Endo 307
Yea Eun Kang 333
Yeoree Yang 200, 300
Yeseul Yang 365
Ye Seul Yang 107, 319, 355
Yongho Jee 364
Yong-Joon Lee 256
Yongmin Lee 314, 315
Yong Sook Kim 359
Yong Tae Kwon 330
Yoosik Youm 321
Yoo-Wook Kwon 344
You Ji Kim 344
Youjin Lee 347
You Jin Lee 347, 371
You-Jin Park 360
Young-Hoon Jeong 354
Young Hoon Seo 314, 315
Youngkeun Ahn 359
Young Mi Park 327
Young-Sang Lyu 171, 296
Young Yang 82
Youssef Soliman 353
Yu-Chen Wang 122
Yueying Yao 348
Yujin Ko 328
Yuliia Kovalova 372
Yun-Gyeong Lee 360, 374
Yun-Hee Lee 170
Yun Kyung Cho 113, 258

Z

Zavia Putri Salsabila 346, 349, 366
Zhang Jing 333
Zhu P 321
Zulfania Rahmah 346, 349, 366

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

발행일 2026년 9월 3일

인쇄일 2026년 9월 10일

발행처 한국지질 · 동맥경화학회
[04168] 서울시 마포구 마포대로 68 마포아크로타워 707호

전 화 02-3272-5330

팩 스 02-3272-5331

이메일 ksla@lipid.or.kr

편집제작 플랜베어
[07806] 서울시 강서구 공항대로 220 우성에스비타워II 11층 1108호

전 화 02-6953-0582

SAVE THE DATE

ICoLA 2027

The 16th International Congress on Lipid & Atherosclerosis

September
9 (Thu)-11 (Sat), 2027
Conrad Seoul, Republic of Korea

ICoLA | by KSoLA International Congress on Lipid & Atherosclerosis

KSoLA The Korean Society of Lipid and Atherosclerosis

한국지질·동맥경화학회
The Korean Society of Lipid and Atherosclerosis

SAVE THE DATE

SoLA 2027

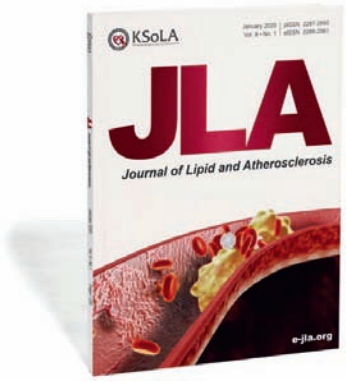
한국지질·동맥경화학회 춘계학술대회

2027 Spring Congress on Lipid and Atherosclerosis of KSoLA

2027. 4. 2. (금)-3. (토)

경주 화백컨벤션센터 (HICO)

Submit to JLA!
KSoLA's Official Journal



The JLA is an official journal of the Korean Society of Lipid and Atherosclerosis. The journal is devoted to the dissemination of knowledge of the mechanisms responsible for lipid metabolism and atherosclerosis and enhance insight into prevention, treatment, and ultimate cure for cardiovascular diseases.
The JLA covers basic, translational, and clinical research of lipid and atherosclerosis. The JLA also encourages articles on broad aspects of vascular biology, thrombosis, metabolism, nutrition, or cardiovascular health.
Only manuscripts written in English are accepted. This journal includes review article, editorial, original article, brief report, opinion, and letter to the editor.

Submit your manuscipt

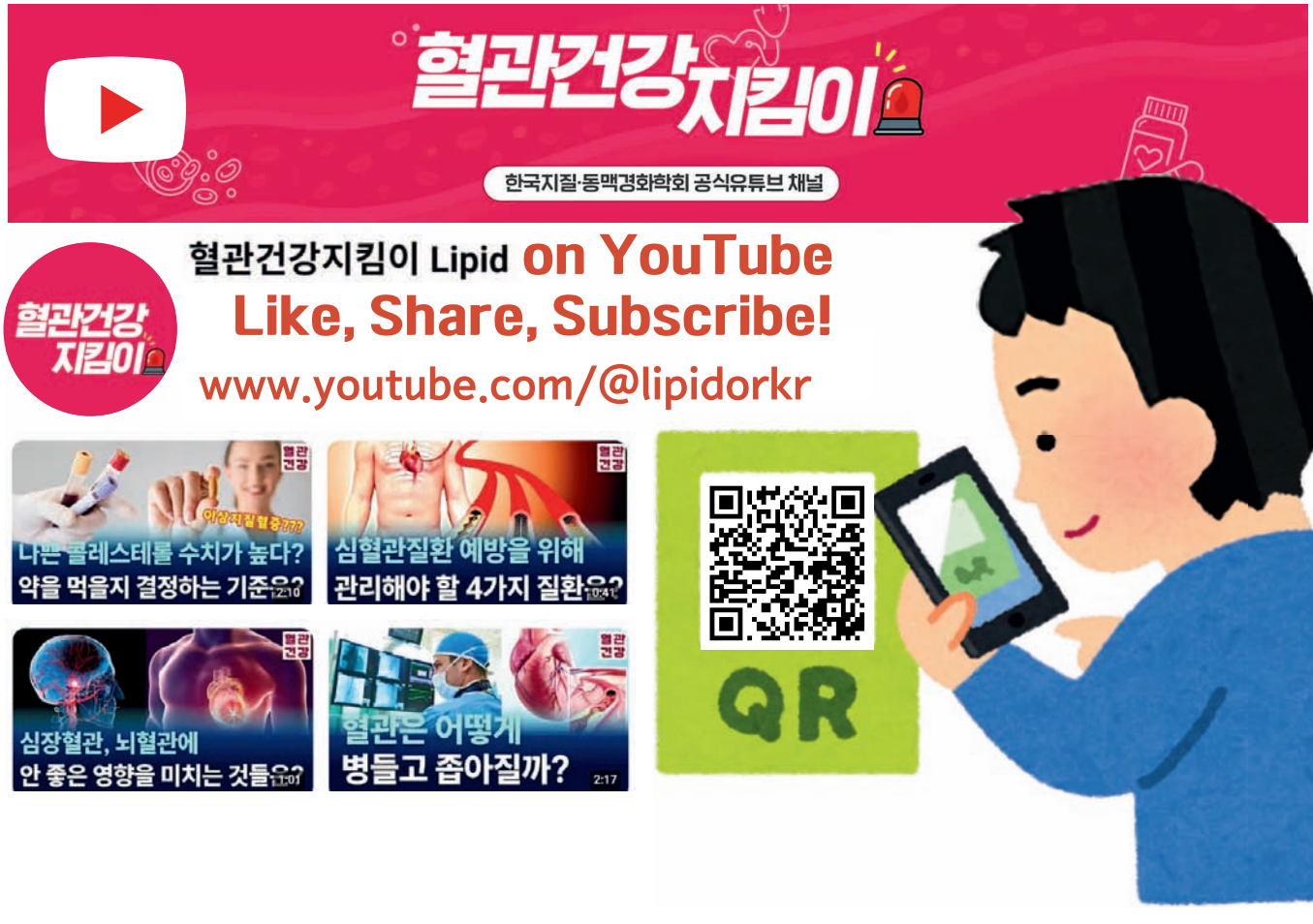
We invite you to submit your papers to JLA. The Journal is published on the official website of the JLA (**https://e-jla.org/**), and you can submit your work through the online submission system (**https://www.editorialmanager.com/la/**).

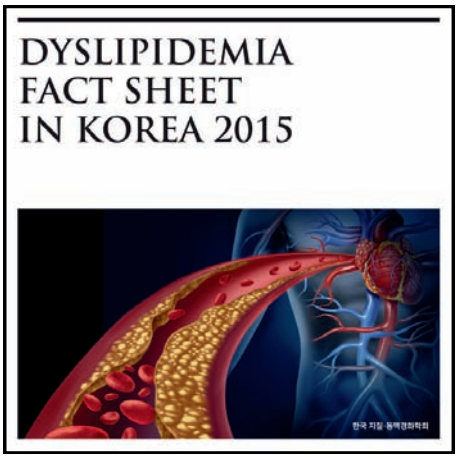
Recent articles

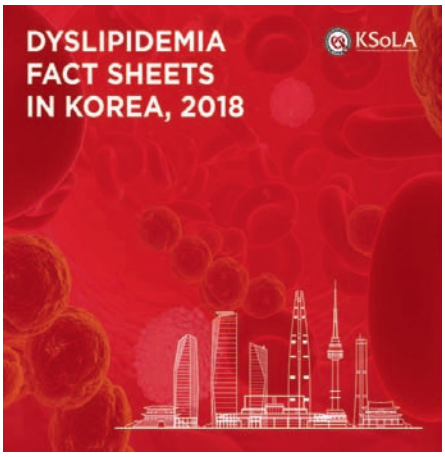
Check out our recently published articles.
https://www.e-jla.org/index.php

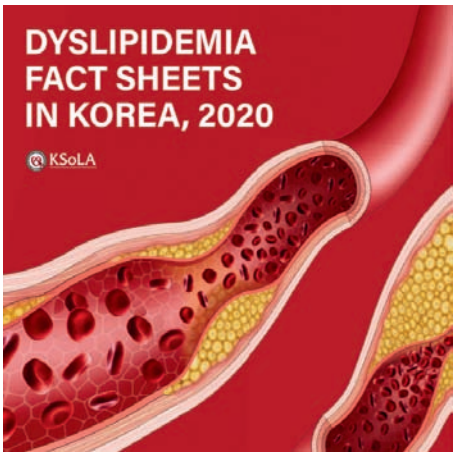
No submission fee

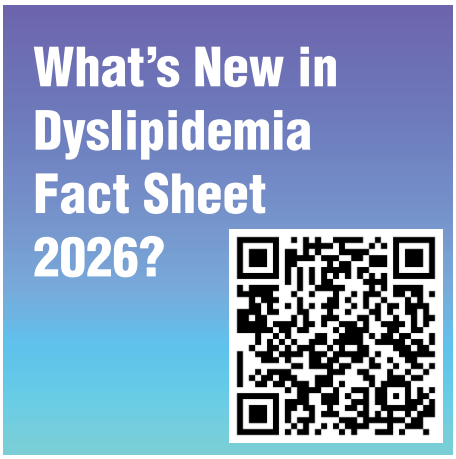
No author's submission fee or other publication-related fee since all article processing costs is supported by KSoLA.











Korean Guidelines for the Management of Dyslipidemia 2026

Committee of Clinical Practice Guideline of
the Korean Society of Lipid and Atherosclerosis

KSoLA
The Korean Society of Lipid and Atherosclerosis

For Healthcare Professionals Only
Rx Only

Vascepa®
(icosapent ethyl)

Reducing Residual CV Risk in High-Risk Patients*[1]

CV, cardivascular
* In adult statin-treated patients with triglycerides ≥150 mg/dL, who are at high CV risk due to: established CV disease, or diabetes mellitus and at least one other CV risk factor

Illustration is an artistic rendition. Not necessarily representative of clinical effects.

VASCEPA® is indicated to reduce the risk of cardiovascular events (cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, coronary revascularization, or hospitalization for unstable angina) in adult statin-treated patients with triglycerides ≥150 mg/dL, who are at high cardiovascular risk due to: established cardiovascular disease, or diabetes mellitus and at least one other cardiovascular risk factor.

[Reference]
1. Bhatt DL, Steg PG, Miller M, et al; REDUCE-IT Investigators. Cardiovascular risk reduction with icosapent ethyl for hypertriglyceridemia. N Engl J Med. 2019;380(1):11-22.

Manufactured for
AMARIN®

Imported and Marketed by
Alvogen

[Ingredients] Active Ingredient: Icosapent Ethyl 1,000 mg (equivalent to EPA 998.0 mg) per capsule (1,499 mg); Inactive Ingredients: Gelatin, Purified Water, Glycerin, Maltitol Liquid, Sorbitol Solution (Non-crystallizing). **[Indications]** Reduction of the risk of cardiovascular events (cardiovascular death, myocardial infarction, stroke, coronary revascularization, or hospitalization for unstable angina) in adult statin-treated patients with triglycerides ≥150 mg/dL, who are at high cardiovascular risk due to: established cardiovascular disease, or diabetes mellitus and at least one other cardiovascular risk factor. **[Dosage and Administration]** Adults: Take 2 capsules twice daily with meals or immediately after meals. If a dose is missed, take it as soon as remembered. Do not take a double dose to make up for a missed dose. Swallow capsules whole; do not chew, crush, or dissolve. **[Storage]** Store in a tightly closed container at room temperature (1-30°C). **[Package]** 120 capsules per bottle.

Scan for VASCEPA® Product Information

082028-VSC-26-4030603

소중한 환자의 건강한 혈관을 위해

프레탈이 걸어온 길,

프레탈이 걸어갈 길.

Efficacy

Safety

and

Trust

The 1st & Original Cilostazol **PLETAAL**

PLETAAL® SR cap. (cilostazol)

Otsuka
한국오츠카제약
Under license of Otsuka pharmaceutical co., Ltd

[효능·효과] 1. 만성동맥폐색증(버거씨병, 폐색성 동맥경화증, 당뇨병성 말초혈관병증 등)에 따른 궤양, 동통 및 냉감 등 허혈성 제증상의 개선 2. 뇌경색(심인성뇌색전증 제외) 발증 후 재발억제

[용법·용량] **프레탈®정**은 성인 1회 100mg을 1일 2회 경구 투여합니다. 단, 연령, 증상에 따라 적절히 증감합니다. **프레탈®서방캡슐**은 성인 1회 200mg을 1일 1회 경구 투여합니다. 이 약은 식사를 피하여 공복 상태에서 복용합니다.

PLT-25-002 | 20250728 approved

* 뉴스타(Neusta) 자리
뉴스타패밀리는 LDL-C, TG, HDL-C를 종합적으로 관리하는
치료 전략으로 이상지질혈증 환자의 밝은 미래를 염원합니다.

Light your star, Neusta

통합적인 지질 관리, 뉴스타 패밀리로 한번에

이상지질혈증 치료제
뉴스타틴™ 에이정
(아토르바스타틴칼슘삼수화물)

이상지질혈증 치료제
뉴스타틴™ 알정
(로수바스타틴칼슘)

이상지질혈증 치료제
뉴스타젯® 알정
(에제티미브 + 로수바스타틴칼슘)

복합형 이상지질혈증 치료제
뉴스타틴™ 듀오캡슐
(피타바스타틴칼슘 + 페노피브레이트과립)

SAMJIN
SAMJIN PHARM.

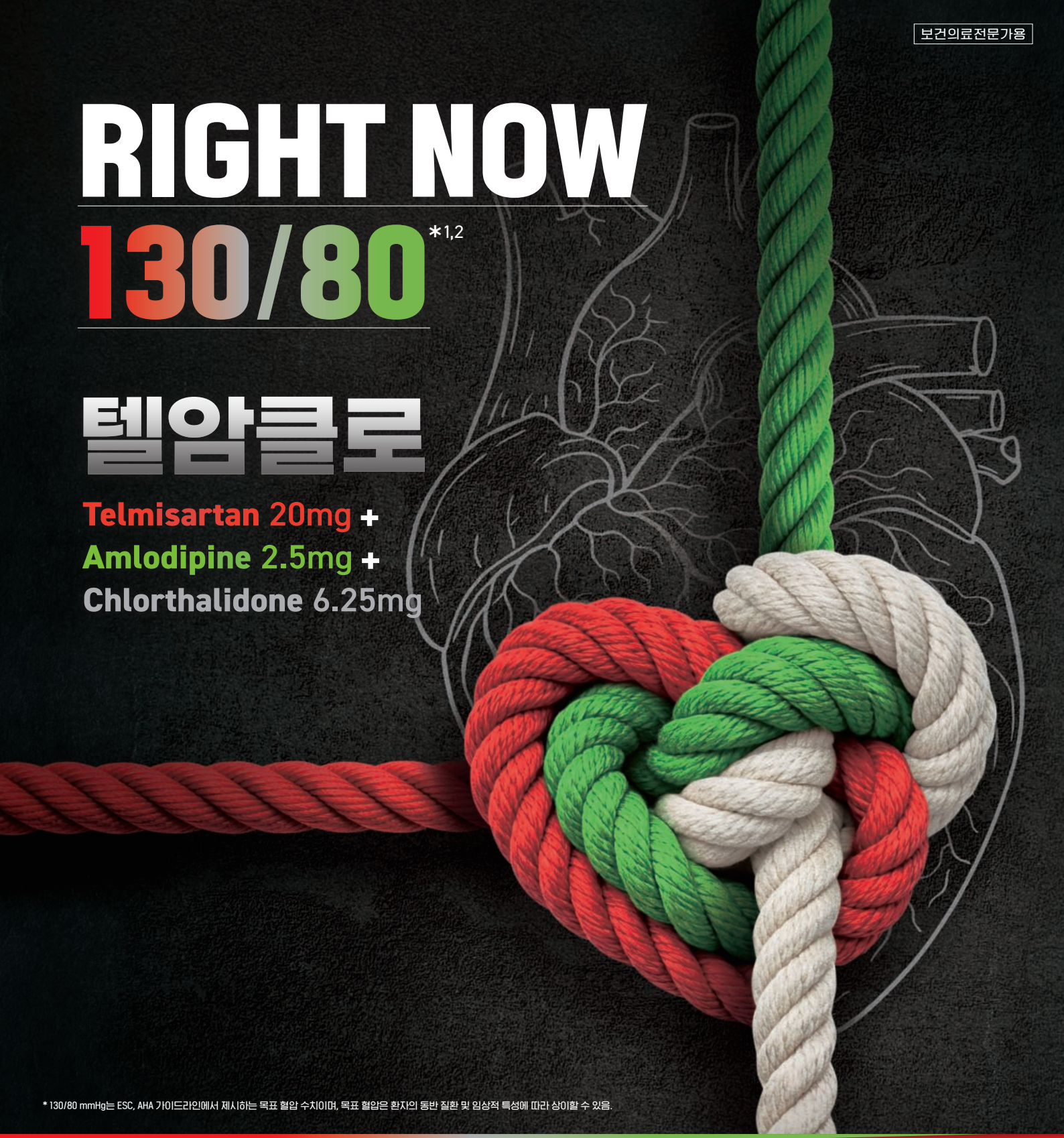
보건의료전문가용

RIGHT NOW

130/80 *1,2

텔암클로

Telmisartan 20mg +
Amlodipine 2.5mg +
Chlorthalidone 6.25mg



* 130/80 mmHg는 ESC, AHA 가이드라인에서 제시하는 목표 혈압 수치이며, 목표 혈압은 환자의 동반 질환 및 임상적 특성에 따라 상이할 수 있음.

References **1.** McEvoy JW, et al. Eur Heart J 2024;45(38):3912–4018. **2.** Jones DW, et al. Hypertension 2025:82(10):e212–e316.

제품요약정보 텔암클로정20/2.5/6.25밀리그램 제품요약정보 **[원료약품 및 그 분량]** 1정 중 텔미사르탄(EP) 20mg, 암로디핀베실산염(EP) 3.4675mg(암로디핀으로서 2.5mg), 클로르탈리돈(불규) 6.25mg · 기타 첨가제: 수산화나트륨, 글리세릴베헤네이트, 포비돈, D-만니톨, 스테아르산마그네슘, 메글루민, 전호화전분, 전분글리콜산나트륨, 미결정셀룰로오스, 콜로이드성이산화규소, 인산수소칼슘수화물 **[효능·효과]** 본태성 고혈압 **[용법·용량]** 이 약(텔미사르탄/암로디핀/클로르탈리돈 20/2.5/6.25mg)은 본태성 고혈압 환자의 초기요법으로 사용되며, 1일 1회, 1회 1정을 식사와 관계없이 복용한다. 가능하면 매일 같은 시간(예: 아침)에 복용하는 것이 권장된다. **[사용상의 주의사항]** 1. 경고 1) 이 약과 같이 레닌-안지오텐신계에 직접적으로 작용하는 약물을 임부에 투여하는 경우 자궁내는 태아 또는 신생아에게 병적 상태 및 사망을 일으킬 수 있다. 특히 임신 2기 3개월 및 3기 3개월 중의 이러한 약물의 사용은 저혈압, 신생아 두개골 형성 저하증, 무뇨증, 가역적 또는 비가역적인 신부전 및 사망을 포함한 태아 또는 신생아의 손상과 관련성이 있다. 태아의 신기능의 저하가 원인일 것으로 추정되는 양수과소증이 보고된 바 있으며, 양수과소증은 태아의 사지연축, 두개안면기형, 폐의 형성저하증과 관련성이 있다. 미숙, 자궁내 성장 지연, 동맥관열림증이 보고된 바 있으나, 이 약 노출에 의한 것인지 여부는 명확하지 않다. 임신이 진단된 경우에는 가능한 빨리 이 약의 투여를 중단해야 한다. 2) 간기능 손상 또는 진행성 간질환 환자: 체액과 전해질 균형의 경미한 변화로 인하여, 특히 간경변환자에 있어 간성혼수를 유발할 가능성이 있다. 3) 중증의 신장질환 환자: 티아지드계 이뇨제들은 중증의 신장질환 환자에 있어 고질소혈증을 유발할 수 있으며, 반복된 투여에 따른 축적작용을 나타낼 수 있다. 2. 다음 환자에는 투여하지 말 것. 1) 이 약 또는 이 약에 함유된 성분, 디하드로피리딘계 유도체, 티아지드계 이뇨제 또는 설폰아미드계열 약물에 과민증 또는 그 병력이 있는 환자 2) 임부 또는 임신하고 있을 가능성이 있는 여성 3) 수유부 4) 담도폐쇄성 질환자 5) 중증의 간질애 환자 6) 좌심실 유출로 폐쇄 [예: 높은 등급(high grade)의 대동맥협착] 7) 중증의 저혈압 환자 8) 속 환자(심장성 쇼크 포함) 9) 급성 심근경색 이후 혈류역학적으로 불안정한 심부전 환자 10) 유전성 혈관부종 환자이거나, ACE억제제 혹은 안지오텐신 II 수용체 차단제 치료시 혈관부종의 병력이 있는 환자 11) 당뇨병이나 중등도~중증의 신장애 환자(사구체여과율 <60mL/min/1.73m2)에서 알리스키렌 함유제제와의 병용 12) 무뇨증 환자 13) 중증 신부전 환자(크레아티닌청소율<30 mL/min) 14) 불응성 저칼륨혈증 환자 15) 저니트륨혈증, 고칼슘혈증 환자 16) 증상이 있는 고요산혈증 환자(통풍 병력 또는 요산결석증) 17) 치료되지 않은 매디슨증후군 환자 18) 리튬요법을 받고 있는 환자 19) 테르페나딘 또는 아스테미졸을 투여 중인 환자(QT 연장, 심실부정맥을 일으킬 수 있다.) **[제조자]** ㈜유한양행 충청북도 청주시 청원구 오창읍 연구단지로 219 **[판매자]** 에스케이 케미칼㈜ 경기도 성남시 분당구 판교로 310

※처방하시기 전 제품설명서 전문을 참고하십시오. 최신 허가사항에 대한 정보는 '식품의약품 안전처 의약품안전나라(https://nedrug.mfds.go.kr/index)'에서 확인할 수 있습니다.

Product Information 텔암클로정20/2.5/6.25밀리그램
최신 허가사항에 대한 정보는 QR 코드 또는 식품의 약품안전처 의약품 안전나라(https://nedrug.mfds.go.kr)에서 확인할 수 있습니다

TAC-HA01-202603-01

SK 케미칼

SK케미칼㈜ 경기도 성남시 분당구 판교로 310 TEL 080-021-3131 www.skchemicals.com/ls

TELAMCHLOW Tab.
20/2.5/6.25mg
(Telmisartan/Amlodipine/Chlorthalidone)

보건의료전문가용

DISCOVER THE POWER OF SEMAGLUTIDE

Once-weekly Wegovy® pen[1]

Up to **~17%**[†] mean weight loss[1,2]
~1 in 3 patients achieved **≥20%** weight loss[2,*]

Once-weekly Ozempic® pen[3]

2.7% HbA1c reduction in T2D patients
with **baseline HbA1c > 9.0%**[4]

The benefit of mono-therapy GLP-1 RA

Weight loss-fat mass reduction.[2,5,#] **Protect the heart and kidneys**[6-8,‡]

ONCE-WEEKLY
wegovy®
semaglutide injection

For people living with **obesity**[1]

OZEMPIC®
semaglutide injection

For people living with **type 2 diabetes**[3]

[Study abstract'] STEP 5 was a 104-week, multinational, randomized, double-blind, placebo-controlled, phase 3 trial conducted in adults with obesity (BMI ≥30 kg/m²) or overweight (BMI ≥27 kg/m²) with at least one weight-related comorbidity, without diabetes. Participants were assigned to receive once-weekly subcutaneous semaglutide 2.4 mg (Wegovy®) (n=152) or placebo (n=152), each as an adjunct to behavioral intervention. Co-primary endpoints were percentage change in body weight and achievement of ≥5% weight loss at week 104. At week 104, the mean change in body weight from baseline was −15.2% in the Wegovy® group versus −2.6% in the placebo group (Treatment policy estimand: ETD −12.6%; 95% CI, −15.3 to −9.8; P<0.0001) and −16.7% versus −0.6% (Trial product estimand: ETD −16.0%; 95% CI, −18.6 to −13.5). 77.1% of participants in the Wegovy® group achieved weight loss ≥5% from baseline at week 104 versus 34.4% with placebo (P<0.0001). Gastrointestinal disorders were more frequent in the Wegovy® group than in the placebo group (Wegovy® 82.2% vs. placebo 53.9%).

[Study abstract'] This pooled analysis of the SUSTAIN 1–5 trials included subjects with inadequately controlled type 2 diabetes from the Phase 3a, multinational, randomized, controlled studies. The SUSTAIN 1–5 trials compared semaglutide subcutaneous once weekly (0.5 mg and/or 1.0 mg) with placebo or active comparators. Across the SUSTAIN 1–5 trials, 2,465 participants were assigned to semaglutide 0.5 mg(n=1,031) or 1.0 mg(n=1,434). The primary endpoint was the change in HbA1c (%) from baseline to end of treatment, assessed across clinically relevant patient subgroups defined by baseline HbA1c . Week 30 was selected as the cut-off for these analyses. HbA1c reductions with semaglutide 0.5 mg were −0.9%, −1.2%, −1.5%, −1.7%, and −2.3% , and with semaglutide 1.0 mg were −1.1%, −1.4%, −1.9%, −2.1%, and −2.7% in patients with baseline HbA1c ≤7.5%, >7.5% to ≤8.0%, >8.0% to ≤8.5%, >8.5% to ≤9.0%, and >9.0%, respectively. HbA1c reductions differed significantly across baseline HbA1c subgroups for semaglutide 1.0 mg (p=0.045), but not for semaglutide 0.5 mg (p=0.247). The incidence of adverse events was similar across baseline HbA1c subgroups with Ozempic® 0.5 mg and 1.0 mg.

[Study abstract'] SEMALEAN was a prospective, longitudinal, real-world cohort study conducted at a university hospital under the temporary use authorization and early access program, evaluating the effects of semaglutide (Wegovy®) on lean mass, muscle function, and metabolic adaptation in adults with obesity. The study enrolled 106 adults with grade 3 obesity (BMI ≥40 kg/m²), at least one obesity-related comorbidity (obstructive sleep apnea syndrome, hypertension, dyslipidemia, or cardiovascular disease), and documented inadequate response to lifestyle interventions, including dietary modification and increased physical activity. Body weight, body composition by DXA, muscle function assessed by handgrip strength, and resting energy expenditure (REE) were evaluated at baseline (M0), month 7 (M7), and month 12 (M12). At month 12, mean changes from baseline in body weight, fat mass, visceral adipose tissue, and lean mass were −15.6 kg, −12.1 kg, −0.78 kg, and −3.27 kg, respectively (p<0.0001). Mean handgrip strength increased by 4.1 kg from baseline, and the proportion of patients with sarcopenic obesity decreased from 49% at month 7 to 33% at month 12.

[Study abstract'] SUSTAIN 6 was a randomised, double-blind, placebo-controlled, parallel-group, multinational phase 3b clinical trial was conducted on adult patients with type 2 diabetes with cardiovascular risk factor. Participants were assigned to receiving either Ozempic® 0.5 mg(n=826) or 1.0 mg(n=822) or placebo 0.5 mg(n=824) or 1.0 mg(n=825). The primary composite outcome was the first occurrence of cardiovascular death, nonfatal myocardial infarction, or nonfatal stroke. Median follow-up was 2.1 years. Ozempic® demonstrated a 26% RRR vs. placebo (HR, 0.74; 95% CI, 0.58 to 0.95; P<0.001 for noninferiority; P = 0.02 for superiority). Gastrointestinal disorders were more frequent in the Ozempic® group than in the placebo group(Ozempic® 0.5mg group 50.7%, Ozempic® 1.0mg group 52.3%, Placebo 0.5mg group 35.7%, Placebo 1.0mg group 35.2%)

[Study abstract'] FLOW was a randomised, double-blind, parallel-group, multinational phase 3b clinical trial was conducted on adult patients with CKD and type 2 diabetes. Participants were assigned to receiving either Ozempic® 1.0 mg or placebo. The primary outcome was major kidney disease events, a composite of the onset of kidney failure, a sustained reduction of at least 50% in eGFR from baseline, or death from kidney-related or cardiovascular causes. Median follow-up was 3.4 years. Ozempic® demonstrated a 24% RRR vs. placebo(HR, 0.76; 95% CI, 0.66 to 0.88; P = 0.0003). Serious adverse events were reported in fewer patients in the Ozempic® group than in the placebo group (49.6% vs. 53.8%).

[Study abstract'] SELECT trial was a multicenter, randomized, double-blind, placebo-controlled, event-driven superiority study conducted in adults aged 45 years or older with established cardiovascular disease and overweight or obesity (BMI≥27kg/m²), but without diabetes. A total of 17,604 participants were randomized 1:1 to receive once-weekly subcutaneous semaglutide 2.4 mg(Wegovy®) (n=8,803) or placebo (n=8,801). The primary cardiovascular endpoint, a composite of death from cardiovascular causes, nonfatal myocardial infarction, or nonfatal stroke, occurred in 569 patients (6.5%) in the Wegovy® group and 701 patients (8.0%) in the placebo group, representing a significant risk reduction with Wegovy® versus placebo (hazard ratio (HR), 0.80; 95% CI, 0.72 to 0.90; p<0.001 for superiority). The first confirmatory secondary endpoint, death from cardiovascular causes, occurred in 223 patients (2.5%) in the Wegovy® group and 262 patients (3.0%) in the placebo group; however, this difference did not reach statistical significance (HR, 0.85; 95% CI, 0.71 to 1.01; P=0.07; nominal significance level for superiority, 0.023). Adverse events leading to permanent discontinuation of the trial product were more frequent in the Wegovy® group than in the placebo group, occurring in 1,461 patients (16.6%) and 718 patients (8.2%), respectively (P<0.001).

† Trial product estimand

* p<0.0001 for odds ratio for the 5%, 10% and 15% thresholds; the 20% threshold was not part of the statistical testing hierarchy.

‡ 위고비®는 한국 식품의약품안전처로부터 성인 비만 환자 또는 체중 관련 동반질환이 있는 성인 과체중 환자와 12세 이상 비만 청소년(체중 60 kg 초과)의 체중 관리 보조요법, 그리고 확증된 심혈관계 질환이 있으면서 BMI가 27kg/m² 이상인 성인 환자에서 주요 심혈관계 사건 위험을 감소시키는 용도로 허가받았습니다.

‡ 오젬픽®은 한국 식품의약품안전처로부터 2형 당뇨병 성인 환자의 혈당 조절 및 동반된 심혈관계 질환 또는 만성 신장 질환 관련 위험을 감소시키는 용도로 허가받았습니다.

BMI, body mass index; **CI**, confidence interval; **CKD**, chronic kidney disease; **eGFR**, estimated glomerular filtration rate; **ETD**, estimated treatment difference; **GLP-1 RA**, glucagon-like peptide-1 receptor agonist; **HbA1c**, glycated hemoglobin; **HR**, hazard ratio; **RRR**, relative risk reduction; **T2D**, type 2 diabetes

References 1. 위고비®프리필드펜 0.25, 0.5, 1.0, 1.7, 2.4 mg (세마글루티드). 국내제품설명서. Available via: https://nedrug.mfds.go.kr/pbp/CCBBB01/getItemDetailCache?cacheSeq=202301384aupdateTs2026-06-13%2015:01:33.0b. Accessed on 23 Jul 2026. **2.** Garvey WT, Batterham RL, Bhatta M, et al. Two-year effects of semaglutide in adults with overweight or obesity: the STEP 5 trial. *Nat Med.* 2022;28(10):2083-91. **3.** 오젬픽®프리필드펜 (세마글루티드). 국내제품설명서. Available via: https://nedrug.mfds.go.kr/pbp/CCBBB01/getItemDetailCache?cacheSeq=202201737aupdateTs2025-12-12%2010:32:14.0b. Accessed on 23 Jul 2026. **4.** Aroda VR, Capehorn MS, Chaykin L, et al. Impact of baseline characteristics and beta-cell function on the efficacy and safety of subcutaneous once-weekly semaglutide: A patient-level, pooled analysis of the SUSTAIN 1-5 trials. *Diabetes Obes Metab.* 2020;22(3):303-14. **5.** Alissou M, Demangeat T, Folope V, et al. Impact of semaglutide on fat mass, lean mass and muscle function in patients with obesity: the SEMALEAN study. *Diabetes Obes Metab.* 2026;28(1):112-21. **6.** Marso SP, Bain SC, Consoli A, et al. Semaglutide and cardiovascular outcomes in patients with Type 2 Diabetes. *N Engl J Med.* 2016;375(19):1834-44. **7.** Perkovic V, Tuttle KR, Rossing P, et al. Effects of semaglutide on chronic kidney disease in patients with Type 2 Diabetes. *N Engl J Med.* 2024;391(2):109-21. **8.** Lincoff AM, Brown-Frandsen K, Colhoun HM, et al. Semaglutide and cardiovascular outcomes in obesity without diabetes. *N Engl J Med.* 2023;389(24):2221-32.

※처방하시기 전 QR코드 또는 식품의약품안전처 의약품정보시스템 (https://nedrug.mfds.go.kr)을 통해 상세정보를 참조하시기 바랍니다.

[수입의약품·전문의약품] 위고비®프리필드펜0.25(세마글루티드), 위고비®프리필드펜0.5(세마글루티드), 위고비®프리필드펜1.0(세마글루티드), 위고비®프리필드펜1.7(세마글루티드), 위고비®프리필드펜2.4(세마글루티드), 오젬픽®프리필드펜(세마글루티드)

본 자료는 의약학적 전문가에게 노보 노디스크의 위고비®프리필드펜(이하 "위고비®로도 약칭"), 오젬픽®프리필드펜(이하 "오젬픽®로도 약칭")의 제품 정보를 설명하기 위한 목적으로 제공되는 자료입니다. 노보 노디스크의 사전 동의 없이 본 자료를 무단 편집 또는 활용할 수 없으며, 본 자료를 제3자에게 배포, 제공하거나 일반 대중의 접근이 가능한 공개된 장소에 비치하여서는 안 됩니다.

Wegovy® 0.25 | Wegovy® 0.5 | Wegovy® 1.0 | Wegovy® 1.7 | Wegovy® 2.4 | Ozempic®

novo nordisk®
노보노디스크제약(주)
서울시 송파구 올림픽로35길 137. (신천동, 한국광고문화회관 16층) TEL: 02-2188-8900 FAX: 02-2188-8980 www.novonordisk.co.kr

All rights reserved by Novo Nordisk Pharma Korea, Ltd.

KR26SEMO00222, Aug 2026

BORYUNG

*Atorvastatin,Rosuvastatin 대비

NODM, New-Onset Diabetes Mellitus

References **1.** Choi JY et al. Am J Cardiol. 2018;122(6):922-928. **2.** 약학정보원 [ezetimibe 성분 상세정보]. 약학정보원. accessed on 2025.12.16

BR-1-LZET-26-002

에제페노® 정

(에제티미브 / 페노피브레이트)

EZEFEND®

(Ezetimibe / Fenofibrate)

New Combination
for Satisfying Unmet- Needs
in Dyslipidemia

We are always together
for your better life

[References] 1. DL Tribble et al. Metabolism. 2008 Jun;57(6):796-801. 2. M Farnier et al. Eur Heart J 2005 May;26(9):897-905. 3. SS Kumar et al. Lipids Health Dis. 2009 Dec 17;8:56. 4. Nagula Jayababu. JMSCR. 2019 Oct; 7(10):963-970. 5. A Shinnakasu et al. J Atheroscler Thromb. 2017 Jul 1;24(7):735-748. 6. M Farnier et al. Am Heart J. 2007 Feb;153(2):335.e1-8. 7. M Farnier et al. Diab Vasc Dis Res. 2012 Jul;9(3):205-15. 8. S Oikawa et al. J Atheroscler Thromb. 2017 Jan 1;24(1):77-94. 9. JM McKenney et al. J Am Coll Cardiol. 2006 Apr 18;47(8):1584-7.

[제품요약정보] **제품명** 에제페노 정 **주성분함량** 1정(515mg) 중 에제티미브 10mg 및 페노피브레이트 160mg **성상** 흰색의 원형 필름 코팅정 **효능효과** 혼합형 고지혈증 환자의 상승된 총콜레스테롤(total-C), 저밀도지단백 콜레스테롤(LDL-C), 아포지단백 B(Apo B) 및 비-고밀도지단백 콜레스테롤(non-HDL-C)을 감소시키기 위한 식이요법의 보조제 **용법용량** 성인 : 이 약은 1일 1회 1정을 식후 즉시 복용한다. 이 약은 반드시 식이요법과 병행하여 투여한다. 이 약 성분 중 페노피브레이트는 빈속에 흡수가 덜 될 수 있으므로 반드시 식후 즉시 투여한다. 간장애환자에는 투여하지 않는다. 신장애환자는 중등도~중증 신장애 환자(혈청 크레아티닌치 2.5 mg/dL 이상)의 경우 이 약을 투여하지 않는다. 고령자는 신기능이 감소되지 않은 경우 일반적으로 용량 감량이 필요하지 않다. 에제티미브와 페노피브레이트를 병용으로 복용하고 있는 환자의 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있다. **사용상의 주의사항(일부)** 1. 다음 환자에는 투여하지 말 것 1) 이 약 및 이 약의 구성성분에 과민증이 있는 환자 2) 활성 간질환 환자 혹은 설명되지 않는 혈청 아미노전이효소 수치 증가가 지속되는 환자에게는 이 약과 HMG-CoA 환원효소 억제제를 병용투여하지 않는다. 3) 임부 또는 임신하고 있을 가능성이 있는 여성 및 수유부 4) 간장애환자 5) 중등도~중증 신장애 환자(혈청 크레아티닌치 2.5 mg/dL 이상)(횡문근융해증이 나타날 수 있다.) 6) 담낭질환 환자(선재성 담낭 질환 환자 포함) 7) 피브레이트 또는 케토프로펜으로 치료하는 동안 광알레르기 또는 광독성을 경험한 환자 8) 소아 9) 담관간경화증 환자 10) 췌장염 환자(중증 고중성지질형증으로 인한 급성 췌장염 제외) 11) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), lapp(유당 분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galatose-malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안된다. 2. 다음 환자에게는 신중히 투여할 것 1) 경증 신장애 또는 그 병력이 있는 환자(혈청 크레아티닌치 1.5mg/dL 이상 2.5mg/dL 미만)(횡문근융해증이 나타날 수 있으므로 투여량을 감량 또는 투여간격을 연장하여 사용한다.) 2) 간기능조사에 이상이 있는 환자 또는 그 병력이 있는 환자(간기능 검사값의 이상변동이 나타날 수 있다.) 3) 저알부민혈증(신증후군) 환자 4) 담석의 병력이 있는 환자(담석형성이 보고되었다.) 5) 혈액응고저지제를 투여중인 환자 6) HMG-CoA 환원효소 저해제(예, 프라바스타틴, 심바스타틴 등)를 투여중인 환자 7) 고령자 3. 이상반응: 이 약은 각 단일제인 에제티미브 및 페노피브레이트의 이상반응을 포함한다. **포장단위** 30정/PTP (10정/PTP X3) **저장방법** 기밀용기, 실온(1~30℃) 보관 제품에 대한 자세한 사항은 사용설명서를 참고하시기 바랍니다. 가장 최근 개정된 제품설명서의 내용은 현대약품 홈페이지를 통해 확인하실 수 있습니다. 설명서 작성년월: 2021년 10월

현대약품(주) 서울특별시 강남구 봉은사로 135 현대약품빌딩 | 대표전화(고객센터) : 1666-9979 | www.hyundaipharm.co.kr

안국약품

New Standard
Beyond Statin Monotherapy[1]

Pevarozet® 1/10 mg

Pitavastatin/Ezetimibe

- ✓ Approximately 48% reduction in LDL-C[2]
- ✓ No significant changes in HbA1c and FPG[2]
- ✓ Compact 4.9x9.9 mm tablet size designed for patient compliance[3,4]

LDL-C, low density lipoprotein cholestero; HbA1c, hemoglobin A1c; FPG, fasting plasma glucose.

References 1. Jun JE, et al. Combination of low - or moderate-intensity statin and ezetimibe vs. high-intensity statin monotherapy on primary prevention of cardiovascular disease and all-cause death: a propensity matched nationwide cohort study. *Eur J Prev Cardiol*. 2024;31:1205-13. 2. Data on file. 임상시험 결과보고서. 원발성 고콜레스테롤혈증 환자를 대상으로 AG2304T와 AG2304R의 유효성과 안전성을 비교평가하기 위한 다기관, 무작위배정, 이중 눈가림, 활성 대조, 제3상 임상시험. 안국약품. 2025. 3. 의약품안전나라. 의약품 등 제품정보 검색(제품명: 페바로젯정). Available at: https://nedrug.mfds.go.kr/searchDrug. Accessed on 16 Jul, 2026. 4. Kabeya K, et al. Threshold size of medical tablets and capsules: Based on information collected by Japanese medical wholesaler. *Patient Prefer Adherence*. 2020;14:1251-8.

[제품명] 페바로젯정1/10 mg **[성분·함량]** 1정 중 피타바스타틴칼슘(별규) 1 mg, 에제티미브(별규) 10 mg **[성상]** 흰색 또는 거의 흰색의 타원형 필름코팅정 **[효능·효과]** 원발성 고콜레스테롤혈증: 원발성 고콜레스테롤혈증(이형접합 가족형 및 비가족형) 또는 혼합형 이상지질혈증 환자의 상승된 총 콜레스테롤(total-C), LDL-콜레스테롤(LDL-C), 아포 B 단백(Apo B), 트리글리세라이드(TG) 및 non-HDL-콜레스테롤을 감소시키고, HDL-콜레스테롤(HDL-C)을 증가시키기 위한 식이요법의 보조제로서 이 약을 투여한다. **[용법·용량]** 성인: 이 약은 식사와 관계없이 1일 1회 투여한다. 이 약을 투여하기 전 또는 투여 중인 환자는 반드시 표준 콜레스테롤 저하식을 지속적으로 해야 한다. 이 약의 투여량은 환자의 LDL-콜레스테롤의 기저치, 권장되는 치료목표치 및 환자의 반응에 따라 조절되어야 한다. 이 약은 초회용량으로 1일 1회 1/10 mg-2/10 mg이 권장된다. LDL-콜레스테롤치의 저하효과가 충분하지 않은 경우 1일 최대 4/10 mg까지 증량할 수 있다. LDL-콜레스테롤치, 치료 목표 및 환자의 반응에 따라 4주 또는 그 이상의 간격을 두고 용량을 적절히 조절한다. 피타바스타틴칼슘과 에제티미브를 병용으로 복용하고 있는 환자인 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있다. 간장애 환자의 경우, 피타바스타틴칼슘의 초회용량으로 1일 1 mg이 권장되므로, 이 약(피타바스타틴칼슘/에제티미브 복합제) 초회용량으로 1/10mg 이 권장된다. 최대 투여량은 1 일 2/10mg 으로 한다. 중등증 간장애 환자의 경우 이 약(피타바스타틴칼슘/에제티미브 복합제) 투여가 권장되지 않는다. 중증 간장애 환자의 경우 이 약(피타바스타틴칼슘/에제티미브 복합제)을 투여하지 않는다. **[금기]** 1) 이 약의 구성성분에 과민증이 있거나, 그 병력이 있는 환자 2) 활동성 간질환 환자 또는 원인이 밝혀지지 않은 아미노전이효소수치의 지속적 상승이 있는 환자 3) 중증의 간장애 또는 담도폐쇄가 있는 환자 및 담즙울체 환자 4) 사이클로스포린을 투여중인 환자 5) 근육병증 환자 6) 임부 또는 임신의 가능성이 있는 부인 및 수유부 7) 소아(사용경험이 없다.) 8) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당분해 효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안된다. **[포장단위]** 30정/병 **[저장방법]** 차광기밀용기, 실온(1~30℃)보관 **[사용기한]** 제조일로부터 12개월 **[제조자/판매자]** 안국약품(주)/안국약품(주)

※제품 정보는 축약된 내용이므로, 자세한 정보는 최신의 제품설명서를 참고하시기 바랍니다.
※ QR코드를 스캔하시면 해당 제품 정보를 확인하실 수 있습니다.

안국약품 13840 경기도 과천시 과천대로7길 12 안국약품빌딩
T. 02-3289-4200 W. www.ahn-gook.com

PVZ-C029-A20260720

페바로젯정 1/10 mg

YOOYOUNG PHARMACEUTICAL
유영제약

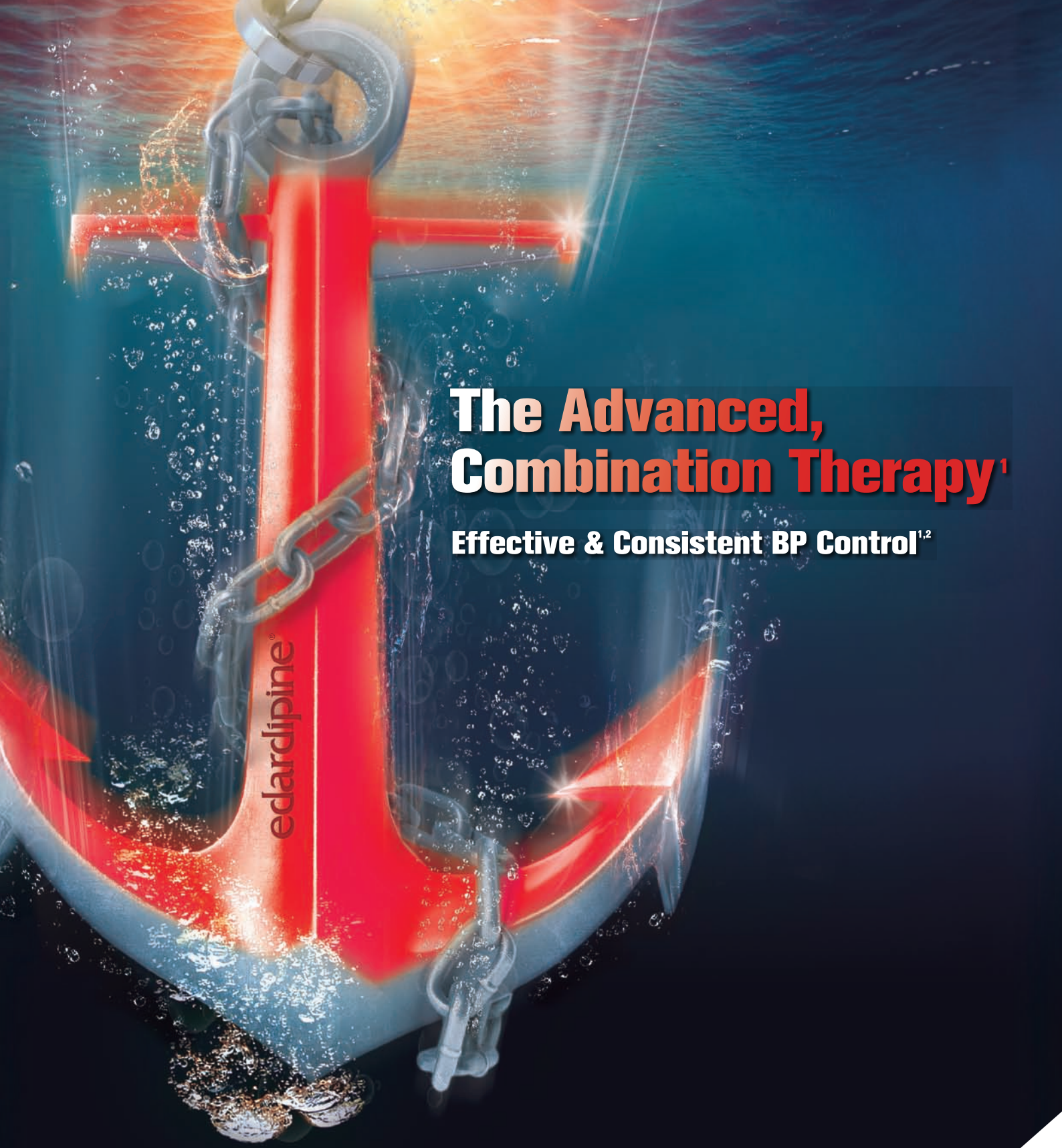
프라바페닉스
(Pravastatin 40mg+Fenofibrate 160mg)

프라바페닉스

The 1st Choice of Mixed Dyslipidmia

당뇨병발생률부터
근육관련 부작용과
약물상호작용의 위험까지 적은

프라바페닉스는
당근약입니다.



The Advanced, Combination Therapy[1]

Effective & Consistent BP Control[1,2]

References 1. Kim DH, *et al.*, A Phase III Randomized Controlled Trial Evaluating the Efficacy and Safety of Azilsartan Medoxomil and Amlodipine Combination Therapy in Patients With Mild-to-Moderate Essential Hypertension Inadequately Controlled on Monotherapy, *Clin Ther.* 2025;12:S0149-2918(25)00285-1. 2. Weber MA, *et al.*, Effects of combining azilsartan medoxomil with amlodipine in patients with stage 2 hypertension, *Blood Press Monit.* 2014 ;19(2):90-7.

Prescribing Information [제품명] 이달디핀정(아질사르탄메독소밀칼륨/암로디핀베실산염) 40/5, 40/10, 80/5, 80/10 mg **[효능·효과]** 아질사르탄메독소밀 또는 암로디핀 단독요법으로 혈압이 적절히 조절되지 않는 본태성 고혈압 **[용법·용량] 1. 성인:** 이 약은 1일 1회 1정을 식사와 관계없이 물과 함께 복용한다. 가능하면 매일 같은 시간(예: 아침)에 복용하는 것이 권장된다. 1) 40/5 mg: 아질사르탄메독소밀 40 mg 또는 암로디핀 5 mg 단독요법으로 혈압이 적절하게 조절되지 않는 환자에게 투여한다. 2) 40/10 mg: 아질사르탄메독소밀 40 mg 또는 암로디핀 10 mg 단독요법 또는 이 약 40/5 mg으로 혈압이 적절하게 조절되지 않는 환자에게 투여한다. 3) 80/5 mg: 아질사르탄메독소밀 80 mg 단독요법으로 혈압이 적절하게 조절되지 않는 환자에게 투여한다. 4) 80/10 mg: 아질사르탄메독소밀 80 mg 또는 이 약 80/5 mg 로 혈압이 적절하게 조절되지 않는 환자에게 투여한다. 5) 각 단일제를 병용하고 있는 환자의 경우, 복용의 편리함을 위해 이 약(개개의 주성분 함량이 동일한 복합제)로 전환할 수 있다. **2. 고령자:** 일반적으로 투여량 조절이 필요하지 않으나 75세 이상 고령자의 경우 초회량으로서 아질사르탄메독소밀 20 mg를 고려하므로, 이 약으로 투여를 시작하는 것은 권장되지 않는다. **3. 신장애 환자:** 경증 및 중등증 신장애 환자에서 투여량 조절이 필요하지 않다. **4. 간장애 환자:** 경증-중등증 간장애 환자의 경우 초회량으로서 아질사르탄메독소밀 20 mg을 고려하므로, 이 약으로 투여를 시작하는 것은 권장되지 않는다. **5. 혈관내 유효혈액량감소 환자:** 염이 감소되거나 유효혈액량감소 가능성이 있는 환자(예: 구토, 설사 증상이 있거나 고용량의 이뇨제투여 환자)의 경우 초회량으로서 아질사르탄메독소밀 20 mg를 고려하므로, 이 약으로 투여를 시작하는 것은 권장되지 않는다. **6. 소아:** 만 18세 미만의 소아 및 청소년에 대한 이 약의 안전성 및 유효성은 확립되지 않았다. **[사용상의 주의사항] 1. 경고:** 임신 2, 3기인 임부에 레닌-안지오텐신계(Renin-Angiotensin System, RAS)에 직접적으로 작용하는 약물 투여 시, 태아 및 신생아에게 손상 및 사망까지도 일어날 수 있다. 따라서 만일 임신으로 확인될 경우 즉시 이 약의 투여를 중단해야 한다(7. 임부 및 수유부에 대한 투여 항 참조). **2. 다음 환자에는 투여하지 말 것** 1) 이 약 또는 이 약의 구성성분 또는 다른 디히드로피리딘계 유도체에 과민증이 있는 환자. 2) 임부 또는 임신하고 있을 가능성이 있는 여성 및 수유부(7. 임부 및 수유부에 대한 투여 항 참조). 3) 다음의 환자에게 이 약과 알리스키렌 제제의 병용투여: 당뇨병 환자 또는 중등증 – 중증의 신장애(사구체여과율 < 60 mL/min/1.73 m²) 환자 (5. 상호작용 항 참조). 4) 중증의 간기능장애 환자. 5) 중증의 대동맥판협착증 환자. 6) 쇼크 환자(심장성 쇼크 포함). **[저장방법]** 기밀용기, 실온(1~30℃)보관

※ 이달디핀정 국내허가 사항 전문은 식약처 의약품통합정보시스템(http://nedrug.mfds.go.kr)을 참고하시기 바랍니다.

셀트리온제약 CELLTRION

Daewon 대원제약

edardipine®

Azilsartan Medoxomil Potassium/Amlodipine Besylate

40/5 mg • 40/10 mg • 80/5 mg • 80/10 mg tablets

P_AZL_LIF_2511005

네시나®
(alogliptin) tablets

하루한 알로 (alogliptin)
당뇨병* 치료의
검증된 승리[1]

* 2형당뇨병

Ref 1. Del Prato S, et al. *Diabetes Obes Metab.* 2014;16(12):1239–46

셀트리온제약
CELLTRION

셀트리온제약 [케미컬국내사업부문] 경기도 과천시 과천대로7길 20 (스마트K) A타워 12층
[소비자상담실] 080-850-3611~2 / www.celltrionph.com

QR코드 또는 식품의약품안전처 의약품통합정보시스템 (https://nedrug.mfds.go.kr)을 통해 상세제품 정보를 참조하시기 바랍니다.

네시나® 정 12.5mg 네시나® 정 25mg

OT2025-125

THEY SURVIVED A CV EVENT.*
NOW PROTECT THEM FURTHER WITH PRALUENT®[1-4]

Redefining expectations for those at risk of cardiovascular events

Treatment options for high CV risk patients with ASCVD[4] – Praluent®

PRALUENT® demonstrated a significant reduction in risk of MACE and a favourable safety profile in CV high-risk patients.[1-4]

STUDY DESIGN ODYSSEY OUTCOMES was a randomised, double-blind, placebo-controlled phase 3 study. Patients with a recent MI or unstable angina, and on high-intensity statin (40 mg or 80 mg atorvastatin or 20 mg or 40 mg rosuvastatin, or maximally tolerated dose of one of these agents) +/- other lipid-lowering therapy but not at predefined target LDL-C were enrolled.

*Defined as hospitalised with an acute coronary syndrome (myocardial infarction or unstable angina).
ASCVD, atherosclerotic cardiovascular disease; **CV,** cardiovascular; **LDL-C,** low-density lipoprotein cholesterol; **MACE,** major adverse cardiovascular event; **MI,** myocardiac infarction.

References 1. PRALUENT® FDA label, 2021. **2.** Schwartz GG, et al. N Engl J Med. 2018;379(22):2097-2107. **3.** Steg GP, et al. J. Am. Heart Assoc. 2019;140:103-112. doi:httpp://10.1161/CIRCILATIONAHA.118.038840. **4.** 프랄런트®펜주 식약처 허가사항(75 mg 개정년월일: 2024.09.30/150 mg 개정년월일: 2022.12.20/300 mg 개정년월일: 2023.02.13)

Praluent®
alirocumab

sanofi

(주)사노피-아벤티스 코리아 서울시 서초구 반포대로 235
대표 전화 : 02-2136-9000, 팩스 : 02-2136-9588

프랄런트®펜주 75 mg/150 mg/300 mg (알리로쿠맙) | 피하주사용 | 전문의약품
※프랄런트®의 제품정보는 하단 QR코드를 통해 확인하시기 바랍니다.
프랄런트®펜주 75 mg(문안개정년월일: 2024.09.30)
프랄런트®펜주 150 mg(문안개정년월일: 2022.12.20)
프랄런트®펜주 300 mg(문안개정년월일: 2023.02.13)

75 mg 제품설명서 바로보기 | 150 mg 제품설명서 바로보기 | 300 mg 제품설명서 바로보기

MAT-KR-2500653-1.0-03/2025

For Healthcare Professionals Only

LG Chem

Zemidapa®
(Gemigliptin/Dapagliflozin)

One & Only†

Zemidapa® Tab.

The only available† FDC of Gemigliptin + Dapagliflozin,
New treatment option for Effective glycemic control.[1-3]

[PIVOTAL STUDY]

Solution II Study
HbA1c reduction of 1.34%†† with
Gemi/Dapa dual add-on to Met[3]

Solution I Study
HbA1c reduction of 0.86%†† with
Zemiglo® add-on to Met/Dapa[2]

†† Change in HbA1c from baseline at week 24

Met, Metformin; **Gemi,** Gemigliptin; **Dapa,** Dapagliflozin; **HbA1c,** Glycated hemoglobin.

[References] 1. MFDS, Pharmaceutical Integrated intelligence system (https://nedrug.mfds.go.kr). 2. Lee BW et al., *Endocrinol Metab (Seoul).* 2023;38(3):328-337. 3. Data on file, Clinical Phase III trial in Korea (Pivotal Study), LG-GLCL001(2023), LG Chem.

Zemidapa® Tab. (Gemigliptin/Dapagliflozin) 50/10 mg (Product Launch Date: 2023. Apr. 08/ Full product information can be confirmed in the QR code to the right position)

■ **Therapeutic Indication** Zemidapa® is indicated as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus. ■ **Dosage and Administration** The recommended dose is one tablet once daily in patients who need 50 mg of gemigliptin and 10 mg of dapagliflozin. [Special population] 1) Renal impairment: The efficacy and safety of Zemidapa® is dependent on renal function, and renal function should be evaluated prior to initiation of Zemidapa® and periodically thereafter. For patients with eGFR≥45 mL/min/1.73m2, no dose adjustment is required. For patients with eGFR < 45 mL/min/1.73m2, Zemidapa® is not recommended. 2) Hepatic impairment: No dose adjustment is required for patient with mild or moderate hepatic impairment. The safety and efficacy of Zemidapa® in patients with severe hepatic impairment have not yet been established. ■ **Contraindication** Zemidapa® is contraindicated in patients with/on: 1) hypersensitivity to the active substances or to any of the excipients or a history of serious hypersensitivity reactions, i.e., angioedema or anaphylaxis, to another dipeptidyl peptidase-4 (DPP-4) inhibitor or sodium glucose cotransporter (SGLT) 2 inhibitor, 2) type 1 diabetes or diabetic ketoacidosis, 3) rare hereditary problems of galactose intolerance, total lactase deficiency or glucose-galactose malabsorption, and 4) hemodialysis. ■ **Precaution** 1) Gemigliptin: patient taking with medicinal products known to cause hypoglycemia, patient with cardiac impairment, patient with severe hepatic impairment, patient with acute pancreatitis. 2) Dapagliflozin: patient with volume depletion and impaired renal function. ■ **Safety Profile** The most common adverse events reported in ≥ 1.0% in patients treated with Gemigliptin once daily in add-on combination of Metformin and Dapagliflozin in a 24 weeks study (Regardless of Investigator Assessment of Causality): Lipase increased, Chronic gastritis, Dizziness, Urinary tract infection, Cough, Diabetic nephropathy, Gastritis, Gingivitis, Large intestine polyp, Urticaria, Dyspepsia. ■ **Manufacturer** LG Chem.

Zemiglo® Tab. (Gemigliptin) 50 mg (Full product information can be confirmed in the QR code to the right position)

■ **Indication and Usage** Zemiglo® 50 mg is a dipeptidyl peptidase-4 (DPP-4) inhibitor indicated as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus. Zemiglo® can be administered 1) Monotherapy, 2) Combination therapy. ■ **Dosage and Administration** The maximum daily recommended dose of Zemiglo® is 50 mg once daily. Zemiglo® can be taken without regard to food. When used in combination with a sulfonylurea or insulin, a lower dose of the sulfonylurea or insulin may be required to reduce the risk of hypoglycemia. No dosage adjustment is required for patients with impaired renal function. No dosage adjustment is required for patients with mild to severe hepatic impairment function. ■ **Contraindication** Zemiglo® is contraindicated in patients with 1) a history of serious hypersensitivity reactions, i.e., angioedema or anaphylaxis, to another dipeptidyl peptidase-4 (DPP-4) inhibitor or 2) type 1 diabetes or diabetic ketoacidosis. ■ **Precaution** Zemiglo® is used carefully in patients with 1) taking sulfonylurea, 2) cardiac impairment, 3) hepatic impairment, 4) acute pancreatitis, 5) hypersensitive reactions. ■ **Adverse Reactions** 1) Most common adverse reactions reported ≥ 3% of patients treated with Zemiglo® once daily in monotherapy: arthralgia, nasopharyngitis, and bacteriuria, 2) Most common adverse reactions reported in ≥ 3% of patients treated with Zemiglo® once daily in add-on combination therapy: upper respiratory tract infection, nasopharyngitis, blood amylase increased, lipase increased, and pyrexia. ■ **Manufacturer** LG Chem.

※ For more information, please refer to recently updated full prescribing information, including WARNINGS and MEDICATION GUIDE. As approved indications differ by country, consult the local prescribing information available with the manufacture before prescribing this medication.

CDP-PRI-001-01-1-KR-EN

Aligning two strengths toward effective LDL-C management.

Dual Power
One Direction

DUONON® Tab. 10/5mg 10/10mg 10/20mg

(ezetimibe / rosuvastatin calcium) tablets

한림제약

EXPANDING AND SHINING ACROSS THE CV ZONE
WITH HANLIM'S STAFEN CONSTELLATION

The Original STAFEN

Shining from the Origin

pitava**STA**tin+**FEN**ofibrate

성분/함량	피타바스타틴칼슘 2mg + 페노피브레이트160mg	용법용량	1일 1회 1캡슐 식사 직후 복용
효능효과	관상동맥심질환(CHD) 고위험이 있는 성인환자에서 피타바스타틴 2mg 단일치료요법시 LDL-콜레스테롤 수치는 적절히 조절되지만 트리글리세라이드 수치는 높고 HDL-콜레스테롤 수치는 낮은 복합형이상지질혈증의 치료	성상	흰색의 원형 필름코팅정과 흰색의 구형 미세과립을 함유 하고 있는 상부 담녹색, 하부 흰색의 경질캡슐
		저장방법	기밀용기, 실온(1~30℃)보관
약가	872원 / 보험코드 645306120	포장단위	30캡슐/상자(10캡슐/PTPX3), 300캡슐/병 / 제조/판매원 한림제약

* Original STAFEN
STAFEN은 Pitavastatin과 Fenofibrate의 복합제로서 최초로 승인된 한림제약의 개량신약입니다.

BEYOND LDL-C lowering STAtin+FENofibrate
스타펜®
Pitavastatin/Fenofibrate 2/160mg

Rovazet® Tab.
Ezetimibe+Rosuvastatin

전문의약품

The Lower, The Better

THE SMALLER, THE BETTER.

로바젯®

[전문의약품] [주성분 함량] 로바젯®정10/5밀리그램 1정(139mg) 중 로수바스타틴칼슘 5.2mg (로수바스타틴으로서 5.0mg), 에제티미브 10.0mg, 로바젯®정10/10밀리그램 1정(160mg) 중 로수바스타틴칼슘 10.4mg (로수바스타틴으로서 10.0mg), 에제티미브 10.0mg, 로바젯®정10/20밀리그램 1정(268mg) 중 로수바스타틴칼슘 20.8mg (로수바스타틴으로서 20.0mg), 에제티미브 10.0mg **[효능 · 효과] 원발성 고콜레스테롤혈증** 원발성 고콜레스테롤혈증(이형접합 가족형 및 비가족형) 또는 혼합형 이상지질혈증 환자의 상승된 총 콜레스테롤 (total-C), LDL-콜레스테롤(LDL-C), 아포 B 단백(Apo B), 트리글리세라이드(TG) 및 non-HDL-콜레스테롤을 감소시키고, HDL-콜레스테롤(HDL-C)을 증가시키기 위한 식이요법의 보조제로서 이 약을 투여한다. 고콜레스테롤혈증에 기인한 동맥경화성 혈관 질환의 위험성이 증가한 환자에게 지질조절약물을 투여할 때에는 많은 위험 인자를 고려해야 한다. 지질조절약물은 적절한 식이요법(포화지방 및 콜레스테롤 제한을 포함)과 함께 사용하고, 식이요법 및 다른 비약물학적 조치에 대한 반응이 불충분한 경우에 사용해야 한다. 이 약 투여에 앞서 이상지질혈증의 다른 이차적 원인(예를 들면, 당뇨, 갑상선기능저하증, 폐쇄성 간질환, 만성신부전, LDL-콜레스테롤을 증가시키는 약물 및 HDL-콜레스테롤을 감소시키는 약물[progestin, anabolic steroid, 및 corticosteroid])을 확인하여야 하며, 필요한 경우 이차적 원인을 치료해야 한다. 지질 검사시에는 총콜레스테롤, LDL-콜레스테롤, HDL-콜레스테롤 및 트리글리세라이드를 포함해야 한다. 트리글리세라이드 수치가 400mg/dL이상(4.5mmol/L 이상)인 경우에는 초원심분리로 LDL-콜레스테롤 농도를 측정해야 한다. 급성관상동맥 사고로 입원할 경우에는 입원 시 혹은 입원 후 24시간 이내에 지질을 측정해야 한다. 환자의 퇴원 전 혹은 퇴원 시에 LDL 저하치료를 시작하는데 있어 이 측정치가 참고가 될 수 있다. **[용법 · 용량]** 이 약은 식사와 관계없이 1일 1회 투여한다. 이 약을 투여하기전 또는 투여 중인 환자는 반드시 표준 콜레스테롤 저하식을 지속적으로 해야 한다. 이 약의 투여량은 환자의 LDL-콜레스테롤의 기저치, 권장되는 치료목표치 및 환자의 반응에 따라 조절되어야 한다. **원발성 고콜레스테롤혈증** 이 약의 용량범위는 1일 10/5mg~10/20mg이다. 초회용량으로 1일 10/5mg이 권장된다. LDL-콜레스테롤 감소가 더 많이 요구되는 환자의 경우 용량을 조절하여 투여할 수 있다. 이 약의 투여를 시작한 후 또는 용량을 적정한 후에는 4주 이상의 간격을 두고 혈중 지질 수치를 확인한 후 그에 따라 용량을 조절하며, 1일 최대 10/20mg까지 증량할 수 있다. 에제티미브와 로수바스타틴을 병용으로 복용하고 있는 환자인 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있다. **[사용상의 주의사항]** 1. 경고 로수바스타틴 및 동일 계열의 다른 약물에서 미오글로빈뇨에 의한 이차적인 급성 신부전을 동반하는 횡문근융해가 드물게 보고되었다. 따라서 급성의 심각한 근육병증을 나타내는 환자 또는 횡문근융해에 의해 이차적으로 신부전이 발생될 수 있는 위험요소(예, 중증 급성 감염, 저혈압, 주요 외과수술, 외상, 중증의 대사, 내분비 및 전해질 장애, 조절되지 않는 발작)를 가진 환자는 이 약의 치료를 일시적으로 보류하거나 또는 중단해야 한다. ('5. 일반적 주의'참조) *기타 자세한 사항은 제품설명서를 참조하십시오.

inno.N
에이치케이이노엔(주)
본사 : 충청북도 청주시 흥덕구 오송읍 오송생명2로 239
판교 스퀘어 : 경기도 성남시 수정구 금토로 79번길 28
http://www.inno-n.com | Tel. 080-700-8802

GC녹십자

고중성지방혈증 치료의

NEW KEY

국내 최초 저용량 Fenofibrate tablet (48mg)

[Fenofibrate 48mg, 145mg의 두 가지 제형으로 환자의 신기능에 따라 선택적 처방 가능]

경증~중등도 신장애 동반 고중성지방혈증 환자에 처방 가능한
국내 유일 저용량 페노피브레이트 제제, GC녹십자 네오페노정 48mg

네오페노정
Fenofibrate

S/N: GCBP-NEOF-KR00241111-BIL (exp. 20261111)

의약전문가용

First Choice
리피로우® 정
아토르바스타틴칼슘삼수화물

TO BE CKD STATIN, 리피로우

Keep More Evidence for Korean

CKD, Chong Kun Dang

리피로우정 (아토르바스타틴칼슘삼수화물)

전문의약품

[조성·성상] 1. 원료약품의 분량 1정 중 유효성분: 리피로우정 5 mg - 아토르바스타틴칼슘삼수화물(별규) 5.425 mg (아토르바스타틴으로서 5mg), 리피로우정 10 mg - 아토르바스타틴칼슘삼수화물(별규) 10.85 mg (아토르바스타틴으로서 10 mg), 리피로우정 20 mg - 아토르바스타틴칼슘삼수화물(별규) 21.70 mg (아토르바스타틴으로서 20 mg), 리피로우정 40 mg - 아토르바스타틴칼슘삼수화물(별규) 43.40 mg(아토르바스타틴으로서 40 mg), 리피로우정 80 mg - 아토르바스타틴칼슘삼수화물(별규) 86.80 mg(아토르바스타틴으로서 80 mg) **2.** 성상: 흰색의 담갈형 필름코팅정제 **[효능·효과] 1.** 다음의 심장혈관 질환에 대한 위험성 감소(10, 20, 40, 80mg에 한함) 1) 관상동맥심장 질환에 대한 임상적 증거는 없으나, 관상동맥 심장 질환의 다중위험요소(55세 이상, 흡연, 고혈압, 낮은 HDL-콜레스테롤치 또는 조기 관상동맥 심장 질환의 가족력 등)가 있는 성인 환자의 (1) 심근경색증에 대한 위험성 감소 (2) 뇌졸중에 대한 위험성 감소 (3) 혈관재생술 및 만성 안정형 협심증에 대한 위험성 감소 2) 관상동맥 심장 질환에 대한 임상적 증거는 없으나, 관상동맥 심질환의 다중위험요소(망막병증, 알부민뇨, 흡연, 또는 고혈압 등)가 있는 제2형 당뇨병 환자의 (1) 심근경색증에 대한 위험성 감소 (2) 뇌졸중에 대한 위험성 감소 3) 관상동맥 심장 질환에 대한 임상적 증거가 있는 성인 환자의 (1) 비치명적 심근경색증에 대한 위험성 감소 (2) 치명적 및 비치명적 뇌졸중에 대한 위험성 감소 (3) 혈관재생술에 대한 위험성 감소 (4) 울혈심부전으로 인한 입원에 대한 위험성 감소 (5) 협심증에 대한 위험성 감소 **2.** 고지혈증 **3.** 식이요법에도 불구하고 여전히 아래의 기준에 해당되는 이형접합 가족형 고콜레스테롤혈증을 가진 10~17세의 소아환자(여성의 경우 초경 이후의 환자)의 총콜레스테롤, LDL-콜레스테롤, 아포-B 단백수치를 감소시키는 식이요법의 보조제(10, 20 mg에 한함): 가. LDL-콜레스테롤이 여전히 190 mg/dL이상 (≥190 mg/dL)이거나 나. LDL-콜레스테롤이 여전히 160 mg/dL이상 (≥160 mg/dL)이고, 조기 심장혈관 질환의 가족력이 있는 경우 또는 해당 소아환자에서 두 가지 이상의 다른 심장혈관 질환의 위험인자가 있는 경우

※ 자세한 내용은 제품설명서를 참고하시기 바랍니다.

종근당

NA3_202601_232

AMGEN

Repatha® (evolocumab)

listen to their heart

ACT NOW ADD Repatha®* to Reduce CV Risk in ASCVD Patients

*Combination therapy with maximally tolerated statins or with statins and other lipid-lowering treatments

ASCVD, atherosclerotic cardiovascular disease; **CV,** cardiovascular.

Reference. Product information_Repath® Inj.Pre-filled pen.

AMGEN

20th Floor, Ferrum Tower, 19 Eulji-ro 5-gil, Jung-gu, Seoul, Republic of Korea (04539).
For inquiries related to pharmaceutical products, please contact the Amgen Medical Information Team.
Phone: 00798-611-3554 (Toll-Free), +82-2-3434-4899 Email: medinfo.JAPAC@amgen.com

Product information
Repath® Inj.Pre-filled pen 140 mg

Before prescribing, please refer to the detailed product information via the QR code or the MFDS Drug Information (https://nedrug.mfds.go.kr/searchDrug).

KOR-145-0225-80023

The First

세계 최초의 Statin, Compactin[1] 으로부터 시작된 메바로친®

Compactin은
Sankyo Co., Ltd. (現, Daiichi Sankyo Co., LTD)에서
1976년 Akira Endo박사에 의해 발견된
전 세계 최초의 Statin이며,
메바로친® [프라바스타틴나트륨]의
모체가 되었습니다[2].
약 50년의 역사를 지닌 메바로친®

이상지질혈증 치료의 첫 번째 선택으로
다시 주목해야 할 분명한 이유입니다.

Reappraise, **메바로친®**

[References] 1. Endo, Akira. "A historical perspective on the discovery of statins." *Proceedings of the Japan Academy. Series B, Physical and biological sciences* vol. 86,5 (2010): 484-93. doi:10.2183/pjab.86.484
2. Kishida, Y et al. *Yakugaku zasshi : Journal of the Pharmaceutical Society of Japan* vol. 111,9 (1991): 469-87. doi:10.1248/yakushi1947.111.9_469

메바로친정 5, 10, 20, 40 mg

[주성분] 프라바스타틴 나트륨 **[효능·효과]** 1. 원발성고지혈증: 고콜레스테롤혈증(IIa형), 고콜레스테롤혈증과 고트리글리세라이드혈증의 복합형(IIb형) 2. 고콜레스테롤혈증 또는 복합성고콜레스테롤혈증을 갖고 있는 환자 중 다음의 고위험군 환자에서 심근경색의 초발, 관상동맥심질환성 사망의 위험성 감소 3. 심근경색 또는 불안정성 협심증의 병력이 있는 환자에서 심근경색, 심혈관재관류술의 필요성, 허혈성 뇌졸중, 일과성 허혈발작 질환의 위험성 감소 **[용법·용량]** 치료를 시작하기 전에, 환자는 저콜레스테롤 식이를 시작해야 하고, 치료 중에도 이를 지속하여야 한다. 통상의 개시용량은 10 mg, 20 mg 혹은 40 mg 단일 용량으로 1일 1회이다. 환자의 반응에 따라 최대 40 mg까지 증량할 수 있다. **[사용상의 주의사항]** 1. 다음 환자에는 투여하지 않는다. 1) 이 약에 과민증 또는 그 병력이 있는 환자 2) 활성 간질환 또는 원인이 밝혀지지 않는 트랜스아미나제의 지속적 상승이 있는 환자 3) 임부 또는 임신하고 있을 가능성이 있는 부인, 수유부 4) 소아 5) 중증의 간·신부전 환자 6) 근병증 환자 7) 담즙울체 환자 8) HDL 콜레스테롤 상승이 동반된 hyperalphalipoproteinaemia에 의한 고콜레스테롤혈증 환자 9) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당분해효소결핍증(Lapplactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption)등의 유전적인 문제가 있는 환자 2. 이상반응 1) 과민증: 발진, 아나필락시스, 혈소판감소, 백혈구 감소, 용혈성 빈혈, 항핵항체(ANA) 양성, 혈액침강속도 증가, 혈관염, 루푸스양증후군, 광과민증, 혈압강하, 혈관부종, 피부근염, 소양증. 2) 소화기계: 설사, 구역, 구토, 변비, 복통, 위부불쾌감, 구내염, 가슴쓰림, 복부팽만감, 식욕부진. 3) 간장: 간기능 이상. 4) 신장: BUN, 혈청 크레아티닌치의 상승. 5) 골격근: 횡문근 융해증, 관절염, 관절통, 근육병변. 6) 정신신경계: 두통, 어지러움, 불면, 말초신경병증, 우울증, 권태감, 피로, 수면장애, 인지장애. 7) 기타: 요산상승, 혈뇨, 부종, 탈모, 발기부전 **[제조원]** HK inno.N **[개정년월일]** 2024년 12월 5일 ※ 본 정보는 요약된 일부의 정보입니다. 따라서 최신 변경된 허가사항이나, 자세한 사항은 당사 홈페이지(www.daiichisankyo.co.kr)나 식품의약품안전처 의약품안전나라(nedrug.mfds.go.kr)를 통해 확인하여 주시기 바랍니다. ※보다 자세한 제품 정보는 QR 코드 또는 식품의약품안전처 의약품안전나라(https://nedrug.mfds.go.kr)를 통해 확인하여 주시기 바랍니다.

[공동판매원]
Daiichi-Sankyo 한국다이이찌산쿄주식회사
서울시 중구 을지로5길 26 미래에셋 센터원빌딩 동관 15층 Tel. 02-3453-3300

[공동판매원]
현대약품(주)
서울시 강남구 봉은사로 135 현대약품빌딩 Tel. 1666-9979

Product info.

Balancing Efficacy and Safety
메바로친정®
(Pravastatin sodium)

P26-153-2028.03-V1

LDL-C GOAL

LIPITOR® Plus Tab.
(Ezetimibe/Atorvastatin calcium trihydrate)

오리지널 Atorvastatin에 Ezetimibe를 더하다![1]

신장애 환자에게 용량조절이 필요하지 않은 리피토®플러스![2]

Abbreviations LDL-C, Low density lipoprotein cholesterol
Reference 1. 리피토®플러스 허가증, Data on file. **2.** 리피토®플러스 국내허가사항, Available at https://nedrug.mfds.go.kr/index Accessed on SEP-02-2025

※ 국내 허가 범위 내 사용하는 경우에 한합니다. 이 약은 신기능 손상 또는 신장애 병력이 있는 환자에는 신중히 투여되어야 합니다.[2]

LIPITOR® Plus Tab.
(Ezetimibe/Atorvastatin calcium trihydrate)

제조 및 판매 제일약품

[공장] [17172] 경기도 용인시 처인구 백암면 청강가창로 7 (B동외) Tel : 080-333-6312 Website : www.jeilpharm.co.kr
리피토플러스 제품 의학정보 문의 medical@jeilpharm.co.kr Tel: 02-549-7451(EXT: 557), Fax: 02-542-7451
[본사] [06543] 서울특별시 서초구 사평대로 343 (반포동, 제일약품) TEL : 02-549-7451~65 FAX : 02-549-4045
https://www.jeilpharm.co.kr 소비자상담 Tel : 080-555-7171 (수신자부담) E-mail : webmaster@jeilpharm.co.kr
리피토플러스 제품 의학정보 문의 medical@jeilpharm.co.kr Tel: 02-549-7451 (EXT: 557), Fax: 02-542-7451

판매 비아트리스코리아㈜

[04527] 서울특별시 중구 세종대로 14, 비동 15층 (남대문로 5가, 그랜드센트럴) / Tel : 02-6411-6200 / Fax : 02-6411-6201
리피토 제품 의학정보 문의 Website : www.viatris.co.kr / Tel : 02-6411-6200 / E-mail : VIATRIS-Korea-MI@Viatris.com

LIPITOR®
atorvastatin calcium trihydrate tablets

제조 및 판매 비아트리스코리아㈜

[04527] 서울특별시 중구 세종대로 14, 비동 15층 (남대문로 5가, 그랜드센트럴) / Tel : 02-6411-6200 / Fax : 02-6411-6201
리피토 제품 의학정보 문의 Website : www.viatris.co.kr / Tel : 02-6411-6200 / E-mail : VIATRIS-Korea-MI@Viatris.com

판매 제일약품

[공장] [17172] 경기도 용인시 처인구 백암면 청강가창로 7 (B동외) Tel : 080-333-6312 Website : www.jeilpharm.co.kr
리피토플러스 제품 의학정보 문의 medical@jeilpharm.co.kr Tel: 02-549-7451(EXT: 557), Fax: 02-542-7451
[본사] [06543] 서울특별시 서초구 사평대로 343 (반포동, 제일약품) TEL : 02-549-7451~65 FAX : 02-549-4045
https://www.jeilpharm.co.kr 소비자상담 Tel : 080-555-7171 (수신자부담) E-mail : webmaster@jeilpharm.co.kr
리피토플러스 제품 의학정보 문의 medical@jeilpharm.co.kr Tel: 02-549-7451 (EXT: 557), Fax: 02-542-7451

※ 제품에 대한 자세한 정보는 제품에 동봉된 제품설명서 및 식품의약품안전처 의약품안전나라 (https://nedrug.mfds.go.kr)를 참고하시기 바랍니다.

KR-LIPP-2025-00064-30NOV2028

LIPITOR®
atorvastatin calcium trihydrate
tablets

신장 보호 리피토

신장애 환자에서 용량 조절이 필요 없는 리피토는 관상동맥 심장 질환 증거는 없으나 다중 위험요소*를 가진 성인 환자의 심근경색 및 뇌졸중 등 심혈관 질환 위험 감소 효과를 확인하였습니다. (*위험 요소: 55세 이상, 흡연, 고혈압, 낮은 HDL-C, 조기 관상동맥 질환 가족력 등)

[Safety info.] 신장 장애는 근병증 및 횡문근 융해의 위험 인자이므로 신장 장애 환자를 대상으로 근병증 발생을 관찰해야 합니다.
References 1. 리피토®정 제품허가정보 (최종개정년월일: 2025.12.03)

[수입자 / 판매자] 비아트리스 코리아㈜
[04527] 서울특별시 중구 세종대로 14, 비동 15층 (남대문로 5가, 그랜드센트럴)
제품 의학정보 문의 **Tel:** 02-6411-6200 | **E-mail:** Viatris-korea-MI@viatris.com
Website: www.viatris.co.kr

[판매자] 제일약품
[06543] 서울특별시 서초구 사평대로 343 (반포동, 제일약품)
Tel: 02-549-7451~65 | **Fax:** 02-549-4045
Website: https://www.jeilpharm.co.kr

[최신개정년월일] 2025.12.03
보다 자세한 제품 정보
확인을 위해 QR code로 연결된
제품 설명서 또는 비아트리스
웹사이트를 참고하시길 바랍니다.

KR-LIPI-2026-00339-30JUN2029

로수바미브
Ez-PAVE Trial,
NEJM 게재!

유한양행

Ez-PAVE는 LDL-C 70 mg/dL을 넘어
55 mg/dL라는 새로운 목표에 대한
근거를 확인하였습니다.

EZ-PAVE, Effects of **EZ**etimibe combination therapy for **P**atients with **A**therosclerotic cardio**V**ascular disease**E**; randomized comparisons of LDL-cholesterol targeting <70 versus <55 mg/dL

KR-RSM-2600006

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

"Bridge to Excellence,
Toward Cardiometabolic Health"

ICoLA | by KSoLA International Congress on Lipid & Atherosclerosis

KSoLA The Korean Society of Lipid and Atherosclerosis