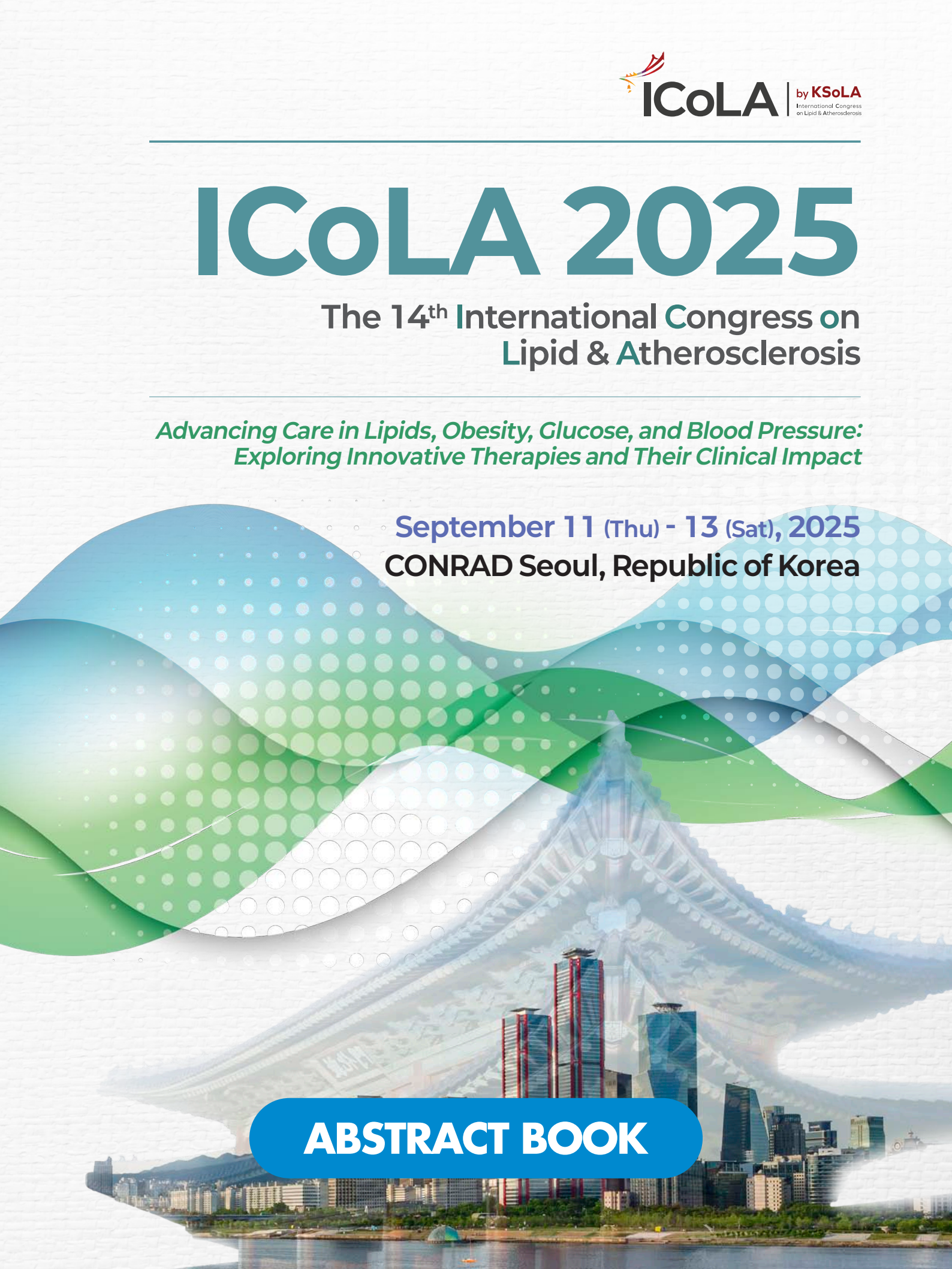


ICoLA 2025

The 14th International Congress on
Lipid & Atherosclerosis

*Advancing Care in Lipids, Obesity, Glucose, and Blood Pressure:
Exploring Innovative Therapies and Their Clinical Impact*

September 11 (Thu) - 13 (Sat), 2025
CONRAD Seoul, Republic of Korea



ABSTRACT BOOK

이상지질혈증 & 고혈압 통합 관리를 위한 Excellent Package Solution

로수젯 정
(에제티미브/로수바스타틴)

- 세계 최초 Rosuvastatin + Ezetimibe 복합제의 **CV Outcome 발표**¹⁾
- 국내사 개발 전문의약품 최초 원외처방조제액 전체 1위²⁾
- 국내 환자 대상 임상 연구 결과 **16편, SCIE급 저널등재** (2025년 3월 기준)
- 한미약품 R&D 및 자체 생산을 통한 Global 진출

R25
10 / 2.5 mg

R5
10 / 5 mg

R10
10 / 10 mg

R20
10 / 20 mg

**다양한 이상지질혈증 환자의 맞춤 처방,
로수젯으로 시작하세요!**

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- 세계 최초 Amlodipine camsylate + Losartan K 복합제³⁾
- 단독요법으로 조절되지 않는 고혈압 환자, 8주 투여 후 약 **90%의 혈압 치료 반응을 입증**^{4,5)}
- 제2기 고혈압 환자의 초기요법 적응증 획득⁶⁾
- 한미약품 R&D 및 자체 생산을 통한 Global 진출

5 / 50 mg

5 / 100 mg

10 / 50 mg

**다양한 고혈압 환자의 병용요법,
아모잘탄으로 시작하세요!**

세계 최초
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Hanmi 한미약품

*SBP(140 mmHg or DBP(90 mmHg 도달 혹은 기저치 대비 SBP)20 mmHg or DBP)10 mmHg 강하
CV, cardiovascular; R&D, research and development; SBP, systolic blood pressure; DBP, diastolic blood pressure; CCB, calcium channel blocker; ARB, angiotensin II receptor blocker
Ref. 1) Kim BK, et al. Lancet. 2022 Jul;400(10349):380-390. 2) UBIST D1 Sales data, 2024년 원외처방조제액 기준. 3) 식품의약품안전처 의약품통합정보시스템.
4) Kang SM, et al. Clin Ther. 2011 Dec;33(12):1953-1963. 5) Hong BK, et al. Am J Cardiovasc Drugs. 2012 Jun;12(3):189-195. 6) 식품의약품안전처, 아모잘탄정 허가사항(2025년 3월 기준)

*Lead
the way* *Atozet®*

변함없이
강력한[#] LDL-C 강하 효과
이상지질혈증 관리의
길을 열어줍니다.^{1,a}

2015 오가온, 아토피으로
새 출발

아토피® 식약처 허가² 2021

2025*

아토젯® 국내 출시
10주년²

*지질치료 경험이 없는 원발성 고콜레스테롤혈증 환자(n=628) 대상 임상 연구 결과. (Primary endpoint result) Mean change in direct LDL-C from baseline: Ezetimibe/Atorvastatin overall (pooled treatment groups) vs. Atorvastatin overall (pooled treatment groups) = -54.5% vs. -49.4%(P<0.01); Mean change in calculated LDL-C from baseline: Ezetimibe/Atorvastatin 10/10 mg vs. Atorvastatin 10 mg = -53% vs. -37%(P<0.01); Mean change in TG from baseline: Ezetimibe/Atorvastatin 10/10 mg vs. Atorvastatin 10 mg = -31% vs. -21%(P<0.05). *식약처 허가(아토르) : 2015.1.23

LDL-C : Low-density lipoprotein cholesterol

Study design. 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 ≤ 500 mg/dL were randomly assigned to receive 1 of the following for 12 weeks: ezetimibe (10 mg/dL; $n=65$); atorvastatin (10 , 20 , 40 , or 80 mg/dL; $n=248$); ezetimibe (10 mg) plus atorvastatin (10 , 20 , 40 , or 80 mg/dL; $n=255$); 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.

References 1. Ballantyne CM, et al. Effect of ezetimibe coadministered with atorvastatin in 628 patients with primary hypercholesterolemia: a prospective, randomized, double-blind trial. *Circulation*. 2003;107:2409-2415. 2. 아토티브 허가사항, 식품의약품안전처

[illegible]

“Safe
and efficient!”



Safe and Effective Treatment for Dyslipidemia

Litorvazet [®] Tab

Litorvazet tab [Ezetimibe / Atorvastatin]

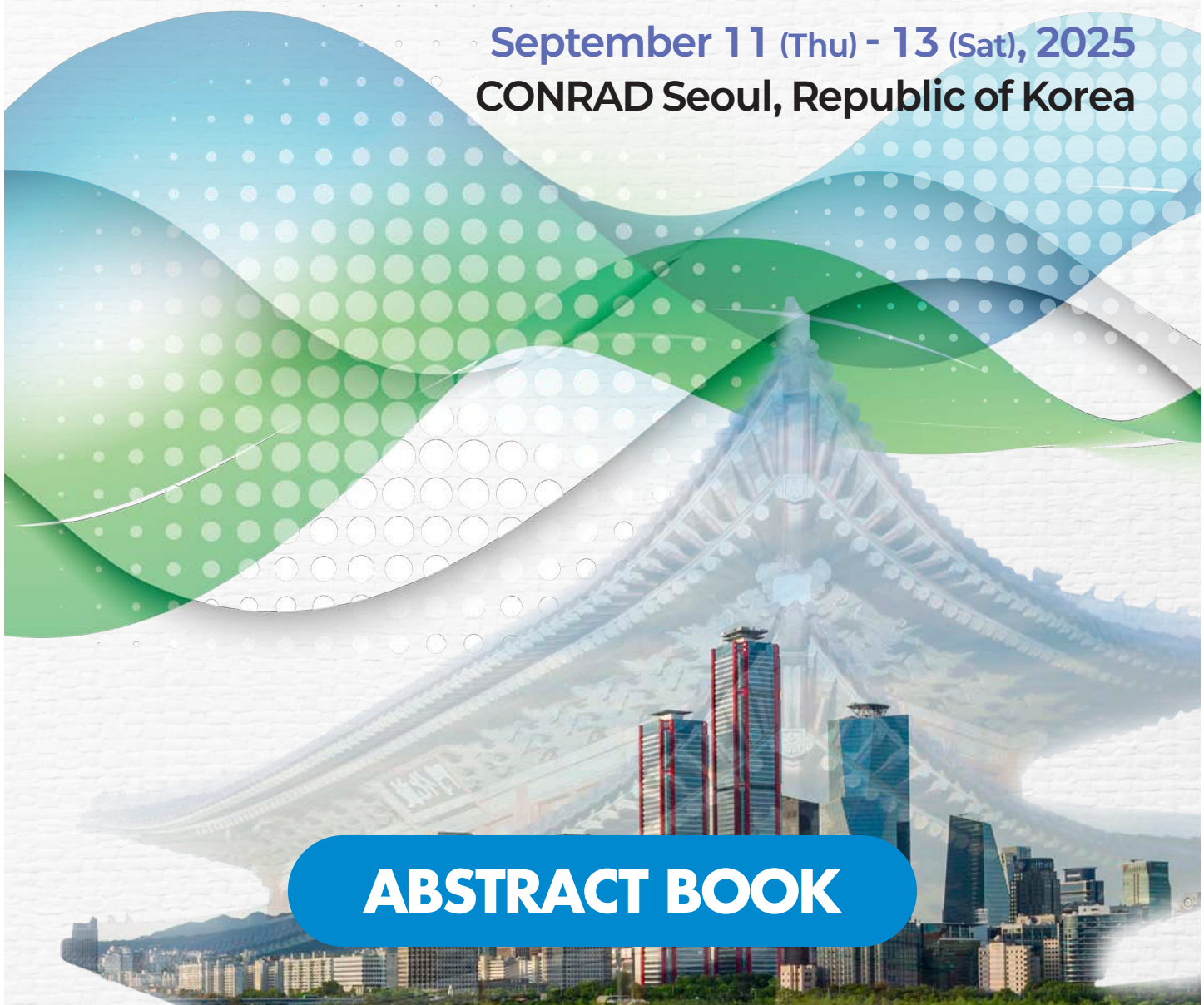
10/5, 10/10, 10/20, 10/40mg

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ABSTRACT BOOK

Welcome Message

Dear Colleagues,

On behalf of the organizing committee, we are pleased to invite you to the 14th International Congress on Lipid & Atherosclerosis (ICoLA 2025), hosted by the Korean Society of Lipid and Atherosclerosis (KSoLA), to be held on September 11 (Thu)–13 (Sat), 2025, at the Conrad Seoul, Republic of Korea.

Under the theme “Advancing Care in Lipids, Obesity, Glucose, and Blood Pressure: Exploring Innovative Therapies and Their Clinical Impact,” ICoLA 2025 expands its scientific vision beyond lipidology to encompass a comprehensive range of cardiometabolic risk factors. This broadened scope reflects our commitment to integrated strategies for the prevention and treatment of cardiovascular and metabolic diseases.

This year’s scientific program includes plenary and keynote lectures by global experts in lipid metabolism, vascular biology, and cardiometabolic disease management. These sessions will highlight recent advances in both basic science and clinical care, offering diverse perspectives from across the globe.

Further enhancing our global relevance, ICoLA 2025 will host joint symposia in collaboration with leading international societies, including the European Atherosclerosis Society (EAS), Japanese Atherosclerosis Society (JAS), and Canadian Society of Atherosclerosis, Thrombosis and Vascular Biology (CSATVB). These collaborative sessions will provide an exceptional opportunity to share scientific insights and foster global academic exchange.

In addition to plenary and keynote lectures, ICoLA 2025 will feature symposia, oral and poster presentations, and educational forums. Special emphasis will be placed on supporting young investigators, providing them with opportunities to present their work and engage with leaders in the field.

We warmly welcome your participation in ICoLA 2025. Join us in Seoul for an engaging and inspiring congress that promises to shape the future of cardiometabolic care.

Sincerely,



A stylized, cursive signature of Ki Hoon Han in black ink.

Ki Hoon Han
President, Board of Directors, KSoLA



A stylized, cursive signature of Sang-Hyun Kim in black ink.

Sang-Hyun Kim
Chairman, Board of Directors, KSoLA

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis



Program at a Glance

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Program in Detail

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Program at a Glance

[Day 1] September 11(Thu), 2025

(K): Korean Session

	Room 1	Room 2	Room 3	Room 4
12:00–	Registration			
13:00–14:30	Symposium 1 (K) 이상지질혈증 관리: 국내 당면 과제와 향후 발전 방향 Management of Dyslipidemia: Current Perspectives and Future Directions in Korea	Symposium 2 Cutting-edge Nutritional Approach for Cardiometabolic Risk Strategies	Symposium 3 Lipoprotein(a) Unmasked: Risk, Paradox, and Measurement	Symposium 4 Lipid Toxicity and Platelet Signaling in Atherosclerotic Inflammation
14:30–14:40	Break			
14:40–16:10	Oral Presentation 1	Symposium 5 Artificial Intelligence in Precision Diagnosis of Atherosclerosis and Cardiovascular Risk	Symposium 6 CVD Risk Prediction in Special Population	Symposium 7 Redefining Cardiovascular Care: PCSK9 Mechanisms and siRNA-based Advances
16:10–16:20	Break			
16:20–17:50	Symposium 8 Recent Prognostic Evidence for the Triglyceride–Glucose (TyG) Index as a Novel Surrogate Marker of Insulin Resistance	JAS-TSLA-KSoLA Joint Symposium Familial Hypercholesterolemia (FH) in the Era of Precision Medicine	Symposium 9 How Low is Low Enough for LDL-C in Diabetes?	Symposium 10 Socioeconomic Status: The Missing Piece in Cardiovascular Risk Stratification
18:10–	Welcome Reception (Lobby, Studio 4–7, 6F)			

Program at a Glance

[Day 2] September 12(Fri), 2025

(K): Korean Session

	Room 1	Room 2	Room 3	Room 4
07:30-08:30	Breakfast Symposium 1	Breakfast Symposium 2	Breakfast Symposium 3	Breakfast Symposium 4
	* Meals will be served from 07:30 to 08:00 on a first-come-first-served basis.			
08:30-10:00	Symposium 11 Genetic Insights into Dyslipidemia and Diabetes: Pathways, Predictions, and Interventions	AAS-KSoLA Joint Symposium Comprehensive Strategies for the Prevention of Atherosclerosis: Current Insights and Future Directions	Symposium 12 Residual CV Risk Era: Remnant Cholesterol	Symposium 13 Shaping the Future of Lipid Management: Insights from the New Consensus and Current Dyslipidemia Trends
10:00-10:10	Break			
10:10-10:20	Opening Address			
10:20-11:00	Plenary Lecture 1 Advances in lipid-lowering therapy: shaping the future of cardiovascular prevention			
11:05-11:45	Special Lecture 1 (K) Integrating ChatGPT into medical and clinical research workflows			
11:45-12:00	Coffee Break			
12:00-13:00	Luncheon Symposium 1 (K)	Luncheon Symposium 2	Luncheon Symposium 3	Luncheon Symposium 4
13:00-14:30	Oral Presentation 2	Symposium 14 What is Important beyond Stenosis in Intracranial Atherosclerosis	Symposium 15 Nontraditional Risk Factors for ASCVD	Symposium 16 Defining the Optimal LDL-C Target for Prevention of Atherosclerotic Cardiovascular and Cerebrovascular Disease
14:40-15:50	Moderated Poster Presentation 1, 3, 5 (Studio 5, 6, 7, 6F)			
15:50-16:30	Keynote Lecture 1 Multi-omics data integration from patients with carotid stenosis illuminates key molecular signatures of atherosclerotic instability			
16:30-16:45	Coffee Break			
16:45-18:15	Symposium 17 Emerging Drugs for Dyslipidemia Management	VAS-KSoLA Joint Symposium Lipid-Lowering Strategies: Targets and Therapies in Clinical Practice	Symposium 18 Sex and Gender Differences in Cardiometabolic Health: From Biology to Treatment	Symposium 19 Immunometabolic Programming of Macrophages in Cardiovascular Injury and Repair

Program at a Glance

[Day 3] September 13(Sat), 2025

(K): Korean Session

	Room 1	Room 2	Room 3	Room 4
07:50-08:50	Breakfast Symposium 5	Breakfast Symposium 6	Breakfast Symposium 7	Breakfast Symposium 8
	* Meals will be served from 07:50 to 08:20 on a first-come-first-served basis.			
08:50-10:20	Oral Presentation 3	CSATVB-KSoLA Joint Symposium Long-term Exposure to LDL-cholesterol and Early Cardiovascular Risk Prevention	Symposium 20 Optimized Management of Cardiometabolic Syndrome in the Elderly: Focus on Multimorbidity and Frailty	Symposium 21 From Treatment to Prevention: A Paradigm Shift in Obesity
10:20-10:40	Coffee Break			
10:40-11:20	Plenary Lecture 2 Cholesterol lowering throughout the life-course to prevent atherosclerotic cardiovascular disease—a pragmatic population health approach			
11:25-12:05	Keynote Lecture 2 The role of smooth muscle cell in atherosclerotic plaque progression			
12:05-12:20	Break			
12:20-13:20	Luncheon Symposium 5 (K)	Luncheon Symposium 6	Luncheon Symposium 7	
13:30-14:40	Special Lecture 2 (K) (Room 1,2,3, 3F) Gardens of Korea (한국의 정원)			
	Moderated Poster Presentation 2, 4, 6 (Studio 5, 6, 7, 6F)			
14:50-16:20	2025 KSoLA Awards for Scientific Excellence & Young Investigator	KoSFoST-KSoLA Joint Symposium Role of Medicinal Foods in Atherosclerosis and Cardiometabolic Disease	Symposium 22 Atherosclerosis beyond Traditional Risks: Emerging Clinical Challenges in Comorbid Conditions	Symposium 23 Publication Committee Session: JLA Award for Outstanding Article
16:20-17:50	Symposium 24 Dyslipidemia Management in Special Populations	EAS-KSoLA Joint Symposium The Impact of Remnant Cholesterol on Various Disease Pathologies and Clinical Outcomes	Symposium 25 Lipid-immune Interactions Shaping Vascular Repair and Disease	MSA-KSoLA Joint Symposium Recent Evidence in Non-invasive Cardiovascular Risk Assessment Techniques
17:50-	Closing Ceremony			

Program in Detail

Key Lectures

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Plenary Lecture 1

Sep 12(Fri) 10:20–11:00 | Room 1,2,3 (3F)

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

10:20–11:00 **Advances in lipid-lowering therapy: shaping the future of cardiovascular prevention**
Ioanna Gouni-Berthold (University of Cologne, Germany)

Plenary Lecture 2

Sep 13(Sat) 10:40–11:20 | Room 1,2,3 (3F)

CHAIRPERSON : Ki Hoon Han (University of Ulsan, Republic of Korea)

10:40–11:20 **Cholesterol lowering throughout the life-course to prevent atherosclerotic cardiovascular disease—a pragmatic population health approach**
Kausik Kumar Ray (Imperial Centre for Cardiovascular Disease Prevention, UK)

Keynote Lecture 1

Sep 12(Fri) 15:50–16:30 | Room 1,2,3 (3F)

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

15:50–16:30 **Multi-omics data integration from patients with carotid stenosis illuminates key molecular signatures of atherosclerotic instability**
Ljubica Matic (Karolinska Institute, Sweden)

Keynote Lecture 2

Sep 13(Sat) 11:25–12:05 | Room 1,2,3 (3F)

CHAIRPERSON : Byung-Chul Oh (Gachon University, Republic of Korea)

11:25–12:05 **The role of smooth muscle cells in atherosclerotic plaque progression**
Jacob Fog Bentzon (Aarhus University, Denmark)

Special Lecture 1 (K)

Sep 12(Fri) 11:05–11:45 | Room 1,2,3 (3F)

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

11:05–11:45 **Integrating ChatGPT into medical and clinical research workflows**
Ki-Hyun Jeon (Seoul National University, Republic of Korea)

Special Lecture 2 (K)

Sep 13(Sat) 13:30–14:40 | Room 1,2,3 (3F)

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

13:30–14:40 **Gardens of Korea (한국의 정원)**

Hong June Yoo (Director General, National Museum of Korea, Republic of Korea)

Main Symposia

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Symposium 1 (K)

Sep 11(Thu) 13:00–14:30 | Room 1 (3F)

이상지질혈증 관리: 국내 당면 과제와 향후 발전 방향

Management of Dyslipidemia: Current Perspectives and Future Directions in Korea

CHAIRPERSONS : 김상현 이사장 (한국지질·동맥경화학회)

조연희 회장 (한국건강검진학회)

13:00–13:10 **인사말**

박주민 위원장 (국회 보건복지위원회)

13:10–13:25 **지질관리! 이상지질혈증 치료 현황과 급여 기준 현실화**

정인경 이사 (한국지질·동맥경화학회)

13:25–13:40 **지질관리! 혈압, 혈당 관리를 넘어 – 만성질환 통합 관리와 이상지질혈증**

백재욱 이사 (대한가정의학과 의사회)

13:40–13:55 **지질관리! 현장의 목소리 – 일차의료 현장의 현실과 지원방안에 대한 요구**

이태인 이사 (한국건강검진학회)

13:55–14:10 **지질관리! 더 빨리, 더 정확히 – 초고령화 시대, 더 오래 더 건강하기 위한 조기검진과 치료의 중요성**

김은지 교수 (가천의대 예방의학과)

14:10–14:30 **패널 토의 및 기자회견**

이창현 이사 (한국건강검진학회)

이금숙 특임이사 (한국의학바이오기자협회)

곽경근 부회장 (대한내과의사회)

전하윤 사무관 (보건복지부)

Symposium 2

Sep 11(Thu) 13:00–14:30 | Room 2 (3F)

Cutting-edge Nutritional Approach for Cardiometabolic Risk Strategies

CHAIRPERSONS : **Hyun-Sook Kim** (Sookmyung Women's University, Republic of Korea)
Eun Mi Kim (Sungkyunkwan University, Republic of Korea)

- | | |
|-------------|---|
| 13:00–13:20 | Interplay between molecular and environmental factors in the new era of precision medicine
Jordi Merino (University of Copenhagen, Denmark) |
| 13:20–13:40 | Human microbiome and the path to cardiometabolic health
Hyun Ju You (Seoul National University, Republic of Korea) |
| 13:40–14:00 | Iron overload, autophagy dysfunction, and ferroptosis: mechanistic insights into cardiometabolic disease
Hyekyoung Sung (York University, Canada) |
| 14:00–14:30 | Panel Discussion
Hyunju Kang (Keimyung University, Republic of Korea)
Jeongseon Kim (National Cancer Center, Republic of Korea)
Hoyoun Won (Chung-Ang University, Republic of Korea) |

Symposium 3

Sep 11(Thu) 13:00–14:30 | Room 3 (3F)

Lipoprotein(a) Unmasked: Risk, Paradox, and Measurement

CHAIRPERSONS : **Myung A Kim** (Seoul National University, Republic of Korea)
Byung Jin Kim (Sungkyunkwan University, Republic of Korea)

- | | |
|-------------|--|
| 13:00–13:20 | Epidemiologic evidence on Lp(a) and ASCVD in Asian populations
Youngwoo Jang (Gachon University, Republic of Korea) |
| 13:20–13:40 | The inverse link between Lp(a) and metabolic risk: signal or noise?
Jung A Kim (Korea University, Republic of Korea) |
| 13:40–14:00 | Lp(a) testing today: mind the method, mind the unit
Sang-Guk Lee (Yonsei University, Republic of Korea) |
| 14:00–14:30 | Panel Discussion
Soo-Jin Kim (Kosin University, Republic of Korea)
Wonjin Kim (CHA University, Republic of Korea)
Joonpyo Lee (Gachon University, Republic of Korea)
Seong Huan Choi (Inha University, Republic of Korea) |

Symposium 4

Sep 11(Thu) 13:00–14:30 | Room 4 (5F)

Lipid Toxicity and Platelet Signaling in Atherosclerotic Inflammation

CHAIRPERSONS : **Jae-Ryong Kim** (Yeungnam University, Republic of Korea)

Hyoung Kyu Kim (Inje University, Republic of Korea)

13:00–13:20 **From nanoparticles to biocides: how toxic chemicals alter lipid homeostasis and accelerate atherosclerotic events**

Ok-Nam Bae (Hanyang University, Republic of Korea)

13:20–13:40 **Immune modulation of lipid metabolism promotes BAT whitening under thermoneutral conditions**

Jun Young Hong (Yonsei University, Republic of Korea)

13:40–14:00 **Role of MCU in human platelet aggregation**

Jin O-Uchi (University of South Florida, USA)

14:00–14:30 **Panel Discussion**

Kyuhoo Kim (The Catholic University of Korea, Republic of Korea)

Jeonghan Kim (The Catholic University of Korea, Republic of Korea)

Chang-Hoon Woo (Yeungnam University, Republic of Korea)

Symposium 5

Sep 11(Thu) 14:40–16:10 | Room 2 (3F)

Artificial Intelligence in Precision Diagnosis of Atherosclerosis and Cardiovascular Risk

CHAIRPERSONS : **Hun Sik Park** (Kyungpook National University, Republic of Korea)

Myung-gon Kim (Catholic Kwandong University, Republic of Korea)

14:40–15:00 **Coronary atherosclerosis and cardiovascular risk prediction through AI-assisted stress echocardiography: insights from the PROTEUS RCT trial**

Paul Leeson (University of Oxford, UK)

15:00–15:20 **AI-enhanced ECG for acute myocardial infarction detection and cardiovascular risk stratification**

Junmyung Kwon (Medical AI, Republic of Korea)

15:20–15:40 **Artificial intelligence-based coronary artery disease screening versus usual care for patients with suspected coronary artery disease: AI-Gatekeeper randomized controlled trial**

SungA Bae (Yonsei University, Republic of Korea)

15:40–16:10 **Panel Discussion**

Dae-Young Kim (Inha University, Republic of Korea)

Seok Oh (Chonnam National University, Republic of Korea)

Hyung Joon Joo (Korea University, Republic of Korea)

Symposium 6

Sep 11(Thu) 14:40–16:10 | Room 3 (3F)

CVD Risk Prediction in Special Population

CHAIRPERSONS : **Hyeon Chang Kim** (Yonsei University, Republic of Korea)
SungHee Choi (Seoul National University, Republic of Korea)

-
- 14:40–15:00 **CVD risk prediction in low-risk population**
Hokyou Lee (Yonsei University, Republic of Korea)
- 15:00–15:20 **CVD risk prediction in older persons**
Sangwoo Park (University of Ulsan, Republic of Korea)
- 15:20–15:40 **CVD risk prediction in diabetes and high-risk patients**
Eu Jeong Ku (Seoul National University, Republic of Korea)
- 15:40–16:10 **Panel Discussion**
Se-eun Park (Sungkyunkwan University, Republic of Korea)
Yong-Jae Lee (Yonsei University, Republic of Korea)
Keum Ji Jung (Yonsei University, Republic of Korea)

Symposium 7

Sep 11(Thu) 14:40–16:10 | Room 4 (5F)

Redefining Cardiovascular Care: PCSK9 Mechanisms and siRNA-based Advances

CHAIRPERSONS : **Jun-Hee Lee** (Hallym University, Republic of Korea)
Sungha Park (Yonsei University, Republic of Korea)

-
- 14:40–15:00 **Deep dive into PCSK9: the master regulator of LDL metabolism and inflammation**
Hyun-Duk Jang (Seoul National University, Republic of Korea)
- 15:00–15:20 **siRNA-based lipid therapies: a revolution in cholesterol management**
Jin Wi (Gachon University, Republic of Korea)
- 15:20–15:40 **Familial hypercholesterolemia treatment beyond statin**
Rodrigo Alonso (Center for Advanced Metabolic Medicine and Nutrition, Chile)
- 15:40–16:10 **Panel Discussion**
Soo-Jin Kim (Kosin University, Republic of Korea)
Ye Seul Yang (Seoul National University, Republic of Korea)
Sangmo Hong (Hanyang University, Republic of Korea)

Symposium 8

Sep 11(Thu) 16:20–17:50 | Room 1 (3F)

Recent Prognostic Evidence for the Triglyceride–Glucose (TyG) Index as a Novel Surrogate Marker of Insulin Resistance

CHAIRPERSONS : **Weon Kim** (Kyung Hee University, Republic of Korea)
Junghwan Park (Hanyang University, Republic of Korea)

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|-------------|---|
| 16:20–16:40 | Prediction of ASCVD using the TyG index: insights from the multinational PURE registry
Patricio Lopez–Jaramillo (Universidad de Santander, Colombia) |
| 16:40–17:00 | Prediction of ASCVD using the TyG index: evidence from Korean data
Yong–Jae Lee (Yonsei University, Republic of Korea) |
| 17:00–17:20 | Prediction of coronary artery calcification progression using the TyG index
Ki–Bum Won (Chung–Ang University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Kyung Woo Park (Seoul National University, Republic of Korea)
Se–eun Park (Sungkyunkwan University, Republic of Korea)
Jun–Hee Lee (Hallym University, Republic of Korea) |

Symposium 9

Sep 11(Thu) 16:20–17:50 | Room 3 (3F)

How Low is Low Enough for LDL–C in Diabetes?

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

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|-------------|---|
| 16:20–16:40 | LDL–C targets for patients with diabetes across major guidelines
Jong Han Choi (Konkuk University, Republic of Korea) |
| 16:40–17:00 | Pro: all patients with diabetes should be treated to LDL–C <55 mg/dL?
Wonjin Kim (CHA University, Republic of Korea) |
| 17:00–17:20 | Cons: LDL–C targets should be personalized in diabetic patients?
Mihye Seo (Soonchunhyang University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Sungha Park (Yonsei University, Republic of Korea)
SungA Bae (Yonsei University, Republic of Korea)
Ye Seul Yang (Seoul National University, Republic of Korea) |

Symposium 10

Sep 11(Thu) 16:20–17:50 | Room 4 (5F)

Socioeconomic Status: The Missing Piece in Cardiovascular Risk Stratification

CHAIRPERSONS : **Sung Nim Han** (Seoul National University, Republic of Korea)
Jeongseon Kim (National Cancer Center, Republic of Korea)

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|-------------|---|
| 16:20–16:40 | Mechanisms linking socioeconomic status and cardiovascular risk
SungHee Choi (Seoul National University, Republic of Korea) |
| 16:40–17:00 | Epidemiologic evidence on socioeconomic status and cardiovascular outcomes
Jong-Ha Baek (Gyeongsang National University, Republic of Korea) |
| 17:00–17:20 | New models for CVD risk assessment: incorporating socioeconomic data
Sadiya S. Khan (Northwestern University, USA) |
| 17:20–17:50 | Panel Discussion
Dae-Young Kim (Inha University, Republic of Korea)
Hyun-Jin Kim (Hanyang University, Republic of Korea)
Yongjoo Park (Chonnam National University, Republic of Korea)
Sangmo Hong (Hanyang University, Republic of Korea) |

Symposium 11

Sep 12(Fri) 08:30–10:00 | Room 1 (3F)

Genetic Insights into Dyslipidemia and Diabetes: Pathways, Predictions, and Interventions

CHAIRPERSONS : **Hyun Jae Kang** (Seoul National University, Republic of Korea)
Byung-Wan Lee (Yonsei University, Republic of Korea)

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|-------------|--|
| 08:30–08:50 | Exploring the genetic landscape of CVD and diabetes: finding and future implication
Soo Heon Kwak (Seoul National University, Republic of Korea) |
| 08:50–09:10 | Multi-omics and multi-trait analysis of metabolic disease
Hong-Hee Won (Sungkyunkwan University, Republic of Korea) |
| 09:10–09:30 | Gene-targeting therapy in familial hypercholesterolemia
Sang-Hak Lee (Yonsei University, Republic of Korea) |
| 09:30–10:00 | Panel Discussion
Yoo-Wook Kwon (Seoul National University, Republic of Korea)
Hee-Dong Kim (Soonchunhyang University, Republic of Korea)
Young Shin Lee (Kyung Hee University, Republic of Korea) |

Symposium 12

Sep 12(Fri) 08:30–10:00 | Room 3 (3F)

Residual CV Risk Era: Remnant Cholesterol

CHAIRPERSONS : **Jeong-Taek Woo** (Kyung Hee University, Republic of Korea)
Jae Hyung Park (Korea University, Republic of Korea)

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|-------------|--|
| 08:30–08:50 | Remnant cholesterol as a residual risk in ASCVD patients under statin-based lipid-lowering therapy
Sung Gyun Ahn (Yonsei University, Republic of Korea) |
| 08:50–09:10 | Association between remnant cholesterol and sarcopenia: implications on muscle health
Soo Yeon Jang (Korea University, Republic of Korea) |
| 09:10–09:30 | Targeting remnant cholesterol in the era of precision lipid management
Pablo Corral (Fasta University, Argentina) |
| 09:30–10:00 | Panel Discussion
Kyung-Soo Kim (CHA University, Republic of Korea)
Hong Seok Lim (Ajou University, Republic of Korea)
Jaehoon Chung (Dongguk University, Republic of Korea) |

Symposium 13

Sep 12(Fri) 08:30–10:00 | Room 4 (5F)

Shaping the Future of Lipid Management: Insights from the New Consensus and Current Dyslipidemia Trends

CHAIRPERSONS : **Keeho Song** (Konkuk University, Republic of Korea)
Sang-Rok Lee (Jeonbuk National University, Republic of Korea)

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|-------------|---|
| 08:30–08:50 | 2024 KSoLA consensus on secondary dyslipidemia
Jae Hyun Bae (Seoul National University, Republic of Korea) |
| 08:50–09:10 | Understanding the progression of obesity-induced organ damage: Insights from the EAS staging system
Dae Young Cheon (Hallym University, Republic of Korea) |
| 09:10–09:30 | Sex-specific gaps in lipid management and ASCVD
Hyun-Jin Kim (Hanyang University, Republic of Korea) |
| 09:30–10:00 | Panel Discussion
Min Kyong Moon (Seoul National University, Republic of Korea)
Jaehyun Bae (Hallym University, Republic of Korea)
Ji-Yong Jang (National Health Insurance Service Ilsan Hospital, Republic of Korea) |

Symposium 14

Sep 12(Fri) 13:00–14:30 | Room 2 (3F)

What is Important beyond Stenosis in Intracranial Atherosclerosis

CHAIRPERSONS : **Eung Gyu Kim** (Inje University, Republic of Korea)
Kwang-Yeol Park (Chung-Ang University, Republic of Korea)

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|-------------|--|
| 13:00–13:20 | Vascular tortuosity and atherosclerosis burden
Mi-Yeon Eun (Kyungpook National University, Republic of Korea) |
| 13:20–13:40 | Vessel wall enhancement and inflammation
Dong-Wan Kang (Seoul National University, Republic of Korea) |
| 13:40–14:00 | Blood viscosity and intracranial atherosclerosis
Ho Geol Woo (Kyung Hee University, Republic of Korea) |
| 14:00–14:30 | Panel Discussion
Hyung-Min Kwon (Seoul National University, Republic of Korea)
Bum Joon Kim (University of Ulsan, Republic of Korea)
Woo-Keun Seo (Sungkyunkwan University, Republic of Korea)
Jin-Man Jung (Korea University, Republic of Korea) |

Symposium 15

Sep 12(Fri) 13:00–14:30 | Room 3 (3F)

Nontraditional Risk Factors for ASCVD

CHAIRPERSONS : **Seok-Min Kang** (Yonsei University, Republic of Korea)
Young Sup Byun (Inje University, Republic of Korea)

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|-------------|--|
| 13:00–13:20 | Environmental pollution as a nontraditional risk factor for atherosclerotic cardiovascular disease: mechanisms and impacts
Sun-Young Kim (National Cancer Center, Republic of Korea) |
| 13:20–13:40 | Health inequity as a nontraditional risk factor for ASCVD
Eunji Kim (Gachon University, Republic of Korea) |
| 13:40–14:00 | Psychiatric disorders: an overlooked nontraditional risk factor in the development of atherosclerotic cardiovascular disease
Seung Jin Han (Ajou University, Republic of Korea) |
| 14:00–14:30 | Panel Discussion
Si-Hyuck Kang (Seoul National University, Republic of Korea)
Ji-Hyun Kim (The Catholic University of Korea, Republic of Korea)
Jaehyun Bae (Hallym University, Republic of Korea)
Jong-Tae Lee (Korea University, Republic of Korea) |

Symposium 16

Sep 12(Fri) 13:00–14:30 | Room 4 (5F)

Defining the Optimal LDL–C Target for Prevention of Atherosclerotic Cardiovascular and Cerebrovascular Disease

CHAIRPERSONS : Jeong–Taek Woo (Kyung Hee University, Republic of Korea)

Min Kyong Moon (Seoul National University, Republic of Korea)

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- 13:00–13:20 **Optimal LDL–C goals for secondary prevention in high–risk patients – a cardiologist’s perspective**
Hyun Sung Joh (Seoul National University, Republic of Korea)
- 13:20–13:40 **Optimal LDL–C level for stroke prevention – insights from a neurologist’s perspective**
Wookjin Yang (University of Ulsan, Republic of Korea)
- 13:40–14:00 **Different LDL–C targets by duration of diabetes: what is the evidence? – an endocrinologist’s perspective**
Young–Sang Lyu (Chosun University, Republic of Korea)
- 14:00–14:30 **Panel Discussion**
Kyung–Soo Kim (CHA University, Republic of Korea)
Minwoo Lee (Hallym University, Republic of Korea)
Jung–Kyu Han (Seoul National University, Republic of Korea)
Joon Ho Moon (Seoul National University, Republic of Korea)

Symposium 17

Sep 12(Fri) 16:45–18:15 | Room 1 (3F)

Emerging Drugs for Dyslipidemia Management

CHAIRPERSONS : Ki Chul Sung (Sungkyunkwan University, Republic of Korea)

Hack–Lyoung Kim (Seoul National University, Republic of Korea)

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- 16:45–17:05 **Bempedoic acid in real world practice, for whom?**
Ioanna Gouni–Berthold (University of Cologne, Germany)
- 17:05–17:25 **Targeting APOC3 for triglyceride management**
Jin Wi (Gachon University, Republic of Korea)
- 17:25–17:45 **ANGPTL3 and ANGPTL8 inhibition: novel strategies to combat atherogenic dyslipidemia**
Janghoon Lee (Kyungpook National University, Republic of Korea)
- 17:45–18:15 **Panel Discussion**
Si–Hyuck Kang (Seoul National University, Republic of Korea)
Yea Eun Kang (Chungnam National University, Republic of Korea)
Joon Ho Moon (Seoul National University, Republic of Korea)
Jung–Kyu Han (Seoul National University, Republic of Korea)

Symposium 18

Sep 12(Fri) 16:45–18:15 | Room 3 (3F)

Sex and Gender Differences in Cardiometabolic Health: From Biology to Treatment

CHAIRPERSONS : **Wook Bum Pyun** (Ewha Womans University, Republic of Korea)
Mi-Seung Shin (Gachon University, Republic of Korea)

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|-------------|--|
| 16:45–17:05 | Biological and hormonal mechanisms linking sex and cardiometabolic risk
Erin D. Michos (Johns Hopkins University, USA) |
| 17:05–17:25 | Sex and gender differences in risk assessment and biomarkers for cardiovascular disease
Ji Eun Lee (Korea University, Republic of Korea) |
| 17:25–17:45 | Do women and men respond differently to cardiometabolic therapies?
Hyun Ju Yoon (Chonnam National University, Republic of Korea) |
| 17:45–18:15 | Panel Discussion
Hyun-Jin Kim (Hanyang University, Republic of Korea)
Ye Seul Yang (Seoul National University, Republic of Korea)
Hyeonju Jeong (Hanyang University, Republic of Korea) |

Symposium 19

Sep 12(Fri) 16:45–18:15 | Room 4 (5F)

Immunometabolic Programming of Macrophages in Cardiovascular Injury and Repair

CHAIRPERSONS : **Goo Taeg Oh** (Ewha Womans University, Republic of Korea)
Yongseek Park (Kyung Hee University, Republic of Korea)

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|-------------|--|
| 16:45–17:05 | Glutamine homeostasis: an overlooked axis in cardiometabolic inflammation
Laurent Yvan-Charvet (INSERM, France) |
| 17:05–17:25 | Myeloid metabolism in the amplification and resolution of cardiac inflammation
Edward Thorp (Northwestern University, USA) |
| 17:25–17:45 | Therapeutic promise of ANGPTL4 in cardiovascular inflammation
Yong Sook Kim (Chonnam National University, Republic of Korea) |
| 17:45–18:15 | Panel Discussion
Jeong-Min Kim (Seoul National University, Republic of Korea)
Jae-Hoon Choi (Hanyang University, Republic of Korea)
Kyung-Sun Heo (Chungnam National University, Republic of Korea) |

Symposium 20

Sep 13(Sat) 08:50–10:20 | Room 3 (3F)

Optimized Management of Cardiometabolic Syndrome in the Elderly: Focus on Multimorbidity and Frailty

CHAIRPERSONS : Kwang-il Kim (Seoul National University, Republic of Korea)

Hiddenori Arai (National Center for Geriatrics and Gerontology, Japan)

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|-------------|--|
| 08:50–09:10 | Strategies for managing hyperlipidemia in fragile elderly patients
Dong-Ho Shin (Yonsei University, Republic of Korea) |
| 09:10–09:30 | Optimizing glucose control in fragile elderly patients with cardiometabolic syndrome
Soo Jin Yun (Kyung Hee University, Republic of Korea) |
| 09:30–09:50 | How to deal with multimorbidity and frailty for the prevention of cardiovascular disease
Hiddenori Arai (National Center for Geriatrics and Gerontology, Japan) |
| 09:50–10:20 | Panel Discussion
Jung-Woo Son (Yonsei University, Republic of Korea)
Min Kyung Lee (Hanyang University, Republic of Korea)
Ja Young Jeon (Ajou University, Republic of Korea)
Jung Yeon Choi (Seoul National University, Republic of Korea) |
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Symposium 21

Sep 13(Sat) 08:50–10:20 | Room 4 (5F)

From Treatment to Prevention: A Paradigm Shift in Obesity

CHAIRPERSONS : Jang-Young Kim (Yonsei University, Republic of Korea)

Cheol-Young Park (Sungkyunkwan University, Republic of Korea)

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|-------------|---|
| 08:50–09:10 | Anti-obesity effects of bioactive compound from natural products: a mechanism-oriented approach focusing on cellular signaling and metabolic regulation
Choon Young Kim (Yeungnam University, Republic of Korea) |
| 09:10–09:30 | Semaglutide for obesity treatment and cardiometabolic benefits
Yun Kyung Cho (University of Ulsan, Republic of Korea) |
| 09:30–09:50 | Tirzepatide a dual GIP GLP-1 receptor agonist in obesity and metabolic care
Sang-Ho Jo (Hallym University, Republic of Korea) |
| 09:50–10:20 | Panel Discussion
Bu Kyung Kim (Kosin University, Republic of Korea)
Hyun-Suk Yang (Konkuk University, Republic of Korea)
Hyemoon Chung (Kyung Hee University, Republic of Korea) |
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Symposium 22

Sep 13(Sat) 14:50–16:20 | Room 3 (3F)

Atherosclerosis beyond Traditional Risks: Emerging Clinical Challenges in Comorbid Conditions

CHAIRPERSONS : **Sang Hong Baek** (The Catholic University of Korea, Republic of Korea)
Masayuki Yoshida (Institute of Science Tokyo, Japan)

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|-------------|--|
| 14:50–15:10 | Atherosclerosis and lipid management in cancer survivors: balancing cardiovascular and oncologic risks
Nuri Lee (Chonnam National University, Republic of Korea) |
| 15:10–15:30 | Metabolic dysfunction–associated steatotic liver disease as a cardiovascular risk equivalent
Won Kim (Seoul National University, Republic of Korea) |
| 15:30–15:50 | Atherosclerosis in chronic inflammatory diseases: focus on rheumatoid arthritis and systemic autoimmunity
Ji-Won Kim (Daegu Catholic University, Republic of Korea) |
| 15:50–16:20 | Panel Discussion
Minjeong Kim (Ewha Womans University, Republic of Korea)
Sungjoon Park (Seoul National University, Republic of Korea)
Sung Kee Ryu (Ewha Womans University, Republic of Korea) |

Symposium 23

Sep 13(Sat) 14:50–16:20 | Room 4 (5F)

Publication Committee Session: JLA Award for Outstanding Article

CHAIRPERSONS : **Hyuk-Sang Kwon** (The Catholic University of Korea, Republic of Korea)
In-Kyung Jeong (Kyung Hee University, Republic of Korea)

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|-------------|---|
| 14:50–15:05 | Discrepant Effect of High-Density Lipoprotein Cholesterol on the Hematologic Malignancy Risk: A Nationwide Cohort Study
Su Youn Nam (Kyungpook National University, Republic of Korea) |
| 15:05–15:20 | Cholesterol and Cardiovascular Risk in Type 2 Diabetes: The Role of Kidney Function
Mee Kyoung Kim (The Catholic University of Korea, Republic of Korea) |
| 15:20–15:35 | Association Between Lipoprotein (a) Levels and Coronary Artery Disease (CAD) Among Patients With or Without CAD Family History
Hayato Tada (Kanazawa University, Japan) |
| 15:35–15:50 | Investigation of the Influence of Lipoprotein(a) and Oxidized Lipoprotein(a) on Plasminogen Activation and Fibrinolysis
Wen-Liang Song (Brown University Health, USA) |
| 15:50–16:05 | Fatty Acids in Childhood Obesity: A Link Between Nutrition, Metabolic Alterations and Cardiovascular Risk
Belen Davico (University of Buenos Aires, Argentina) |

Symposium 24

Sep 13(Sat) 16:20–17:50 | Room 1 (3F)

Dyslipidemia Management in Special Populations

CHAIRPERSONS : **Yong-Jae Kim** (Kim's Neurology Clinic, Republic of Korea)
Young Joon Hong (Chonnam National University, Republic of Korea)

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|-------------|---|
| 16:20–16:40 | Managing dyslipidemia in women before and after menopause
Mi-Na Kim (Korea University, Republic of Korea) |
| 16:40–17:00 | Managing dyslipidemia in patients with liver disease
Byung Sik Kim (Hanyang University, Republic of Korea) |
| 17:00–17:20 | HIV, chronic inflammation, and dyslipidemia
Seonghoon Choi (Hallym University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Sung Kee Ryu (Ewha Womans University, Republic of Korea)
Bom Lee (CHA University, Republic of Korea)
Seung-Hwan Lee (The Catholic University of Korea, Republic of Korea) |

Symposium 25

Sep 13(Sat) 16:20–17:50 | Room 3 (3F)

Lipid-immune Interactions Shaping Vascular Repair and Disease

CHAIRPERSONS : **Chi Dae Kim** (Pusan National University, Republic of Korea)
Young Mi Park (Ewha Womans University, Republic of Korea)

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| 16:20–16:40 | IKKϵ-deficient macrophages impede cardiac repair after myocardial infarction by enhancing the macrophage-myofibroblast transition
Youngkeun Ahn (Chonnam National University, Republic of Korea) |
| 16:40–17:00 | Sensing ceramides by CYSLTR2 and P2RY6 to aggravate atherosclerosis
Wei Kong (Peking University, China) |
| 17:00–17:20 | Targeting the USP35-FASN axis and choline metabolism in cancer: a lipidomics perspective
Hyunbeom Lee (Korea Institute of Science and Technology (KIST), Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Jun Namkung (Yonsei University, Republic of Korea)
Taesik Park (Gachon University, Republic of Korea)
Jae-Han Jeon (Kyungpook National University, Republic of Korea)
Su Myung Jung (Sungkyunkwan University, Republic of Korea) |

2025 KSoLA Awards for Scientific Excellence & Young Investigator

Sep 13(Sat) 14:50–15:25 | Room 1 (3F)

CHAIRPERSONS : **Ki Hoon Han** (University of Ulsan, Republic of Korea)
Sang-Hyun Kim (Seoul National University, Republic of Korea)

- 14:50–14:55 **2025 KSoLA Award Ceremony for Scientific Excellence**
- 14:55–15:00 **2025 KSoLA Award Ceremony for Young Investigator**
- 15:00–15:25 **Triglyceride metabolism, hypertriglyceridemia, and its therapeutics**
Nam Hoon Kim (Korea University, Republic of Korea)
- 15:20–15:25 **Q&A**

Joint Symposia

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JAS–TSLA–KSoLA Joint Symposium

Sep 11(Thu) 16:20–17:50 | Room 2 (3F)

Familial Hypercholesterolemia (FH) in the Era of Precision Medicine

CHAIRPERSONS : **Donghoon Choi** (Yonsei University, Republic of Korea)
Woo Je Lee (University of Ulsan, Republic of Korea)

- 16:20–16:40 **Early detection of FH: role of genetic screening and cascade testing**
Hayato Tada (Kanazawa University, Japan)
- 16:40–17:00 **Cardiovascular risk in FH: how early is early enough for intervention**
Po-Sheng Chen (National Cheng Kung University, Taiwan)
- 17:00–17:20 **Beyond statins: advances in FH management**
Chan Joo Lee (Yonsei University, Republic of Korea)
- 17:20–17:50 **Panel Discussion**
Hoyoun Won (Chung-Ang University, Republic of Korea)
Dae Young Cheon (Hallym University, Republic of Korea)
Seong Huan Choi (Inha University, Republic of Korea)
Hun Jee Choe (Hallym University, Republic of Korea)

AAS-KSoLA Joint Symposium

Sep 12(Fri) 08:30–10:00 | Room 2 (3F)

Comprehensive Strategies for the Prevention of Atherosclerosis: Current Insights and Future Directions

CHAIRPERSONS : Jae Bum Kim (Seoul National University, Republic of Korea)

Hyeong Kyu Park (Soonchunhyang University, Republic of Korea)

08:30–08:50 **Metabolic disease promotes deoxyceramide accumulation in immune cells altering cytokine production**

Andrew Murphy (Baker Heart and Diabetes Institute, Australia)

08:50–09:10 **The role of digital health technology in managing atherosclerosis**

Hojin Choi (Hanyang University, Republic of Korea)

09:10–09:30 **Impact of lipid-lowering therapy on aortic aneurysm progression and cardiovascular outcomes**

Gyu Chul Oh (The Catholic University of Korea, Republic of Korea)

09:30–10:00 **Panel Discussion**

Ung Kim (Yeungnam University, Republic of Korea)

Da Young Lee (Korea University, Republic of Korea)

Jong-Young Lee (Sungkyunkwan University, Republic of Korea)

VAS-KSoLA Joint Symposium

Sep 12(Fri) 16:45–18:15 | Room 2 (3F)

Lipid-Lowering Strategies: Targets and Therapies in Clinical Practice

CHAIRPERSONS : Ki Hoon Han (University of Ulsan, Republic of Korea)

Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea)

16:45–17:05 **Triglyceride and HDL-C focus in Asian populations: rethinking priorities?**

Jaehoon Chung (Dongguk University, Republic of Korea)

17:05–17:25 **Combined lipid-lowering therapy: when statins alone are not enough**

Chang Hee Jung (University of Ulsan, Republic of Korea)

17:25–17:45 **Ethnic differences in managing dyslipidemia: tailoring treatment strategies for Asian and Western populations**

Tien Hoang Anh (Hue University of Medicine and Pharmacy, Vietnam)

17:45–18:15 **Panel Discussion**

Hyun-Suk Yang (Konkuk University, Republic of Korea)

Jong-Young Lee (Sungkyunkwan University, Republic of Korea)

Jung Rae Cho (Hallym University, Republic of Korea)

CSATVB-KSoLA Joint Symposium

Sep 13(Sat) 08:50–10:20 | Room 2 (3F)

Long-term Exposure to LDL-cholesterol and Early Cardiovascular Risk Prevention**CHAIRPERSONS : In-Ho Chae** (Seoul National University, Republic of Korea)**Jeong Hyeon Noh** (Inje University, Republic of Korea)08:50–09:10 **Cumulative LDL-cholesterol burden and cardiovascular risk: the unsuspected role of LDL transcytosis****Warren Lee** (Unity Health Toronto and University of Toronto, Canada)09:10–09:30 **Dyslipidemia in youth: why early intervention matters for lifelong cardiovascular health****Aram Yang** (Sungkyunkwan University, Republic of Korea)09:30–09:50 **Beyond LDL-C: role of ApoB and non-HDL cholesterol in early prevention****Jae Hyoung Park** (Korea University, Republic of Korea)09:50–10:20 **Panel Discussion****Yeoree Yang** (The Catholic University of Korea, Republic of Korea)**Seung Hun Lee** (Chonnam National University, Republic of Korea)**Sang Yup Lim** (Korea University, Republic of Korea)**Jung Rae Cho** (Hallym University, Republic of Korea)**KoSFoST-KSoLA Joint Symposium**

Sep 13(Sat) 14:50–16:20 | Room 2 (3F)

Role of Medicinal Foods in Atherosclerosis and Cardiometabolic Disease**CHAIRPERSONS : Hyojee Joung** (Seoul National University, Republic of Korea)**Min-Jeong Shin** (Korea University, Republic of Korea)14:50–15:10 **Novel health functional products to prevent post-MI pathologic remodeling****Hun-Jun Park** (The Catholic University of Korea, Republic of Korea)15:10–15:30 **Ultrasonicated *Lespedeza cuneata* extract prevents TNF- α -induced early atherosclerosis in vitro and in vivo****Sung Keun Jung** (Kyungpook National University, Republic of Korea)15:30–15:50 **Regulation of mitochondrial metabolic reprogramming during aging****Jiyun Ahn** (Korea Food Research Institute, Republic of Korea)15:50–16:20 **Panel Discussion****Hyunju Kang** (Keimyung University, Republic of Korea)**Bohkyung Kim** (Pusan National University, Republic of Korea)**Dahyun Park** (Korea University, Republic of Korea)**Hyunjung Lim** (Kyung Hee University, Republic of Korea)

EAS-KSoLA Joint Symposium

Sep 13(Sat) 16:20–17:50 | Room 2 (3F)

The Impact of Remnant Cholesterol on Various Disease Pathologies and Clinical Outcomes

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

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|-------------|---|
| 16:20–16:40 | Remnant cholesterol as a key contributor to atherosclerotic cardiovascular disease (ASCVD): mechanisms and clinical implications
Alberto Zambon (University of Padua, Italy) |
| 16:40–16:55 | The role of remnant cholesterol in chronic kidney disease (CKD)
Soo Yeon Jang (Korea University, Republic of Korea) |
| 16:55–17:10 | Association between remnant cholesterol and dementia: potential mechanisms and clinical perspectives
Han Na Jung (Hallym University, Republic of Korea) |
| 17:10–17:30 | Remnant cholesterol in women's cardiometabolic health
Meral Kayikcioglu (Ege University, Türkiye) |
| 17:30–17:50 | Panel Discussion
Mi-Hyang Jung (The Catholic University of Korea, Republic of Korea)
Eun Jeong Cho (Chung-Ang University, Republic of Korea)
Ji Hye Huh (Hallym University, Republic of Korea) |

MSA-KSoLA Joint Symposium

Sep 13(Sat) 16:20–17:50 | Room 4 (5F)

Recent Evidence in Non-invasive Cardiovascular Risk Assessment Techniques

CHAIRPERSONS : **Donghoon Choi** (Yonsei University, Republic of Korea)
Sang-Hyun Lhm (The Catholic University of Korea, Republic of Korea)

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|-------------|---|
| 16:20–16:40 | Recent updates on coronary artery calcium scoring
Nor Ashikin Md Sari (Universiti Malaya, Malaysia) |
| 16:40–17:00 | The role of carotid ultrasonography for the primary prevention of cardiovascular disease
Jun Hwa Hong (Eulji University, Republic of Korea) |
| 17:00–17:20 | The usefulness of brachial-ankle PWV in the risk prediction for Asians
Hack-Lyoung Kim (Seoul National University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Jong Shin Woo (Kyung Hee University, Republic of Korea)
Hyun Sung Joh (Seoul National University, Republic of Korea)
Hyo-In Choi (Sungkyunkwan University, Republic of Korea) |

Satellite Symposia

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Breakfast Symposium 1

Sep 12(Fri) 07:30–08:00 | Room 1 (3F)

CHAIRPERSON : **Sung Rae Kim** (The Catholic University of Korea, Republic of Korea)07:30–07:50 **The cardio–renal–metabolic vicious cycle: choosing the suitable statin for complex risk profiles****Youngwoo Jang** (Gachon University, Republic of Korea)07:50–08:00 **Panel Discussion****So Hee Kwon** (Soonchunhyang University, Republic of Korea)**Woohyeun Kim** (Hanyang University, Republic of Korea)

Breakfast Symposium 2

Sep 12(Fri) 07:30–08:00 | Room 2 (3F)

CHAIRPERSON : **Hyo–Soo Kim** (Seoul National University, Republic of Korea)07:30–07:50 **Lowest is the best: reduction of LDL–C level and CV risk in ASCVD patients****Dae Young Cheon** (Hallym University, Republic of Korea)07:50–08:00 **Panel Discussion****Dong–Hwa Lee** (Chungbuk National University, Republic of Korea)**Oh–Hyun Lee** (Yonsei University, Republic of Korea)

Breakfast Symposium 3

Sep 12(Fri) 07:30–08:00 | Room 3 (3F)

CHAIRPERSON : **Do–Sun Lim** (Korea University, Republic of Korea)07:30–07:50 **Upfront lipid–lowering combination therapy in very high–risk patients: not a choice, a necessity****Kang–Un Choi** (Yeungnam University, Republic of Korea)07:50–08:00 **Panel Discussion****Keewan Lee** (University of Ulsan, Republic of Korea)**A Ram Hong** (Chonnam National University, Republic of Korea)

Breakfast Symposium 4

Sep 12(Fri) 07:30–07:54 | Room 4 (5F)

CHAIRPERSON : **Myung A Kim** (Seoul National University, Republic of Korea)07:30–07:42 **New concepts of dyslipidemia medication: the lower, the smaller****Wonjin Kim** (CHA University, Republic of Korea)07:42–07:54 **Meeting an unmet need: low–dose atorvastatin****Doyeon Hwang** (Seoul National University, Republic of Korea)

Breakfast Symposium 5

Sep 13(Sat) 07:50–08:20 | Room 1 (3F)

CHAIRPERSON : Jeong Euy Park (Drs Park & Kim Heart and Lung International Clinic, Republic of Korea)

07:50–08:10 **Benefits of atorvastatin / ezetimibe combination in diabetic dyslipidemia**

Yun Kyung Cho (University of Ulsan, Republic of Korea)

08:10–08:20 **Panel Discussion**

In Tae Moon (Eulji University, Republic of Korea)

Eunshil Hong (National Medical Center, Republic of Korea)

Breakfast Symposium 6

Sep 13(Sat) 07:50–08:35 | Room 2 (3F)

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

Ade Meidian Ambari (Universitas Indonesia, Indonesia)

07:50–08:05 **Minimizing LDL–C variability via sustained, long–term efficacy**

Youngwoo Jang (Gachon University, Republic of Korea)

08:05–08:20 **The lower the better: Rosuvastatin's next option for dyslipidemia management**

Jina Choi (Seoul National University, Republic of Korea)

08:20–08:25 **Panel Discussion**

Bambang Widyanoro (Universitas Indonesia, Indonesia)

08:25–08:35 **MOU Ceremony – The Indonesian Heart Association (IHA) & The Korean Society of Lipid and Atherosclerosis (KSoLA)**

Breakfast Symposium 7

Sep 13(Sat) 07:50–08:20 | Room 3 (3F)

CHAIRPERSON : Kyong Soo Park (Konkuk University, Republic of Korea)

07:50–08:10 **Safety and efficacy of moderate–intensity statin with ezetimibe in elderly patient with ASCVD**

Jung–Joon Cha (Korea University, Republic of Korea)

08:10–08:20 **Panel Discussion**

Shin–young Woo (Konyang University, Republic of Korea)

Kyu–Sun Lee (Eulji University, Republic of Korea)

Breakfast Symposium 8

Sep 13(Sat) 07:50–08:26 | Room 4 (5F)

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

07:50–08:02 **New approaches to treating hypertriglyceridemia in patients with renal impairment**

Young–Sang Lyu (Chosun University, Republic of Korea)

08:02–08:14 **Profile of Pravastatin and its effects in transplant recipients**

Jae Yoon Park (Dongguk University, Republic of Korea)

08:14–08:26 **Comparative effectiveness of lercanidipine and amlodipine on MACE in hypertensive patients**

Hyung Joon Joo (Korea University, Republic of Korea)

Luncheon Symposium 1 (K)

Sep 12(Fri) 12:00–12:30 | Room 1 (3F)

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

12:00–12:20 **Integrated management of hypertension and dyslipidemia for cardiovascular disease prevention**

Chan Joo Lee (Yonsei University, Republic of Korea)

12:20–12:30 **Panel Discussion**

Ye An Kim (Veterans Health Service Medical Center, Republic of Korea)

Hanbit Park (University of Ulsan, Republic of Korea)

Luncheon Symposium 2

Sep 12(Fri) 12:00–12:30 | Room 2 (3F)

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

12:00–12:20 **A new “standard” in dyslipidemia treatment**

Jeehoon Kang (Seoul National University, Republic of Korea)

12:20–12:30 **Panel Discussion**

Soo Kyoung Kim (Gyeongsang National University, Republic of Korea)

Subin Lim (Ewha Womans University, Republic of Korea)

Luncheon Symposium 3

Sep 12(Fri) 12:00–12:30 | Room 3 (3F)

CHAIRPERSON : Chee Jeong Kim (Chung-Ang University, Republic of Korea)

12:00–12:20 **Recent trends and clinical evidence for optimizing lipid management**

Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

12:20–12:30 **Panel Discussion**

Sang Ah Lee (Jeju National University, Republic of Korea)

Sungsoo Cho (Yonsei University, Republic of Korea)

Luncheon Symposium 4

Sep 12(Fri) 12:00–12:30 | Room 4 (5F)

CHAIRPERSON : Shung Chull Chae (Kyungpook National University, Republic of Korea)

12:00–12:20 **Closing the LDL–C gap in ASCVD: the role of siRNA therapy**

Jung Ho Heo (Kosin University, Republic of Korea)

12:20–12:30 **Panel Discussion**

Youngsook Kim (Soonchunhyang University, Republic of Korea)

Sungjoon Park (Seoul National University, Republic of Korea)

Luncheon Symposium 5 (K)

Sep 13(Sat) 12:20–12:50 | Room 1 (3F)

CHAIRPERSON : Kwang-Won Kim (Gachon University, Republic of Korea)

12:20–12:40 **Cutting edge care of pitavastatin with ezetimibe combination therapy**

Soo Lim (Seoul National University, Republic of Korea)

12:40–12:50 **Panel Discussion**

Ji Yoon Kim (Sungkyunkwan University, Republic of Korea)

Hyun Sung Joh (Seoul National University, Republic of Korea)

Luncheon Symposium 6

Sep 13(Sat) 12:20–12:50 | Room 2 (3F)

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

12:20–12:40 **Clinical efficacy of ultra-low-dose triple combination therapy (Amlodipine/Losartan/Chlorthalidone) in hypertension management**

Kyu-Yong Ko (Inje University, Republic of Korea)

12:40–12:50 **Panel Discussion**

Yu Ji Kim (Jeonbuk National University, Republic of Korea)

Ki Hong Choi (Sungkyunkwan University, Republic of Korea)

Luncheon Symposium 7

Sep 13(Sat) 12:20–12:50 | Room 3 (3F)

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

12:20–12:40 **Reversing atherosclerosis – 20 years of data for CRESTOR**
Kausik Kumar Ray (Imperial Centre for Cardiovascular Disease Prevention, UK)

12:40–12:50 **Panel Discussion**
Seokhun Yang (Seoul National University, Republic of Korea)

Kyong Hye Joung (Chungnam National University, Republic of Korea)

Oral Presentations

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Oral Presentation 1

Sep 11(Thu) 14:40–16:10 | Room 1 (3F)

CHAIRPERSONS : Hyun Kook (Chonnam National University, Republic of Korea)

Kyung Woo Park (Seoul National University, Republic of Korea)

OP1-1 ApoA1 and HDL selectively protect the heart during doxorubicin chemotherapy via hepatic SR-B1

Jeong-Ah Yoo^{1,2*}, Bernardo Trigatti^{1,2}
¹Department of Biochemistry and Biomedical Sciences, McMaster University, Hamilton, Ontario, Canada, ²Thrombosis and Atherosclerosis Research Institute, Hamilton Health Sciences and McMaster University, Hamilton, Ontario, Canada

OP1-2 Lipid profiling of plasma extracellular vesicles from adiponectin deficient mice reveals pro-oxidative and pro-inflammatory lipid signatures driving metabolic dysfunction

Sungji Cho^{1*}, Hyekyoung Sung¹, Khang Nguyen¹, Yubin Lei¹, Eddie Tam¹, Yihan Luo², Evgeniy V. Petrotchenko³, Kacey J. Prentice², Dylan Burger^{4,5}, Gary Sweeney¹
¹Department of Biology, York University, Canada, ²Department of Physiology, University of Toronto, Canada, ³Segal Cancer Proteomics Centre, Lady Davis Institute for Medical Research, Jewish General Hospital, McGill University, Canada, ⁴Kidney Research Centre, Inflammation and Chronic Disease Program, Ottawa Hospital Research Institute, Canada, ⁵Department of Cellular and Molecular Medicine and School of Pharmaceutical Sciences, University of Ottawa, Canada

OP1-4 Atherosclerosis associated with HIV - is it a result of treatment or inflammation

Nuriyat Efendieva*, Alexey Sozykine, Oleg Shevchenko

Cardiology, Pirogov Russian National Research Medical University, Russian Federation

OP1-5 Anti-atherosclerotic effect of thymol extract via regulating inflammation and oxidative stress and EGFR/PI3K/Akt/GSK-3 β pathway in rats

Manvendra Singh^{2*}, Niti Singh³, Deepika Singh¹
¹Maa Gange Research Foundation, Kashi, India, ²Computer Sciences, HMFA-MIET, India, ³Basic Sciences, CSVTU, Bhilai, India

OP1-6 PCSK9 deficiency promotes ANP-mediated protection against post-infarction cardiac inflammation

NaHyeon Yoon*, GooTaeg Oh

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

- OP1-7** Targeting LIG3 attenuates atherosclerosis by modulating inflammatory responses and plaque development
Jing Jin*, Do Hee Yu, Goo Taeg Oh
Department of Life Science, Heart-Immune-Brain Network Research Center, Ewha Womans University, Republic of Korea
- OP1-8** Proteomic profiling of inflammatory and non-lipid determinants of plaque instability in myocardial infarction
Monirujjaman Biswas*
Special Centre for Molecular Medicine, Jawaharlal Nehru University, India
- OP1-9** The role of NLRP3 inflammasome activation in atherosclerosis: a systematic review and meta-analysis
Alaa Ramadan^{1*}, Esraa Abdelhazf², Mostafa A Soliman³
¹Internal Medicine, Faculty of Medicine, South Valley University, Qena, Egypt, ²Pharmacy, Modern University for Technology and Information, Egypt, ³Internal Medicine, Cairo University, Cairo, Egypt

Oral Presentation 2

Sep 12(Fri) 13:00–14:30 | Room 1 (3F)

CHAIRPERSONS : Ju Han Kim (Chonnam National University, Republic of Korea)
Ung Kim (Yeungnam University, Republic of Korea)

- OP2-1** Protective effect of biofabricated curcumin silver nanoparticles against atherosclerosis in rodent model via modulating EGFR/PI3K/Akt/GSK-3 β signaling pathway
Yadav E*
Shalom Institute of Health and Allied Sciences, SHUATS, India
- OP2-2** Extracellular vesicles produced by IL4-polarized macrophages drive atherosclerosis stabilization by reprogramming Ly-6Chi monocytes & enhancing efferocytosis
Martin Ng^{1*}, Ngan Vu¹, Tuan Anh Phu¹, Alex S Gao¹, Robert L Raffai^{1,2}
¹Surgery, UCSF, NCIRE, United States, ²SF VA Medical Center, United States
- OP2-3** Alteration of lipid gene modulation and gut microbiota by gallic acid loaded liposomes in atherosclerosis in apolipoprotein-E deficient mice
Deepika Singh*
Maa Gange Research Foundation, Kashi, India
- OP2-4** Candidates of STAT3-mediated miRNAs in LPS-induced mouse endothelium
Lan Phuong Phan*, Yujin Jin, Kyung-Sun Heo
College of Pharmacy, Chungnam National University, Republic of Korea
- OP2-5** KAI1 on perivascular cells: a key switch regulator of angiogenesis
Taehun Yoon^{1*}, Yoo-Wook Kwon²
¹Molecular Medicine and Biopharmaceutical Sciences, Seoul National University, Republic of Korea, ²Biomedical Research Institute, Seoul National University Hospital, Republic of Korea

OP2-6 **Distribution of LDL-cholesterol and its association with cardiovascular outcomes in young adults under 40 years: a nationwide cohort study**

You-Bin Lee^{1*}, Kyu-Na Lee², Kyungdo Han³

¹Division of Endocrinology and Metabolism, Department of Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine, Republic of Korea, ²Department of Public Health, Catholic University, Republic of Korea, ³Department of Statistics and Actuarial Science, Soongsil University, Republic of Korea

OP2-7 **Lipoprotein(a) variability and risk of major adverse cardiovascular event: insights from a real-world multicenter cohort**

Mi-Na Kim^{1*}, Soon Jun Hong¹, Cheol Woong Yu¹, Seung Yong Shin², Eung Ju Kim³, Hyung Joon Joo¹

¹Cardiology, Korea University Anam Hospital, Republic of Korea, ²Cardiology, Korea University Ansan Hospital, Republic of Korea, ³Cardiology, Korea University Guro Hospital, Republic of Korea

OP2-8 **Discontinuation and non-publication of atherosclerosis clinical studies: a cross-sectional analysis**

Yousef Radwan Alnomani^{1*}, Omnia Samy El-Sayed², Mohamed Mohsen Helal², Menna Marwan⁴, Mazen Negmeldin Aly Yassin⁵, Mohamed Karam Allah Elkholy⁶, Mona Mohsen Nasr Abdelaziz³, Ahmed Abdelaziz⁷

¹Faculty of Medicine, Benha University, Egypt, ²Faculty of Medicine, Zagazig University, Egypt, ³Faculty of Medicine, Minia University, Egypt, ⁴Faculty of Medicine, Port Said University, Egypt, ⁵Faculty of Medicine, Cairo University, Egypt, ⁶Faculty of Pharmacy, Zagazig University, Egypt, ⁷Cardiology, Montefiore Health System/Albert Einstein College of Medicine, Bronx, NY, United States

OP2-9 **Association between dietary antioxidant intake and cardiovascular diseases risk factors in Singapore individuals with eczema**

Liang Yuxin^{1*}, Hu Weili², Amelia Chng³, Jung Eun Kim^{1,4}

¹Nutrition within Food science and technology, National University of Singapore, Singapore, ²Ocular science, Centre for Eye and Vision Research (CEVR) in Hong Kong, China, ³Bioinformatics, A*STAR, Singapore, ⁴Bezos Center for Sustainable Protein, National University of Singapore, Singapore

Oral Presentation 3

Sep 13(Sat) 08:50–10:20 | Room 1 (3F)

CHAIRPERSONS : Dae Jung Kim (Ajou University, Republic of Korea)

Soo Lim (Seoul National University, Republic of Korea)

OP3-1 **Statin use and site-specific cancer risk: a nationwide cohort study using Korean health insurance data**

Yun Kyung Cho^{1*}, Jihoon Seo¹, Jung Yoon Moon¹, Hee Sung Kim¹, Ye Jee Kim², Chang Hee Jung¹, Woo Je Lee¹

¹Department of Internal Medicine, Asan Medical Center, University of Ulsan, College of Medicine, Republic of Korea, ²Department of Clinical Epidemiology and Biostatistics, Asan Medical Center, University of Ulsan College of Medicine, Republic of Korea

OP3-2 **Association between circulating trimethylamine N-oxide levels and carotid perivascular fat density in acute ischemic stroke patients**

Eung-Joon Lee^{*}

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

- OP3-3** **The relationship of inflammatory markers with the development of coronary artery calcification: a multicenter, longitudinal cohort study**
- Kyung An Kim^{1,2*}, Mi-Jeong Kim¹, So-Young Lee², Donghee Han³, Dong-Hyeon Lee², Su-Yeon Choi⁵, Jidong Sung⁴, Eun Ju Chun⁵, Hyuk-Jae Chang⁶, Hae-Ok Jung²
- ¹Cardiology, Incheon St. Mary's Hospital, Republic of Korea, ²Cardiology, Seoul St. Mary's Hospital, Republic of Korea, ³Imaging, Cedars-Sinai Medical Center, Republic of Korea, ⁴Cardiology, Samsung Medical center, Republic of Korea, ⁵Radiology, Seoul National University Bundang Hospital, Republic of Korea, ⁶Cardiology, Severance Hospital, Republic of Korea
- OP3-4** **Saffron as a natural statin alternative: in silico modulation of MMP-9 and TIMP-1 for plaque stability**
- Iman Nabilah Abd Rahim^{1*}, Yasmin Mohd Zainal Abidin Shukri², Nur Aqasyah Amran², Opik Taupiqurrohman³
- ¹Faculty of Medicine, Universiti Teknologi MARA, Malaysia, ²Cardiovascular Advancement and Research Excellence Institute, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, ³Biotechnology Doctoral Program Graduate School, Universitas Padjadjaran, Indonesia
- OP3-5** **Comparative outcomes of moderate-intensity statin with ezetimibe vs high-intensity statin therapy: a retrospective observational cohort study**
- Junpil Yun^{*}, Seokhun Yang, Doyeon Hwang, Hyun-Jae Kang, Jeehoon Kang, Bon-Kwon Koo, Kyung Woo Park, Han-Mo Yang, Jung-Kyu
- Cardiology, Seoul National University Hospital, Republic of Korea
- OP3-6** **Effectiveness and safety of very low-dose rosuvastatin-ezetimibe therapy in dyslipidemia: a multicenter prospective observational study**
- SungA Bae^{1*}, Ji Woong Roh¹, Ji-won Son²
- ¹Division of Cardiology, Department of Internal Medicine, Yongin Severance Hospital, Yonsei University College of Medicine, Republic of Korea, ²Department of Internal Medicine, Son Ji-Won Internal Medicine Clinic, Republic of Korea
- OP3-7** **Clinical outcomes of high intensity lipid lowering agents for patients after complex PCI**
- Youngkwan Kim^{*}, Doyeon Hwang, Jeehoon Kang, Han-Mo Yang, Kyung Woo Park, Hyun-Jae Kang, Bon-Kwon Koo, Hyo-Soo Kim, Jung-Kyu Han
- Cardiovascular Center, Seoul National University Hospital, Republic of Korea
- OP3-8** **Effect of omega-3 fatty acid supplementation on liver function and lipid profile in tobacco Users: a randomized placebo-controlled trial**
- Anjali Singh^{1*}, Shraddha Singh¹, Narsingh Verma²
- ¹Physiology, King Georges Medical University, India, ²Physiology, Hind Institute of Medical Sciences, India
- OP3-9** **Metabolomic patterns of dietary protein intake and their link to cardiometabolic risk: a systematic review and meta-analysis**
- Nicole Min Yee Wong^{1*}, Marcus Ting¹, Jung Eun Kim^{1,2}
- ¹Department of Food Science and Technology, National University of Singapore, Singapore, ²Bezos Center for Sustainable Protein, National University of Singapore, Singapore

Moderated Poster Presentations

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Moderated Poster Presentation 1

Sep 12(Fri) 14:40–15:40 | Studio 5 (6F)

CHAIRPERSON : Jeong–Min Kim (Seoul National University, Republic of Korea)

MPP-01 Hepatic lipase silencing promotes cholesterol excretion with activation of hepatobiliary sterol transportHyeon Ji Lee^{1*}, Soo–Jin Ann², Sang–Hak Lee³¹Graduate school, Yonsei University, Republic of Korea, ²Integrative Research Center for Cerebrovascular and Cardiovascular Diseases, Yonsei University, Republic of Korea, ³Division of Cardiology, Department of Internal Medicine, Yonsei University College of Medicine, Republic of Korea**MPP-02** Cholesterol regulating factor X(CRFX) deficiency exacerbates hypercholesterolemia–induced liver injury by disrupting bile acid synthesis and promoting hepatic cholesterol accumulationHaeun Jang^{1*}, Min ji Cho³, Hye Rang Park^{1,2}, Young Hoon Seo^{1,2}, Yongmin Lee¹, Jimin Lee^{1,2}, Daeun Kim¹, Jong–Gil Park^{1,2}¹Biotherapeutics Translational Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, ²Department of Bioscience, KRIBB School of Bioscience, Korea University of Science and Technology (UST), Republic of Korea, ³Center for Gene and Cell Therapy, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea**MPP-03** The role of cereblon in mitochondrial function and metabolic reprogramming in the aging heartTrong Kha Pham^{1,2*}, Hyeong Rok Yun¹, Hoai T.T. Nguyen¹, Hyoung Kyu Kim¹, Jin Han¹¹Department of Physiology, Cardiovascular and Metabolic Disease Center, Inje University, Republic of Korea, ²Department of Physiology, VNU University, Vietnam National University, Hanoi, Vietnam**MPP-04** Quail egg yolks has an effects on body weight, lipid profiles and liver tissue injury of rats (*rattus norvegicus*)Rafik Prabowo^{1,2*}, Miranti Dewi Pramaningtyas³¹General Practitioner, Community Health Center of Rambah, Rokan Hulu, Indonesia, ²Alumnus of The Faculty of Medicine, Universitas Islam Indonesia, Indonesia ³Department of Physiology, Faculty of Medicine, Universitas Islam Indonesia, Indonesia**MPP-05** Lipoprotein(a) and long-term cardiovascular events in patients with coronary vasospasmMelly Susanti^{1,2*}, Seung–Woon Rha¹¹Cardiovascular Centre, Korea University Guro Hospital, Republic of Korea, ²Cardiovascular Centre, Ministry of Health Central General Surabaya Hospital, Indonesia**MPP-06** Exploring novel lncRNA regulators of macrophage mediated inflammation in atherosclerosis

Dharmendra Kumar Khatri*

Health Sciences Center School of Pharmacy, Texas Tech University, United States

MPP-07 Effects of omega-3 fatty acid supplementation on chylomicron remnant concentrations and vascular inflammatory biomarkers: a systematic review

Afifah Az Zahra^{1*}, Ana Fauziyati², Afifah Az Zahra¹

¹Lecturer Faculty of Medicine, Islamic University of Indonesia, Indonesia, ²Internal Medicine, Islamic University of Indonesia, Indonesia

MPP-08 Cardioprotective effects of β -Lapachone through alleviation of lipotoxicity and fibrosis in type 2 diabetic mice

Nguyen Thi To Hoai^{1*}, Bui Van Nam¹, Pham Trong Kha^{1,2}, Luu Thi Thu Phuong², Vu Thi Thu², Hyoung Kyu Kim¹, Han Jin¹

¹Department of Physiology, Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutic Center, College of Medicine, Inje University, Busan, Republic of Korea, ²Department of Biology, VNU University of Science, Vietnam National University, Hanoi, Vietnam

Moderated Poster Presentation 2

Sep 13(Sat) 13:30–14:30 | Studio 5 (6F)

CHAIRPERSON : Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

MPP-09 Association between COVID-19 and cardiovascular disease through small extracellular vesicles

Hyae Yon Kweon^{*}, Goo Taeg Oh

Life Science, Ewha Womans University, Republic of Korea

MPP-10 Trained immunity (intolerant responses) in patients with atherosclerosis and other non-infectious diseases

Nikolai A. Orekhov^{1*}, Alexander N. Orekhov¹, Anastasia V. Poznyak¹, Natalia V. Elizova¹, Nikolay A. Orekhov^{1,2}

¹Institute for Atherosclerosis Research, Russian Federation, ²Lomonosov Moscow State University, Moscow, Russian Federation

MPP-11 LDLR-independent role of PCSK9 in post-myocardial infarction inflammatory response

Yun Seo Noh^{*}, Na Hyeon Yoon, Goo Taeg Oh

Life science, Ewha Womans University, Republic of Korea

MPP-12 A potential rejuvenation factor C improves metabolic dysfunctions and mitigates atherosclerosis in Ldlr KO mice

Hye Rang Park^{1,3*}, Young Hoon Seo^{1,3}, Jimin Lee^{1,3}, Yongmin Lee¹, Haeun Jang¹, Daeun Kim¹, Min ji Cho², Jong-Gil Park^{1,3}

¹Biotherapeutics Translational Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, ²Center for Gene and Cell Therapy, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, ³Department of Bioscience, KRIBB School of Bioscience, Korea University of Science and Technology (UST), Republic of Korea

MPP-13 Potential rejuvenation factor C overexpression as a novel anti-senescence therapeutic target for atherosclerosis prevention

Yongmin Lee^{1*}, Hye Rang Park^{1,3}, Young Hoon Seo^{1,3}, Jimin Lee^{1,3}, Haeun Jang¹, Daeun Kim¹, Min ji Cho², Jong-Gil Park^{1,3}

¹Biotherapeutics Translational Research Center, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, ²Center for Gene and Cell Therapy, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Republic of Korea, ³Department of Bioscience, KRIBB School of Bioscience, Korea University of Science and Technology (UST), Republic of Korea

MPP-14 Transcriptomic and network-based analysis reveals inflammatory and matrix-degrading pathways in atherosclerotic plaque instability

Nadyatul Husna*

Internship Doctor, dr. Reksodiwiryo Military Hospital Padang, Indonesia

MPP-15 Short variant of mitochondrial calcium uniporter forms plasma-membrane channels in human platelets

Bong Sook Jhun*, Jin O-Uchi

University of South Florida Morsani College of Medicine, United States

MPP-16 CBL-b E3 ligase-mediated neddylation and activation of PARP-1 induce vascular calcification

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

Pharmacology, Chonnam National University Medical School, Republic of Korea

Moderated Poster Presentation 3

Sep 12(Fri) 14:40–15:40 | Studio 6 (6F)

CHAIRPERSON : Jaehoon Chung (Dongguk University, Republic of Korea)

MPP-17 Adherence to statins: a cornerstone for preventing atherosclerotic cardiovascular disease and mortality

Sambit Kumar Pradhan*

LifeScience, National Institute of Technology, Rourkela, India

MPP-18 PCSK7 drives post-infarction inflammation via TNF- α /JNK activation in cardiac macrophages

Shin Hye Moon*, Inyoung Chung, Goo Taeg Oh

Life science, Ewha Womans University, Republic of Korea

MPP-19 Corticosterone mitigates stroke-induced injury via upregulation of antioxidant Prdx1

Huiju Jo*, Goo Taeg Oh

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

MPP-20 Pharmacological assessment of low-dose oral docetaxel for atheroprotection

Senna Choi*, Guanhan Yao, Hong Choi, Isabelle Ruel, Iulia Iatan, Jacques Genest

Cardiology, The Research Institute of the McGill University Health Centre, Canada

MPP-21 The Interplay between oxidative stress and cholesterol in non-alcoholic steatohepatitis (NASH): a key to understanding disease pathogenesis

Hina Parveen^{1*}, Mohammad Kaleem Ahmad², Nasreen Ghazi Ansari³, Sumit Rungta¹

¹Department of Gastroenterology, King George's Medical University, Lucknow, India, ²Department of Biochemistry, King George's Medical University Lucknow, India ³Department of Regulatory Toxicology, Indian Institute of Toxicology Research Lucknow, India

MPP-22 Moringa oleifera root extracts reduces the apoptotic activity in pancreatic Langerhans cells of wistar rats induced metabolic syndrome

Shafira Yasmine Anshari^{1*}, Dyah Ratna Budiani², Novan Adi Setyawan², Riza Noviarta Pesik²

¹Faculty of Medicine, Universitas Sebelas Maret, Indonesia, ²Pathology Anatomy Department, Universitas Sebelas Maret, Indonesia

Moderated Poster Presentation 4

Sep 13(Sat) 13:30–14:30 | Studio 6 (6F)

CHAIRPERSON : Jun Hwa Hong (Eulji University, Republic of Korea)

MPP-26 Different effect of ACE-I vs ARB on TNF- α , IL-1 β , and IL-6 level in type-4 cardio-renal syndrome secondary to diabetic kidney disease

Marcelino Adiska Megantara^{1,2*}

¹Psychosomatic and Palliative Medicine, Cipto Mangunkusumo Hospital, Indonesia, ²Emergency Medicine, Sleman Regional Hospital, Indonesia

MPP-27 Factors associated with vertebral metabolic activity and its implications for cardiovascular disease

Jiyeona Ha^{1*}, Jin Chul Paeng², Wookjin Yang³, Yong Seo Kim⁴, Soo Jin Lee⁵, Ji Young Kim⁵, Yun Young Choi⁵, Kang-Ho Choi⁶, Jahae Kim⁷, Kwang-Yeol Park⁸, Juwon Seok⁹, Jeong-Min Kim¹

¹Department of Neurology, Seoul National University Hospital, Republic of Korea, ²Department of Nuclear Medicine, Seoul National University Hospital, Republic of Korea, ³Department of Neurology, Asan Medical Center, Republic of Korea, ⁴Department of Neurology, Hanyang University School of Medicine, Republic of Korea, ⁵Department of Nuclear Medicine, Hanyang University School of Medicine, Republic of Korea, ⁶Department of Neurology, Chonnam National University Hospital, Republic of Korea, ⁷Department of Nuclear Medicine, Chonnam National University Hospital, Republic of Korea, ⁸Department of Neurology, Chung-Ang National University Hospital, Republic of Korea, ⁹Department of Nuclear Medicine, Chung-Ang National University Hospital, Republic of Korea

MPP-28 Cardiovascular health by life's essential 8 and chronic kidney disease: Korea national health and nutrition examination survey 2019–2021

Enji Kim^{1,2*}, Yeeun Seo³, Dasom Son³, Kyoung Hwa Ha^{4,5,6}, Hyeon Chang Kim^{4,5}, Jong Hyun Jhee⁷, Hokyoo Lee^{4,5}

¹Department of Preventive Medicine, Gachon University College of Medicine, Republic of Korea, ²Artificial Intelligence and Big-Data Convergence Center, Gachon University Gil Medical Center, Republic of Korea, ³Department of Public Health, Yonsei University Graduate School, Republic of Korea, ⁴Department of Preventive Medicine, Yonsei University College of Medicine, Republic of Korea, ⁵Institute for Innovation in Digital Healthcare, Yonsei University, Republic of Korea, ⁶Department of Endocrinology and Metabolism, Ajou University School of Medicine, Republic of Korea, ⁷Division of Nephrology, Department of Internal Medicine, Gangnam Severance Hospital, Yonsei University College of Medicine, Republic of Korea

MPP-29 Repeated non-invasive limb ischemic preconditioning protects against myocardial ischemia-reperfusion injury in type 1 diabetic rats via KLK12 signaling activation

Yuxin Jiang^{1,2*}, Zhengyuan Xia², David Cai¹

¹Department of Health Technology and Informatics, The Hong Kong Polytechnic University, China, ²Anaesthesiology, Guandong Medical University, China

MPP-30 Novel mechanism of KAI1 in angiogenesis inhibition: expression in PVCs and VEGF

Taehun Yoon^{1*}, Yoo-Wook Kwon²

¹Molecular Medicine and Biopharmaceutical Sciences, Seoul National University, Republic of Korea, ²Biomedical Research Institute, Seoul National University Hospital, Republic of Korea

MPP-31 Hidden cardiovascular risks in cancer survivors: atherosclerosis-related mortality trends with implications for lipid-centric surveillance (population based study)

Ahmed Almezaine*, Ibrahim Elbably

Cardiology, Tanta University Hospitals, Egypt

MPP-32 Machine-learning-assessed abdominal aortic calcification and the brain: associations with brain imaging markers and incident dementia in the UK Biobank

Carlos J. Toro-Huamanchumo^{1,2,3*}, Blossom CM Stephan⁴, Mario Siervo^{4,5}, Zhongyang Guan^{4,5}, Afsah Saleem^{1,6}, Syed Zulqarnain Gilani^{1,6,7}, John T. Schousboe^{8,9}, William D. Leslie¹⁰, Nicholas C. Harvey^{11,12}, Joshua R. Lewis^{1,13}, Marc Sim^{1,13}

¹Nutrition & Health Innovation Research Institute, School of Medical and Health Sciences, Edith Cowan University, Australia, ²OBEMET Center for Obesity and Metabolic Health, Peru, ³Research Unit for Health Evidence Generation and Synthesis, Universidad San Ignacio de Loyola, Peru, ⁴Dementia Centre of Excellence, enAble Institute, Curtin University, Australia, ⁵School of Population Health, Curtin University, Australia, ⁶Centre for AI&ML, School of Science, Edith Cowan University, Australia, ⁷Computer Science and Software Engineering, The University of Western Australia, ⁸Park Nicollet Clinic and HealthPartners Institute, HealthPartners, United States, ⁹Division of Health Policy and Management, University of Minnesota, United States, ¹⁰Departments of Medicine and Radiology, University of Manitoba, Canada, ¹¹MRC Lifecourse Epidemiology Centre, University of Southampton, United Kingdom, ¹²NIHR Southampton Biomedical Research Centre, University of Southampton and University Hospital Southampton NHS Foundation Trust, United Kingdom, ¹³Medical School, The University of Western Australia, Australia

Moderated Poster Presentation 5

Sep 12(Fri) 14:40–15:40 | Studio 7 (6F)

CHAIRPERSON : Hyun-Jin Kim (Hanyang University, Republic of Korea)

MPP-33 Global trends of cardiovascular burden and its metabolic risk factors in adolescent and young adult in Southeast Asia: insights from the global burden of disease study

Haidar Ali Hamzah^{1*}, Salma Rasyidah¹, Aulia Budi Agustin³, Pramudito Cahyo Januaryadi¹, Muhammad Patara Aulia Putra¹, Luhur Pribadi², Margono Gatot Suwandi², Muhammad Fakhri Eliansyah Putra⁴

¹Department of Emergency, Indonesia Air Force Center Hospital (RSPAU) dr. S. Hardjolukito, Yogyakarta, Indonesia, ²Department of Cardiology, Indonesia Air Force Center Hospital (RSPAU) dr. S. Hardjolukito, Yogyakarta, Indonesia, ³Department of Emergency, Gadjah Mada University Hospital, Sleman, Yogyakarta, Indonesia, ⁴Department of Medicine, Sriwijaya University, Indonesia

MPP-34 Disease burden of cardiovascular disease and metabolic risk factors among elderly in Asian population: a systematic analysis from the global burden of disease study 2021

Putri Mahirah Afladhanti^{1*}, Haidar Ali Hamzah², Muhammad Fakhri Eliansyah Putra³

¹Family Medicine, Universitas Sriwijaya, Indonesia, ²Department of Emergency, Indonesia Air Force Center Hospital (RSPAU) dr. S. Hardjolukito, Yogyakarta, Indonesia, ³Medical Profession, Universitas Sriwijaya, Indonesia

MPP-35 Burden of systemic inflammation and associated health outcomes in adults with atherosclerotic cardiovascular disease managed in routine care

KS Won^{1*}, F. Mazhar², A.L. Faucon², E.L. Fu², K.E. Szummer³, J. Mathisen⁴, S. Gerward⁴, S.B. Reuter⁴, N. Marx⁵, R. Mehran⁶, J.J. Carrero²

¹Novo Nordisk Pharma Korea Ltd, Seoul, Republic of Korea, ²Karolinska Institute, Department of Medical Epidemiology and Biostatistics, Stockholm, Sweden, ³Karolinska Institute, Department of Cardiology, Stockholm, Sweden, ⁴Novo Nordisk, Copenhagen, Denmark, ⁵RWTH Aachen University, Department of Internal Medicine I, Aachen, Germany, ⁶Mount Sinai School of Medicine, New York, United States

- MPP-36** Insulin resistance assessed by the triglyceride-glucose index and left-ventricular function in non-diabetic ST-elevation myocardial infarction patients: an echocardiographic study from the UK Biobank
Javad Alizargar*
Medicine, Kashan University, Iran
- MPP-37** Comparative outcomes of drug-coated balloon versus uncoated balloon angioplasty in the treatment of femoropopliteal artery disease
Ulil Albab Habibah*
Faculty of Medicine, Islamic University Indonesia, Indonesia
- MPP-38** Predictors of significant high-sensitivity C-reactive protein re-reduction after use of rosuvastatin/amlodipine and atorvastatin/amlodipine
Haewon Jung*, Jaeyong Lee
Cardiology, Daegu Catholic Medical Center, Republic of Korea
- MPP-39** Vasoprotective effect of moringa oleifera root ethanolic extract on thrombospondin-1 expression in aortic tissue of metabolic syndrome-induced Wistar rats
Salman Alfarys^{1*}, Riza Novierta Pesik², Endang Listyaningsih³, Dyah Ratna Budiani²
¹Faculty of Medicine, Sebelas Maret University, Indonesia, ²Department of Pathology Anatomy, Sebelas Maret University, Indonesia, ³Department of Histology, Sebelas Maret University, Indonesia

Moderated Poster Presentation 6

Sep 13(Sat) 13:30–14:30 | Studio 7 (6F)

CHAIRPERSON : Jong Shin Woo (Kyung Hee University, Republic of Korea)

- MPP-40** Effects of cilostazol on prognosis of peripheral arterial disease in patients with diabetes mellitus in Korea: a nationwide population-based study
Shinje Moon^{1*}, Sangmo Hong², Kyungdo Han³, Cheol-Young Park⁴
¹Department of Internal Medicine, College of Medicine, Hanyang University, Republic of Korea, ²Department of Internal Medicine, Guri Hospital, College of Medicine, Hanyang University, Republic of Korea, ³Department of Statistics and Actuarial Science, Soongsil University, Republic of Korea, ⁴Department of Internal Medicine, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Republic of Korea
- MPP-41** Associations between multiple levels of advanced glycation end products and cardiovascular disease risk factors in Singapore older adults
Marcus Ting^{1*}, Ian En Kai Mak¹, Yueying Yao¹, Clarinda Nataria Sutanto¹, Zi Ning Leong¹, Chin Meng Khoo², Jung Eun Kim^{1,3}
¹Department of Food Science and Technology, National University of Singapore, Singapore, ²Department of Medicine, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, ³Bezos Center for Sustainable Protein, National University of Singapore, Singapore
- MPP-42** Effects of portulaca oleracea L. extract on inflammation and mitochondrial biogenesis in rats fed a high-fat diet
Jumi Lee^{1,2*}, Mak-Soon Lee¹, Minji Kim^{1,2}, Gayoung Kim^{1,2}, Jungeun Kim^{1,2}, Yangha Kim^{1,2}
¹Department of Nutritional Science and Food Management, Ewha Womans University, Republic of Korea, ²Graduate Program in System Health Science and Engineering, Ewha Womans University, Republic of Korea

MPP-43 Impact of gut microbiota-based therapies on cardio-metabolic risk factors induced by atypical antipsychotics: a network meta-analysis

Youssef Soliman^{1*}, Nada Ibrahim Hendi², John Magdy³, Moamen Emara⁴, Nour Maher⁵

¹Faculty of Medicine, Assiut University, Egypt, ²Faculty of Medicine, Ain Shams University, Egypt, ³Faculty of Medicine, Menofia University, Egypt, ⁴Faculty of Medicine, PortSaid University, Egypt, ⁵Faculty of Medicine, Alexandria University, Egypt

MPP-44 Ellagic acid alleviates MASLD by reducing hepatic lipogenesis and fibrosis in cholesterol-fed apoE-deficient mice

Yulim Choi^{1*}, Eunhyeong Kim¹, Sin-Hye Park¹, Young-Hee Kang¹, Sookyoung Jeon²

¹Department of Food and Nutrition, Hallym University, Republic of Korea, ²Department of Food and Nutrition, Kookmin University, Republic of Korea

MPP-45 Adiponectin, dyslipidemia, nutritional indices and atherogenic indices in relation to muscle quality in sarcopenic and non-sarcopenia elderly diabetic patients

Aruna Raju^{*}

Department of Physiology, All India Institute of Medical Science, Kalyani, West Bengal, India

MPP-46 Effects of RAS and SGLT2 inhibitors alone or in combination on end-stage kidney disease and/or all-cause death in patients with both diabetes and hypertension: a nationwide cohort studyRenin-angiotensin-aldosterone system (RAS) inhibitors and sodium-glucose cotransporter 2 (SGLT2) inhibitors are key

Sangmo Hong^{1*}, Kyungdo Han², Kyung-Soo Kim³, Cheol-Young Park⁴

¹Endocrinology and Metabolism, Department of Internal Medicine, Guri Hospital, Hanyang University, College of Medicine, Republic of Korea, ²Department of Statistics and Actuarial Science, Soongsil University, Republic of Korea, ³Endocrinology and Metabolism, Department of Internal Medicine, CHA Bundang Medical Center, CHA University School of Medicine, Republic of Korea, ⁴Endocrinology and Metabolism, Department of Internal Medicine, Samsung Kangbuk Hospital, Republic of Korea

- PE-01** Integrative fine-mapping and functional annotation identify novel genetic determinants of LDL cholesterol regulation in Koreans
Yongho Jee^{1*}, Tae-Jin Song³, Wes Spiller²
¹Advanced Biomedical Research Institute, Ewha Womans University Seoul Hospital, Republic of Korea, ²Institute for Health Promotion, Graduate School of Public Health, Yonsei University, Republic of Korea, ³Department of Neurology, Seoul Hospital, Ewha Womans University College of Medicine, Republic of Korea
- PE-02** Torin-1 administration enhances cognitive function by regulating autophagy and cholesterol metabolism in hepatic encephalopathy
So Yeong Cheon^{*}
Department of Biotechnology, College of Biomedical & Health Science, Konkuk University, Republic of Korea
- PE-04** Precision hepatometabolic therapy: integrating PPAR α agonists and omega-3 fatty acids in the treatment of diabetic lipotoxic liver disease
Roland Helmizar^{1,2*}, Wisda Widiastuti^{1,2}, Vina Tri Septiana^{3,4}
¹Internal Medicine, Baiturrahmah University, Indonesia, ²Internal Medicine, Siti Rahmah Hospital, Indonesia, ³Radiology, Baiturrahmah University, Indonesia, ⁴Radiology, Siti Rahmah Hospital, Indonesia
- PE-05** Chronic intermittent hypoxia induces aortic inflammation via NLRP3 inflammasome activation in normal C57BL/6 mice
Parag Rane^{*}
Pharmacy, JD Pawar College of Pharmaceutical Sciences, India
- PE-06** Perivascular adipose tissue browning in early atherosclerosis
Jiyeon Park^{*}, Yong Joo Ahn
Medical Science and Engineering, Pohang University of Science and Technology (POSTECH), Republic of Korea
- PE-07** Dietary flaxseed oil restores lipid homeostasis and reduces atherogenic index in fructose-induced metabolic syndrome in Wistar rats
Shatrughna U. Nagrik^{*}
Pharmacy, Satya University, India
- PE-08** Mucosal invariant T-Cells link CCBE1 expression to clinically actionable CAD
Weiqian Lee^{1,2*}, Gemma Figtree^{1,2}
¹Medicine and Health, University of Sydney, Australia, ²Cardiology, Kolling Institute, Australia
- PE-09** Elucidating the renoprotective effects of Boerhaavia diffusa in a high-fat diet-induced chronic kidney disease mouse model
Lee On Ying^{*}, Martin Ho Yin Yeung
The Department of Health Technology and Informatics, The Hong Kong Polytechnic University, China

- PE-10** **Boerhaavia diffusa ameliorates renal lipid deposition and attenuates kidney injury in high-fat diet-induced chronic kidney disease mouse models**
Ching In Chung^{1*}, On Ying Lee¹, Angela Zaneta Chan², Martin Ho Yin Yeung^{1,2}
¹Department of Health Technology and Informatics, The Hong Kong Polytechnic University, Hong Kong, China, ²Department of Anatomical and Cellular Pathology, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, China
- PE-11** **Lactoferrin alleviates renal oxidative stress and lipid accumulation in high-fat diet-induced diabetic nephropathy**
Martin Ho Yin Yeung^{1,2,3*}, Angela Zaneta Chan²
¹Curriculum and Learning, Education, English Schools Foundation, Hong Kong SAR, China, ²Department of Anatomical and Cellular Pathology, The Chinese University of Hong Kong, Hong Kong SAR, China, ³The Department of Health Technology and Informatics, The Hong Kong Polytechnic University, Hong Kong SAR, China
- PE-12** **Smad7 deletion in smooth muscle cells promotes the contractile phenotype and results in more stable atherosclerotic plaques**
Alonso-Herranz L^{1,2*}, Albarrán-Juárez J¹, Markov A¹, Lewis E¹, Izquierdo-Serrano R⁴, Matchkov V³, Bentzon JF^{1,2,4}
¹Department of Clinical Medicine, Aarhus University, Denmark, ²Steno Diabetes Center Aarhus, Aarhus University Hospital, Denmark, ³Department of Biomedicine, Aarhus University, Denmark, ⁴Novel Mechanisms of Atherosclerosis, Centro Nacional de Investigaciones Cardiovasculares Carlos III (CNIC), Spain
- PE-13** **Adenylyl cyclase-associated protein 1 and caveolin complex regulates adhesion molecules on endothelial cells**
You Ji Kim^{1*}, Cheong-Whan Chae³, Taehun Yoon², Gun Choi², Yoo-Wook Kwon^{1,3}
¹Department of Medicine, Seoul National University College of Medicine, Seoul National University, Seoul, Republic of Korea, ²Molecular Medicine and Biopharmaceutical Sciences, Graduate School of Convergence Science and Technology, Seoul National University, Seoul, Republic of Korea, ³Strategic Center of Cell and Bio Therapy for Heart, Diabetes & Cancer, Biomedical Research Institute, Seoul National University Hospital, Seoul, Republic of Korea
- PE-14** **ANGPTL4 suppresses endothelial inflammation and EndMT to preserve vascular integrity in atherosclerosis**
Dong Im Cho^{1*}, Bo Gyeong Kang¹, In Joo Hwang¹, Meeyoung Cho¹, Jin Yoo¹, Soo Ji Yoo¹, Yong Sook Kim^{1,2}, Youngkeun Ahn^{1,3}
¹Cell Regeneration Research Center, Chonnam National University Hospital, Republic of Korea, ²Biomedical Research Institute, Chonnam National University Hospital, Republic of Korea, ³Department of Cardiology, Chonnam National University Hospital, Republic of Korea
- PE-15** **Eicosapentaenoic acid suppresses vascular smooth muscle cell calcification through regulation of miR-30c1-3p**
Da Yeon Kyeon^{1*}, Yeon Woo Lee¹, Hyeon Ji Lee¹, Soo-jin Ann², Sang Hak Lee³
¹Graduate School, Yonsei University, Republic of Korea, ²Integrative Research Center for Cerebrovascular and Cardiovascular Diseases, Yonsei University College of Medicine, Republic of Korea, ³Division of Cardiology, Department of Internal Medicine, Yonsei University College of Medicine, Republic of Korea
- PE-16** **Effects of semaglutide on glucose metabolism in the streptozotocin-high fat diet-induced diabetic mouse model**
Kyuho Kim^{1*}, Ye-Jee Lee², Ji-Won Kim², Jae-Seung Yun¹, Yu-Bae Ahn¹, Seung-Hyun Ko¹
¹Division of Endocrinology and Metabolism, Department of Internal Medicine, St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Republic of Korea, ²Department of Internal Medicine, College of Medicine, The Catholic University of Korea, Republic of Korea

- PE-17** **Multitarget modulation of LOX-1, 15-LOX-1, and NLRP3 by thiazole-coumarin-azomethine derivatives: a pharmacoinformatic strategy to disrupt lipid peroxidation-driven inflammation in atherosclerosis**
 Rizki Rachmad Saputra^{1*}, Muhammad Priyadi²
¹Department of Chemistry, Universitas Palangka Raya, Indonesia, ²Department of Pharmacy, Universitas Palangka Raya, Indonesia
- PE-18** **Mathematical modeling of palmitic acid-induced super-enhancer and noncoding RNA crosstalk regulating atherogenic inflammation in human monocytes**
 Prihantini Prihantini^{1*}, Rini Winarti², Sahnaz Vivinda Putri³, Rifaldy Fajar¹
¹AI-BioMedicine Research Group, IMCDS-BioMed Research Foundation, Indonesia, ²Biology, Yogyakarta State University, Indonesia, ³Health Management Enthusiast, Indonesia Open University, Indonesia
- PE-19** **MicroRNA-17, -21, and -92a as novel predictive biomarkers for cardiovascular disease risk: A STAT3-mediated pathway analysis**
 Nurul Izzati Abdullah^{1*}, Fatin Syazwani Abd Malek¹, Fitri Kahar¹, Amrina Mohamad Amin¹, Md Parvez Eusof Izzudin², Sazlina Shariff Ghazali³, Norshariza Nordin⁴, Sabariah Md Noor¹
¹Department of Pathology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Malaysia, ²Family Medicine Clinic, Hospital Sultan Abdul Aziz Shah, Malaysia, ³Department of Family Medicine, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Malaysia, ⁴Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Malaysia
- PE-20** **Non-coding RNAs differently expressed in cell lines with normal and defective mitophagy**
 Elizaveta Pleshko^{*}
 Institute for Atherosclerosis Research, Russian Federation
- PE-21** **HDAC inhibitor YAK577 mitigates vascular calcification through regulation of MMP14**
 Hae Jin Kee^{1*}, Hongyan Zhou¹, Young Joon Hong², Doo Sun Sim², Myung Ho Jeong³
¹Heart Research Center, Chonnam National University Hospital, Republic of Korea, ²Cardiology, Chonnam National University Hospital, Republic of Korea, ³Cardiology, Gwangju Veterans Hospital, Republic of Korea
- PE-22** **Prdx1-dependent stroke-associated microglia attenuate ischemic damage in a photothrombotic stroke model**
 Hyemin Park^{*}, Huiju Jo, Goo Taeg Oh
 Life Science, Ewha Womans University, Republic of Korea
- PE-23** **Comparing cost-effectiveness of obesity interventions: insights into surgery, medications, and diets**
 Rosinta Purba^{2*}, Yesika Simbolon^{1,2}, Hepri Ardianson², Lintong Simbolon², Ester Purba^{2,3}, Silmi Rahmani²
¹Accounting, Atmajaya University, Indonesia, ²Health Economics, The Pranala Institute, Indonesia, ³Hospitality and Care, Raff Tindal NT, Australia
- PE-24** **Healthy fat expansion is induced by extracorporeal shockwave treatment (ESW-treatment) under high-fat diet (HFD) in mouse**
 Wonkyoung Cho^{*}, Young Mi Park
 Department of Molecular Medicine, Ewha Womans University, School of Medicine, Republic of Korea

- PE-26** Trends and factors associated with LDL-C treatment target attainment rates among patients with secondary prevention setting of atherosclerotic cardiovascular disease: assessments from Japanese health checkup data
Atsushi Furukawa*, Hayato Tada, Kenji Sakata, Soichiro Usui, Masayuki Takamura
Department of Cardiovascular Medicine, Kanazawa University Graduate School of Medicine, Japan
- PE-27** Effect of statin and ezetimibe combination therapy on small dense LDL in patients undergoing coronary angiography
Soo-Jin Kim^{1*}, Bong-Joon Kim¹, Sung-Il Im¹, Hyunyoung Hwang², Jung-Ho Heo¹
¹Division of Cardiology, Kosin University Gospel Hospital, Kosin University College of Medicine, Busan, Republic of Korea, ²Department of Laboratory Medicine, Kosin University Gospel Hospital, Kosin University College of Medicine, Busan, Republic of Korea
- PE-28** Bibliometric mapping of single nucleotide polymorphisms and lipoprotein(a) towards cardiovascular disease: evolution, contribution, and knowledge gaps
Khairul Nisa' Ishak^{1*}, Fauziah Md Tahib², Hazirah Watikah Abdah², Noor Alicezah Mohd Kasim^{2,3}, Siti Hamimah Sheikh Abdul Kadir^{1,2}, Yung-An Chua^{1,2}
¹Department of Biochemistry and Molecular Medicine, Faculty of Medicine, Universiti Teknologi MARA, Sungai Buloh, Selangor, Malaysia, ²Cardiovascular Advancement and Research Excellence Institute (CARE Institute), Universiti Teknologi MARA, Selangor, Malaysia, ³Department of Pathology, Faculty of Medicine, Universiti Teknologi MARA, Sungai Buloh, Selangor Malaysia
- PE-29** HLA DR genome editing with TALENs in human iPSCs produced immune-tolerant dendritic cells
Hunji Choi^{2*}, Yoo-Wook Kwon¹
¹Biomedical Research Institute, Seoul National University Hospital, Republic of Korea, ²College of Medicine, Seoul National University, Republic of Korea
- PE-30** FFAR-Based polygenic risk score and its interaction with macronutrient intake in relation to MAFLD risk in Korean adults
Yoonji Ryu^{1*}, Jinyoung Shon², Yoonjung Park²
¹Graduate School of Clinical Biohealth, Ewha Womans University, Republic of Korea, ²Department of Nutritional Science and Food Management, Ewha Womans University, Republic of Korea
- PE-32** Mapping the scientific landscape of sitosterolemia: a bibliometric and visualization study
Aldona Akhira Susanto^{1*}, Akmal Nur Setyawan²
¹Faculty of Medicine, Sebelas Maret University, Indonesia, ²Faculty of Medicine and Health Sciences, Muhammadiyah University of Yogyakarta, Indonesia
- PE-33** Eight-year trends in random urine sodium-to-potassium ratio among Korean adults: findings from KNHANES 2016-2023
Rihwa Choi^{1,2*}, Gayoung Chun⁴, Sung-Eun Cho³, Sang Gon Lee¹
¹Laboratory Medicine, GC Labs, Republic of Korea, ²Laboratory Medicine and Genetics, Sungkyunkwan University School of Medicine, Republic of Korea, ³Endocrine Substance Analysis Center, GC Labs, Republic of Korea, ⁴Infectious Disease Research Center, GC Labs, Republic of Korea
- PE-34** Cardioprotective effects of β -Lapachone on Isoproterenol-Induced heart failure mice
Mario Albino Sozinho Indarua*, Hyoung Kyu Kim, To Hoai T. Nguyen, Trong Kha Pham, Jin Han
Department of Physiology, College of Medicine, Inje University, Republic of Korea

- PE-35** **ASR-ZnO nanoparticles mitigate oxidative and inflammatory stress in human umbilical vein endothelial cells**
 Omilla Ragavan^{1*}, Yoke Keong Yong¹, Muhammad Nazrul Hakim Abdullah², Lai Yen Fong³, Omilla Ragavan¹
¹Human Anatomy, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Malaysia, ²Biomedical Science, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Malaysia, ³Pre-Clinical Sciences, Faculty of Medicine and Health Sciences, Universiti Tuanku Abdul Rahman, Malaysia
- PE-36** **A novel mannose receptor agonist enhances self-renewal and therapeutic potential of mesenchymal stem cells**
 Gun Choi^{*}, Yoo-Wook Kwon
 CardioVascular medicine, Seoul National University Hospital, Republic of Korea
- PE-37** **Elevated lipoprotein(a) levels are associated with increased arterial stiffness in hypertension**
 Hack-Lyoung Kim^{*}, Hyun Sung Joh, Myung-A Kim, Sang-Hyun Kim
 Division of Cardiology, Department of Internal Medicine, Seoul National University Boramae Medical Center, Seoul, Republic of Korea
- PE-38** **The impact of high protein diet with different source on cardiovascular disease risk factors in good and poor sleepers: a randomized controlled trial**
 Lingyin Yu^{1*}, Yueying Yao¹, Ian Mak En Kai¹, Clarinda Nataria Sutanto¹, Zi Ning Leong¹, Khoo Chin Meng², Jung Eun Kim^{1,3}
¹Department of Food Science and Technology, National University of Singapore, Singapore, ²Division of Endocrinology, Department of Medicine, National University Hospital, Singapore, ³Bezos Center for Sustainable Protein, National University of Singapore, Singapore
- PE-39** **Prognostic significance of endothelial dysfunction in coronary heart disease patients**
 Valentyna Romanova^{1*}, Nataliia Kuzminova¹, Lidiia Romanova¹, Mykhailo Repetenko², Anastasiia Ivankova¹, Iryna Gunko¹
¹Internal medicine #1, National Pirogov Memorial Medical University, Vinnytsya, Ukraine, ²Marketing, Limited Liability Company "KRKA Ukraine", Ukraine
- PE-41** **Patient-specific hemodynamic evaluation of coronary stenting for atherosclerosis**
 Ayeon Hwang^{*}, Nari Kim
 Department of Physiology, College of Medicine, Inje University, Republic of Korea
- PE-42** **Predicting future cardiovascular events by lipid core burden index (LCBI)**
 Farrukh Malik^{1*}, Shahzad Wahid², Muhammad Asif¹
¹Adult Cardiology, National Institute of Cardiovascular Diseases Pakistan, Pakistan, ²Bolan University of Health Sciences, Pakistan
- PE-43** **Ginsenoside compound K, a panax ginseng metabolite, attenuates macrophage phagocytosis by inhibiting histone deacetylase 6**
 Chae Young Moon^{*}, Jimin Park, Hyunju Kang
 Department of Food and Nutrition, Keimyung University, Republic of Korea

PE-44 Association between dietary niacin intake and dyslipidemia in Korean adults: a cross-sectional analysis based on KNHANES 2016-2018

Eunmi Joo*, Sunhye Shin

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

PE-45 Association between laver consumption and dyslipidemia in Korean adults: a community-based prospective cohort study

Yuna Jeong^{1,2*}, Minji Kim^{1,2}, Nayeon Do^{1,2}, Eunpyeong Yang^{1,2}, Yangha Kim^{1,2}

¹Department of Nutritional Science and Food Management, Ewha Womans University, Republic of Korea, ²Graduate program in System Health Science and Engineering, Ewha Womans University, Republic of Korea

PE-46 Biglycan enhances muscle regeneration and function in dystrophic muscle: evidence from mdx mice and human population data

Sehyun Yoon^{1*}, Hyesun Han¹, Shieon Kim², Hee Ju Jun², Min-Jeong Shin³

¹Department of Integrated Biomedical and Life Sciences, Korea University, Republic of Korea, ²Interdisciplinary Program in Precision Public Health, Korea University, Republic of Korea, ³School of Biosystems and Biomedical Sciences, Korea University, Republic of Korea

PE-47 Development of polyvinyl alcohol nanofiber-covered stents for coronary perforation

Mi Hyang Na^{1,2*}, Dae Young Hyun^{1,2,3,4}, Myung-Han Yoon⁶, Doo Sun Sim^{1,2,3,4}, Dae Sung Park^{1,2}, Yu Jeong Jin^{1,2}, Chan Woo Kim^{1,2}, Jaell Park⁶, Jeong Ha Kim^{1,2}, Young Joon Hong^{1,3,4}, Kyung Hoon Cho^{1,2,3,4}, Seok Oh^{1,2,3}, Jeong Hun Kim³, Myung Ho Jeong^{1,4,5}

¹The Korea Cardiovascular Stent Research Institute, Chonnam National University, Republic of Korea, ²The Cardiovascular Convergence Research Center, Chonnam National University Hospital, Republic of Korea, ³Department of Cardiology, Chonnam National University Hospital, Republic of Korea, ⁴Department of Cardiovascular Medicine, Chonnam National University Medical School, Republic of Korea, ⁵Department of Cardiovascular Center, Gwangju Veterans Hospital, Republic of Korea, ⁶School of Materials Science and Engineering, Gwangju Institute of Science and Technology, Republic of Korea

PE-48 Safety and efficacy evaluation of bioresorbable vascular scaffolds according to changes in a porcine coronary artery vessel size

Dae Sung Park^{1,2,3*}, Doo Sun Sim^{1,2,3}, Yu Jeong Jin^{1,2}, Mi Hyang Na^{1,2}, Chan Woo Kim^{1,2}, Jeong Ha Kim^{1,2}, Kyung Seob Lim⁵, Young Joon Hong^{1,2,3}, Kyung Hoon Cho^{1,2,3}, Dae Young Hyun^{1,2,3}, Seok Oh^{1,2}, Jung Hoon Kim², Myung Ho Jeong^{1,2,3,4}

¹Department of The Korea Cardiovascular Stent Research Institute, Chonnam National University Medical School, Republic of Korea, ²Department of Cardiology, Chonnam National University Hospital, Republic of Korea, ³Department of Cardiology, Chonnam National University Medical School, Republic of Korea, ⁴Department of Cardiovascular Center, Gwangju Veterans Hospital, Republic of Korea, ⁵Department of The Futuristic Animal Research Center, Korea Research Institute of Bioscience and Biotechnology, KRIBB, Republic of Korea

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Key Lectures



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Plenary Lecture 1

Sep 12(Fri) 10:20–11:00 | Room 1,2,3 (3F)

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

10:20–11:00 **Advances in lipid-lowering therapy: shaping the future of cardiovascular prevention**

Ioanna Gouni-Berthold (University of Cologne, Germany)



CURRICULUM VITAE**Ioanna Gouni-Berthold**

Professor, Head of the Lipid Clinic and Lipid Research Clinic, Center for Endocrinology, Diabetes and Preventive Medicine, University of Cologne, Germany

**Education and Training**

1984 Aristotle University, Thessaloniki, Greece, M.D, Medicine

Employment and Position

1985–1986	Boston University, Research Fellow
1986–1989	Franklin Square Hospital Center, Resident
1989–1992	Baylor College of Medicine, Fellow
1992–1993	Harvard Medical School, Fellow
1994–1996	Aristotle University, Attending physician
1996–1999	University of Bonn, Attending physician
1999–2002	University of Bonn, Lecturer
2002–2020	University of Cologne, Professor, Senior Consultant
2020–	University of Cologne, Head Lipid Clinic and Lipid Research Clinic

Important Publications

1. Volanesorsen to prevent acute pancreatitis in hypertriglyceridemia. Alexander VJ, Karwatowska-Prokopczuk E, Prohaska TA, Li L, Geary RS, Gouni-Berthold I, Oral EA, Hegele RA, Stroes ESG, Witztum JL, Tsimikas S. N Engl J Med. 2024 Feb 1;390(5):476–477.
2. Association Between Achieved Low-Density Lipoprotein Cholesterol Levels and Long-Term Cardiovascular and Safety Outcomes: An Analysis of FOURIER-OLE. Gaba P, O'Donoghue ML, Park JG, Wiviott SD, Atar D, Kuder JF, Im K, Murphy SA, De Ferrari GM, Gaciong ZA, Toth K, Gouni-Berthold I, Lopez-Miranda J, Schiele F, Mach F, Flores-Arredondo JH, López JAG, Elliott-Davey M, Wang B, Monsalvo ML, Abbasi S, Giugliano RP, Sabatine MS. Circulation. 2023 Apr 18;147(16):1192–1203.
3. Efficacy and safety of volanesorsen in patients with multifactorial chylomicronaemia (COMPASS): a multi-centre, double-blind, randomised, placebo-controlled, phase 3 trial. Gouni-Berthold I, Alexander VJ, Yang Q, Hurh E, Steinhagen-Thiessen E, Moriarty PM, Hughes SG, Gaudet D, Hegele RA, O'Dea LSL, Stroes ESG, Tsimikas S, Witztum JL; COMPASS study group. Lancet Diabetes Endocrinol. 2021 May;9(5):264–275.
4. Lipoprotein(a) reduction in persons with cardiovascular disease. Tsimikas S, Karwatowska-Prokopczuk E, Gouni-Berthold I, Tardif JC, Baum SJ, Steinhagen-Thiessen E, Shapiro MD, Stroes ES, Moriarty PM, Nordestgaard BG, Xia S, Guerriero J, Viney NJ, O'Dea L, Witztum JL; AKCEA-APO(a)-LRx Study Investigators. N Engl J Med. 2020 Jan 16;382(3):244–255.

Advances in lipid-lowering therapy: shaping the future of cardiovascular prevention

Ioanna Gouni-Berthold

University of Cologne, Germany

There is a number of very promising lipid-lowering therapies for cardiovascular prevention under development such a new CETP inhibitor, lipoprotein(a) lowering therapies, and two oral PCSK9 inhibitors. The new CETP inhibitor obicetrapib has shown promising results in decreasing LDL-C in various patient populations such as in patients with heterozygous FH or with a history of atherosclerotic cardiovascular disease. The LDL-C decrease ranges between about 20%–30% as monotherapy and reaches an approximately 50% reduction as a combination with ezetimibe. A cardiovascular outcomes trial is ongoing and expected to be completed in November 2026.

There are currently 5 therapeutic agents that have shown a significant decrease in lipoprotein(a) levels. Pelacarsen is an antisense oligonucleotide targeting LPA, the gene expressing lipoprotein(a), is applied subcutaneously once a month and decreases lipoprotein(a) concentrations by up to 80%. A cardiovascular outcomes trial is ongoing and expected to be completed in February 2026. Olpasiran is the first siRNA targeting LPA, is applied subcutaneously

every 3 months and has been shown to decrease Lp(a) levels by about 100%. A cardiovascular outcomes trial is ongoing and expected to be completed in December 2026. Lepodisiran, another siRNA targeting LPA, has been shown to decrease lipoprotein(a) by about 95% when injected twice a year. A cardiovascular outcomes trial is ongoing and expected to be completed in March 2029. Other substances in development that significantly decrease Lp(a) is zerlasiran, another siRNA targeting LPA and an oral inhibitor of the formation of lipoprotein(a), muvalaplin.

There are two oral PCSK9 inhibitors in phase 3 development, enlicitide and AZD0780. Enlicitide decanoate (previously MK-0616) has been shown to decrease LDL-C levels by up to about 65%. A cardiovascular outcomes trial is ongoing and expected to be completed in November 2029. The second one is AZD0780, which has been shown to decrease LDL-C by up to about 50%. A cardiovascular outcomes trial with AZD0780 is currently in planning and expected to be completed in October 2029.

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Plenary Lecture 2

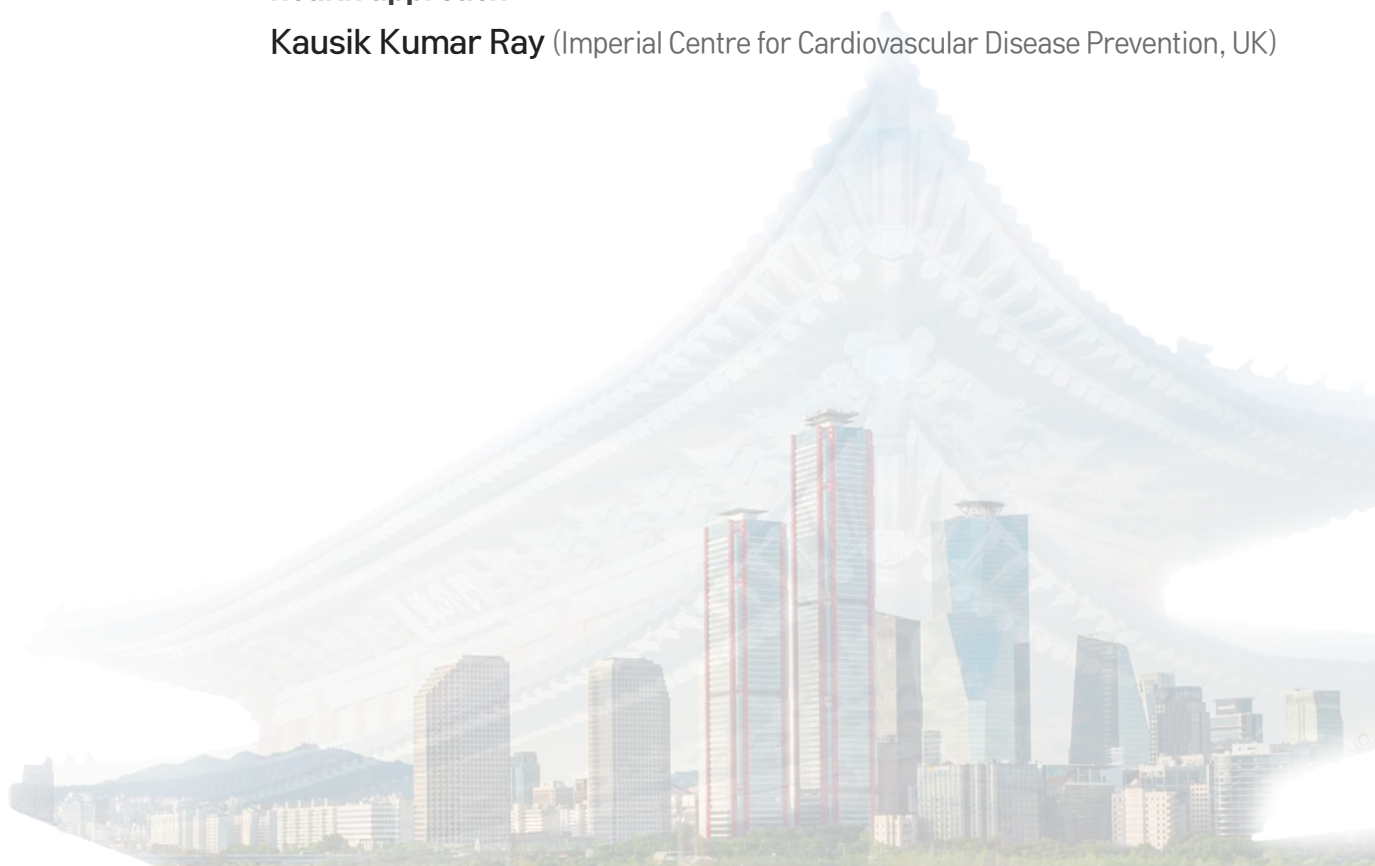
Sep 13(Sat) 10:40–11:20 | Room 1,2,3 (3F)

CHAIRPERSON : Ki Hoon Han (University of Ulsan, Republic of Korea)

10:40–11:20

Cholesterol lowering throughout the life-course to prevent atherosclerotic cardiovascular disease—a pragmatic population health approach

Kausik Kumar Ray (Imperial Centre for Cardiovascular Disease Prevention, UK)



CURRICULUM VITAE**Kausik Kumar Ray**

Professor, Imperial Centre for Cardiovascular Disease Prevention, UK

**Degrees and Qualifications Held**

2023	FMEDSci
2015	FRCP (Ed)
2013	FAHA
2011	FRCP (Lon)
2008	FESC
2008	FACC
2007	MPhil (epidemiology), University of Cambridge
2004	MD, University of Sheffield
1994	MRCP, UK
1991	MBChB, University of Birmingham (Medical School)

Education

University	
1990	Elective, Anaesthetics
1988	Intercalated BSc, University of Birmingham
1985–1991	Medical School, University of Birmingham
School	
1985	'A' Levels • 3 Grade A • 1 Grade B
	'S' Level • 1 Grade 1
1983	'O' Levels • 6 Grade A • 3 Grade B
1978–1985	Queen Mary's Grammar School, Walsall

Prizes and Distinctions

1990	Recommendation for Elective Report
1985	J.F. Crump Chemistry Prize
1984	Michael Bollen Chemistry Prize

Cholesterol lowering throughout the life-course to prevent atherosclerotic cardiovascular disease—a pragmatic population health approach

Kausik Kumar Ray

Imperial Centre for Cardiovascular Disease Prevention, UK

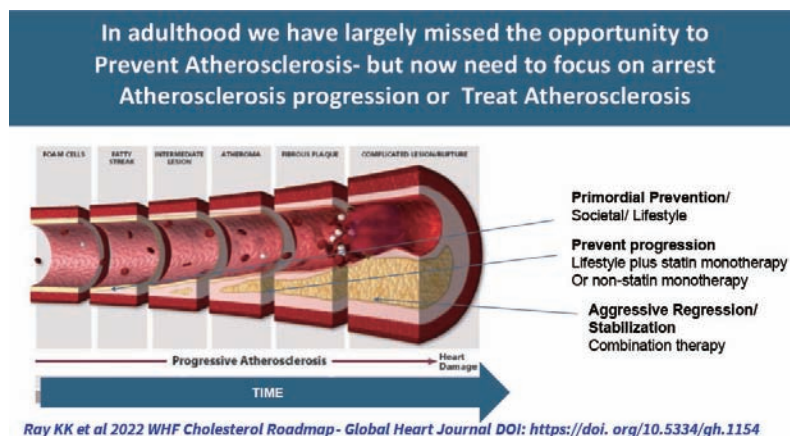
Atherosclerotic cardiovascular disease (ASCVD) occurs from the retention and accumulation of apoB containing lipoproteins and their cholesterol cargo in the walls of arteries over time. This is accelerated via risk factors such as diabetes, smoking and hypertension which result in arterial wall injury and in people with inherited genetic vulnerability. Thus the vast majority of individuals who develop ASCVD do so without extreme levels of cholesterol in the blood. The majority of apoB containing lipoproteins in the circulation consist of LDL particles hence LDL cholesterol (LDL-C) makes up the vast majority of the atherogenic cholesterol content in blood.

Irrespective of whether someone has high medium or low LDL-C levels the relative risk reduction per 1mmol/L LDL-C lowering is approximately 22% in major adverse cardiovascular events. Absolute benefits depend upon baseline risk. This benefit

per mmol/L is observed with statins, ezetimibe, bempedoic acid or PCSK9i, meaning there are no magical extra benefits from any approach and benefit relates to LDL-C lowering.

The only scenario where 1mmol/L reduction in LDL-C offers greater relative risk reduction per 1mmol/L lowering is in lower risk primary prevention patients with annualised risk < 1% where per 1mmol/L the benefits approach ~39% falling per 1 mmol/L as event rates increase. This means among those with more advanced atherosclerosis 2mmol/L lowering is needed to reduce risk by 40%.

Therefore, pragmatic solutions throughout the life-course, could be primordial prevention with lifestyle/ policy to reduce the likelihood of development of atherosclerosis, in early adult life consider statin or other monotherapy as the relative risk reduction 1mmol/L is greater and in later life use combination therapy as an initial approach.



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Keynote Lecture 1

Sep 12(Fri) 15:50–16:30 | Room 1,2,3 (3F)

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

15:50–16:30 **Multi-omics data integration from patients with carotid stenosis illuminates key molecular signatures of atherosclerotic instability**

Ljubica Matic (Karolinska Institute, Sweden)



CURRICULUM VITAE

Ljubica Matic

Associated Professor, Principal Researcher,
Group Leader, Karolinska Institute (KI), Stockholm, Sweden

**Education and Training**

1998–2003	Faculty of Biology, Belgrade University, Belgrade, Serbia, MSc, Molecular Biology and Physiology
2004–2012	Department of Medical Biochemistry and Biophysics (MBB), Karolinska Institute, Stockholm, Sweden, PhD, Medical Biochemistry, kidney disease, podocyte biology
2012–2014	Department of Molecular Medicine and Surgery (MMK), Karolinska Institute, Stockholm, Sweden, Postdoc, Atherosclerosis biomarkers
2019–2021	Department of Molecular Medicine and Surgery (MMK), Karolinska Institute, Stockholm, Sweden, Assist Prof, Smooth muscle cell biology/atherosclerosis/vascular disease
2022–	Department of Molecular Medicine and Surgery (MMK), Karolinska Institute, Stockholm, Sweden, Assoc Prof, Molecular Medicine

Current Positions

2025–	Assoc Editor Atherosclerosis journal
2024–	PI in several European Horizon Widera program and European Horizon TOOL program projects
2024–	Management Committee member EU COST actions AtheroNET and CardioPharmaGENET
2023–	Group Leader for Translational Vascular Medicine, Center for Molecular Medicine, KI
2023–	Assoc Editor Vascular Pharmacology journal
2021–	Assoc Prof and Principal Researcher, Vascular Surgery, Department of Molecular Medicine, KI
2021–	Co-Director and Examiner Global MSc Program in Biomedicine, elective track in Circulation, Metabolism and Endocrinology, KI
2013–	Director and Examiner, PhD course in Vascular Cell Biology, Cardiovascular Program, KI
2013–	Research Coordinator and co-PI in the Biobank of Karolinska Endarterectomies (BiKE), KI

Previous Positions

2020–2023	Steering Board member and co-founder, KI-Novo Nordisk Cardiovascular Program
2019–2021	Assistant Prof and Senior Researcher with Swedish Heart-Lung Foundation fellowship, KI
2017–2019	Researcher with Swedish Heart-Lung Foundation fellowship, Department of Molecular Medicine, KI
2012–2016	Postdoc with Swedish Society for Medical Research fellowship, Vascular Surgery, Department of Molecular Medicine, KI

Multi-omics data integration from patients with carotid stenosis illuminates key molecular signatures of atherosclerotic instability

Ljubica Matic

Vascular Surgery, Department of Molecular Medicine and Surgery, Karolinska Institute, Sweden

Background: Understanding the pathophysiology of unstable atherosclerosis is imperative to prevent myocardial infarction and stroke. We used multi-omics integration to identify key molecular targets with diagnostic and therapeutic potential.

Methods: Biobank of Karolinska Endarterectomies encompassing patients with symptomatic (S) and asymptomatic (AS) carotid atherosclerosis, was the main resource. Plaques, peripheral blood monocytes and plasma sampled locally from around plaque or periphery of $n > 700$ individuals, were profiled by transcriptomics, proteomics and metabolomics. A supervised machine learning method DIABLO was used for *per patient* data integration. Multi-omics layers were integrated separately across local and peripheral disease sites, and their intersection, with stratification for symptomatology. Identified analytes were investigated using scRNA-seq, clinical and outcome data.

Results: In peripheral circulation, FABP4, IL6, Bilirubin and Sphingomyelin were the most prom-

inent analytes. F11, ANGPTL3, ICOSLG, ITGB1 and Sphingomyelin were enriched in the local disease site, while FABP4, C1R, IL6, Bilirubin and Sphingomyelin appeared at the intersection. Coagulation, necroptosis, inflammation and cholesterol metabolism were confirmed as key pathways determining symptomatology. Clinical analyses showed an impact of lipid-lowering therapy on ICOSLG expression, anti-hypertensives on plasma FABP4 and BLVRB levels, anti-diabetics on plasma Sphingomyelins, while no medications affected ANGPTL3. Association with future adverse events was shown for plasma Bilirubin, Sphingomyelin, ANGPTL3 and ICOSLG plaque levels. Open-source target genetic analyses suggested causal involvement of F11, C1S, EGFR, IL6, ANGPTL3 in the disease.

Conclusions: Using an innovative, deep-data framework, this study provides confirmatory and novel information on mechanisms behind atherosclerotic instability. The findings raise possibilities for translational prioritizations to aid personalized medicine.

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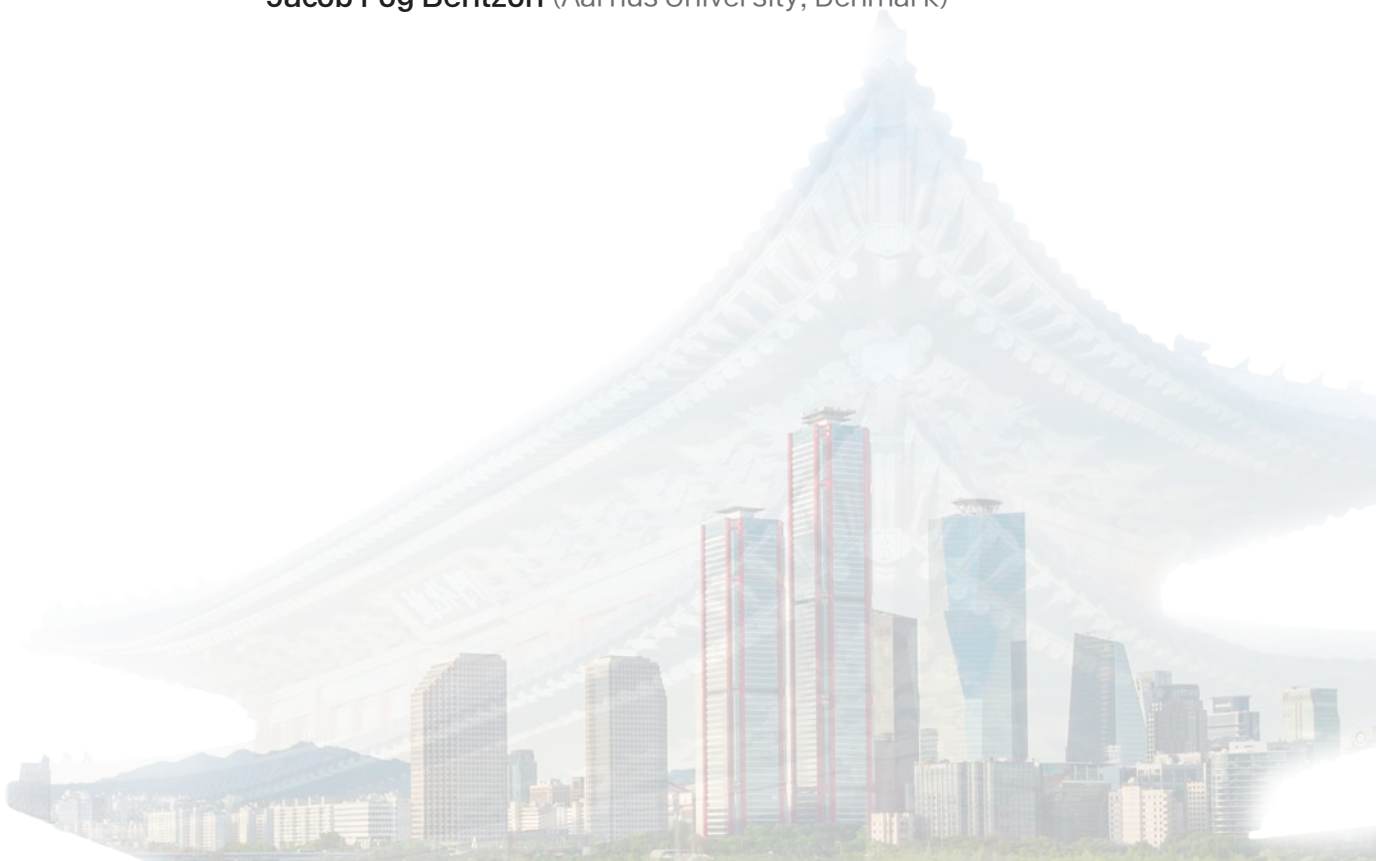
Keynote Lecture 2

Sep 13(Sat) 11:25–12:05 | Room 1,2,3 (3F)

CHAIRPERSON : Byung-Chul Oh (Gachon University, Republic of Korea)

11:25–12:05 **The role of smooth muscle cells in atherosclerotic plaque progression**

Jacob Fog Bentzon (Aarhus University, Denmark)



CURRICULUM VITAE

Jacob Fog Bentzon

Professor, Aarhus University, Denmark



Education and Training

2003.01	Aarhus University, Denmark, M.D, Medicine
2007.02	Aarhus University, Denmark, Ph.D., Health Sciences

Employment and Position

2004–2007	Aarhus University, PhD Student
2007–2010	Aarhus University, Postdoc/Assistant Professor
2010–2019	Aarhus University, Associate Professor
2015–2025	Spanish National Cardiovascular Research Center, Full Professor
2019–	Aarhus University, Professor

Important Publications

1. Carramolino L, Albarrán-Juárez J, Markov A, Hernández-SanMiguel E, Sharysh D, Vanessa Cumbicus V, Morales-Cano D, Labrador V, Møller PL, Nogales P, Benguria A, Dopazo A, Sanchez-Cabo F, Torroja C, Bentzon JF. Cholesterol lowering depletes atherosclerotic lesions of smooth muscle cell-derived fibromyocytes and chondromyocytes. *Nature Cardiovasc Res.* 2024; 3:203–220.
2. Martos-Rodríguez CJ, Albarrán-Juárez J, Morales-Cano D, Caballero A, MacGrogan D, de la Pompa JL, Carramolino L, Bentzon JF. Fibrous Caps in Atherosclerosis Form by Notch-Dependent Mechanisms Common to Arterial Media Development. *Arterioscler Thromb Vasc Biol.* 2021 Sep;41(9):e427–e439.
3. Al-Mashhadi RH, Al-Mashhadi AL, Nasr ZP, Mortensen MB, Lewis EA, Camafeita E, Ravlo K, Al-Mashhadi Z, Kjær DW, Palmfeldt J, Bie P, Jensen JM, Nørgaard BL, Falk E, Vázquez J, Bentzon JF. Local pressure drives LDL accumulation and atherosclerosis in the coronary arteries of hypertensive minipigs. *J Am Coll Cardiol.* 2021;77(5):575–589.
4. Jacobsen K, Lund MB, Shim J, Gunnarsen S, Füchtbauer EM, Kjolby M, Carramolino L, Bentzon JF. Diverse cellular architecture of atherosclerotic plaque derives from clonal expansion of a few medial SMCs. *JCI Insight.* 2017;2(19). pii: 95890.
5. Bjørklund MM, Hollensen AK, Hagensen MK, Dagnæs-Hansen F, Christoffersen C, Giehm Mikkelsen J, Bentzon JF. Induction of atherosclerosis in mice and hamsters without germline genetic engineering. *Circ Res.* 2014;114(11):1684–9.

Awards and Honors

2021	Blue Flame Award, Addgene.org, for research reagent sharing
2019	ERC consolidator grant awardee
2019	Top reviewer award from Arteriosclerosis, Thrombosis and Vascular Biology journal

Research Interest

Our research group investigates the mechanisms of atherosclerosis, with a special focus on the role of smooth muscle cells. Ongoing work aims to uncover the mechanisms underlying the fate decisions of smooth muscle cells in plaques and their responses to therapy.

The role of smooth muscle cells in atherosclerotic plaque progression

Jacob Fog Bentzon

Aarhus University, Denmark

Atherosclerosis is a leading cause of death and disability worldwide, driven by high blood cholesterol, hypertension, obesity, and diabetes. The disease affects the arterial intima, where plaques develop that can eventually obstruct blood flow through stenosis or trigger thrombosis. While atherosclerosis is initiated by lipoprotein-driven inflammation, plaque growth is sustained by a maladaptive response of local arterial smooth muscle cells (SMCs), which proliferate and produce a heterogeneous connective tissue matrix.

Recent work by our group and others has revealed that most mesenchymal cells in atherosclerotic plaques originate from the clonal expansion of a few preexisting SMCs. During this process, the clonally expanding cells undergo phenotypic transitions, losing their contractile identity and acquiring features of other mesenchymal cell types,

including fibroblasts and osteochondrogenic cells. This broad differentiation potential resembles that of embryonic mesenchymal progenitors, suggesting that developmental mechanisms may be reactivated in disease.

In this lecture, I will present recently published and unpublished data from murine models of atherosclerosis that combine conditional gene targeting and single-cell RNA sequencing to dissect the embryonic pathways controlling SMC fate in the plaque. I will also discuss how SMC-derived cells respond to cholesterol-lowering therapy and how SMC-derived cells in experimental models compare to those in human disease. Together, these findings support a model in which SMCs drive lesion growth through developmental programs that could be targeted to inhibit lesion size or vulnerability.

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Special Lecture 1 (K)

Sep 12(Fri) 11:05–11:45 | Room 1,2,3 (3F)

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

11:05–11:45 **Integrating ChatGPT into medical and clinical research workflows**
Ki-Hyun Jeon (Seoul National University, Republic of Korea)



CURRICULUM VITAE**Ki-Hyun Jeon**

Assistant Professor, Division of Cardiology, Department of Internal Medicine,
Seoul National University Bundang Hospital, Seongnam, Republic of Korea

**Education and Training**

1999.03–2005.02	Chung-Ang University College of Medicine, Korea, M.D., Medicine
2009.03–2014.02	Graduate School, Chung-Ang University, Korea, Master of Medical Science, Internal Medicine
2006.03–2010.02	Seoul National University Hospital, Korea, Residency, Internal Medicine
2013.05–2015.02	Seoul National University Hospital, Korea, Fellowship, Cardiology (Intervention)
2010.03–2013.04	Republic of Korea Army, Military Medical Officer, Captain, General Medicine

Employment and Position

2015–2017	Sejong General Hospital, Interventional Cardiologist
2017–2021	Mediplex Sejong Hospital, Interventional Cardiologist
2021–Present	Seoul National University Bundang Hospital, Assistant Professor

Important Publications

1. Jeon KH, Lee HS, Kang S, et al. AI-enabled ECG index for predicting left ventricular dysfunction in patients with ST-segment elevation myocardial infarction. *Sci Rep.* 2024;14(1):16575. doi:10.1038/s41598-024-67532-6.
2. Jeon KH, Jang JH, Kang S, et al. Identifying Atrial Fibrillation With Sinus Rhythm Electrocardiogram in Embolic Stroke of Undetermined Source: A Validation Study With Insertable Cardiac Monitors. *Korean Circ J.* 2023;53(11):e63.
3. Jeon KH, Jeong YH, Chae IH, et al. Implication of diabetic status on platelet reactivity and clinical outcomes after drug-eluting stent implantation: results from the PTRG-DES consortium. *Cardiovasc Diabetol.* 2023;22(1):245.
4. Jeon KH, Kim HL, Lim WH, et al. Associations between measurements of central blood pressure and target organ damage in high-risk patients. *Clin Hypertens.* 2021;27:23.
5. Kwon JM, Jeon KH, et al. A deep learning algorithm to detect anaemia with ECGs. *Lancet Digit Health.* 2020;2(7):e358–e367.

Awards and Honors

1. Best Lecturer Award, Korean Society of Interventional Cardiology (KSIC), 2025
2. Academic Excellence Award, National Health Forum, 2023
3. Best Reviewer Award, Asia-Pacific CardioMetabolic Syndrome (APCMS), 2023

Research Interest

Artificial intelligence in cardiology, digital biomarkers from electrocardiography and imaging, interventional cardiology, coronary physiology, and clinical decision support systems.

Integrating ChatGPT into medical and clinical research workflows

Ki-Hyun Jeon

Division of Cardiology, Seoul National University Bundang Hospital, Republic of Korea

Generative artificial intelligence (AI) is rapidly transforming the landscape of medical research. Unlike traditional AI used in diagnostics or imaging, generative models such as ChatGPT are now actively assisting in literature review, data analysis, manuscript drafting, and study design. This shift is especially meaningful for clinician-scientists who struggle with time constraints and limited access to technical resources.

Generative AI lowers the barrier to complex statistical analysis by enabling non-experts to perform tasks through natural language prompts. It also helps structure research ideas, improve academic writing, and overcome language barriers for non-native English speakers. As a result, research that once required intensive support from statisticians or programmers can now be initiated and refined through interactive dialogue with AI.

However, the adoption of AI in research requires

caution. Inaccuracies, misinterpretations, and ethical issues such as plagiarism or data privacy violations must be carefully managed. Researchers must maintain critical oversight and avoid uncritical acceptance of AI-generated outputs.

The rise of generative AI demands a paradigm shift in research education. Beyond tool usage, future training must focus on AI literacy, critical thinking, and ethical judgment. While AI serves as a powerful collaborator, human researchers must retain ownership of scientific reasoning and decision-making.

Ultimately, generative AI is not replacing the researcher but empowering them. It redefines the research space as a collaborative, dynamic environment—where asking the right questions and interpreting answers wisely become the core competencies of modern scientific inquiry.

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Special Lecture 2 (K)

Sep 13(Sat) 13:30–14:40 | Room 1,2,3 (3F)

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

13:30–14:40 **Gardens of Korea (한국의 정원)**

Hong June Yoo (Director General, National Museum of Korea, Republic of Korea)



CURRICULUM VITAE**Hong June Yoo**

Professor Emeritus, Department of Art History, Myongji University, Republic of Korea

**Major Career Highlights**

- B.A. in Aesthetics, Seoul National University (1967-1980)
- M.A. in Art History, Hongik University Graduate School, 1983
- Ph.D. in East Asian Philosophy, Sungkyunkwan University Graduate School, 1998
- Began career as an art critic after being selected in the 1981 Dong-A Ilbo New Year's Literary Contest (Art Criticism category)
- Former Co-representative of the Korean People's Art Association (1984)
- Commissioner, 1st Gwangju Biennale

Academic and Public Service

- Professor and Director of the University Museum, Yeungnam University (1991-2002)
- Professor and Dean of the Graduate School of Cultural Arts, Myongji University (2002-2004)
- Administrator, Cultural Heritage Administration of Korea (Sep. 2004-Feb. 2008)
- Honorary Director, Chusa Memorial Museum, Jeju (Oct. 2015-Dec. 2018)
- Chairman of the Board, Academy of Korean Studies (Jul. 2021-Jun. 2024)
- Currently serves as Professor Emeritus, Department of Art History, Myongji University

Major Publications

- My Cultural Heritage Exploration (12 volumes)
- Pilgrimage to the National Museum (2 volumes)
- My Cultural Heritage Exploration - Japan & China Editions (8 volumes)
- Lectures on the History of Korean Art by Hong-june Yoo (6 volumes)
- Lives of Great Artists (2 volumes)
- Critical Biography of Wandang (3 volumes)
- A Study on Theories of Painting in the Joseon Dynasty
- Pilgrimage to National Treasures, Pilgrimage to Masterpieces, Sense of Discernment, Pilgrimage to Korean Buddhist Temples
- Chusa Kim Jeong-hui

Honors and Awards

- Publication Award, Korea Publication Ethics Commission (1998)
- 17th Manhae Literature Award (2003)

Gardens of Korea (한국의 정원)

Hong June Yoo

Director General, National Museum of Korea, Republic of Korea

정원(庭園, Jeong-won, Garden), 원림(園林, Won-rim, Natural forest gardens), 별서(別墅, Byeol-seo, Countryside retreats), 정사(精舍, Jeong-sa, Scholarly or meditative dwellings), 누정(樓亭, Nu-jeong, Pavilions and towers), 구곡(九曲, Gu-gok, Nine-bend valleys designed for reflection and rest)

정원(庭園)의 사전적 정의는 '집(건물)이나 프락에 조성된 꽃밭'

꽃밭을 가꾸는 인간의 마음은 다 같지만 그 형식은 동서양이 다르고 한중일이 달랐다. 서양의 정원은 베르사유 궁전의 정원처럼 인공과 자연이 완전히 구분되어 꽃밭을 다양한 기하학적 형태로 아름답게 꾸며 시각적으로 즐기는 형식이다.

동양의 정원은 인공과 자연이 하나로 되어 그 속에서 삶을 즐기는 형식으로 되어 있다. 중국의 경우는 쭈저우의 <拙정원>처럼 인공적인 공간 안에 이상적인 자연을 화려하고 웅장하게 재현하였다. 일본의 정원은 교토의 <류안지(龍安寺)> 석정(石庭)처럼 자연을 디자인하여 관조하는 형식으로 발전하였다.

한국의 정원은 인공적 공간 안에 자연을 재현하는 것이 아니라 자연 속에 인공적인 건물을 적절히 배치하여 앉은 자리(location) 전체를 정원으로 삼았다. 다른 나라 정원과 비교하여 인공과 자연의 관계가 역전되었다.

이는 전국토의 70%가 산이고 모든 산에는 아름답고 그윽한 계곡이 발달하였기 때문에 이를 정원으로 삼은 것이다.

예술철학자 조요한은 한중일 정원을 비교하여 "중국 정원처럼 인공에 의하여 창조하는 것도 아니고, 일본 정원처럼 자연을 주택의 마당에 끌어들이어서 주인 행세를 하는 것도 아니며, 한국 정원은 자연 속의 일부분으로 풍광과 어울

리는 것이다."라고 하였다.

한국의 정원은 쓰임새에 따라 원림(園林) 별서(別墅), 정사(精舍), 누정(樓亭), 구곡(九曲) 등 여러 가지 형태로 구현되었다.

The dictionary definition of a "garden" (정원) is "a flowerbed created in or around a house or yard." While the human desire to cultivate and enjoy flowers is universal, its expression varies between East and West, and even among China, Japan, and Korea. Western gardens, such as those at Versailles, are characterized by a strict separation between nature and artificiality, with geometrically arranged flowerbeds designed for visual enjoyment.

In contrast, Eastern gardens seek harmony between the artificial and the natural, integrating them into a space for living. Chinese gardens, like the Humble Administrator's Garden (拙政園) in Suzhou, recreate an idealized version of nature within an artificial space—splendid and grand. Japanese gardens, such as the rock garden at Ryoan-ji (龍安寺) in Kyoto, distill nature into minimalist compositions meant for contemplation.

Korean gardens, however, reverse this relationship. Rather than reproducing nature within an artificial space, they place artificial structures thoughtfully within the natural landscape, making the location itself the garden. This unique approach stems from Korea's mountainous terrain—70% of the country consists of mountains with tranquil, scenic valleys that were often embraced as natural

gardens.

Art philosopher Yo Han Cho once said of East Asian gardens: “The Korean garden is not created through artificial means like the Chinese garden, nor does it domesticate nature into a courtyard like the Japanese garden. Rather, it harmonizes with the natural scenery as part of the landscape.” Depend-

ing on their function and context, Korean gardens took various forms, including Won-rim (園林, natural forest gardens), Byeol-seo (別墅, country retreats), Jeong-sa (精舍, scholarly or meditative dwellings), Nu-jeong (樓亭, pavilions and towers), and Gu-gok (九曲, nine-bend valleys designed for reflection and rest).

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Main Symposia



ICoLA 2025

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Symposium 1 (K)

이상지질혈증 관리: 국내 당면 과제와 향후 발전 방향

Management of Dyslipidemia: Current Perspectives and Future Directions in Korea

Sep 11(Thu) 13:00–14:30 | Room 1 (3F)

CHAIRPERSONS : 김상현 이사장 (한국지질·동맥경화학회)

조연희 회장 (한국건강검진학회)

13:00–13:10

인사말

박주민 위원장 (국회 보건복지위원회)

13:10–13:25

지질관리! 이상지질혈증 치료 현황과 급여 기준 현실화

정인경 이사 (한국지질·동맥경화학회)

13:25–13:40

지질관리! 혈압, 혈당 관리를 넘어 – 만성질환 통합 관리와 이상지질혈증

백재욱 이사 (대한가정의학과 의사협회)

13:40–13:55

지질관리! 현장의 목소리 – 일차의료 현장의 현실과 지원방안에 대한 요구

이태인 이사 (한국건강검진학회)

13:55–14:10

지질관리! 더 빨리, 더 정확히 – 초고령화 시대, 더 오래 더 건강하기 위한 조기검진과 치료의 중요성

김은지 교수 (가천의대 예방의학과)

14:10–14:30

패널 토의 및 기자회견

이창현 이사 (한국건강검진학회)

이금숙 특임이사 (한국의학바이오기자협회)

곽경근 부회장 (대한내과 의사협회)

전하윤 사무관 (보건복지부)

CURRICULUM VITAE

정인경 이사

경희의대 강동경희대학교병원 내분비대사내과



학력

1988.03-1994.02 경희대학교 의과대학 학사
 1995.03-1997.02 경희대학교 의과대학 석사
 1997.03-1999.02 경희대학교 의과대학 박사

경력

1999.03-2002.04 성균관대의대 삼성서울병원 전임의
 2002.05-2005.12 한림대학교 의과대학 한강성심병원 전임강사, 조교수
 2006.01-2010.02 경희대학교 의과대학 강동경희대학교병원 조교수
 2008.03-2009.07 Harvard University Joslin Diabetes Center, Visiting researcher
 2010.03-2015.02 경희대학교 의과대학 강동경희대학교병원, 부교수
 2015.03-현재 경희대학교 의과대학 강동경희대학교병원 교수

관심분야

- Glucose and lipid metabolism
- Clinical therapeutics of diabetes and metabolism
- Non-alcoholic fatty liver disease and energy metabolism
- Vascular biology and diabetic vascular complication
- Pancreatic beta cell biology and insulin secretion.

논문

1. Yoo J, Jun JE, Jeong IK, Ahn KJ, Chung HY, Lee MS, Hwang YC. DA-1241, a GPR119 Agonist, Ameliorates Fatty Liver Through the Upregulation of TFEB-Mediated Autophagy. Diabetes. 2025 Jul 1;74(7):1107-1120.
2. Jun JE, Choi SH, Moon MK, Ko SH, Kim HJ, Hur KY, Rhee EJ, Noh JH, Jeong IK. Efficacy and safety of ANagliptin when added to iNsinulin and metformin therapy in patients with uncontrolled type 2 diabetes; randomized, placebo-controlled, double-blind, multicentre trial (CANNA study). Diabetes Obes Metab. 2025 Sep;27(9):5351-5355.

지질관리! 이상지질혈증 치료 현황과 급여 기준 현실화

우리나라 이상지질혈증의 현황에 대해, 한국지질동맥경화 학회는 2015년 이후 2-3년 간격으로 Dyslipidemia Fact Sheet (DFS) 형태로 보고해 왔다. 가장 최근에 발표된 2024년도 DFS에 따르면, 고콜레스테롤혈증(총콜레스테롤 240 mg/dl 이상이거나 지질강화제 복용) 유병률은 27.4% (남 23.8%, 여 31%)로, 20세 이상 성인 4명중 1명에 해당되며, 2007년에 비해 2.5배 증가하였다. 고콜레스테롤혈증에 대한 인지율은 68%로, 과거에 비해 증가하였으나, 여전히 고콜레스테롤혈증 환자의 30%는 본인이 고콜레스테롤혈증인지 인지하지 못하고 있다. 치료율은 61.2%였으며, 지질강화제 복용한 사람 중에 치료율은 87.4%이었다. 이상지질혈증은 다음 3가지 중 1가지 이상일때로 정의하였다: ① LDL-C $\geq$ 160 mg/dL 또는 지질강화제 복용, ② TG $\geq$ 200 mg/dL, ③ HDL-C $<$ 40 mg/dL. 하지만 HDL-C를 남녀 모두 40 mg/dl 미만으로 할 경우 여성의 이상지질혈증 유병률이 남자에 비해 매우 낮으며, 실제 HDL-C은 여자에서 남자에 비해 높고, 이미 대사증후군 진단 기준에서도 남녀 HDL-C 기준을 남자 HDL-C $<$ 40 mg/dL, 여자 HDL-C $<$ 50 mg/dL로 달리 하고 있어서 2022년 DFS부터는 이상지질혈증 유병률

을 두가지 모두로 제시하고 있다. 그 결과 과거 HDL-C 기준 시 이상지질혈증 유병률은 40.9% (남 47.1%, 여 34.7%)이나 HDL-C을 각기 달리하면, 47.4% (남 47.1%, 여자 47.7%)로 남녀 유병률이 비슷하다. 따라서, 20세 이상 성인 대상(심혈관위험인자가 있는 경우라면 더 젊은 연령부터) 남녀 모두, 지속적으로 지질대사에 대한 검사가 필요하다.

우리나라 이상지질혈증에 대한 치료 지침은 한국지질동맥경화학회에서 1996년 1판을 시작으로 최근 2022년 5번째 개정판이 보고되었다. 이상지질혈증의 치료는 1단계) 심혈관 질환 위험도 평가를 통해 환자의 상태에 맞는 LDL-C 치료 목표를 파악한 후, 2단계) 치료 목표에 도달이 가능한 지질강화제를 선택하는데, 스타틴을 1차 치료약제로 투여하고, 치료 목표에 도달 못하거나 스타틴 불내성이면 그외 에제티미브나 PCSK9 억제제의 병합요법을 고려한다. 하지만, 우리나라 지질강화제에 대한 급여기준은 2018년 이후 현재까지 변함이 없어 좀더 치료 지침에 근거한 현실적인 급여 기준의 개정이 필요하다. 이 강의에서는 우리나라 이상지질혈증의 치료 현황과 급여기준 현실화에 대한 심도 깊은 내용을 다룰 예정이다.

CURRICULUM VITAE

백재욱 이사

동동가정의학과



학력

2002	고려대의대 졸업
2008	고대 안암병원 가정의학과 전문의 취득

경력

2011-	가정의학회 보험이사
2020-	대한의사협회 보험자문위원
2025-	서울시 의사회 의무정책 부회장

관심분야

커뮤니티 케어

논문

1. World Health Organization Integrated Care for Older People (ICOPE) and the Integrated Care of Older Patients with Frailty in Primary Care (ICOOP_Frail) Study in Korea (2021).
2. Efficacy of Combined Exercise and Nutrition Intervention Study for Possible Sarcopenia in Community-Based Primary Care Clinics (ENdSarC): study protocol for a multicenter single-blinded randomized controlled trial (2024).

지질관리! 혈압, 혈당 관리를 넘어 - 만성질환 통합 관리와 이상지질혈증

일차의료의 필요성이 강조되고 있으며 최근 일차의료 강화 특별 법안이 발의된 상황에서도 현장에서는 현실적으로 반드시 행하여야하는 검사들이 등안 시 되고 있다.

이는 우리나라 일차의료에서 진행되고 있는 관련 사업들을 되돌아보면서 그 바탕이 무엇인지 확인해본다.

CURRICULUM VITAE

이태인 이사

한국건강검진학회(박앤김내과의원)



학력

2007.2 경희대학교 의과대학 의학과 학사
2016.8 경희대학교대학원 의학과 의학석사

경력

2007.3-2008.2 강동경희대병원 수련의
2011.5-2015.2 경희의료원 내과 전공의
2015.3-2016.2 경희의료원 소화기내과 전임의
2016.3-2020.2 김현우내과의원 부원장
2020.3-현재 박앤김내과의원 대표원장

관심분야

내과, 소화기내과, 검진

논문

1. Oxyntic gland adenoma endoscopically mimicking a gastric neuroendocrine tumor: A case report, World J Gastroenterol 2015 April 28; 21(16): 5099-5104.

지질관리! 현장의 목소리 - 일차의료 현장의 현실과 지원방안에 대한 요구

본 고는 이상지질혈증의 현황과 일차의료 현장의 현실을 조망하며, 그 효과적인 조절을 위해 일차의료기관 의료진 입장에서 가장 필요하다 여기는 제안과 요구를 담았다. 현재 이상지질혈증은 조기 발견 및 적절한 투약 시 85% 이상 조절 가능하지만, 확진검사의 부재와 국가검진 주기의 한계로 조기 진단률이 낮다. 이에 고위험군을 국가검진주기를 4년에서

2년으로 환원하고, 국가검진 시 확인된 이상지질혈증에 대한 확진진료의 개념으로 진찰·상담의 본인부담금 면제를 제안한다. 또한 '만성질환의 예방 및 관리에 관한 법률' 등의 제정을 통해 컨트롤타워를 구축하고, 일차의료 만성질환관리 본사업에 이상지질혈증을 포함해 통합적 관리체계를 마련하는 것을 정책 방향으로 제시한다.

CURRICULUM VITAE

김은지 교수

가천의대 예방의학과



학력

2024	연세대학교 의과대학 예방의학 박사학위
2019	University College London, UK 국제보건 석사학위
2015	동국대학교 의학전문대학원 의무석사
2011	고려대학교 생명과학 학사

경력

2024-현재	가천대학교 의과대학 예방의학 조교수
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관심분야

Cardiovascular disease, Non-communicable disease, Health equity, Preventive medicine, Epidemiology, Public health.

논문

1. Kim E, Hong J, Shin H, Moon JY, Sunwoo W. Prophylactic antibiotic use in closed basilar skull fractures: A nationwide cohort study. Journal of Trauma and Acute Care Surgery. In press.
2. Kim E, Lee HH, Kim EJ, Cho SM, Kim HC, Lee H. Factors associated with medication adherence among young adults with hypertension. Clin Hypertens. 2025 May;31(1):e18.
3. Joo H, Kim E, Huh K, et al. Risk of postacute sequelae of COVID-19 and oral antivirals in adults aged over 60 years: A nationwide retrospective cohort study. International Journal of Infectious Diseases, Volume 154, 107850.
4. Kwon J, Kim E. Lifelong impact of elevated blood pressure from childhood to adulthood. Clin Exp Pediatr. 2025;68(4):278-286.
5. Kim E, Lee H, Lloyd-Jones D, Ko YG, Kim BG, Kim HC. Area deprivation and premature cardiovascular mortality: a nationwide population-based study in South Korea. BMJ Public Health. 2024;2:e000877.

지질관리! 더 빨리, 더 정확히 - 초고령화 시대, 더 오래 더 건강하기 위한 조기검진과 치료의 중요성

대한민국은 2024년 말 초고령사회에 진입하였다. 고령 인구의 확대와 함께 이상지질혈증의 유병과 의료이용 증가가 관찰된다. 그러나 연령구조를 보정해도 유병이 상승한다는 점은 질병부담이 단순한 고령화만으로 설명되지 않음을 시사한다. 특히 젊은 성인은 유병 자체는 낮으나 일부 지질지표의 악화되는 경향이 있으며, 다른 연령대에 비해 인지-치료-조절률이 상대적으로 매우 낮아 조기 발견-치료-조절의 이행 간극이 지속되고 있다. 이러한 간극은 누적 노출을 통해 장기 위험을 증가시키므로 전 생애주기 관점의 개입이 필요하다.

LDL 콜레스테롤은 죽상경화의 인과적 원인이며, LDL-C를 1 mmol/L 낮추면 주요 심혈관사건 위험이 약 20-25% 감소한다. 더 나아가 조기 개입을 통해 '콜레스테롤-연수'를 줄이고, 평생 누적 노출을 낮출수록, 후기 개입 대비 장기 이득이 크다는 근거가 축적되고 있다. 본 발표는 전 연령대에서 죽상동맥경화성 심혈관질환의 위험을 실질적으로 낮추고 건강수명을 연장하기 위한, 이상지질혈증의 조기 검진과 치료의 중요성을 검토하고자 한다.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Symposium 2

Cutting-edge Nutritional Approach for Cardiometabolic Risk Strategies

Sep 11(Thu) 13:00–14:30 | Room 2 (3F)

CHAIRPERSONS : Hyun-Sook Kim (Sookmyung Women's University, Republic of Korea)
Eun Mi Kim (Sungkyunkwan University, Republic of Korea)

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- | | |
|-------------|--|
| 13:00–13:20 | Interplay between molecular and environmental factors in the new era of precision medicine
Jordi Merino (University of Copenhagen, Denmark) |
| 13:20–13:40 | Human microbiome and the path to cardiometabolic health
Hyun Ju You (Seoul National University, Republic of Korea) |
| 13:40–14:00 | Iron overload, autophagy dysfunction, and ferroptosis: mechanistic insights into cardiometabolic disease
Hyekyoung Sung (York University, Canada) |
| 14:00–14:30 | Panel Discussion
Hyunju Kang (Keimyung University, Republic of Korea)
Jeongseon Kim (National Cancer Center, Republic of Korea)
Hoyoun Won (Chung-Ang University, Republic of Korea) |

CURRICULUM VITAE

Jordi Merino

Associate Professor, University of Copenhagen, Denmark



Education and Training

2013.11	Rovira i Virgili University, Spain, Ph.D., Metabolism and atherosclerosis
2008.11	Rovira i Virgili University, Spain, B.S, Nutrition

Employment and Position

2022–	University of Copenhagen, Associate Professor
2022–	Massachusetts General Hospital, Adjunct Research Associate
2018–2022	Massachusetts General Hospital, Research Associate
2018–2022	Harvard University, Instructor
2015–2022	Broad Institute of MIT and Harvard, Research Scientist
2015–2018	Massachusetts General Hospital, Postdoc

Important Publications

1. Guasch-Ferre M, [···], Merino J (2025). Precision nutrition for cardiometabolic diseases. *Nat Med.* 31:1444–1453. PMID: 40307513.
2. Tobias DK, Merino J [···], Franks PW (2023) Second international consensus report on gaps and opportunities for the clinical translation of precision diabetes medicine. *Nat Med.* 10:2438–2457. PMID: 37794253.
3. Merino J, [···], Hu FB (2022). Polygenic scores, diet quality, and type 2 diabetes risk: An observational study among 35,759 adults from 3 US cohorts. *PLoS Med.* 19:e1003972. PMID: 35472203.
4. Wu P, [···], Merino J (2022). Obesity partially mediates the diabetogenic effect of lowering LDL cholesterol. *Diabetes Care*, 45:232–40. PMID: 34789503.
5. Merino J, [···] Florez JC (2019). Quality of dietary fat and genetic risk of type 2 diabetes: individual participant data meta-analysis. *BMJ*, 366:l4292. PMID:31345923.

Awards and Honors

2023	Future Leader Award, EFSD/Novo Nordisk Foundation
2023	Early-Career Researcher Award in Diabetes, Danish Diabetes and Endocrine Academy
2016	Levine-Riggs Young Investigator Award, USA

Interplay between molecular and environmental factors in the new era of precision medicine

Motivated by the need to reduce the global burden of type 2 diabetes (T2D), my research focuses on how genetic and environmental factors shape T2D risk and influence responses to preventive strategies. I have led several projects within large-scale initiatives such as the U.S. Diabetes Prevention Program, the CHARGE Consortium, and other international collaborations. Our findings show that both genetic and lifestyle factors independently contribute to T2D risk, with limited evidence of strong interactions. These insights have paved the way for studies that integrate genetics with multi-omics and environmental markers to identify

high-risk individuals who may benefit from tailored interventions, advancing the field toward precision diabetes medicine. In this presentation, I will discuss how emerging approaches—leveraging omics technologies, deep phenotyping, wearable devices, and digital health tools—are uncovering differential responses to diet and lifestyle interventions. I will also review the current evidence for precision nutrition in cardiometabolic disease, highlight its potential to transform healthcare, and outline key knowledge gaps and implementation challenges that must be addressed before precision nutrition can deliver on its promise for population health.

CURRICULUM VITAE

Hyun Ju You

Assistant Professor, Seoul National University, Republic of Korea



Education and Training

1997.03	Seoul National University, Korea, B.Sc., Food and Nutrition
2001.08	Seoul National University, Korea, M.Sc., Food and Nutrition
2011.02	Seoul National University, Korea, Ph.D., Food and Nutrition

Employment and Position

2011–2022	Graduate School of Public Health, Seoul National University, Research Professor
2022–2023	Center for Microbiome Research, KoBioLabs Inc., Board Director
2023–	Department of Food and Nutrition, Seoul National University, Assistant Professor

Important Publications

1. You HJ*, Si J*, Kim J*, Yoon S*, Cha KH, Yoon HS, Lee G, Yu J, Choi JS, Jung M, Kim DJ, Lee Y, Kim M, Vázquez-Castellanos JF, Sung J, Park JM, Ko G. (2023) *Bacteroides vulgatus* SNUG 40005 Restores *Akkermansia* Depletion by Metabolite Modulation. *Gastroenterology*. 164(1):103–116.
2. Kang H*, You HJ*, Lee G, Lee SH, Yoo T, Choi M, Joo SK, Park JH, Chang MS, Lee DH, Kim W, Ko G; Innovative Target Exploration of NAFLD (ITEN) consortium. (2022) Interaction effect between NAFLD severity and high carbohydrate diet on gut microbiome alteration and hepatic de novo lipogenesis. *Gut Microbes*.14(1): 2078612. *co-first authors.
3. Si J, Kang H, You HJ§, Ko G§. (2022) Revisiting the role of *Akkermansia muciniphila* as a therapeutic bacterium. *Gut Microbes*. 14(1): 2078619. §co-corresponding authors.
4. Yoon HS, Cho CH, Yun MS, Jang SJ, You HJ, Kim JH, Han D, Cha KH, Moon SH, Lee K, Kim YJ, Lee SJ, Nam TW, Ko G. (2021) *Akkermansia muciniphila* secretes a glucagon-like peptide-1-inducing protein that improves glucose homeostasis and ameliorates metabolic disease in mice. *Nature Microbiology*. 6(5):563–573.
5. Lee G*, You HJ*, Bajaj JS, Joo SK, Yu J, Park S, Kang H, Park JH, Kim JH, Lee DH, Lee S, Kim W, Ko G. (2020) Distinct signatures of gut microbiome and metabolites associated with significant fibrosis in non-obese NAFLD. *Nature Communications*. 5: 11(1): 4982. *co-first authors.

Research Interest

Human Microbiome, Precision Nutrition, Metabolome, Immuno-metabolic Diseases, Microbiome Therapeutics

Human microbiome and the path to cardiometabolic health

The gut microbiome is a key regulator of metabolic homeostasis and cardiometabolic diseases (CMD), including hypertension, dyslipidemia, atherosclerosis, and type 2 diabetes. Diet-driven shifts in microbial composition profoundly affect host metabolism, immune signaling, and vascular function. Beneficial taxa such as *Akkermansia muciniphila*, *Faecalibacterium prausnitzii*, *Bifidobacterium*, and *Roseburia* are linked to improved lipid metabolism and reduced inflammation, largely via production of microbial metabolites like short-chain fatty acids. These compounds enhance hepatic lipid regulation and promote anti-inflammatory pathways. Conversely, metabolites such as trimethylamine N-oxide, derived from microbial conversion of dietary choline and carnitine, contribute to endothelial dysfunction and atherosclerosis progression. Secondary bile acids, generated by gut microbes, also regulate lipid absorption and modulate host metabolic signaling via nuclear receptors includ-

ing FXR and TGR5. Dietary components such as fiber, polyphenols, omega-3 fatty acids, and fermented foods promote beneficial microbial configurations and suppress the production of pro-atherogenic metabolites. Precision nutrition approaches that tailor dietary recommendations based on individual microbiome profiles represent a promising strategy for CMD prevention and therapy. However, significant inter-individual variability in microbiome composition and function complicates clinical translation. Advances in multi-omics profiling, machine learning, and human cohort studies now enable mechanistic insight into host-microbe-diet interactions. These tools facilitate the identification of predictive microbial signatures and the development of personalized interventions targeting the gut microbiome. This presentation will summarize how the gut microbiome shapes cardiometabolic risk and explore how microbial metabolites can be leveraged for prevention and treatment.

CURRICULUM VITAE

Hyekyoung Sung

Senior Research Scientist, York University, Canada



Education and Training

2006.02	Sungkyunkwan University, Korea, MPharm, Pharmacy
2017.08	York University, Canada, Ph.D., Medical Biology
2020.03	York University, Canada, Postdoctoral Fellow, Medical Biology
2023.06	Sunnybrook Research Institute, University of Toronto, Postdoctoral Fellow, Medicine

Employment and Position

2017–2020	York University, Postdoctoral Fellow
2020–2023	Sunnybrook Research Institute, University of Toronto, Postdoctoral Fellow
2023–	York University, Senior Research Scientist

Important Publications

1. Cho S, Tam E, Nguyen K, Lei Y, Fillebeen C, Pantopolous K, Sung HK*, Sweeney G* (2025). ω -6 PUFA-enriched membrane phospholipid composition of cardiomyocytes increases susceptibility to iron-induced ferroptosis and inflammation. * co-correspondence, Apoptosis. 2025 Jun;30(5-6):1614-1627. doi: 10.1007/s10495-025-02121-0. Epub 2025 May 17.
2. Nguyen K, Tang J, Gatica D, Russell RC, Sung HK*, Sweeney G* (2025). ALY688 attenuates iron-induced ER stress and insulin resistance via activation of ER-phagy. *co-correspondence, Diabetes.
3. Sung HK*, Cho SJ*, Nguyen K, Lei Y, Tam E, Yihan Luo, Evgeniy V. P, Kacey J. P, Scott A S, Dylan B, Sweeney G. Lipidomic profiling of plasma extracellular vesicles from adiponectin knockout mice reveals a pro-inflammatory and pro-oxidative lipid signature linked to metabolic dysfunction. JEV. Revision.
4. Sung HK*, Tang J, Jahng JWS, Chan YK, Cho HH, Song E and Sweeney G (2024). Ischemia-Induced Cardiac Dysfunction Is Exacerbated In Adiponectin-knockout Mice Due To Impaired Autophagy Flux. Clin Transl Sci. 2024 Mar;17(3): e13758. doi: 10.1111/cts.13
5. Sung HK*, Murugathansan M, Abdul-Sater AA and Sweeney G (2023). Autophagy deficiency exacerbates iron overload induced reactive oxygen species production and apoptotic cell death in skeletal muscle cells. Cell Death Dis. 2023 Apr 7;14(4):252. doi: 10.1038/s41419-022-05484-3.

Iron overload, autophagy dysfunction, and ferroptosis: mechanistic insights into cardiometabolic disease

Disrupted iron homeostasis has emerged as a critical contributor to the pathogenesis of cardiometabolic disease (CMD); however, the molecular mechanisms linking iron overload (IO) to metabolic dysfunction and myocardial injury remain incompletely defined. Through integrative analyses in both skeletal and cardiac muscle systems, we delineate a mechanistic axis wherein IO perturbs insulin signaling via oxidative stress, autophagy impairment, and enhanced ferroptosis susceptibility.

We demonstrate that IO attenuates insulin-stimulated Akt phosphorylation and glucose uptake in cardiomyocytes and skeletal muscle cells, with particularly pronounced effects in primary adult cardiomyocytes. These defects are mediated by elevated mitochondrial and cytosolic reactive oxygen species (ROS), impaired mitochondrial function, and disruption of autophagic flux. Notably, IO induces a block at the lysosomal degradation stage of autophagy, associated with suppression of mTORC1-UVRAG signaling and defective autophagosome-lysosome regeneration. Autophagy-de-

ficient models further reveal exacerbation of ROS accumulation and apoptotic cell death, underscoring the essential cytoprotective role of functional autophagy in maintaining metabolic homeostasis under iron stress.

In parallel, we show that phospholipid remodeling in cardiomyocytes, specifically enrichment in ω -6 polyunsaturated fatty acid (PUFA)-containing phospholipids, amplifies IO-induced ferroptotic susceptibility. This is characterized by mitochondrial lipid peroxidation, release of mitochondrial DNA, and induction of sterile inflammation via activation of the cGAS-STING/type I interferon axis.

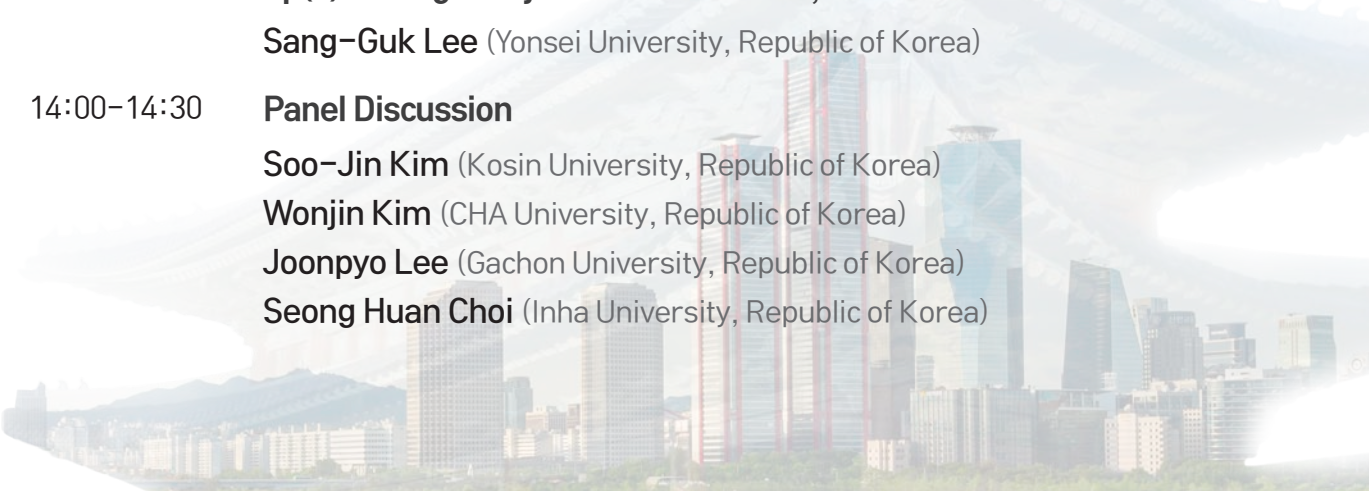
Together, these findings define a coordinated pathophysiological cascade of iron-induced oxidative injury, autophagy dysfunction, and ferroptosis-driven inflammation that contributes to CMD progression. This work provides a mechanistic framework for targeting iron metabolism, autophagic regulation, and lipid composition to mitigate metabolic dysregulation and cardiac injury in CMD.

Symposium 3

Lipoprotein(a) Unmasked: Risk, Paradox, and Measurement

Sep 11(Thu) 13:00–14:30 | Room 3 (3F)

CHAIRPERSONS : Myung A Kim (Seoul National University, Republic of Korea)
Byung Jin Kim (Sungkyunkwan University, Republic of Korea)

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- | | |
|-------------|--|
| 13:00–13:20 | Epidemiologic evidence on Lp(a) and ASCVD in Asian populations
Youngwoo Jang (Gachon University, Republic of Korea) |
| 13:20–13:40 | The inverse link between Lp(a) and metabolic risk: signal or noise?
Jung A Kim (Korea University, Republic of Korea) |
| 13:40–14:00 | Lp(a) testing today: mind the method, mind the unit
Sang-Guk Lee (Yonsei University, Republic of Korea) |
| 14:00–14:30 | Panel Discussion
Soo-Jin Kim (Kosin University, Republic of Korea)
Wonjin Kim (CHA University, Republic of Korea)
Joonpyo Lee (Gachon University, Republic of Korea)
Seong Huan Choi (Inha University, Republic of Korea) |
- 

CURRICULUM VITAE

Youngwoo Jang

Gachon University, Republic of Korea



Education & Career

2008–2012	School of Medicine, Gachon University, Doctor of Medicine
2017–2018	Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
2018–2019	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Fellow
2019–2021	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
2022–	Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

Research Interests

Cardiovascular intervention, Atherosclerosis, Acute myocardial infarction, Angina and heart failure, Pulmonary hypertension, Atrial fibrillation

Important Publications

1. Jang Y, Park SD, Lee JP, et al. One-month dual antiplatelet therapy followed by prasugrel monotherapy at a reduced dose: the 4D-ACS randomised trial. *EuroIntervention* 2025.
2. Kang SH, Pack KY, Kim JH, Jang Y (corresponding author). The effect of sarpogrelate compared to aspirin in high- or very-high-risk diabetes for primary prevention. *Sci Rep*. 2025 Jan 29;15(1):3616. doi: 10.1038/s41598-025-87868-x.
3. Kim S, Jang Y, Inflammation in Atherosclerotic Cardiovascular Diseases: Biomarkers to Therapeutics in Clinical Settings. *J Cardiovasc Interv*. 2024 Oct;3(4):199-215.
4. Jang Y, Han SH, Sohn IS, Oh PC, Koh KK. Lipoprotein(a) and Cardiovascular Diseases — Revisited —. *Circ J* 2020; 84: 867 – 874 doi: 10.1253/circj.CJ-20-0051.
5. Jang Y, Kim M; Oh, PC, Suh SY, Lee K, Kang WC, and Han SH, Long-term Clinical Outcomes and Its Predictors Between 1- and 2-stent Strategy in Coronary Bifurcation Lesions: A Baseline Clinical and Lesion Characteristic Matched Analysis, *Circ J*. 2022 Aug 25;86(9):1365-1375. doi: 10.1253/circj.CJ-22-0163.2.
6. Jang Y, Lee HH, Lee H, Kim HC and Chung WJ. Epidemiology of PAH in Korea: An Analysis of the National Health Insurance Data, 2002–2018. *Korean Circ J*. 2023;53:313-327.
7. Jang Y, Kim B-G, Kwon S, Seo J, Kim HK, Chang H-J, et al. (2020) Prevalence and clinical features of bone morphogenetic protein receptor type 2 mutation in Korean idiopathic pulmonary arterial hypertension patients: The PILGRIM explorative cohort. *PLoS ONE* 15(9): e0238698. Sept. 2020. <https://doi.org/10.1371/journal.pone.0238698>.

Epidemiologic evidence on Lp(a) and ASCVD in Asian populations

Lipoprotein(a) [Lp(a)] is increasingly recognized as an independent, causal risk factor for atherosclerotic cardiovascular disease (ASCVD). However, most large-scale data originate from Western populations, while epidemiologic evidence in Asian cohorts has been relatively limited until recently. This lecture will review current knowledge on the prevalence and distribution of Lp(a) across Asian populations, highlighting ethnic and regional differences compared with Western counterparts. Special attention will be given to the relationship between elevated Lp(a) levels and incident ASCVD, including myocardial infarction, stroke, and peripheral artery disease, in both general and high-risk Asian groups. Emerging paradoxes—such

as the relatively lower median Lp(a) concentrations observed in Asians but the persistence of significant risk at comparable thresholds—will be discussed. Methodological challenges in measuring Lp(a), including assay standardization and unit conversion, also remain critical in interpreting epidemiologic findings. Finally, implications for risk assessment, guideline integration, and future therapeutic strategies tailored to Asian populations will be considered. By synthesizing available evidence, this presentation aims to provide a comprehensive overview of how Lp(a) contributes to ASCVD risk in Asia and to identify key research gaps for advancing precision prevention in this region.

CURRICULUM VITAE

Jung A Kim

Clinical Assistant Professor, Korea University, Republic of Korea



Education and Training

2013.02	Korea University, Korea, M.D, Medicine
2019.09	Korea University, Korea, Ph.D., Internal Medicine

Employment and Position

2018–2021	Korea University Guro Hospital, Fellow
2021–2023	Bundang Jaeseng General Hospital, Staff
2024–	Korea University Anam Hospital, Clinical Assistant Professor

Important Publications

1. 2025.07 Diabetes & Metabolism journal, Exploring the side effects of GLP-1 receptor agonist: to ensure its optimal positioning.
2. 2025.05 Journal of Lipid and Atherosclerosis, Lipoprotein(a) and Cardiovascular Risk in Asian Populations: A Comprehensive Review.
3. 2025.04 Journal of Cachexia, Sarcopenia and Muscle, Impact of muscle quality on muscle strength and physical performance beyond muscle mass or diabetes status.
4. 2025.02 Endocrinology and metabolism, Unveiling risk factors for treatment failure in patients with Graves disease: A nationwide cohort study in Korea.

Awards and Honors

2024	Outstanding Oral Presentation Award, International Congress of Diabetes and Metabolism
2020	Boryung Fellowship Academic Award, Korean Medical Women's Association

Research Interest

Diabetes, Dyslipidemia, Metabolism

The inverse link between Lp(a) and metabolic risk: signal or noise?

Lipoprotein(a) [Lp(a)] is largely determined by genetic factors, including copy number variation in the Kringle-IV type 2 domain and single-nucleotide polymorphisms within the LPA gene. While high Lp(a) is a well-established causal risk factor for atherosclerotic cardiovascular disease (ASCVD), an inverse relationship has been observed between Lp(a) and metabolic diseases, particularly type 2 diabetes (T2D), metabolic syndrome, and fatty liver disease. Prospective studies have shown that low Lp(a) levels predict increased risk of T2D, especially at very low concentrations, although Mendelian randomization studies have produced inconsistent findings.

Mechanistic insights suggest that hyperinsulinemia may underlie this paradox. Experimental studies demonstrate that high insulin concentrations reduce hepatic apo(a) secretion in a dose- and time-dependent manner, resulting in lower circulating Lp(a) levels. Recent Mendelian randomization analyses are consistent with this, reporting a causal link between elevated insulin and reduced Lp(a). These observations imply

that the inverse association between Lp(a) and T2D may not reflect a direct causal effect of low Lp(a) increasing diabetes risk, but rather an epiphenomenon of insulin resistance and metabolic dysregulation—more likely to reflect noise than a true causal signal.

Clinically, this distinction is important. Although low Lp(a) is associated with T2D, high Lp(a) continues to confer substantial cardiovascular risk, including in patients with diabetes. Potent Lp(a)-lowering agents such as PCSK9 inhibitors, antisense oligonucleotides, and siRNA therapies raise theoretical concerns regarding diabetogenic effects. However, current evidence suggests that the cardiovascular benefits of lowering Lp(a) clearly outweigh any modest or uncertain risk of diabetes.

In conclusion, Lp(a) serves as a robust signal for ASCVD risk. In contrast, its inverse association with metabolic disease is more likely to reflect noise arising from insulin resistance rather than a direct causal role. Further long-term studies are needed to clarify these relationships.

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.

Research Interest

His research interests are in clinical metabolomics, clinical mass spectrometry, metabolic disorders and laboratory results standardization. He is currently focusing on the research to find new metabolite biomarkers of various diseases and evaluate metabolic mechanism for disease development.

Lp(a) testing today: mind the method, mind the unit

The Lp(a) kringle IV type 2 (KIV2) element can be repeated more than 40 times, leading to significant 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 against apo(a) used in immunoturbidometric or nephelometric assays are polyclonal, often targeting repetitive motifs within the apo(a) protein. If an antibody is directed against such a repetitive motif, the protein may be recognized multiple times by a single antibody, making accurate molar quantification challenging.

As these polyclonal antibodies in nearly all Lp(a) assays detect the repeating KIV2 structure of apo(a), these assays are intrinsically isoform dependent. The apo(a) isoform-dependent assays, particularly when combined with serially diluted single-point calibrators, typically result in underestimation of high Lp(a) levels (associ-

ated with low molecular weight apo(a) isoforms) and overestimation of low Lp(a) concentrations (associated with high molecular weight apo(a) isoforms). However, in most current Lp(a) assays, the impact of KIV2 dependence has been largely minimized within acceptable limits by using multiple (at least five), independent, and carefully selected calibrators with representative isoform compositions across the measuring range.

Providing manufacturers with easily accessible reference materials, characterized using molar-based reference methods, will be a crucial next step toward the standardization of Lp(a) measurement. Raising antibodies against apo(a) that recognize a unique motif in the protein is a challenging task, and only a few antibodies targeting unique epitopes of apo(a) have been identified and characterized. Nevertheless, the ultimate goal of performing Lp(a) measurements in molar terms should not be abandoned.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Symposium 4

Lipid Toxicity and Platelet Signaling in Atherosclerotic Inflammation

Sep 11(Thu) 13:00–14:30 | Room 4 (5F)

CHAIRPERSONS : Jae-Ryong Kim (Yeungnam University, Republic of Korea)
Hyoung Kyu Kim (Inje University, Republic of Korea)

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|-------------|--|
| 13:00–13:20 | From nanoparticles to biocides: how toxic chemicals alter lipid homeostasis and accelerate atherosclerotic events
Ok-Nam Bae (Hanyang University, Republic of Korea) |
| 13:20–13:40 | Immune modulation of lipid metabolism promotes BAT whitening under thermoneutral conditions
Jun Young Hong (Yonsei University, Republic of Korea) |
| 13:40–14:00 | Role of MCU in human platelet aggregation
Jin O-Uchi (University of South Florida, USA) |
| 14:00–14:30 | Panel Discussion
Kyuhoo Kim (The Catholic University of Korea, Republic of Korea)
Jeonghan Kim (The Catholic University of Korea, Republic of Korea)
Chang-Hoon Woo (Yeungnam University, Republic of Korea) |

CURRICULUM VITAE

Ok-Nam Bae

Professor, Hanyang University, Republic of Korea



Education and Training

1999.02	Seoul National University, Korea, B.S., Pharmacy
2001.02	Seoul National University, Korea, Ph.D., Pharmacology and Toxicology
2006.02	Seoul National University, Korea, Ph.D., Pharmacology and Toxicology

Employment and Position

2011-Present	Hanyang University, Assistant/ Associate/ Full Professor
2019-2025	Animal Facility Hanyang University ERICA, Director

Important Publications

1. Polyhexamethylene guanidine-phosphate enhances pro-coagulant activity of human erythrocytes and venous thrombosis in rats through phosphatidylserine externalization. Choi S, Kim EH, Kim D, Park HJ, Gil J, Bian Y, Bae ON. J Hazard Mater. 2025;492:138303.
2. Polystyrene nanoplastics promote the blood-brain barrier dysfunction through autophagy pathway and excessive erythrophagocytosis. Kim EH, Baek SM, Park HJ, Bian Y, Chung HY, Bae ON. Ecotoxicol Environ Saf. 2025;289:117471.
3. Promoted coagulant activity and disrupted blood-brain barrier depending on phosphatidylserine externalization of red blood cells exposed to ZnO nanoparticles. Kim EH, Baek SM, Choi S, Cho J, Tahmasebi S, Bae ON. Environ Pollut. 2024;362:124921.
4. A systematic review and BMD modeling approach to develop an AOP for humidifier disinfectant-induced pulmonary fibrosis and cell death. Kim D, Shin Y, Park JI, Lim D, Choi H, Choi S, Baek YW, Lim J, Kim Y, Kim HR, Chung KH, Bae ON. Chemosphere. 2024;364:143010.
5. Amine-modified nanoplastics promote the procoagulant activation of isolated human red blood cells and thrombus formation in rats. Kim EH, Choi S, Kim D, Park HJ, Bian Y, Choi SH, Chung HY, Bae ON. Part Fibre Toxicol. 2022;19(1):60.

Awards and Honors

2024	Commendation from the Minister of Environment (환경부 장관 표창-화학물질안전관리 유공)
2022	Seokoh Life Science Award, Women Bioscience Forum
2021	Commendation from Minister of Gender Equality and Family (여성가족부 장관 표창-선도여성과학자 유공)

From nanoparticles to biocides: how toxic chemicals alter lipid homeostasis and accelerate atherosclerotic events

Environmental toxicants such as nanoplastics and biocidal chemicals are emerging as key disruptors of lipid homeostasis in blood components, contributing to thrombotic complications and atherosclerotic progression. We examined how polystyrene nanoplastics (PS-NPs) and polyhexamethylene guanidine-phosphate (PHMG-p) impair red blood cell (RBC) membrane stability and promote vascular dysfunction. Amine-modified PS-NPs induced phosphatidylserine (PS) externalization, microvesicle formation, and pro-coagulant activation of RBCs, enhancing thrombin

generation and thrombus formation. PS-NP-damaged RBCs also triggered erythrophagocytosis in brain endothelial cells, leading to iron accumulation, ferroptosis, and blood-brain barrier (BBB) disruption via autophagy inhibition. Similarly, PHMG-p exposure caused calcium-dependent PS externalization and reduced RBC deformability, which increased pro-thrombotic activity both in vitro and in vivo. These findings demonstrate that structurally diverse toxicants converge on RBC lipid asymmetry to promote thrombotic risk and vascular inflammation.

CURRICULUM VITAE

Jun Young Hong

Assistant Professor, Yonsei University, Republic of Korea



Education and Training

2007.02	Seoul National University, Korea, B.S., Oceanography
2009.02	Seoul National University, Korea, M.S., Oceanography
2015.02	University of Michigan, Ann Arbor, US, Ph.D., Physiology

Employment and Position

2015–2021	Yale University, Postdoctoral Fellow
2021–	Yonsei University, Assistant Professor

Important Publications

1. Hong JY* and Medzhitov R*. On Developmental Programming of the Immune System. *Trends Immunol* 44(11):877–889 (2023). (*co-correspondence)
2. Lim J[†], Lin EV[†], Hong JY^{†*}, Vaidyanathan B, Erickson SA, Annicelli C, Medzhitov R* Induction of natural IgE by glucocorticoids. *J Exp Med* 219:e20220903 (2022). (†co-first authors, *co-correspondence)
3. Hong JY. Developmental programming by perinatal glucocorticoids. *Mol Cells*. 45:685–691 (2022).
4. Im S, Kim H, Jeong M, Yang H, Hong JY. Integrative understanding of immune-metabolic interaction *BMB Rep* 55:259–266 (2022).
5. Hong JY, Lim J, Carvalho F, Cho JY, Vaidyanathan B, Yu S, Annicelli C, IP EWK, Medzhitov R, Long-term programming of CD8 T cell immunity by perinatal exposure to glucocorticoids. *Cell* 180: 847–861.e15 (2020). [Pre-viewed in the issue, Highlighted in Science, Previewed in Immunity].

Awards and Honors

2024	Young Medical Scientist Research Grant through the Daewoong Foundation
2020	KOLIS Award, Korean American Bioscience Forum 2020
2018–2021	Cancer Research Institute Irvington Postdoctoral Fellowship

Research Interest

Endocrine, Immunometabolism, Epigenetics

Immune modulation of lipid metabolism promotes BAT whitening under thermoneutral conditions

Brown adipose tissue (BAT) is a thermogenic organ that adapts to environmental temperature, becoming activated in the cold and promoting energy expenditure. While cold-induced activation of BAT has been extensively studied, much less is known about how BAT structure and function are remodeled under thermoneutral (TN) conditions—conditions more relevant to human physiology. Here, we apply integrated spatial transcriptomics and single-cell RNA sequencing to reveal coordinated transcriptional and spatial remodeling of BAT during TN acclimation. We identify distinct immune-enriched zones that expand in BAT over time at thermoneutrality, marked by elevated expression of genes related to immune activation and tissue remodeling.

eling.

Functionally, we show that immune cell activity plays a key role in TN-associated BAT involution. Mice lacking adaptive immune cells or subjected to localized immune modulation retain thermogenic gene expression and exhibit resistance to tissue whitening under TN conditions. In vitro and in vivo experiments further demonstrate that immune-derived signals can suppress thermogenic activity in brown adipocytes.

Together, our findings highlight the critical involvement of immune-adipose interactions in thermoneutral remodeling of BAT and suggest new avenues for modulating energy metabolism in physiologically relevant conditions.

CURRICULUM VITAE

Jin O-Uchi

Associate Professor, University of South Florida Morsani College of Medicine, USA



Education and Training

2001.03	Jikei University, Japan, M.D, Medicine
2006.08	Jikei University, Japan, Ph.D., Medicine/Physiology

Employment and Position

2001–2003	Jikei University, Residency
2006–2008	Jikei University, Postdoctoral Research Associate
2008–2011	University of Rochester, Postdoctoral Research Associate
2011–2014	Thomas Jefferson University, Instructor (Research)
2015	Thomas Jefferson University, Assistant Professor (Research)
2016–2018	Brown University, Assistant Professor
2018–2024	University of Minnesota, Assistant Professor
2024–Present	University of South Florida, Associate Professor

Important Publications

Vang A et al, JCI Insight. 2021 PMID: 33974567
Jhun BS et al, J Physiol. 2018 PMID: 29313986
O-Uchi J et al, Antioxid Redox Signal. 2014 PMID: 24800979
Jhun BS, et al, Circ Res. 2012 PMID: 22076634
Jons C et al, Sci Transl Med. PMID: 21451124

Awards and Honors

2024	Dale J. Benos Professional Service Award, American Physiological Society (APS)
2015	New investigator Award, APS, Cell and Molecular Physiology Section
2010	Richard J. Bing Award, International Society for Heart Research (ISHR)

Research Interest

Cardiac excitation-contraction coupling
Calcium signaling
Mitochondrial ion channels/transporters
Cardiac arrhythmia and heart failure

Role of MCU in human platelet aggregation

Ca²⁺ concentration at the mitochondrial matrix is a crucial signaling component for cell life and death. A pore subunit of mitochondrial Ca²⁺ uniporter complex (mtCUC), MCU, which is encoded by *CCDC109A*, is well characterized as a protein located at the inner mitochondrial membrane responsible for controlling mitochondrial Ca²⁺ influx in various cell types, including platelets. MCU at the mitochondria is derived from the "full-length" transcript of *CCDC109A*, which contains a mitochondria-targeted sequence. However, the roles of the other variants from this gene have not been fully investigated. Here, we report a novel short variant derived from this gene (termed *MCU-S*) that

lacks the mitochondria-targeted sequence and forms a Ca²⁺-permeable channel outside of mitochondria such as plasma membrane, which exhibited similar channel properties to those observed in mtCUC. The *MCU-S* was ubiquitously expressed in all cell-types/tissues, with particularly high expression in human platelets. Our data suggest that MCU-S channels at the plasma membrane serve as an additional Ca²⁺ influx pathway that regulates platelet activation. Our findings are completely distinct from the originally reported MCU function and provide novel insights into the molecular basis of Ca²⁺-dependent activation of aggregation/coagulation pathways in platelets.

Symposium 5

Artificial Intelligence in Precision Diagnosis of Atherosclerosis and Cardiovascular Risk

Sep 11(Thu) 14:40–16:10 | Room 2 (3F)

CHAIRPERSONS : Hun Sik Park (Kyungpook National University, Republic of Korea)
Myung-gon Kim (Catholic Kwandong University, Republic of Korea)

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|-------------|---|
| 14:40–15:00 | Coronary atherosclerosis and cardiovascular risk prediction through AI-assisted stress echocardiography: insights from the PROTEUS RCT trial
Paul Leeson (University of Oxford, UK) |
| 15:00–15:20 | AI-enhanced ECG for acute myocardial infarction detection and cardiovascular risk stratification
Junmyung Kwon (Medical AI, Republic of Korea) |
| 15:20–15:40 | Artificial intelligence-based coronary artery disease screening versus usual care for patients with suspected coronary artery disease: AI-Gatekeeper randomized controlled trial
SungA Bae (Yonsei University, Republic of Korea) |
| 15:40–16:10 | Panel Discussion
Dae-Young Kim (Inha University, Republic of Korea)
Seok Oh (Chonnam National University, Republic of Korea)
Hyung Joon Joo (Korea University, Republic of Korea) |

CURRICULUM VITAE**Paul Leeson**

Professor, University of Oxford, UK

**Education and Training**

1998	University of Cambridge, MB, Medicine
1998	University of Cambridge, PhD, Medicine

Employment and Position

2013–	University of Oxford, Professor of Cardiovascular Medicine
2007–	Oxford University Hospitals, Consultant Cardiologist

Important Publications

1. Johnson CL et al Changes in coronary disease management decisions in real-world practice between 2015 and 2023: insights from the EVAREST/BSE-NSTEP observational study EHJ CV Imaging 2025.
2. Upton et al PROTEUS: A Prospective RCT Evaluating Use of AI in Stress Echocardiography NEJM AI 2024.
3. Fletcher AJ et al Artificial intelligence and innovation of clinical care: the need for evidence in the real world. EHJ 2024.
4. Upton et al Automated Echocardiographic Detection of Severe Coronary Artery Disease Using Artificial Intelligence. JACC Imaging 2022.

Awards and Honors

Fellow Royal College of Physicians
Fellow British Society of Echocardiography
Fellow European Society of Cardiology

Research Interest

Application of AI within imaging and cardiovascular prevention to improve the management of cardiovascular disease

Coronary atherosclerosis and cardiovascular risk prediction through AI-assisted stress echocardiography: insights from the PROTEUS RCT trial

Artificial intelligence (AI) in cardiovascular imaging may potentially augment clinical decision but there is a lack of prospective randomized controlled trials to evaluate the impact of AI on cardiovascular disease. randomized controlled trials provide the best evidence base for deciding which advances in AI may have a benefit for patients. Within this talk, background work on development of AI innovations within the field of cardiovascular imaging, and in particular echocardiography, will be presented. Furthermore, some of the

first randomized controlled trials within the field of AI-Cardiology will be discussed with a specific focus on PROTEUS, a multi-centre, prospective study that enrolled 2500 patients across 20 hospitals within the UK national health service. The study looked at how use of AI within stress echocardiography interpretation might impact clinical outcomes and decision making. The trial highlighted important factors that need to be considered when implementating AI into healthcare systems.

CURRICULUM VITAE

Junmyung Kwon

Medical AI, Republic of Korea



- Emergency medicine specialist (MD.) and Computer scientist (MS.)
<https://scholar.google.co.kr/citations?user=DMd-2NEAAAAJ&hl=ko>
- CEO and Founder of Medical AI Co., Ltd. (2019-)
<https://www.medicalai.com>
<https://www.youtube.com/watch?v=N1Llop7RdX4>
- Chief Director, Department of Emergency medicine and Digital healthcare research center, Sejong Hospital Group (2015-)
<https://incheonsjh.co.kr/medical/team/info?orddrid=160>

Research Interests

I am a critical care and emergency medicine physician and data scientist researching the application of artificial intelligence (AI) in medical domains. I am the CEO of Medical AI Co., Ltd. and the Chief Director of the AI and Big Data Center at Sejong Medical Research Institute. I am focused on the following research fields: raw signal electrocardiography (single-lead or 12-lead) processing, development of AI algorithm-based electrocardiography, endpoint prediction in critical care, emergency medicine, resuscitation, cardiology using AI, and AI-based clinical decision support systems.

In recent study, I have published several papers that artificial intelligence could diagnosis heart failure, left ventricular hypertrophy, aortic valve stenosis, mitral valve stenosis, pulmonary hypertension, atrial fibrillation, anemia, and myocardial infarction using electrocardiography.

Education

2004-2005	Premedical College, Seoul National University, Seoul, Korea
2006-2009	School of Medicine, Seoul National University, Seoul, Korea (MD)
2017-2020	Bioinformatics and Statistics, Korean National Open University Graduate School, Seoul, Korea (MS)

AI-enhanced ECG for acute myocardial infarction detection and cardiovascular risk stratification

Heart failure and myocardial infarction are leading cardiovascular diseases with nonspecific early symptoms and rapidly worsening outcomes when diagnosis is delayed. In high-acuity settings such as emergency departments and intensive care units, fast and accurate diagnosis is crucial, yet conventional tools are often insufficient.

Medical AI, founded in 2019, developed two AI-based ECG analysis software tools—AiTiALVSD and AiTiAMI—to address these gaps. These devices analyze raw 12-lead ECG signals with high-resolution, deep learning models, offering high diagnostic performance in real-world settings.

AiTiALVSD detects left ventricular systolic dysfunction (LVSD) from ECGs and demonstrated AUROC 0.971, sensitivity 94.0%, and specificity 91.9% in a Korean regulatory clinical trial—outperforming

NT-proBNP testing. AiTiAMI supports the diagnosis of both STEMI and NSTEMI. Its efficacy was validated in the ROMIAE study (European Heart Journal, IF 39.3), a prospective trial involving over 8,500 chest pain patients across 18 centers. AiTiAMI achieved AUROC >0.93 for all AMI types, surpassing initial physician ECG readings and troponin tests.

Both products are deployed on-premise within hospital IT systems, ensuring high data security and real-time integration with existing ECG machines and EMRs. Currently, over 90 hospitals in Korea utilize the technology with more than 100,000 monthly paid prescriptions.

This presentation will illustrate how a clinical need led to real-world innovation and demonstrate how AI-ECG solutions are transforming everyday cardiovascular care.

CURRICULUM VITAE

SungA Bae

Assistant Professor, Yonsei University, Republic of Korea



Education and Training

2016–2021	Doctor of Philosophy, Chonnam National University School of Medicine
2010–2014	M.D, Master, Chonnam National University School of Medicine
2003–2010	Bachelor, Chonnam National University College of Engineering

Employment and Position

2021–Present	Assistant Professor, Yongin Severance Hospital, Yonsei University College of Medicine
2020	Echocardiography Fellowship in Cardiology, Korea University Anam Hospital
2019	Clinical Instructor, Department of Cardiology, Chonnam National University School of Medicine and Hospital
2015–2018	Resident, Department of Internal Medicine, Chonnam National University School of Medicine and Hospital

Important Publications

1. Bae S, et al. Early Invasive Strategy Based on the Time of Symptom Onset of Non-ST-Segment Elevation Myocardial Infarction. JACC Cardiovasc Interv. 2023 Jan 9;16(1):64-75.
2. Cha JJ, Bae S, et al. Clinical Outcomes in Patients With Delayed Hospitalization for Non-ST-Segment Elevation Myocardial Infarction. J Am Coll Cardiol. 2022 Feb 1;79(4):311-323.
3. Han C, Kim DW, Kim S, Chan You S, 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 Jan 24;27(2):109022.

Artificial intelligence-based coronary artery disease screening versus usual care for patients with suspected coronary artery disease: AI-Gatekeeper randomized controlled trial

Background: Coronary artery disease (CAD) remains the leading cause of cardiovascular mortality globally, yet current diagnostic approaches often fail to accurately identify patients who would benefit from early intervention. More than half of patients undergoing invasive coronary angiography show normal or non-obstructive disease, representing substantial healthcare costs and unnecessary procedural risks.

Methods: This multicenter, prospective, randomized, open-label trial compared artificial intelligence (AI)-Gatekeeper-assisted care with usual care in patients with suspected CAD. AI-Gatekeeper integrated chest radiography, electrocardiography, echocardiography, and clinical risk factors to predict significant CAD. The co-primary endpoints were major adverse cardiovascular events (MACE) and unnecessary utilization of advanced cardiac imaging at 24 weeks.

Results: A total of 450 participants were randomly assigned to AI-Gatekeeper-assisted care (n=221) or usual care (n=229). MACE occurred in 0.5% of par-

ticipants in the AI-Gatekeeper group compared to 3.7% in the usual care group. Unnecessary utilization of advanced cardiac imaging occurred in 8.8% of participants in the AI-Gatekeeper group compared with 37.0% in the usual care group (odds ratio, 0.24; 95% confidence interval [CI], 0.15 to 0.40; P<0.001). Moreover, the total healthcare costs were significantly lower in the AI-Gatekeeper group (median difference, -\$122.5; 95% CI, -\$131.0 to -\$23.9; P<0.001). AI-Gatekeeper demonstrated 89.8% diagnostic accuracy with 93.0% specificity for detecting obstructive coronary artery disease.

Conclusions: AI-Gatekeeper-assisted care markedly reduced MACE and unnecessary cardiac imaging, while lowering healthcare costs in patients with suspected CAD. These findings support the clinical implementation of multimodal AI systems for cardiovascular diagnostic evaluation.

Trial registration number: NCT06178900.

Symposium 6

CVD Risk Prediction in Special Population

Sep 11(Thu) 14:40–16:10 | Room 3 (3F)

CHAIRPERSONS : Hyeon Chang Kim (Yonsei University, Republic of Korea)
SungHee Choi (Seoul National University, Republic of Korea)

- | | |
|-------------|---|
| 14:40–15:00 | CVD risk prediction in low-risk population
Hokyou Lee (Yonsei University, Republic of Korea) |
| 15:00–15:20 | CVD risk prediction in older persons
Sangwoo Park (University of Ulsan, Republic of Korea) |
| 15:20–15:40 | CVD risk prediction in diabetes and high-risk patients
Eu Jeong Ku (Seoul National University, Republic of Korea) |
| 15:40–16:10 | Panel Discussion
Se-eun Park (Sungkyunkwan University, Republic of Korea)
Yong-Jae Lee (Yonsei University, Republic of Korea)
Keum Ji Jung (Yonsei University, Republic of Korea) |

CURRICULUM VITAE**Hokyou Lee**

Associate Professor, Yonsei University, Republic of Korea

**Education and Training**

2008	UC Berkeley, CA, USA, BS, Chemistry
2013	Yonsei University, Korea, MD, Medicine
2022	Yonsei University, Korea, PhD, Preventive Medicine

Employment and Position

2013–2018	Internal Medicine, Severance Hospital, Intern/Resident
2018–2024	Preventive Medicine, Yonsei University, Fellow
2022–2024	Preventive Medicine, Yonsei University, Assistant Professor
Present	Preventive Medicine, Yonsei University, Associate Professor

Important Publications

1. Very High Prevalence of Non-Optimally Controlled Traditional Risk Factors At the Onset of Cardiovascular Disease. J Am Coll Cardiol 2025 Oct.
2. Association of Cumulative Cardiovascular Health Score Through Young Adulthood with Cardiovascular and Kidney Outcomes in Midlife. JAMA Cardiol 2025 Oct.
3. Risk prediction of cardiovascular disease in the Asia-Pacific region: the SCORE2 Asia-Pacific model. Eur Heart J 2025;46(8):702–715.
4. Blood Pressure Classification Using the 2017 ACC/AHA Guideline and Heart Failure in Patients With Cancer. J Clin Oncol 2023;41(5):980–990.
5. 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.

Awards and Honors

2023	Wunsch Medical Award for Young Medical Scientist, awarded by the Korean Academy of Medical Science
2023	100 Next Leaders 2023, selected by the Sisa Journal
2022	Daewoong Scientific Award, by Daewoong Foundation

Research Interest

Preventive Cardiology, Clinical Epidemiology, Risk Prediction

CVD risk prediction in low-risk population

CURRICULUM VITAE

Sangwoo Park

Department of Cardiology, Associate Professor, Ulsan University Hospital, Republic of Korea



Education and Training

2010.02	Inje University, College of Medicine, Korea, Medicine
2015.02	Ulsan University, Korea, M.D., Internal Medicine
2023.02	Ulsan University, Korea, Ph.D., Internal Medicine

Employment and Position

2011–2015	Asan Medical Center, Residency, Internal medicine
2018–2019	Asan Medical Center, Fellowship, Department of Cardiology
2019–2025.8	Ulsan University Hospital, Assistant professor, Department of Cardiology
2025.9–	Ulsan University Hospital, Associate professor, Department of Cardiology

Important Publications

1. Park S, Jeon YJ, Ann SH, Kim YG, Lee Y, Choi SH, Han S, Park GM. Comprehensive Prediction of Subclinical Coronary Atherosclerosis in Subjects Without Traditional Cardiovascular Risk Factors. *Am J Cardiol.* 2023 Jul 1;198:64–71.
2. Park S, Kim YG, Ann SH, Cho YR, Kim SJ, Han S, Park GM. Prediction of the 10-year risk of atherosclerotic cardiovascular disease in the Korean population. *Epidemiol Health.* 2023;45:e2023052.
3. Park S, Park SJ, Park DW. Percutaneous Coronary Intervention for Left Main Coronary Artery Disease: Present Status and Future Perspectives. *JACC Asia.* 2022 Mar 15;2(2):119–138.
4. Park S, Ahn JM, Kim TO, Park H, Kang DY, Lee PH, Jeong YJ, Hyun J, Lee J, Kim JH, Yang Y, Choe K, Park SJ, Park DW. Revascularization in Patients With Left Main Coronary Artery Disease and Left Ventricular Dysfunction. *J Am Coll Cardiol.* 2020 Sep 22;76(12):1395–1406.

Research Interest

Atherosclerosis, Primary prevention, Optimal medical therapy

CVD risk prediction in older persons

With global population aging, accurately assessing cardiovascular risk in older adults is a growing priority. However, cardiovascular risk prediction in older adults presents unique challenges due to atypical disease presentations, prevalent multimorbidity, polypharmacy, and the significant impact of frailty. Traditional risk models like the Framingham Risk Score and Pooled Cohort Equations, originally developed for middle-aged populations, often over- or underestimate risk in the elderly. Newer models (such as SCORE2-OP for Europeans aged ≥ 70) and efforts to incorporate factors like novel biomarkers and functional status, have slightly improved prediction, but important challenges remain.

Studies consistently find that the predictive performance of these models decreases when used in diverse elderly cohorts, especially outside their development populations. Many models classify nearly all individuals over 75 as "very high risk" based primarily on age, which limits meaningful individualization. Guidelines now increasingly recognize these short-

comings. The European Society of Cardiology recommends SCORE2-OP for older adults but acknowledges its age-dominant bias. American guidelines continue to advise PCE use, but with caution due to calibration issues, particularly in older patients and those with diabetes. Across all guidelines, there is a call for risk models that incorporate geriatric-specific factors such as frailty, multimorbidity, and functional status. However, practical barriers—like limited external validation and integration of new variables into everyday clinical workflows—slow progress.

In summary, while new tools have advanced cardiovascular risk assessment in older adults, further improvement is urgently needed. Models must move beyond age alone, integrating clinical, functional, and social variables, and leveraging advanced approaches like Artificial Intelligence to process complex, multi-omics data and enable more personalized, accurate predictions. However, careful validation across diverse elderly populations is crucial for safer, more personalized prevention strategies.

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, Korea, M.D., Medicine
2013.08	Seoul National University, Korea, M.S., Clinical Medical Sciences
2021.02	Seoul National University, Korea, Ph.D., Translational Medicine

Employment and Position

2008–2009	Seoul National University Hospital, Intern
2009–2013	Seoul National University Hospital, Resident
2013–2015	Seoul National University Bundang Hospital, Instructor (Fellowship)
2015–2023	Chungbuk National University Hospital, Assistant/Associate/Professor
2023–Present	Seoul National University Hospital Healthcare System Gangnam Center, Associate Professor

Important Publications

1. Ku EJ, et al. Fenofibrate to prevent amputation and reduce vascular complications in patients with diabetes: FENO-PREVENT. *Cardiovasc Diabetol*. 2024 Sep 3;23(1):329.
2. Ku EJ, et al. Long-Term Effectiveness of Quadruple Combination Therapy with Empagliflozin Versus Basal Long-Acting Insulin Therapy in Patients with Type 2 Diabetes: 3-Year Retrospective Observational Study. *Diabetes Ther*. 2023 Sep;14(9):1471–1479.
3. Choi SH and Ku EJ, et al. Grave-to-cradle: human embryonic lineage tracing from the postmortem body. *Exp Mol Med*. 2023 Jan;55(1):13–21.
4. Ku EJ, et al. The Anti-Atherosclerosis Effect of Anakinra, a Recombinant Human Interleukin-1 Receptor Antagonist, in Apolipoprotein E Knockout Mice. *Int J Mol Sci*. 2022 Apr 28;23(9):4906.
5. Ku EJ, et al. Discovery of plasma biomarkers for predicting the severity of coronary artery atherosclerosis by quantitative proteomics. *BMJ Open Diabetes Res Care*. 2020 Apr;8(1):e001152.

Awards and Honors

AASD Presentation Award, Asian Association for the Study of Diabetes, 2021
Best Poster Oral Presentation Award, Korean Endocrine Society, 2023
Research Award, Adrenal Disease Working Group, Korean Endocrine Society, 2023

CVD risk prediction in diabetes and high-risk patients

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality in patients with diabetes and other high-risk conditions. Traditional risk calculators, such as the Framingham Risk Score and pooled cohort equations, frequently underestimate risk in individuals with diabetes, chronic kidney disease, or multiple comorbidities. Findings from large cohort studies and real-world data suggest that more specific approaches are required for these populations.

In this presentation, I will review current evidence on CVD risk prediction in diabetes and high-risk groups, highlighting the limitations of established models and potential avenues for refinement. I will address diabetes-related factors, including disease du-

ration, glycemic variability, hypoglycemia, and complications such as nephropathy, along with emerging biomarkers. Imaging modalities such as coronary artery calcium scoring and carotid intima-media thickness also provide valuable insights for risk assessment.

Improved prediction should translate into better patient care. Individualized risk stratification can guide the use of cardioprotective therapies, including statins, SGLT2 inhibitors, and GLP-1 receptor agonists, as well as inform comprehensive management strategies. I will emphasize that moving beyond general approaches to individualized strategies may improve cardiovascular outcomes in patients with diabetes and other high-risk conditions.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Symposium 7

Redefining Cardiovascular Care: PCSK9 Mechanisms and siRNA-based Advances

Sep 11(Thu) 14:40–16:10 | Room 4 (5F)

CHAIRPERSONS : Jun-Hee Lee (Hallym University, Republic of Korea)

Sungha Park (Yonsei University, Republic of Korea)

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- | | |
|-------------|--|
| 14:40–15:00 | Deep dive into PCSK9: the master regulator of LDL metabolism and inflammation
Hyun-Duk Jang (Seoul National University, Republic of Korea) |
| 15:00–15:20 | siRNA-based lipid therapies: a revolution in cholesterol management
Jin Wi (Gachon University, Republic of Korea) |
| 15:20–15:40 | Familial hypercholesterolemia treatment beyond statin
Rodrigo Alonso (Center for Advanced Metabolic Medicine and Nutrition, Chile) |
| 15:40–16:10 | Panel Discussion
Soo-Jin Kim (Kosin University, Republic of Korea)
Ye Seul Yang (Seoul National University, Republic of Korea)
Sangmo Hong (Hanyang University, Republic of Korea) |
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CURRICULUM VITAE

Hyun-Duk Jang

Associate Professor, Seoul National University Hospital, Republic of Korea



Education and Training

1997.02	Ewha Womans University, Korea, B.S., Science Education (Biology)
1999.02	Ewha Womans University, Korea, M.S., Genetics
2005.08	Ewha Womans University, Korea, Ph.D., Immunology

Employment and Position

2003–2005	University of Pennsylvania, Immunology, Visiting student
2005–2007	UCLA, Hematology-Oncology, Post-doctoral fellow
2008–2011	Ewha Womans University, Molecular Biopharmaceuticals, Research Professor
2012–2014	Seoul National University Hospital, Biomedical Research Institute, Research Professor
2015–Current	Seoul National University Hospital, Biomedical Research Institute, Assistant/Associate Professor
2022–Current	Seoul National University Hospital, Biomedical Research Institute, Director of Biomarker Center

Important Publications * First or Corresponding author

1. Shin D, Kim S, Lee H, Lee HC, Lee J, Park HW, Fukai M, Choi E, Choi S, Koo BJ, Yu JH, No G, Cho S, Kim CW, Han D, Jang HD*, Kim HS. PCSK9 stimulates Syk, PKC δ , and NF- κ B, leading to atherosclerosis progression independently of LDL receptor. *Nat Commun.* 2024 Mar 30;15 (1):2789.
2. Yang HM, Kim J, Shin D, Kim JY, You J, Lee HC, Jang HD, Kim HS. Resistin impairs mitochondrial homeostasis via cyclase-associated protein 1-mediated fission, leading to obesity-induced metabolic diseases. *Metabolism.* 2023 Jan;138:155343.
3. Jang HD*, Lee SE, Yang J, Lee HC, Shin D, Lee H, Lee J, Jin S, Kim S, Lee SJ, You J, Park HW, Nam KY, Lee SH, Park SW, Kim JS, Kim SY, Kwon YW, Kwak SH, Yang HM, Kim HS. Cyclase-associated protein-1 (CAP1) is a binding partner of proprotein convertase subtilisin/kexin type-9 (PCSK9) and is required for the degradation of low-density lipoprotein receptors by PCSK9. *Eur Heart J.* 2020 Jan 7;41 (2):239–252.
4. Kim YC, Lee SE, Kim SK, Jang HD*, Hwang I, Jin S, Hong EB, Jang KS, Kim HS. Toll-like receptor-mediated inflammation requires FASN-dependent MYD88 palmitoylation. *Nat Chem Biol.* 2019 Sep; 15 (9):907–916.
5. Jang HD*, Hwang HZ, Kim HS, Lee SY. c-Cbl negatively regulates TRAF6-mediated NF- κ B activation by promoting K48-linked ubiquitination of TRAF6. *Cell Mol Biol Lett.* 2019 May 14; 24:29.

Deep dive into PCSK9: the master regulator of LDL metabolism and inflammation

Proprotein convertase subtilisin/kexin type-9 (PCSK9), which degrades low-density lipoprotein receptor (LDLR) and elevates LDL-cholesterol (LDL-C) levels, is used for atherosclerosis treatment. Recently, we demonstrated that PCSK9 interacts with adenylyl cyclase-associated protein 1 (CAP1) that mediates endocytosis and degradation of LDLR. Here, we investigated whether PCSK9 binding to CAP1 induces inflammation directly or independently of LDLR. The direct inflammatory action of PCSK9 is examined *in vitro* in monocytes and endothelial cells, as well as via an *in vivo* atherosclerosis animal model. PCSK9 exacerbates atherosclerosis in LDLR^{-/-} mice independently of the LDLR pathway. Here we show that CAP1 is the main binding partner of PCSK9 and indispensable for the inflammatory action of PCSK9, including in-

duction of cytokines, Toll like receptor 4 (TLR4), and scavenger receptors, enhancing the uptake of oxidized LDL. We find spleen tyrosine kinase (Syk) and protein kinase C delta (PKC δ) to be the key mediators of inflammation after PCSK9-CAP1 binding. In human peripheral blood mononuclear cells (PBMCs), serum PCSK9 levels are positively correlated with Syk, PKC δ , and p65 phosphorylation. The CAP1-fragment crystallizable region (CAP1-Fc) shows superior efficacy in mitigating PCSK9-mediated inflammatory signal transduction when compared with the PCSK9 inhibitor, evolocumab.

Keywords: Proprotein convertase subtilisin/kexin type-9 (PCSK9), adenylyl cyclase-associated protein 1 (CAP1), low-density lipoprotein receptor, LDL-cholesterol (LDL-C), inflammation, atherosclerosis.

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

2005–2010	Severance Hospital, Intern & Resident, Internal Medicine
2010–2013	Severance Hospital, Clinical Fellow, Cardiology
2013–2019	Severance Hospital, Assistant Professor, Cardiology
2019–	Gil Medical Center, Professor, Cardiology

Important Publications

1. Dose Optimization of Meropenem in Patients on Veno-Arterial Extracorporeal Membrane Oxygenation in Critically Ill Cardiac Patients: Pharmacokinetic/Pharmacodynamic Modeling. *J Clin Med* 2022;11:6621 (IF 5.098).
2. 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).
3. 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).
4. Association of vitamin D deficiency with profound cardiogenic shock in patients resuscitated from sudden cardiac arrest. *Shock* 2020;53:717-722 (IF 3.533).
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

siRNA-based lipid therapies: a revolution in cholesterol management

The burden of atherosclerotic disease worldwide necessitates implementing the treatment of its risk factors. Among them, hypercholesterolemia has a central role. There is an unmet clinical need to reduce residual cardiovascular risk attributable to apolipoprotein B-containing lipoproteins, particularly low-density lipoprotein and remnant particles. In addition to conventional small organic compounds, the recently introduced monoclonal antibodies and new technologies are arising.

Pharmacological targeting of mRNA represents an emerging, innovative approach. Two major classes of agents have been developed – antisense oligonucleotides (ASO) and small interfering RNA (siRNAs). Early problems with their use have been overcome by conjugation with N-acetylgalactosamine, an adduct that

targets their delivery to the primary site of action in the liver. Using these agents to inhibit the translation of key regulatory proteins such as PCSK9, apolipoprotein CIII, apolipoprotein(a), and angiopoietin-like 3 has been shown to be effective in attenuating dyslipidemic states. Cardiovascular outcome trials with RNA-targeting drugs are ongoing. The advantages of these agents include long dosing intervals and the potential to regulate the abundance of any disease-related protein. Long-term safety has yet to be demonstrated in large-scale clinical trials.

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

Rodrigo Alonso

CoDirector, Physician staff, Center for Advanced Metabolic Medicine and Nutrition, Chile



Education and Training

1987.12	Universidad de Valparaíso, Chile, M.D, Medicine
1992.12	Universidad de Valparaíso, Chile, Specialty, Internal Medicine
1994.12	Universidad Autónoma Madrid, Spain, PhD, Medicine
1994	Universidad Autónoma Madrid, Spain, Magister, Clinical Nutrition
2022	National Lipid Association, USA, Fellow, Lipids
2022	CONACEM, Chile, Specialty, Clinical Nutrition

Employment and Position

1997–2014	Fundación Jiménez Díaz, Madrid, Associate physician (Lipid Clinic)
2014–2020	Clínica Las Conde, Santiago Chile, Staff, Head Department Clinical Nutrition
2020–Today	Center for Advanced Metabolic Medicina, CoDirector, Staff
2016–Today	AterosChile Corporation, President

Important Publications

1. de Isla LP, Vallejo-Vaz AJ, Watts GF, Muñoz-Grijalvo O, Alonso R, et al. Long-term sex differences in atherosclerotic cardiovascular disease in individuals with heterozygous familial hypercholesterolaemia in Spain: a study using data from SAFEHEART, a nationwide, multicentre, prospective cohort study. *Lancet Diabetes Endocrinol.* 2024;12:643–652.
2. Alonso R, Arroyo-Olivares R, Díaz-Díaz JL, et al. Improved lipid-lowering treatment and reduction in cardiovascular disease burden in homozygous familial hypercholesterolemia: The SAFEHEART follow-up study. *Atherosclerosis* 2024 Mar 16;16:117516. Doi: 10.1016/j.atherosclerosis.2024.117516.
3. Alonso R, Arroyo-Olivares R, Muñoz-Grijalvo O, et al. Persistence with long-term PCSK9 inhibitors treatment and its effectiveness in familial hypercholesterolemia: Data from the SAFEHEART study. *Eur J Prev Cardiol* 2022; doi: 10-1093/eurjpc/zwac277.
4. Ray K, Ference B, Severin T, Blom D, Nichols S, Shiba M, Almahmeed W, Alonso R, et al. World Heart Federation Cholesterol Roadmap 2022. *Global Heart* 2022;17(1). DOI:https://doi.org/10.5334/gh.1154.

Familial hypercholesterolemia treatment beyond statin

Familial hypercholesterolemia (FH) is a common genetic disorder characterized by lifelong elevated LDL-cholesterol and markedly increased risk of premature atherosclerotic cardiovascular disease (ASCVD). Statins remain the cornerstone of therapy in FH, and observational studies have shown, that early initiation—particularly in childhood—reduces long-term cardiovascular events. However, most FH patients fail to achieve recommended LDL-C targets with high intensity statins, and require additional medications to further reduce LDL-C levels. Over the past decade, several therapies have emerged and transformed the management of FH. Ezetimibe, an inhibitor of intestinal cholesterol absorption, provides an additional 17–20% reduction in LDL-C levels, with no major safety concerns. More recently, PCSK9 inhibitors (alirocumab, evolocumab) have provided potent and sustained

LDL-C reduction of 50–60% on top of statins, along with demonstrated effects on atherosclerotic disease in FH. Long-term persistence with PCSK9 inhibitors has also proven to be very high in real-world registries. Inclisiran, a small interfering RNA targeting PCSK9, offers similar efficacy with biannual dosing, improving convenience and adherence. Bempedoic acid is an additional oral option, particularly valuable in statin-intolerant patients.

For homozygous FH, where LDL receptor function is severely impaired, therapies independent of the LDLR pathway—such as lomitapide, evinacumab, and lipoprotein apheresis—are essential. Emerging therapies targeting PCSK9, ANGPTL3, CETP, and lipoprotein(a) promise further advances. Early diagnosis, access to novel therapies, and comprehensive management strategies are critical to improving outcomes in FH.

Symposium 8

Recent Prognostic Evidence for the Triglyceride–Glucose (TyG) Index as a Novel Surrogate Marker of Insulin Resistance

Sep 11(Thu) 16:20–17:50 | Room 1 (3F)

CHAIRPERSONS : **Weon Kim** (Kyung Hee University, Republic of Korea)
Junghwan Park (Hanyang University, Republic of Korea)

- | | |
|-------------|---|
| 16:20–16:40 | Prediction of ASCVD using the TyG index: insights from the multinational PURE registry
Patricio Lopez-Jaramillo (Universidad de Santander, Colombia) |
| 16:40–17:00 | Prediction of ASCVD using the TyG index: evidence from Korean data
Yong-Jae Lee (Yonsei University, Republic of Korea) |
| 17:00–17:20 | Prediction of coronary artery calcification progression using the TyG index
Ki-Bum Won (Chung-Ang University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Kyung Woo Park (Seoul National University, Republic of Korea)
Se-eun Park (Sungkyunkwan University, Republic of Korea)
Jun-Hee Lee (Hallym University, Republic of Korea) |

CURRICULUM VITAE

Patricio Lopez-Jaramillo

General Rector and Professor Masira Research Institute, Medical School,
Universidad de Santander (UDES), Colombia



Education and Training

1978.07 Universidad Central del Ecuador, M.D., Medicine
1984.09 Universidad de Sao Paulo, Brasil, PhD., Pharmacology

Employment and Position

2022- Universidad de Santander (UDES), General Rector
2005- Masira Research Institute UDES, Professor and Scientific Director

Important Publications

1. Cardiovascular disease in the Americas: the epidemiology of cardiovascular disease and its risk factors. Joseph P, Lanas F, Roth G, Lopez-Jaramillo P, Lonn E, Miller V, Mente A, Leong D, Schwalm JD, Yusuf S. Lancet Reg Health Am. 2025 Feb 14;42:100960. doi: 10.1016/j.lana.2024.100960. eCollection 2025 Feb.PMID: 40034110.

2. External validation and comparison of six cardiovascular risk prediction models in the Prospective Urban Rural Epidemiology (PURE)-Colombia study. Lopez-Lopez JP, Garcia-Pena AA, Martinez-Bello D, Gonzalez AM, Perez-Mayorga M, Muñoz Velandia OM, Ruiz-Urbe G, Campo A, Rangarajan S, Yusuf S, Lopez-Jaramillo P. Eur J Prev Cardiol. 2025 May 12;32(7):564-572. doi: 10.1093/eurjpc/zwae242.PMID: 39041366.

3. Physical Activity and Obesity Risk in Adults in Colombia: The Prospective Urban Rural Epidemiology (PURE) Study. O'Donovan G, Martínez D, López-López JP, Otero J, Urina M, Vasquez T, Niño M, Narvaez C, Campo MC, Perez-Mayorga M, Rodríguez S, Arcos E, Sanchez G, García H, Rangarajan S, Yusuf S, López-Jaramillo P. Med Sci Sports Exerc. 2024 Jul 1;56(7):1291-1296. doi: 10.1249/MSS.0000000000003413. Epub 2024 Feb 23.PMID: 38648672.

Prediction of ASCVD using the TyG index:
insights from the multinational PURE registry

The TyG index is an easily accessible surrogate marker of insulin resistance (IR). We assessed the association of IR with mortality and cardiovascular diseases (CVD) in individuals from five continents at different levels of economic development. We also examined whether the associations differed according to the country's economic development. Fasting triglycerides and fasting plasma glucose were measured at the baseline visit in 141 243 individuals aged 35-70 years from 22 countries in the Prospective Urban Rural Epidemiology (PURE) study. The TyG index was calculated as $\ln(\text{fasting triglycerides [mg/dL]} \times \text{fasting plasma glucose [mg/dL]}/2)$. We calculated hazard ratios (HRs) using a multivariable Cox frailty model with random effects to test the associations between the TyG index and risk of CVD and mortality. The primary outcome was the composite of mortality or major cardiovascular events (defined as death from cardiovascular causes, and non-fatal myocardial infarction, or stroke). Secondary outcomes were non-cardiovascular mortality, cardiovascular mortality, all myocardial in-

farctions, stroke, and incident diabetes. We also did subgroup analyses to examine the magnitude of associations between IR and outcome events according to the income level of the countries. During a median follow-up of 13.2 years, we recorded 6345 composite events, 2030 cardiovascular deaths, 3038 cases of MI, 3291 cases of stroke, and 5191 incident cases of DM2. After adjusting for all other variables, the risk of developing CVD increased across tertiles of the baseline TyG index. Compared with the lowest tertile, the highest tertile was associated with a greater incidence of the composite outcome, MI, stroke, and DM2. In LICs and MICs, the highest tertile was associated with increased hazards for the composite outcome, cardiovascular mortality, MI, stroke, and DM2. In contrast, in HICs, higher tertiles were only associated with an increased DM2, but not of CVD or mortality. The TyG index is significantly associated with future cardiovascular mortality, MI, stroke, and DM2 suggesting that insulin resistance plays a promoting role in the pathogenesis of cardiovascular and metabolic diseases.

CURRICULUM VITAE

Yong-Jae Lee

Professor, Yonsei University, Republic of Korea



Education and Training

1999.02	Yonsei University College of Medicine, Seoul, Korea, M.D, Medicine
2005.08	Yonsei University Graduate School of Public Health, Seoul, M.P.H, Public Health
2013.08	Yonsei University Graduate School of Medicine, Seoul, Ph.D, Medical Science

Employment and Position

2009–2015	Yongin-Severance Hospital, Clinical Assistant Professor
2015–2019	Gangnam Severance Hospital, Associate Professor
2019–	Gangnam Severance Hospital, Professor

Important Publications

1. Triglyceride to HDL-cholesterol ratio and the incidence risk of type 2 diabetes in community dwelling adults: A longitudinal 12-year analysis of the Korean Genome and Epidemiology Study. *Diabetes Research and Clinical Practice* 2020;163: e108150.
2. Triglyceride glucose (TyG) index as a predictor of incident type 2 diabetes among nonobese adults: a 12-year longitudinal study of the Korean Genome and Epidemiology Study cohort. *Translational Research* 2021;228(2):42–51.
3. The triglyceride-glucose (TyG) index is a more powerful surrogate marker for predicting the prevalence and incidence of type 2 diabetes mellitus than the homeostatic model assessment of insulin resistance. *Diabetes Research and Clinical Practice* 2021;180:e109042.
4. Triglyceride Glucose-Waist Circumference (TyG-WC) Is a Reliable Marker to Predict Non-Alcoholic Fatty Liver Disease. *Biomedicines* 2022;10(9):2251.
5. Modified triglyceride-glucose index (TyG) indices are reliable markers for predicting risk of metabolic dysfunction-associated fatty liver disease (MAFLD): a cross-sectional study. *Frontiers in Endocrinology*, 2024;14(1):1308265.

Awards and Honors

Outstanding Achievement Professor (Research), Yonsei University College of Medicine (2019)
 Professor of the Year, Yonsei University College of Medicine (2022)
 Outstanding Achievement Professor (Education), Yonsei University College of Medicine (2025)

Research Interest

Clinical epidemiology of cardiometabolic diseases

Prediction of ASCVD using the TyG index: evidence from Korean data

The triglyceride-glucose (TyG) index has been identified as a reliable alternative biomarker of insulin resistance. Recently, a considerable number of studies have provided robust statistical evidence suggesting that the TyG index is associated with the development and prognosis of cardiovascular disease (CVD) including stable coronary artery disease, acute coronary syndrome, arterial stiffness, coronary calcification, and heart failure in particularly among East Asian populations. Based on the current evidence from ob-

servational studies in South Korea, a high TyG index may have a significant relationship with the incidence of coronary artery disease, acute coronary syndrome and CVD but not cardiovascular mortality or all-cause mortality in the general population. Considering the limited evidence from non-Asian populations and possible bias due to diabetes, further prospective studies in non-diabetes and non-Asian populations are needed to explore the association of the TyG index with CVD incidence and mortality.

CURRICULUM VITAE**Ki-Bum Won**

Associate Professor, Chung-Ang University College of Medicine, Republic of Korea

**Education and Training**

2005.02	Keimyung University, Korea, M.D, Medicine
2021.02	Keimyung University, Korea, Ph.D., Internal Medicine

Employment and Position

2016–2022	Ulsan University, Assistant Professor
2022–2024	Dongguk University, Associate Professor
2024–Present	Chung-Ang University, Associate Professor

Important Publications

1. Different associations of atherogenic index of plasma, triglyceride glucose index, and hemoglobin A1C levels with the risk of coronary artery calcification progression according to established diabetes. *Cardiovasc Diabetol* 2024; 23: 418.
2. Body mass index and major adverse events during chronic antiplatelet monotherapy after percutaneous coronary intervention with drug-eluting stents - Results from the HOST-EXAM Trial. *Circ J* 2023; 87: 268–276.
3. Glycemic control is independently associated with rapid progression of coronary atherosclerosis in the absence of a baseline coronary plaque burden: a retrospective case-control study from the PARADIGM registry. *Cardiovasc Diabetol* 2022; 21: 239.
4. Atherogenic index of plasma and the risk of rapid progression of coronary atherosclerosis beyond traditional risk factors. *Atherosclerosis* 2021; 324: 46–51.
5. Independent role of low-density lipoprotein cholesterol in subclinical coronary atherosclerosis in the absence of traditional cardiovascular risk factors. *Eur Heart J Cardiovasc Imaging* 2019; 20: 866–872.

Awards and Honors

Professor of Excellence in Research at University of Ulsan College of Medicine (2020)

Research Interest

Cardiac CT, Coronary intervention, Metabolic syndrome, diabetes

Prediction of coronary artery calcification progression using the TyG index

The triglyceride glucose (TyG) index has emerged as a more practical and reliable predictor of insulin resistance (IR) compared to the traditional methods including the hyperinsulinemic-euglycemic clamp test and the homeostasis model assessment of IR index. Recent data have shown a significant association between TyG index and subclinical atherosclerosis in the field of primary prevention of cardiovascular (CV) disease. In the asymptomatic adult population, the coronary artery calcium score (CACS) has been widely

used to stratify the risk of major adverse CV events, providing additional prognostic values beyond traditional CV risk factors. Among biomarkers for assessing IR, TyG index is known to have a strong association with the risk of coronary artery calcification (CAC) progression irrespective of established diabetes. The predictive value of TyG index for CAC progression is more significant in the asymptomatic population of adults without heavy CAC at baseline.

Symposium 9

How Low is Low Enough for LDL-C in Diabetes?

Sep 11(Thu) 16:20–17:50 | Room 3 (3F)

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

- | | |
|-------------|--|
| 16:20–16:40 | LDL-C targets for patients with diabetes across major guidelines
Jong Han Choi (Konkuk University, Republic of Korea) |
| 16:40–17:00 | Pro: all patients with diabetes should be treated to LDL-C <55 mg/dL?
Wonjin Kim (CHA University, Republic of Korea) |
| 17:00–17:20 | Cons: LDL-C targets should be personalized in diabetic patients?
Mihye Seo (Soonchunhyang University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Sungha Park (Yonsei University, Republic of Korea)
SungA Bae (Yonsei University, Republic of Korea)
Ye Seul Yang (Seoul National University, Republic of Korea) |

CURRICULUM VITAE**Jong Han Choi**

Assistant Professor, Division of Endocrinology and Metabolism,
Konkuk University Medical Center, Republic of Korea

**Education and Training**

2010.02	Chonnam National University, Korea, M.D, Medicine
2024.02	Konkuk University, Korea, Ph.D., Internal Medicine

Employment and Position

2010–2015	Asan Medical Center, Intern / Residency
2018–2020	Asan Medical Center, Clinical Fellow
2020–	Konkuk University Medical Center, Assistant Professor

Important Publications

1. JH Choi, BK Koo, YS Yang, SH Min, JS Park, SY Rhee, HJ Kim, MK Moon, Initial Pharmacological Strategies in People with Early Type 2 Diabetes Mellitus: A Systematic Review and Network Meta-Analysis. *Diabetes Metab J*. Published online Apr 29, 2025.
2. JH Choi, MK Moon, HJ Kim, KA Lee, HJ Kim, JH Ko, J-S Yun, S-H Ko, S Chon, NH Kim, Current Status of Delay in Injectable Therapy among Type 2 Diabetes Mellitus Patients in South Korea: Multicenter Retrospective Study (2015–2021), *Endocrinol Metab*. Published online May 29, 2025.
3. JH Choi, C Lulu, S-J Park, H-J Lee, Adherence to the nutritional recommendations according to diabetes status in Korean adults: a cross-sectional study, *BMC Public Health* 2024; 24: 2647.
4. JH Choi, KA Lee, JH Moon, S Chon, et al., 2023 Clinical Practice Guidelines for Diabetes Mellitus of the Korean Diabetes Association. *Diabetes Metab J*. 2023; 47(5): 575–594.
5. JH Choi, M-S Kim, Homeostatic Regulation of Glucose Metabolism by the Central Nervous System, *Endocrinol Metab*. 2022;37(1):9–25.

Awards and Honors

April 2019	Korean Endocrine Society, Plenary Oral Presentation Award
October 2022	Korean Endocrine Society, Big Data Research Award
April 2023	Korean Endocrine Society, Outstanding Abstract Award

Research Interest

Diabetes, Obesity, Methodology of clinical practice guidelines

LDL-C targets for patients with diabetes across major guidelines

Optimal management of low-density lipoprotein cholesterol (LDL-C) is a cornerstone in reducing atherosclerotic cardiovascular disease (ASCVD) risk in patients with diabetes. However, treatment targets for LDL-C vary across international guidelines, reflecting differences in the interpretation of risk stratification, evidence from randomized trials, and regional practice patterns. This presentation provides a comprehensive overview of major contemporary guidelines—including those from the American Diabetes Association (ADA), European Society of Cardiology (ESC), and Korean Dia-

betes Association (KDA)—highlighting their similarities and differences in LDL-C target recommendations for diabetic patients. Special emphasis will be placed on how each guideline defines high or very high risk in diabetes, the thresholds for initiating statin or combination therapy, and the emerging role of non-statin agents such as ezetimibe and PCSK9 inhibitors. Understanding the rationale behind these guidelines is essential for tailoring lipid-lowering strategies that maximize cardiovascular protection while considering individual patient profiles and healthcare system contexts.

CURRICULUM VITAE

Wonjin Kim

Associate Professor, CHA University, Republic of Korea



Education and Training

2008.02	Wonju College of Medicine, Yonsei University, M.D, Medicine
2023.02	Yonsei University, Korea, Ph.D., Internal Medicine

Employment and Position

2009–2013	Severance Hospital, Resident
2015–2025	CHA Gangnam Medical Center, Assistant Professor
2025–Current	CHA Gangnam Medical Center, Associate Professor

Important Publications

1. W Kim, SK Park, YL Kim. Fetal Abdominal Overgrowth is Already Present at 20–24 Gestational Weeks Prior to Diagnosis of Gestational Diabetes Mellitus. *Scientific Reports*. 2021, 11:23821.
2. JH Jang, W Kim, JS Moon, E Roh, JG Kang, SJ Lee, SH I, JH Huh. Association between Sleep Duration and Incident Diabetes Mellitus in Healthy Subjects: A 14-Year Longitudinal Cohort Study. *Journal of Clinical Medicine*. 2023, 12, 2899.
3. HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, KH Chun, SH Han, KK Koh. Metabolic Syndrome Fact Sheet 2024: Executive Report. *Cardiometab Syndr J*. 2024;4:e14.
4. KH Chun, HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, SH Han, KK Koh. Sex-Specific Impact of the COVID-19 Outbreak on the Incidence of Metabolic Syndrome: A Comparative Study of 2018–2019 and 2020–2021. *Korean J Intern Med* 2025 Mar;40(2):262–274. doi: 10.3904/kjim.2024.288. Epub 2025 Mar 1.
5. W Kim, MK Seo, YJ Kim, SH Choi, CR Ku, S Kim, EJ Lee, JS Yoon. Role of the suppressor of cytokine signaling-3 in the pathogenesis of Graves' orbitopathy. *Front. Endocrinol.*, 04 March 2025. <https://doi.org/10.3389/fendo.2025.1527275>.

Pro: all patients with diabetes should be treated to LDL-C <55 mg/dL?

Patients with diabetes are at markedly increased risk for atherosclerotic cardiovascular disease (ASCVD), and intensive low-density lipoprotein cholesterol (LDL-C) reduction has emerged as a cornerstone of risk mitigation. Evidence from large-scale statin and non-statin lipid-lowering trials, including CARDS, HPS, TNT, IMPROVE-IT, FOURIER, and ODYSSEY OUTCOMES, has consistently demonstrated that greater absolute and relative reductions in LDL-C yield proportional declines in major adverse cardiovascular events, without evidence of a threshold below which benefit is lost. Recent analyses suggest a log-linear relationship, whereby each 1 mmol/L ($\approx$ 39 mg/dL) LDL-C reduction confers a $\sim$ 20–25% relative risk reduction, regardless of baseline LDL-C.

In diabetes-specific subgroups, including those from IMPROVE-IT and FOURIER, the magnitude of benefit was even greater, supporting more aggressive treatment goals. Importantly, safety signals such as

hemorrhagic stroke, neurocognitive impairment, or hormonal imbalance have not been substantiated, even at LDL-C levels <20 mg/dL achieved with PCSK9 inhibitors. Consequently, the European Society of Cardiology/European Atherosclerosis Society (ESC/EAS) 2019 guidelines endorse LDL-C <55 mg/dL as the therapeutic target for all patients at very high risk, including those with diabetes and target organ damage or multiple risk factors.

This lecture will critically review the major outcome trials, meta-analyses, and contemporary guideline recommendations, making the case that an LDL-C target <55 mg/dL should not be reserved for a subset of patients with diabetes, but rather represents the optimal strategy for all. The accumulating evidence supports the principle that, for LDL-C in diabetes, “lower is better, and lowest is best”—with <55 mg/dL as a safe and evidence-based universal goal.

CURRICULUM VITAE

Mihye Seo

Associate Professor, Soonchunhyang University College of Medicine, Republic of Korea



Education and 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

- Division of Endocrinology and Metabolism, Department of Internal Medicine, Kangbuk Samsung Hospital, Sungkyunkwan University College of Medicine, Clinical Fellow
- Division of Endocrinology and Metabolism, Department of Internal Medicine, Soonchunhyang University Hospital, Gumi, Soonchunhyang University College of Medicine, Assistant Professor
- Division of Endocrinology and Metabolism, Department of Internal Medicine, Soonchunhyang University Hospital, Seoul, Soonchunhyang University College of Medicine, Associate Professor

Important Publications

1. Mihae Seo, Hyoungnae Kim, Hyunjin Noh, Jin Seok Jeon, Dong Won Byun, Sang Hyun Kim, Hye Jeong Kim, Kyoil Suh, Hyeong Kyu Par, Soon Hyo Kwon, 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.
2. Mi Hae Seo, Yang-Hyun Kim, Kyungdo Han, Jin-Hyung Jung³, Yong-Gyu Park, Seong-Su Lee, HyukSang Kwon, WonYoung Lee, Soon Jib Yoo; on Behalf of the Taskforce Team of the Obesity Fact Sheet of the Korean Society for the Study of ObesityPrevalence of Obesity and Incidence of ObesityRelated Comorbidities in Koreans Based on National Health Insurance Service Health Checkup Data 2006–2015, *Journal of Obesity & Metabolic Syndrome* 2018;27:46–52.
3. Mi Hae Seo, Yang-Hyun Kim, Kyungdo Han, Jin-Hyung Jung³, Yong-Gyu Park, Seong-Su Lee, Hyuk-Sang Kwon, Won Young Lee, Soon Jib Yoo; on Behalf of the Taskforce Team of the Obesity Fact Sheet of the Korean Society for the Study of Obesity, 2018 Korean Society for the Study of Obesity Guideline for the Management of Obesity in Korea *Journal of Obesity & Metabolic Syndrome* 2019;28(1):40–45.

Research Interest

Diabetes, Dyslipidemia, Obesity, Aging, Cardiovascular disease

Cons: LDL-C targets should be personalized in diabetic patients?

It is well recognized that patients with diabetes mellitus (DM) are at increased risk for atherosclerotic cardiovascular disease (ASCVD), and lowering low-density lipoprotein cholesterol (LDL-C) remains a cornerstone of preventive therapy. However, the recent trend toward a universal stringent LDL-C target, such as <55 mg/dL for all individuals with diabetes, warrants critical reassessment. Emerging evidence and clinical experience suggest that rigid, “one-size-fits-all” LDL-C thresholds may not universally benefit all patients with diabetes and may, in certain cases, expose patients to unnecessary medication burden, cost, or adverse events.

This lecture will review the heterogeneous nature of cardiovascular risk among individuals with diabetes, influenced by factors such as age, comorbidity burden,

diabetes duration, and the presence or absence of established ASCVD. The limitations of applying intensive LDL-C lowering indiscriminately will be discussed in light of real-world observational data and secondary analyses of major trials, which indicate that absolute risk reduction and benefit from aggressive lipid-lowering therapy is greatest in those at highest cardiovascular risk. For some patients—particularly young individuals with short diabetes duration, no other risk factors, and no evidence of subclinical atherosclerosis—exceedingly low LDL-C targets may have only marginal benefit, while increasing polypharmacy.

In this presentation, we will specifically focus on the counterpoint that LDL-C targets should be personalized in diabetic patients.

Symposium 10

Socioeconomic Status: The Missing Piece in Cardiovascular Risk Stratification

Sep 11(Thu) 16:20–17:50 | Room 4 (5F)

CHAIRPERSONS : Sung Nim Han (Seoul National University, Republic of Korea)
Jeongseon Kim (National Cancer Center, Republic of Korea)

- 
- | | |
|-------------|---|
| 16:20–16:40 | Mechanisms linking socioeconomic status and cardiovascular risk
SungHee Choi (Seoul National University, Republic of Korea) |
| 16:40–17:00 | Epidemiologic evidence on socioeconomic status and cardiovascular outcomes
Jong-Ha Baek (Gyeongsang National University, Republic of Korea) |
| 17:00–17:20 | New models for CVD risk assessment: incorporating socioeconomic data
Sadiya S. Khan (Northwestern University, USA) |
| 17:20–17:50 | Panel Discussion
Dae-Young Kim (Inha University, Republic of Korea)
Hyun-Jin Kim (Hanyang University, Republic of Korea)
Yongjoo Park (Chonnam National University, Republic of Korea)
Sangmo Hong (Hanyang University, Republic of Korea) |

CURRICULUM VITAE

SungHee Choi

Chief Professor, College of Medicine, Seoul National University &
Seoul National University Bundang Hospital
Endocrinology & Metabolism, Internal Medicine
Director of International Health Care Center, SNUBH, Republic of Korea



Education / Career

- Professor (2017–Present), College of Medicine, Seoul National University, Seoul, Korea & SNUH Bundang Hospital
- Associate Professor (2012–2017), College of Medicine, Seoul National University, Seoul, Korea & SNUH Bundang Hospital
- Medical Instructor/Assistant Professor (2004–2012), College of Medicine, Seoul National University, Korea
- Research Scholar (2009–2011), College of Physicians & Surgeons, Columbia University, NYC, NY, USA : Irving clinical and translational research center, Columbia University Medical Center
- PhD (2006), MSc (2001), MD (1997) Yonsei University College of Medicine
- 1997–2002 Intern, Resident, Clinical Fellow, Yonsei University College of Medicine

Recent Publications List (Orcid 0000-0003-0740-8116)

1. Park SE*, Ko SH*, Kim JY, Kim K, Moon JH, Kim NH, Han KD, Choi SH*, Cha BS*. Diabetes Fact Sheet in Korea 2024. *Diabetes Metab J* 2025;49(1):24–33.
2. Kim HJ, Hur KY, Lee YH, Choi SH. Effect of pravastatin on kidney function and type 2 diabetes mellitus: A multicenter prospective observational study. *Advances in Therapy*, vol. 41, 3119–3137, 2024.
3. Lee YK, Oh TJ, Choi SH. Complementary effects of dapagliflozin and lobeglitazone on metabolism in a diet-induced obese mouse model, *Eur J Pharmacol*, 2023 Aug 2;857:17946.
4. Moo JH, Kim Y, Oh TJ, Jang HC, Choi SH. Triglyceride-Glucose index predicts future atherosclerotic cardiovascular diseases: A 16 year follow up in a prospective, community-dwelling cohort study. *Endocrinol Metab* 2023;38(4):406–417.
5. Kim KH, Choi SH. New, novel lipid lowering agents for reducing cardiovascular risk: beyond statins. *Diabetes Metab J*. 2022 July;46(4):517–532.

Mechanisms linking socioeconomic status and cardiovascular risk

Socioeconomic status (SES) is a powerful determinant of cardiovascular disease (CVD). Lower SES is consistently associated with higher incidence, worse outcomes, and greater mortality, yet the biological and systemic mechanisms linking social disadvantage to cardiovascular risk remain incompletely understood.

The relationship between SES and CVD can be assumed through three interconnected domains. First, behavioral and lifestyle factors—including smoking, dietary quality, and physical inactivity—contribute substantially but do not fully explain SES-related disparities. Second, biological embedding of chronic stress and adverse neighborhood exposures (such as pollution, food insecurity, and noise) induce low-grade inflammation, endothelial dysfunction, and epigenetic modifications that accelerate vascular aging. Third, inequities in access to health care and preventive

therapies amplify these risks by limiting adherence to guideline-based treatment and secondary prevention.

Chronic psychosocial stress and environmental deprivation activate the sympathetic-HPA system and promote maladaptive immunity in myeloid cells. This, in turn, engages NLRP3-IL-1 β signaling, driving fibro-inflammatory responses in vascular and adipose tissue. The resulting insulin resistance, impaired endothelial nitric oxide bioavailability, and accelerated vascular fibrosis converge to promote atherosclerosis and heart failure.

Through behavioral, biologic, and health care pathways. Interventions that combine environmental and policy-level reforms with targeted biological therapies may offer the greatest potential to reduce persistent inequities in cardiovascular outcomes.

CURRICULUM VITAE

Jong-Ha Baek

Associate Professor, MD, PhD, Department of Internal Medicine,
Gyeongsang National University Changwon Hospital,
Gyeongsang National University, Changwon, Republic of Korea



Education and Training

2006	Gyeongsang National University, Korea, M.D, Medicine
2006–2010	Gyeongsang National University Hospital, Residents
2014–2015	Samsung Medical Center, Fellow
2018	Pusan National University, Korea, Ph.D., Internal Medicine

Employment and Position

2016–	Gyeongsang National University Changwon Hospital, Associate Professor
2023–2025	University of Arkansas for Medical Sciences (UAMS), Visiting Professor

Important Publications

1. Income Variability and Incident Cardiovascular Disease in diabetes: A Population-based Cohort Study. Eur Heart J. 2024 Jun 1;45(21): 1920–1933.
2. Artificial Light at Night and Type 2 Diabetes, Diabetes Metab J. 2024 Sep;48(5): 847–863.
3. Comparison of Operational Definition of Type 2 Diabetes Based on Data from Korean National Health Insurance Service and Korea National Health and Nutritional Examination Survey. Diabetes Metab J. 2023 Mar;47(2): 201–210.
4. Real-world Prescription Patterns and Barriers Related to the Use of Sodium-glucose Cotransporters 2 Inhibitors Among Korean Patients with Type 2 Diabetes and Cardiovascular Disease. Diabetes Metab J. 2022 Sep;46(5): 701–712.

Research Interest

Diabetes, Epidemiology

Epidemiologic evidence on socioeconomic status and cardiovascular outcomes

While traditional cardiovascular (CV) risk factors are well-established, the profound impact of socioeconomic status (SES) on CV outcomes is often underestimated in clinical risk assessment. Historically, research examining the influence of SES on CV disease outcomes has predominantly focused on the general population, often utilizing cross-sectional income data derived from area-based or survey-based sources. This approach fails to account for the dynamic nature of income, which is characterized by significant temporal variability and is closely linked to health behaviors. This is particularly relevant for individuals with type 2 diabetes (T2D), who not only represent an independent high-risk group for CV disease but are also more susceptible to the adverse health effects of income changes compared to the general population. This presentation will review robust epidemiologic

evidence from large-scale, population-based cohort studies that demonstrate the critical role of income dynamics as a key determinant of CV health. Drawing from a nationwide study of over 1.5 million adults with type 2 diabetes, we will present compelling data showing that high income variability, sustained low-income status, and sharp income declines are independently associated with a significantly increased risk of incident CV disease, including myocardial infarction, heart failure, and stroke. These findings persist even after adjusting for a comprehensive set of traditional risk factors. The evidence underscores the limitations of current CV risk models that omit socioeconomic data and highlights the urgent need to integrate SES metrics into novel risk assessment tools, such as the PREVENT equations, to achieve more equitable and accurate risk stratification in clinical practice.

CURRICULUM VITAE

Sadiya S. Khan

Magerstadt Professor of Cardiovascular Epidemiology
Associate Professor of Medicine and Preventive Medicine
Director, Center for Population Science and Aging
Northwestern University Feinberg School of Medicine, USA



Education and Training

2005	Northwestern University, Evanston, IL, BS, Biomedical Engineering
2009	Northwestern University, Chicago, IL, MD, Medicine
2014	Northwestern University, Chicago, IL, MSc, Clinical Investigation

Employment and Position

2012–2013	Northwestern University Feinberg School of Medicine (NUFSM), Department of Medicine, Clinical Instructor
2016–2017	NUFSM, Division of Cardiology, Department of Medicine, Clinical Instructor
2017–2023	NUFSM Division of Cardiology, Departments of Medicine and Preventive Medicine, Assistant Professor
2023–	NUFSM Departments of Medicine (Cardiology), Preventive Medicine (Epidemiology) and Medical Social Sciences (Determinants of Health), Magerstadt Professor of Cardiovascular Epidemiology, Associate Professor

Important Publications

1. Khan SS, Matsushita K, Sang Y, Ballew SH, Grams ME, Surapaneni A, Blaha MJ, Carson AP, Chang AR, Ciemins E, Go AS, Gutierrez OM, Hwang SJ, Jassal SK, Kovesdy CP, Lloyd-Jones DM, Shlipak MG, Palaniappan LP, Sperling L, Virani SS, Tuttle K, Neeland IJ, Chow SL, Rangaswami J, Pencina MJ, Ndumele CE, Coresh J. Development and Validation of the American Heart Association's PREVENT Equations. *Circulation*. 2024 Feb 6;149(6):430–449. PubMed Central PMCID: PMC10910659.
2. Khan SS, Coresh J, Pencina MJ, Ndumele CE, Rangaswami J, Chow SL, Palaniappan LP, Sperling LS, Virani SS, Ho JE, Neeland IJ, Tuttle KR, Rajgopal Singh R, Elkind MSV, Lloyd-Jones DM. Novel Prediction Equations for Absolute Risk Assessment of Total Cardiovascular Disease Incorporating Cardiovascular-Kidney-Metabolic Health: A Scientific Statement From the American Heart Association. *Circulation*. 2023 Dec 12;148(24):1982–2004. PubMed PMID: 37947094.
3. Glynn P, Lloyd-Jones DM, Feinstein MJ, Carnethon M, Khan SS. Disparities in Cardiovascular Mortality Related to Heart Failure in the United States. *J Am Coll Cardiol*. 2019 May 14;73(18):2354–2355. PubMed PMID: 31072580.
4. Khan SS, Ning H, Wilkins JT, Allen N, Carnethon M, Berry JD, Sweis RN, Lloyd-Jones DM. Association of Body Mass Index With Lifetime Risk of Cardiovascular Disease and Compression of Morbidity. *JAMA Cardiol*. 2018 Apr 1;3(4):280–287. PubMed Central PMCID: PMC5875319.

New models for CVD risk assessment: incorporating socioeconomic data

In the last century, there has been a global upsurge in the burden of cardiovascular, kidney, and metabolic disease leading to the growing recognition of the interrelated cardiovascular-kidney-metabolic (CKM) syndrome. New risk prediction models, specifically the PREVENT risk equations, were developed to better address the multifaceted, complex, risk factors, including adverse social factors and CKM syndrome, that can impact a patient's risk for cardiovascular dis-

ease (CVD). The PREVENT models have proven to be more accurate in predicting the risk of cardiovascular disease, atherosclerotic cardiovascular disease, and heart failure when compared to previous risk equations. The accuracy of the PREVENT models, along with the emergence of novel CKM therapies, have the potential to transform approaches to primary prevention of cardiovascular disease in a more equitable and comprehensive way.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Symposium 11

Genetic Insights into Dyslipidemia and Diabetes: Pathways, Predictions, and Interventions

Sep 12(Fri) 08:30–10:00 | Room 1 (3F)

CHAIRPERSONS : Hyun Jae Kang (Seoul National University, Republic of Korea)
Byung-Wan Lee (Yonsei University, Republic of Korea)

08:30–08:50 **Exploring the genetic landscape of CVD and diabetes: finding and future implication**

Soo Heon Kwak (Seoul National University, Republic of Korea)

08:50–09:10 **Multi-omics and multi-trait analysis of metabolic disease**

Hong-Hee Won (Sungkyunkwan University, Republic of Korea)

09:10–09:30 **Gene-targeting therapy in familial hypercholesterolemia**

Sang-Hak Lee (Yonsei University, Republic of Korea)

09:30–10:00 **Panel Discussion**

Yoo-Wook Kwon (Seoul National University, Republic of Korea)

Hee-Dong Kim (Soonchunhyang University, Republic of Korea)

Young Shin Lee (Kyung Hee University, Republic of Korea)

CURRICULUM VITAE

Soo Heon Kwak

Professor, Seoul National University Hospital, Republic of Korea



Education and Training

2002.02	Seoul National University College of Medicine, Korea, M.D, Medicine
2012.08	Seoul National University, Graduate School, Korea, Ph.D, Internal Medicine

Employment and Position

2011-2015	Seoul National University Hospital, Assistant Professor
2015-2021	Seoul National University Hospital, Associate Professor
2021-	Seoul National University Hospital, Professor

Important Publications

1. Lee H, Fernandes M, Lee J, Merino J, Kwak SH. Exploring the shared genetic landscape of diabetes and cardiovascular disease: findings and future implications. *Diabetologia*. 2025 Jun;68(6):1087-1100. doi: 10.1007/s00125-025-06403-9. Epub 2025 Mar 15. PMID: 40088285; PMCID: PMC12069157.
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.

Exploring the genetic landscape of CVD and diabetes: finding and future implication

Diabetes is rapidly becoming a significant global health challenge, with projections indicating that by 2045, approximately one in eight adults, or around 783 million people, will be affected. The substantial metabolic changes associated with hyperglycemia greatly increase the risk of cardiovascular disease (CVD). While genetic predisposition is a key factor in the development of both diabetes and its vascular complications, identifying specific genes and molecular mechanisms that influence both conditions simultaneously has proven difficult. This challenge largely stems from the pathophysiologic heterogeneity of these diseases, where various processes contribute to different forms of diabetes and specific cardiovascular outcomes. Such molecular diversity has limited the effectiveness of large-scale genome-wide association studies (GWAS) in identifying shared underlying mechanisms.

Recent studies have made significant progress in this area. We have conducted a time-to-event GWAS in a large, multi-ethnic cohort of individuals with type 2 diabetes (T2D) who had not yet developed CVD at enrollment. This study, involving 49,230 participants from the CHARGE Consortium and a median follow-up of 10 years, identified three distinct ge-

netic variants (rs147138607 near CACNA1E/ZNF648, rs77142250 near HS3ST1, and rs335407 near TFB1M/NOX3) associated with an increased risk of incident CVD in T2D patients. Notably, these variants did not significantly influence CVD risk in the general population, highlighting the importance of genetic studies focused on specific subgroups. These findings illustrate how a systematic approach can uncover mechanisms underlying both diabetes and CVD, as well as identify individuals with a higher genetic risk for developing cardiovascular complications.

In this presentation I will review recent advancements in genetic epidemiology, emphasizing the importance of studies that explore causal associations to identify genetic and molecular factors influencing both hyperglycemia and cardiovascular complications. I will also discuss how disease subtyping approaches can be crucial in identifying the unique molecular signatures associated with both diabetes and cardiovascular disease. Finally, critical research gaps and future opportunities will be discussed to enhance our understanding of these diseases and translate these findings into improved patient care and public health outcomes.

CURRICULUM VITAE

Hong-Hee Won

Professor, Sungkyunkwan University & Samsung Medical Center, Republic of Korea



Education and Training

2002.02	Yonsei University, Korea, B.S., Computer Science
2004.02	Yonsei University, Korea, M.S., Bioinformatics
2011.08	KAIST, Korea, Ph.D., Genomics

Employment and Position

2012–2015	Harvard Medical School & Massachusetts General Hospital, Research Fellow
2016–2020	Sungkyunkwan University & Samsung Medical Center, Assistant Professor
2020–2025	Sungkyunkwan University & Samsung Medical Center, Associate Professor
2025–	Sungkyunkwan University & Samsung Medical Center, Professor

Important Publications

1. Park S, et al. Multivariate genomic analysis of 5 million people elucidates the genetic architecture of shared components of the metabolic syndrome. *Nature Genetics*, 1–12, 2024.
2. Kim MS, et al. Association of genetic risk, lifestyle, and their interaction with obesity and obesity-related morbidities. *Cell Metabolism*, 36(7), 1494–1503.e3, 2024.
3. Kim MS, et al. Association between adiposity and cardiovascular outcomes: an umbrella review and meta-analysis of observational and Mendelian randomization studies. *European Heart Journal*, 42(34), 3388–3403, 2021.
4. Khera A, et al. Association of rare and common variation in the lipoprotein lipase gene with coronary artery disease. *Journal of the American Medical Association*, 317(9), 937–46, 2017.
5. Stitzel NO, et al. Inactivating mutations in NPC1L1 and protection from coronary heart disease. *New England journal of medicine New England Journal of Medicine*, 371(22), 2072–2082, 2014.

Awards and Honors

1. SKKU Fellowship, Sungkyunkwan University, 2024
2. Commendation, Commissioner of the Korea Disease Control and Prevention Agency (for contributions to the Korea Biobank Project), 2022
3. Next-Generation Member, Y-KAST, The Korean Academy of Science and Technology, 2022

Multi-omics and multi-trait analysis of metabolic disease

Observational studies can suggest associations between risk factors and disease, but confounding, reverse causality, and study heterogeneity often limit their ability to establish causality. In medicine, however, causality is essential for developing effective therapies. Randomized controlled trials (RCTs) are the gold standard for testing causal relationships and play a central role in drug development. Yet when exposures cannot be ethically or practically manipulated (e.g., smoking and lung cancer), RCTs are infeasible. Moreover, drug targets derived from observational associations frequently fail in costly trials when no therapeutic benefit is observed.

Mendelian randomization (MR) provides a powerful alternative by using genetic variants, randomly allocated during meiosis, as instrumental variables to infer causal effects of risk factors on disease. By comparing

disease incidence between individuals with high versus low genetically determined risk, MR can evaluate whether modifying a factor may reduce disease risk, without direct intervention. Recent advances extend this framework by incorporating multi-omics resources such as expression quantitative trait loci (eQTL) and protein quantitative trait loci (pQTL). These approaches enable the systematic investigation of how genetic variation influences molecular traits, and how these traits in turn affect disease outcomes, thereby improving causal inference and drug target validation.

This presentation will introduce MR-based studies, including applications that integrate multi-omics data, and will discuss the potential of causality-driven strategies to identify and validate therapeutic targets more efficiently, ultimately enhancing the success of drug development.

CURRICULUM VITAE

Sang-Hak Lee

Professor, Yonsei University, Republic of Korea



Education and Training

1994.02	Yonsei University, Korea, M.D, Medicine
2005.08	Yonsei University, Korea, Ph.D., Internal Medicine

Employment and Position

2003–2006	Hallym University, Instructor, Assistant Professor
2010–2011	UCSD, USA, Visiting Scholar, Postdoc Fellow
2007–2025	Yonsei University, Assistant Professor, Associate Professor, Professor

Important Publications

1. Jin IT, et al. Aspirin and clinical outcomes in individuals with incidentally diagnosed coronary artery stenosis. *Am J Med* 2025;138:994–1000.
2. Kim J, et al. Statin therapy in individuals with intermediate cardiovascular risk. *Metabolism* 2024;150:155723.
3. An DB, et al. Hepatic Cdkal1 deletion regulates HDL catabolism and promotes reverse cholesterol transport. *Atherosclerosis* 2023;375:21–29.
4. Lee CJ, et al. Cardiovascular risk and treatment outcomes in severe hypercholesterolemia: a nationwide cohort study. *J Am Heart Assoc* 2022;11:e024379.
5. Ann SJ, et al. Role of lncRNA HSPA7 in human atherosclerotic plaque in sponging miR-223 and promoting proinflammatory vascular smooth muscle cell transition. *Exp Mol Med* 2021;53:1842–1849.

Awards and Honors

2024	Academic award of Korean Society of Lipid and Atherosclerosis
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Research Interest

Lipoprotein metabolism, Preventive cardiology, Vascular biology, Cardiovascular genetics

Gene-targeting therapy in familial hypercholesterolemia

Patients with familial hypercholesterolemia (FH) have apparently elevated risk of atherosclerotic cardiovascular diseases. However, patients' adherence to medical preventive measure is far from perfect and subgroup of the patients shows suboptimal response even after maximal pharmacological therapy. Therefore, this population has been frequently a candidate group for new lipid-lowering agents.

A few therapeutics based on small interfering RNA were developed and include inclisiran and zolasiran. These agents target *PCSK9* and *ANGPTL3* genes, respectively, and showed considerable additional LDL-C

reduction.

To date, gene editing has been approved for treatment of genetic conditions such as sickle cell disease. However, many researchers are trying to develop efficient gene editing for treatment of FH patients. Several issues may be needed to be answered before wide use of gene editing in this population: short and long-term safety, best target gene for editing, optimal editing technique. In addition, the cost is important to be included in treatment guidelines and to gain sufficient marketability.

Symposium 12

Residual CV Risk Era: Remnant Cholesterol

Sep 12(Fri) 08:30–10:00 | Room 3 (3F)

CHAIRPERSONS : Jeong-Taek Woo (Kyung Hee University, Republic of Korea)
Jae Hyung Park (Korea University, Republic of Korea)

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|-------------|---|
| 08:30–08:50 | Remnant cholesterol as a residual risk in ASCVD patients under statin-based lipid-lowering therapy
Sung Gyun Ahn (Yonsei University, Republic of Korea) |
| 08:50–09:10 | Association between remnant cholesterol and sarcopenia: implications on muscle health
Soo Yeon Jang (Korea University, Republic of Korea) |
| 09:10–09:30 | Targeting remnant cholesterol in the era of precision lipid management
Pablo Corral (Fasta University, Argentina) |
| 09:30–10:00 | Panel Discussion
Kyung-Soo Kim (CHA University, Republic of Korea)
Hong Seok Lim (Ajou University, Republic of Korea)
Jaehoon Chung (Dongguk University, Republic of Korea) |

CURRICULUM VITAE

Sung Gyun Ahn

Professor, Wonju Severance Christian Hospital, Republic of Korea



Education and Training

1996.02 Wonju College of Medicine, Korea, M.D., Medicine
2014.08 Ajou University, Korea, Ph.D., Internal Medicine

Employment and Position

2010-Current Wonju College of Medicine, Professor
2025- ICoLA Team, Staff

Important Publications

1. Non-culprit lesion location and FFR-guided revascularization in acute myocardial infarction with multivessel disease: FRAME-AMI substudy. Korean Circ J 2025 May 13 Online ahead of print.
2. Remnant cholesterol as a residual risk in atherosclerotic cardiovascular disease patients under statin-based lipid-lowering therapy: A Post Hoc Analysis of the RACING trial. J Clin Lipidol 2024 Nov-Dec;18(6):e905-e914.
3. Failed thrombus aspiration and reduced myocardial perfusion in patients with STEMI and large thrombus burden. JACC Cardiovasc Interv 2024;7:2216-2225.
4. High Platelet Reactivity Combined with CYP2C19 Genotype in Predicting Outcomes in East Asian Patients Undergoing Percutaneous Coronary Intervention. Clin Pharmacol Ther 2023 Nov;114(5):1104-1115.
5. Remnant cholesterol, LDL cholesterol and incident cardiovascular disease among Koreans: a national population-based study. Eur J Prev Cardiol 2023 Aug 21;30(11):1142-1150.

Research Interest

Remnant cholesterol, coronary physiology, individualized anti-platelet therapy, myocardial perfusion

Remnant cholesterol as a residual risk in ASCVD patients under statin-based lipid-lowering therapy

Remnant cholesterol (remnant-C) has emerged as a potential marker of residual cardiovascular risk even in patients receiving intensive lipid-lowering therapy (LLT). While lowering low-density lipoprotein cholesterol (LDL-C) with statins remains the cornerstone of therapy in atherosclerotic cardiovascular disease (ASCVD), many patients continue to experience major adverse cardiovascular events despite achieving target LDL-C levels. This observation highlights the need to explore alternative lipid parameters that may better reflect residual risk.

In this post hoc analysis of the RACING trial, we examined the prognostic significance of on-treatment remnant-C levels in 3,348 patients with established ASCVD. Lipid profiles obtained at 1 year after randomization were used to calculate remnant-C as total cholesterol minus LDL-C minus high-density lipoprotein cholesterol. Based on on-treatment values, patients were divided into tertiles: low (≤ 14.0 mg/dL), intermediate (14–20.5 mg/dL), and high (>20.5 mg/dL). The primary endpoint was a composite of cardiovas-

cular death, major cardiovascular events, or nonfatal stroke over 3 years.

Patients in the high remnant-C tertile experienced the highest cumulative incidence of the primary endpoint, followed by the intermediate and low tertiles (11.0%, 10.3%, and 7.5%, respectively; $p=0.009$). Importantly, multivariable analysis demonstrated that high on-treatment remnant-C levels were independently associated with adverse outcomes, whereas achievement of LDL-C thresholds below 55 or 70 mg/dL did not predict event rates. A remnant-C cut-off of 17 mg/dL, corresponding to the median value, effectively discriminated between higher- and lower-risk patients (HR 1.42; 95% CI: 1.14–1.78; $p=0.002$).

These findings suggest that remnant-C levels during statin-based therapy provide meaningful prognostic information and may serve as an additional biomarker to capture residual cardiovascular risk in ASCVD. Monitoring and potentially targeting remnant-C could represent a new strategy to further reduce adverse outcomes beyond traditional LDL-C-centered approaches.

CURRICULUM VITAE

Soo Yeon Jang

Clinical Assistant Professor, Korea University, Republic of Korea



Education and Training

2018.02	Korea University, Korea, M.D, Medicine
2025.08	Korea University, Korea, Ph.D., Internal Medicine

Employment and Position

2018-2019	Korea University Medical Center, Intern
2020-2023	Korea University Guro Hospital, Resident
2023-2025	Korea University Guro Hospital, Fellow
2025-	Korea University Guro Hospital, Clinical Assistant Professor

Important Publications

1. Jang SY, Choi KM. Impact of Adipose Tissue and Lipids on Skeletal Muscle in Sarcopenia. *J Cachexia Sarcopenia Muscle*. 2025;16(4):e70000.
2. Jang SY, Hwang SY, Jang A, Kim KJ, Yu JH, Kim NH, Yoo HJ, Kim NH, Baik SH, Choi KM. Association of remnant cholesterol with sarcopenia in Korean adults: a nationwide population-based study using data from the KNHANES. *Front Endocrinol (Lausanne)*. 2024 Aug 23;15:1391733.
3. Jang SY, Choi KM. Bidirectional crosstalk between bone and muscle: the role of RANKL pathway in osteosarcopenia. *J Endocrinol*. 2024 Jul 18;262(3):e240093.
4. Jang SY, Kang M, Song E, Jang A, Choi KM, Baik SH, Yoo HJ. Remnant cholesterol is an independent risk factor for the incidence of chronic kidney disease in newly-diagnosed type 2 diabetes: A nationwide population-based study. *Diabetes Res Clin Pract*. 2024 Apr;210:111639.

Research Interest

Diabetes, Sarcopenia, Dyslipidemia

Association between remnant cholesterol and sarcopenia: implications on muscle health

Sarcopenia, characterized by decreased muscle mass and function, represents a substantial health concern in aging societies. Despite the clinical importance of sarcopenia, pharmacological treatments remain limited. Identifying novel therapeutic targets based on a deeper understanding of its pathophysiology is crucial.

Fatty infiltration into skeletal muscle-both intermuscular and intramyocellular-could disrupt muscle structure, impair muscular metabolism, induce oxidative stress and mitochondrial dysfunction, ultimately leading to loss of muscle mass and performance.

Remnant cholesterol (remnant-C) has attracted attention as a contributor to the residual risk of cardiovascular diseases, and previous studies have

demonstrated its significant impact on various metabolic diseases, such as type 2 diabetes and Metabolic dysfunction-Associated Steatotic Liver Disease. We recently reported that higher remnant-C levels were significantly associated with increased risk of low muscle mass in a nationwide population-based study. Remnant-C may play a detrimental role in development of sarcopenia and muscle health through its pro-inflammatory and atherogenic features in skeletal muscle, which is highly vascularized.

This lecture will explore the role of remnant-C in sarcopenia and muscle dysfunction, discuss its potential as a promising marker for the prediction and therapeutic target of sarcopenia.

CURRICULUM VITAE

Pablo Corral

Medical Doctor, ICM Institute Medical Clinic, Medical Clinic office and Internal Medicine, Lipids2019 and Atherosclerosis Clinic, Argentina
 Medical Doctor. Investigator, Instituto de Investigaciones Clínicas Mar del Plata, Argentina



Main University Degree(s)

1998 Medical Doctor, Universidad del Salvador, Buenos Aires, Argentina

Postgraduate Education/Specialisation/Training

2009 Internal Medicine Consultant Specialist, College of Physicians IX district, Mar del Plata, Argentina

Previous Appointments

1/02/2014 to Date Full Pharmacology Professor, Fasta University, School of Medicine, Mar del Plata, Argentina

Clinical Research Experience

- National Leader & Steering Committee CORAL-REEF Trial, MK-0616, MSD
- National Lead Investigator (NLI) EAS FH Studies Collaboration (FHSC) Familial Hypercholesterolemia
- Principal Investigator COLCOVID Phase III Trial (Colchicine in COVID-19) published 2022, JAMA
- Principal Investigator in PREPARE-IT 1I and 2 Phase III Trial (EPA in COVID-19) 2021–2022
- Principal Investigator DA VINCI Study (Familial Hypercholesterolemia Argentina)
- Principal Investigator (PI) Assessing the Impact of Lipoprotein (a) Lowering With TQJ230 on Major Cardiovascular Events in Patients With CVD (Lp(a)HORIZON), 2022
- National Lead Investigator (NLI) Study of Inclisiran to Prevent Cardiovascular (CV) Events in Participants With Established Cardiovascular Disease (VICTORION-2P), 2022
- Sub-Investigator, Phase III, Chronic Heart Failure, 2019–2020
- Sub-Investigator, Phase IV, Chronic Heart Failure, 2019–to date

Targeting remnant cholesterol in the era of precision lipid management

Despite significant advances in lowering LDL cholesterol and widespread use of statins, a substantial residual cardiovascular risk remains in many patients. Among the key contributors to this residual risk is remnant cholesterol (RC)—the cholesterol content of triglyceride-rich lipoproteins such as very-low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), and chylomicron remnants. RC is now recognized not merely as a marker but as a causal and modifiable risk factor for atherosclerotic cardiovascular disease (ASCVD).

In the current era of precision lipid management, a shift from a one-size-fits-all approach to more targeted, mechanism-based interventions has highlighted the need to incorporate RC assessment into routine cardiovascular risk evaluation. RC levels are particularly relevant in individuals with obesity, insulin resistance, metabolic syndrome, and type 2 diabetes—conditions where triglyceride metabolism is disrupted and residual risk remains high despite optimal LDL-C control.

Recent genetic and epidemiological data have demonstrated that elevated RC levels are independently associated with increased risk of myocardial infarction and stroke, even in patients with low LDL-C. Moreover, non-HDL cholesterol and apolipoprotein B measurements can help capture the atherogenic burden of remnant particles, offering practical clinical tools when direct RC measurement is unavailable.

Novel therapies, including agents targeting ANGPTL3, APOC3, and lipoprotein lipase modulation, as well as lifestyle interventions, are emerging as potential strategies to lower RC and reduce cardiovascular events. Integrating RC into clinical practice represents a crucial step toward comprehensive risk reduction and individualized lipid management.

As our understanding of lipid biology evolves, targeting remnant cholesterol may redefine therapeutic priorities in patients with mixed dyslipidemia and persistent ASCVD risk, offering a new frontier in the quest for precision cardiovascular prevention.

Symposium 13

Shaping the Future of Lipid Management: Insights from the New Consensus and Current Dyslipidemia Trends

Sep 12(Fri) 08:30–10:00 | Room 4 (5F)

CHAIRPERSONS : Keeho Song (Konkuk University, Republic of Korea)
Sang-Rok Lee (Jeonbuk National University, Republic of Korea)

08:30–08:50 **2024 KSoLA consensus on secondary dyslipidemia**

Jae Hyun Bae (Seoul National University, Republic of Korea)

08:50–09:10 **Understanding the progression of obesity-induced organ damage: Insights from the EAS staging system**

Dae Young Cheon (Hallym University, Republic of Korea)

09:10–09:30 **Sex-specific gaps in lipid management and ASCVD**

Hyun-Jin Kim (Hanyang University, Republic of Korea)

09:30–10:00 **Panel Discussion**

Min Kyong Moon (Seoul National University, Republic of Korea)

Jaehyun Bae (Hallym University, Republic of Korea)

Ji-Yong Jang (National Health Insurance Service Ilsan Hospital, Republic of Korea)

CURRICULUM VITAE

Jae Hyun Bae

Associate Professor, Seoul National University Hospital, Republic of Korea



Education and Training

2008.02	Seoul National University College of Medicine, Korea, M.D, Medical Science
2020.02	Seoul National University College of Medicine, Korea, Ph.D., Medical Science

Employment and Position

2016-2017	Seoul National University Hospital, Fellow
2018-2024	Korea University Anam Hospital, Assistant Professor, Associate Professor
2024-	Seoul National University Hospital, Associate Professor

Important Publications

1. Hong B, Lee H, Bae JH, Cho YM, Shin JY. Risk for Angioedema With the Use of Dipeptidyl Peptidase 4 Inhibitors: A Population-Based Cohort Study. *J Allergy Clin Immunol Pract*. 2025;13(8):2025-2032.
2. Bae JH. SGLT2 Inhibitors and GLP-1 Receptor Agonists in Diabetic Kidney Disease: Evolving Evidence and Clinical Application. *Diabetes Metab J*. 2025;49(3):386-402.
3. Ko HY, Bea S, Yoon D, Hong B, Bae JH, Cho YM, Shin JY. Incretin-based drugs and the risk of gallbladder or biliary tract diseases among patients with type 2 diabetes across categories of body mass index: a nationwide cohort study. *Lancet Reg Health West Pac*. 2025;56:101242.
4. Bae JH, Choi J, Kim NH, Kim SG. Cardiovascular Outcomes With Antidiabetic Drugs in People With Type 2 Diabetes and a Prior Stroke. *Mayo Clin Proc*. 2025;100(2):235-248.
5. Bea S, Ko HY, Bae JH, Cho YM, Chang Y, Ryu S, Byrne CD, Shin JY. Risk of hepatic events associated with use of sodium-glucose cotransporter-2 inhibitors versus glucagon-like peptide-1 receptor agonists, and thiazolidinediones among patients with metabolic dysfunction-associated steatotic liver disease. *Gut*. 2025;74(2):284-294.

2024 KSoLA consensus on secondary dyslipidemia

Secondary dyslipidemia, defined as abnormal cholesterol and triglyceride levels arising from identifiable and often reversible causes, is an important yet frequently overlooked component of lipid management. The 2024 KSoLA Consensus on Secondary Dyslipidemia highlights the need to evaluate and correct these contributing factors before starting conventional lipid-lowering therapy. The consensus classifies secondary causes into three major domains. Dietary influences are particularly prominent: excessive caloric intake, diets high in saturated and trans fats, refined carbohydrates, added sugars, alcohol, and ultra-processed foods all contribute to elevations in LDL cholesterol and triglycerides. Beyond food composition, the timing of caloric intake also affects lipid metabolism, with evidence showing that shifting even modest amounts of energy from late evening meals to earlier in the day can reduce LDL-C levels. A second domain includes underlying medical conditions such as hy-

pothyroidism, nephrotic syndrome, cholestasis, and autoimmune disorders, each of which can disrupt lipid synthesis or clearance and should be considered in individuals with unexplained lipid abnormalities. The third domain comprises drug-induced dyslipidemia, caused by agents including thiazide diuretics, glucocorticoids, immunosuppressants such as cyclosporine, antiretroviral therapies, anticonvulsants, and certain hormonal or dermatologic medications. Whenever possible, modifying or substituting these treatments is encouraged. The consensus also proposes a practical clinical pathway: identify and correct the most relevant secondary factor first, then proceed with guideline-directed lipid-lowering therapy once lipid levels have stabilized. By promoting this stepwise, person-centered approach, the 2024 KSoLA Consensus provides clinicians with a framework to improve the precision of dyslipidemia management and enhance cardiovascular risk reduction.

CURRICULUM VITAE

Dae Young Cheon

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



Education and Training

2012.02	Hallym University, Korea, M.D, Medicine
2005.08	Korea National Open University, Korea, M.S, Bioinformatics & Statistics

Employment and Position

2020–2021	Seoul National University Hospital, Division of Cardiology, Fellowship
2021–2023	Division of Cardiology Dongtan Sacred Heart Hospital, Clinical Assistant Professor
2023–	Division of Cardiology Dongtan Sacred Heart Hospital, Assistant Professor

Important Publications

1. Association between Metabolic Syndrome and Young-Onset Dementia: A Nationwide Population-Based Study. *Neurology*, 2025.4.
2. Diabetes status, duration, and risk of dementia among ischemic stroke patients, *Alzheimer's Research & Therapy*, 2025.3.
3. Korea Hypertension Fact Sheet 2024: nationwide population-based analysis with a focus on young adults. *Clinical Hypertension*, 2025.01.
4. Depression and Risk of Stroke and Mortality after Percutaneous Coronary Intervention: A Nationwide Population Study, *Journal of Internal Medicine*, 2024.09.
5. Associations between migraine and major cardiovascular events in type 2 diabetes mellitus. *Cardiovasc Diabetol*, 2022.12.

Awards and Honors

2025	Asia-Pacific Cardiometabolic Syndrome Congress (APCMS), Distinguished Young Investigator Award
2024	Korean Society of Hypertension, Young Investigator Award

Understanding the progression of obesity-induced organ damage:
Insights from the EAS staging system

The EAS proposes a pathophysiology-based staging system for systemic metabolic disorders (SMD)—a cluster of metabolic abnormalities involving multiple organs, primarily driven by high-risk obesity and insulin resistance, and leading to cardiovascular, renal, and hepatic complications. Recognizing SMD as a progressive, multi-organ condition allows for earlier, stage-specific intervention to reduce the burden of obesity-related comorbidities and improve long-term outcomes.

Stages of SMD:

Stage 1: Metabolic abnormalities without organ damage (e.g., overweight/dysfunctional adiposity plus dyslipidaemia, hypertension, or liver steatosis; pre-diabetes alone).

Stage 2: Early organ damage (e.g., type 2 diabetes, metabolic-associated steatohepatitis/fibrosis, CKD stage 1-2, asymptomatic diastolic dysfunction, sub-clinical atherosclerosis).

Stage 3: Advanced, symptomatic organ damage (e.g., HFpEF, cirrhosis, CKD stage 3-5, clinical ASCVD).

Management principles:

Stage 1: Lifestyle interventions (diet quality, physical activity, weight reduction), risk factor monitoring.

Stage 2: Add pharmacotherapy (e.g., glucose-lowering, lipid-lowering, antihypertensive agents); consider targeted therapies for MASLD or CKD.

Stage 3: Intensive multidisciplinary management; treat organ-specific complications; consider metabolic surgery where appropriate.

CURRICULUM VITAE

Hyun-Jin Kim

Associate Professor, Hanyang University Guri Hospital, Republic of Korea



Education and Training

2014.3–2017.2	Postgraduate School, College of Medicine, Seoul National University
2011.9–2013.8	Master of Medical Science, Postgraduate School, College of Medicine, Seoul National University
2004.3–2008.2	Doctor of Medicine, Department of Medicine, EwhaWomans University School of Medicine

Employment and Position

2008–2009	Medical Internship, Seoul National University Hospital
2009–2013	Residency in Internal Medicine, Seoul National University Hospital
2013–2015	Clinical & Research Fellow, Seoul National University Hospital
2015–2018	Assistant professor, Department of Internal Medicine, Division of Cardiology, Myoungji Hospital
2018–2019	Assistant professor, Department of Internal Medicine, Division of Cardiology, Chungbuk University Hospital, Chungbuk University
2019–Present	Associate professor, Department of Internal Medicine, Division of Cardiology, Hanyang University Guri Hospital, Hanyang University

Important Publications

1. Kim HJ, Shin JH, Kim BS, Kang J, Lee H, Sung KC. Age-related annual changes in arterial stiffness in healthy adults: Insights from a large Korean cohort study. *Atherosclerosis*. 2024;398:118592. doi: 10.1016/j.atherosclerosis.2024.118592.
2. Kim HJ, Kim BS, Lee Y, Ahn SB, Kim DW, Shin JH. Harnessing Metabolic Indices as a Predictive Tool for Cardiovascular Disease in a Korean Population without Known Major Cardiovascular Event. *Diabetes Metab J*. 2024;48:449–462. doi: 10.4093/dmj.2023.0197.
3. Kim H, Shin M, Kim K, Jung M, Cho D, Lee J, Koh K. Metabolic syndrome awareness in the general Korean population: results from a nationwide survey. *KOREAN JOURNAL OF INTERNAL MEDICINE*. 2024;39:272–282. doi: 10.3904/kjim.2023.363.

Sex-specific gaps in lipid management and ASCVD

Atherosclerotic cardiovascular disease (ASCVD) remains the leading cause of death globally for both sexes. However, mounting evidence indicates that women face unique and underappreciated risks across the continuum of ASCVD care—from diagnosis and risk stratification to treatment and follow-up. Despite similar LDL-C-associated cardiovascular risk in both sexes, women are less likely to be diagnosed early, to receive guideline-directed statin therapy, and to attain lipid targets, particularly among those with familial hypercholesterolemia. Hormonal transitions—such as pregnancy, menopause, and lactation—introduce dynamic shifts in lipid profiles and are often overlooked in risk modeling. Furthermore, the burden of remnant cholesterol and elevated lipoprotein(a) [Lp(a)] appears to be more consequential in women after midlife, yet current guidelines recommend only a single lifetime Lp(a) measurement. Additionally, traditional cardiovascular

risk prediction models underestimate lifetime ASCVD risk in women due to biases in outcome detection and the exclusion of female-specific risk enhancers such as hypertensive pregnancy disorders, premature menopause, and autoimmune diseases. Sociocultural barriers—such as delayed health-seeking behavior, caregiver prioritization, and therapeutic discontinuation—exacerbate these inequities. This session will explore how sex-based differences in lipid trajectories and biomarker expression influence cardiovascular risk, and how female-specific life events impact the progression of ASCVD. It will review emerging data on undertreatment in lipid-lowering therapy and discuss the limitations of current risk prediction models that fail to incorporate sex- and gender-specific variables. Finally, the presentation will outline potential strategies—both clinical and policy-driven—to improve prevention and care for women at risk of cardiovascular disease.

Symposium 14

What is Important beyond Stenosis in Intracranial Atherosclerosis

Sep 12(Fri) 13:00–14:30 | Room 2 (3F)

CHAIRPERSONS : Eung Gyu Kim (Inje University, Republic of Korea)
Kwang-Yeol Park (Chung-Ang University, Republic of Korea)

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- | | |
|-------------|--|
| 13:00–13:20 | Vascular tortuosity and atherosclerosis burden
Mi-Yeon Eun (Kyungpook National University, Republic of Korea) |
| 13:20–13:40 | Vessel wall enhancement and inflammation
Dong-Wan Kang (Seoul National University, Republic of Korea) |
| 13:40–14:00 | Blood viscosity and intracranial atherosclerosis
Ho Geol Woo (Kyung Hee University, Republic of Korea) |
| 14:00–14:30 | Panel Discussion
Hyung-Min Kwon (Seoul National University, Republic of Korea)
Bum Joon Kim (University of Ulsan, Republic of Korea)
Woo-Keun Seo (Sungkyunkwan University, Republic of Korea)
Jin-Man Jung (Korea University, Republic of Korea) |

CURRICULUM VITAE

Mi-Yeon Eun

Assistant Professor, Department of Neurology, Kyungpook National University, Republic of Korea



Education and Training

2006.02	Korea University College of Medicine, Korea, M.D, Medicine
2016.02	Korea University College of Medicine, Korea, M.S., Medical Science (Neurology)
2019.03–Present	Korea University College of Medicine, Korea, Ph.D., Medical Science (Neurology)

Employment and Position

2011–2012	Korea University Guro Hospital, Clinical Fellow
2012–2018	Hanil General Hospital, Staff Neurologist
2018–2024	Kyungpook National University Chilgok Hospital, Clinical Professor
2024–Present	Kyungpook National University Chilgok Hospital, Assistant Professor

Important Publications

1. Mi-Yeon Eun, Ha-Na Song, Jong-Un Choi, Hwan-Ho Cho, Hyung Jun Kim, Jong-Won Chung, Tae-Jin Song, Jin-Man Jung, Oh-Young Bang, Gyeong-Moon Kim, Hyunjin Park, David S Liebeskind, Woo-Keun Seo. Global intracranial arterial tortuosity is associated with intracranial atherosclerotic burden. *Sci Rep.* 2024;14(1):11318.
2. Jeon ET, Lee SH, Eun MY, Jung JM. Center of Pressure- and Machine Learning-based Gait Score and Clinical Risk Factors for Predicting Functional Outcome in Acute Ischemic Stroke. *Arch Phys Med Rehabil.* 2024;105(12):2277–2285.
3. Mi-Yeon Eun, Eun-Tae Jeon, Jin-Man Jung. Machine learning application in ischemic stroke diagnosis, management, and outcome prediction: a narrative review. *J Med Life Sci.* 2023;20(4):141–157.
4. Ahn JW, Hwang J, Lee M, Kim JH, Cho HJ, Lee HW, Eun MY. Serum neurofilament light chain levels are correlated with the infarct volume in patients with acute ischemic stroke. *Medicine (Baltimore).* 2022;101(39):e30849.

Vascular tortuosity and atherosclerosis burden

Vascular tortuosity, characterized by arterial elongation and abnormal curvature, has emerged as a critical geometric risk factor in atherosclerotic disease development. Recent evidence demonstrates that tortuosity is not merely a consequence of aging or atherosclerosis but represents an independent pathophysiological mechanism driving cardiovascular disease progression through complex hemodynamic alterations.

Arterial tortuosity disrupts blood circulation, increases vessel wall shear stress, and can lead to complications such as atherosclerosis and cerebrovascular strokes. The underlying mechanisms involve disturbed flow patterns, oscillatory wall shear stress, and altered endothelial mechanoreceptor signaling. Local hemodynamics play an essential role in the initiation and progression of coronary artery disease, with areas exposed to low wall shear stress indicating regions of plaque growth. These geometric changes fundamentally alter blood flow dynamics, creating microenvironments that promote inflammatory responses and accelerated atherosclerotic plaque formation.

Contemporary research reveals significant associations between tortuosity and atherosclerotic burden across multiple vascular territories. Global intracranial arterial tortuosity is independently associated with in-

tracranial atherosclerotic burden, with adjusted odds ratio of 1.33 after controlling for traditional risk factors. Tortuosity index of coronary bifurcations appears to be an emergent geometric risk factor, with strong linear correlations between low wall shear stress and arterial segment tortuosity. In femoral arteries, tortuosity influences atherosclerosis development more than atherosclerosis causes tortuosity, suggesting tortuosity as a primary pathogenic factor.

Advanced computational fluid dynamics and imaging technologies now enable precise quantification of tortuosity parameters and their hemodynamic consequences. Noninvasive tests of vascular function provide additional information for cardiovascular risk stratification and offer opportunities for timely intervention. Clinical implications include enhanced risk stratification, procedural planning considerations, and potential therapeutic targeting of geometric risk factors alongside traditional interventions.

Future directions focus on developing standardized tortuosity metrics, elucidating molecular mechanisms linking vascular geometry to atherosclerosis, and integrating geometric parameters into personalized cardiovascular risk prediction models for precision medicine approaches.

CURRICULUM VITAE

Dong-Wan Kang

Assistant Professor, Seoul National University Bundang Hospital, Republic of Korea



Education and Training

2012.02	Seoul National University, Korea, MD, Medicine
2017.02	Seoul National University Hospital, Korea, Board certified Neurologist, Neurology
2021.02	Seoul National University, Korea, MSc, Department of Clinical Medical Sciences

Employment and Position

2012–2013	Seoul National University Hospital, Intern
2013–2017	Seoul National University Hospital, Residency in Neurology
2017–2020	Gangjin Public Health Center, Public Health Doctor
2020–2022	Seoul National University Hospital, Fellowship (Stroke and Intensive Care Medicine)
2022–2024	Seoul National University Bundang Hospital, Assistant Professor, Department of Public Health
2024–Present	Seoul National University Bunhang Hospital, Assistant Professor, Division of Intensive Care Medicine, Department of Neurosurgery and Neurology

Important Publications

1. Kang DW, Kim J, Kim DY, Baik SH, Jung C, Menon BK, Song JW, Han MK, Bae HJ, Kim BJ*. Temporal changes in symptomatic intracranial arterial disease: a longitudinal high-resolution vessel wall imaging study. *Front Neurol*. 2025 Jun 16;16:1583857. (IF=3.4)
2. Kang DW, Kim DY, Kim J, Baik SH, Jung C, Singh N, Song JW, Bae HJ, Kim BJ*. Emerging Concept of Intracranial Arterial Diseases: The Role of High Resolution Vessel Wall MRI. *J Stroke*. 2024 Jan 26(1): 26–40. (IF=8.2)

Vessel wall enhancement and inflammation

Unlike extracranial atherosclerosis, intracranial artery disease (ICAD) includes a wide range of different conditions that go far beyond the idea of atherosclerosis. Recognizing this heterogeneity is crucial, as grouping all lesions under “intracranial atherosclerosis” or “intracranial stenosis” oversimplifies pathophysiology and risks overlooking mechanistic insights essential for tailored therapy.

With advances in high-resolution vessel wall magnetic resonance imaging (HR-VWMRI), direct visualization of the arterial wall has become possible, shifting attention from luminal narrowing to wall pathology. Among the most debated findings is vessel wall enhancement. Enhancement is often interpreted as a marker of inflammation, yet the relationship is nuanced and dependent on the underlying etiology. In intracranial atherosclerosis, enhancement may reflect vasa vasorum proliferation, neovascularization, increased endothelial permeability, or gadolinium leakage through fragile microvasculature. In contrast, dissections, moyamoya disease, and vasculitis demonstrate distinct enhancement patterns associated with intramural hematoma, intimal hyperproliferation, or immune-mediated injury.

Our studies using serial HR-VWMRI demonstrate that the natural history of vessel wall enhancement varies significantly across ICAD etiologies. Atherosclerotic and dissecting lesions, in particular, show divergent temporal trajectories, suggesting different inflammatory dynamics and healing responses.

In intracranial atherosclerotic stenosis, both luminal narrowing and vessel wall enhancement independently contribute to the risk of recurrent ischemic stroke, emphasizing the complementary clinical implications of stenosis and inflammation. Inflammatory activity can be assessed through circulating biomarkers as well as advanced imaging features, providing opportunities for refined risk stratification and monitoring of treatment response. Integrating vessel wall enhancement into the diagnostic framework highlights the importance of moving beyond stenosis alone. This lecture will review the mechanistic basis of vessel wall enhancement and inflammation, findings from serial imaging analyses, and discuss how multiple imaging approaches can refine diagnosis and prognosis in intracranial arterial disease, as well as how advances in imaging techniques and processing may enable quantitative interpretation in the future.

CURRICULUM VITAE**Ho Geol Woo**

Assistant Professor, Department of Neurology, Kyung Hee University College of Medicine,
Kyung Hee University Hospital, Republic of Korea

**Education and Training**

Mar 2018–Feb 2020	Kyung Hee University, College of Medicine, Seoul, Korea, Ph.D., Neuroscience
Sep 2014–Feb 2017	Kyung Hee University, College of Medicine, Seoul, Korea, M.S., Neuroscience
2007–Feb 2011	Kyung Hee University, College of Medicine, Seoul, Korea, M.D.
Mar 2005–Feb 2007	Kyung Hee University, College of Medicine, Seoul, Korea, B.S.
Mar 2011–Feb 2012	Kyung Hee University Hospital, Seoul, Internship
Mar 2012–Feb 2016	Kyung Hee University Hospital, Seoul, Department of Neurology, Residency
Apr 2016–Feb 2017	Seoul National University Bundang Hospital, Department of Radiology, Interventional Neuroradiology, Fellow
Mar 2017–Feb 2018	Seoul National University Bundang Hospital, Department of Neurology, Neurology, Stroke, Neuro critical care, Fellow

Employment and Position

Mar 2018–Feb 2019	Department of Neurology, Soonchunhyang University College of Medicine, Cheonan Hospital, Cheonan, South Korea, Clinical Assistant Professor
Mar 2019–May 2020	Department of Neurology, Seoul Hospital, Ewha Womans University College of Medicine, Seoul, Korea, Clinical Assistant Professor
Jun 2020–Aug 2021	Department of Neurology, Kyung Hee University Hospital, Kyung Hee University College of Medicine, Seoul, Korea, Clinical Assistant Professor
Sep 2021–Current	Department of Neurology, Kyung Hee University Hospital, Kyung Hee University College of Medicine, Seoul, Korea, Assistant Professor

Important Publications

1. Global burden of vaccine-associated multiple sclerosis, 1967–2022: A comprehensive analysis of the international pharmacovigilance database. *Journal of Medical Virology*. First. 2024.04.01. 12.7
2. Clinical characteristics and outcomes of patients with mpox during the 2022 mpox outbreak compared with those before the outbreak: A systematic review and meta-analysis. *Reviews in Medical Virology*. Corresponding. 2024.01.01. 11.1

Blood viscosity and intracranial atherosclerosis

Intracranial atherosclerosis is a major cause of ischemic stroke, particularly in Asian, African, and Hispanic populations. While traditional risk factors such as hypertension, diabetes, and dyslipidemia are well-established, increasing attention is being directed toward hemorheological parameters, including blood viscosity, as potential contributors to the pathogenesis and progression of intracranial arterial disease. This lecture will explore the relationship between blood viscosity and intracranial atherosclerosis, emphasizing the mechanistic interplay between elevated blood viscosity, endothelial dysfunction, and impaired cerebral perfu-

sion. We will review current evidence from clinical and translational studies that demonstrate how alterations in blood rheology may contribute to plaque formation, vessel narrowing, and recurrent cerebrovascular events. In addition, we will discuss the clinical implications of these findings, including the potential role of hemorheological markers in risk stratification, prognosis, and treatment strategies for patients with intracranial atherosclerotic disease. The session aims to provide a comprehensive overview for neurologists, stroke specialists, and researchers interested in advancing precision medicine approaches to cerebrovascular health.

Symposium 15

Nontraditional Risk Factors for ASCVD

Sep 12(Fri) 13:00–14:30 | Room 3 (3F)

CHAIRPERSONS : Seok–Min Kang (Yonsei University, Republic of Korea)
Young Sup Byun (Inje University, Republic of Korea)

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|-------------|--|
| 13:00–13:20 | Environmental pollution as a nontraditional risk factor for atherosclerotic cardiovascular disease: mechanisms and impacts
Sun–Young Kim (National Cancer Center, Republic of Korea) |
| 13:20–13:40 | Health inequity as a nontraditional risk factor for ASCVD
Eunji Kim (Gachon University, Republic of Korea) |
| 13:40–14:00 | Psychiatric disorders: an overlooked nontraditional risk factor in the development of atherosclerotic cardiovascular disease
Seung Jin Han (Ajou University, Republic of Korea) |
| 14:00–14:30 | Panel Discussion
Si–Hyuck Kang (Seoul National University, Republic of Korea)
Ji–Hyun Kim (The Catholic University of Korea, Republic of Korea)
Jaehyun Bae (Hallym University, Republic of Korea)
Jong–Tae Lee (Korea University, Republic of Korea) |

CURRICULUM VITAE

Sun-Young Kim

Professor, National Cancer Center Graduate School of Cancer Science and Policy,
Republic of Korea



Education and Training

1998.02	Ewha Womans University, Korea, B.A., Health Education
2002.02	Seoul National University, Korea, M.P.H., Public Health (Biostatistics major)
2005.02	Seoul National University, Korea, Ph.D., Public Health (Biostatistics major)

Employment and Position

2005–2014	University of Washington, Research Fellow/Research Scientist
2014–2017	Seoul National University, Research Assistant Professor
2017–	National Cancer Center Graduate School, Assistant/Associate/Full Professor

Important Publications

1. Jun YB, Song I, Kim OJ, Kim SY. The impact of limited residential address on health effect analysis of predicted air pollution in a simulation study. *Journal of Exposure Science and Environmental Epidemiology* 2022.
2. Kim SY, Blanco M, Bi Z, Larson TV, Sheppard L. Exposure assessment to air pollution epidemiology: a scoping review of emerging monitoring platforms and designs. *Environmental Research* 2023;223:115451.
3. Kim SY, Gassett AJ, Blanco M, Sheppard L. Ultrafine particle mobile monitoring study designs for epidemiology: Cost and performance comparison. *Environmental Health Perspectives* 2025;113(3–4):047010.
4. Kim OJ*, Kim SY. Regional difference in the association between long-term PM and cardiovascular disease incidence and potential determinants of the difference *Geohealth* 2025;19:9(3):e2024GH001245.
5. Park J, Kim OJ, Shin M, Kang S, Hwang SS, Cho YJ, Kim SY. Long-term exposure to air pollution and lung cancer incidence: findings from improved exposure assessment and extended population. *Cancer Causes & Control* (in press).

Research Interest

My research interest lies in epidemiological and methodological studies on air pollution and human health. In epidemiological research, I am interested in investigating various characteristics of the association between particulate matter air pollution and health. Methodologically, I focus on conditions to produce more accurate health effect estimates from predicted air pollution.

Environmental pollution as a nontraditional risk factor for atherosclerotic cardiovascular disease: mechanisms and impacts

There has been accumulating evidence of the association between exposure to air pollution and atherosclerosis. Many epidemiological reported that long-term exposure to particulate matter air pollution contributed to the risk of having and progressing atherosclerosis. Toxicological studies supported biological

mechanisms including inflammatory mediation and epigenetic modification. However, there are remaining work to elucidate the role of various pollutants, to improve exposure and outcome assessment, and to integrate epidemiological and toxicological evidence.

CURRICULUM VITAE

Eunji Kim

Assistant Professor, Gachon University College of Medicine, Republic of Korea



Education and Training

2011.02	Korea University, Korea, BSc, Life Science
2015.02	Dongguk University, Korea, MD, Medicine
2019.11	University College London, UK, MSc, Global Health and Development
2024.02	Yonsei University, Korea, Ph.D., Preventive Medicine

Employment and Position

2024–Present	Gachon University College of Medicine, Assistant Professor
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Important Publications

1. Kim E, Lee HH, Kim EJ, Cho SM, Kim HC, Lee H. Factors associated with medication adherence among young adults with hypertension. *Clin Hypertens*. 2025 May;31(1):e18.
2. Kim E, Lee H, Lloyd-Jones D, Ko YG, Kim BG, Kim HC. Area deprivation and premature cardiovascular mortality: a nationwide population-based study in South Korea. *BMJ Public Health* 2024;2:e000877.
3. Kim E, Lee GB, Yon DK, Kim HC. Trends in socioeconomic inequalities in obesity among Korean adolescents: the Korea Youth Risk Behavior Web-based Survey (KYRBS) 2006 to 2020. *Epidemiol Health*. 2023;45:e2023033.
4. Kim E, Sung K, Kim CO, Youm Y, Kim HC. The Effect of Cognitive Impairment on the Association Between Social Network Properties and Mortality Among Older Korean Adults. *J Prev Med Public Health*. 2023;56(1):31–40.
5. Kim E, Baek J, Kim M, Lee H, Bae JW, Kim HC. Trends in Regional Disparity in Cardiovascular Mortality in Korea, 1983–2019. *Korean Circ J*. 2022 Nov;52(11):829–843.

Health inequity as a nontraditional risk factor for ASCVD

Atherosclerotic cardiovascular disease (ASCVD) remains a leading cause of morbidity and mortality worldwide. While traditional risk factors—such as hypertension, dyslipidemia, diabetes, and smoking—are well established, growing evidence highlights the critical role of nontraditional determinants, particularly health inequity, in shaping ASCVD risk and outcomes.

Health inequity refers to avoidable, systematic, and unjust disparities in health status and access to care across populations, often driven by socioeconomic status, educational attainment, race/ethnicity, geographic location, and other social determinants. These factors influence exposure to traditional risk factors, timely diagnosis, adherence to treatment, and access to preventive care, thereby contributing to disparities in ASCVD incidence, management, and prognosis.

This lecture will review the emerging evidence linking health inequity to the development and progression of ASCVD. Drawing on findings from recent

population-based studies, I will highlight how socioeconomic disadvantage and social marginalization are independently associated with higher ASCVD burden, even after adjusting for conventional clinical risk factors. Particular attention will be paid to vulnerable populations frequently overlooked in traditional cardiovascular frameworks—including individuals with disabilities, those from socioeconomically disadvantaged backgrounds, and residents of medically underserved areas. I will also discuss conceptual frameworks for understanding health inequity as a structural risk factor, emphasizing its role across the life course and the need for multilevel interventions that address upstream determinants alongside clinical care.

This lecture aims to advance the understanding of health inequity as a modifiable, nontraditional risk factor for ASCVD—one that warrants integrated attention from clinicians, researchers, public health practitioners, and policymakers.

CURRICULUM VITAE**Seung Jin Han**

Professor, Department of Endocrinology & Metabolism,
Ajou University School of Medicine, Republic of Korea

**Education and Training**

2001.02	Yonsei University, Korea, M.D, Medicine
2012.02	Yonsei University, Korea, Ph.D., Internal Medicine

Employment and Position

2006–2008	Yonsei University, Fellow
2008–2021	Ajou University School of Medicine, Instructor/ Assistant /Associate Professor
2016–2017	University of Washington, USA, Visiting Professor
2021–Present	Ajou University School of Medicine, Professor

Important Publications

1. Cardiorenal outcomes and safety of SGLT2 inhibitors in patients with diabetes secondary to disorders of the exocrine pancreas: a nationwide population-based study (Corresponding author). *Diabetes & Metabolism* 2025;51:101668.
2. Ipeglimin Inhibits Macrophage Foam Cell Formation and Atherosclerosis in Streptozotocin-Induced Diabetic ApoE-Deficient Mice (Corresponding author). *Cells* 2025;14:472.
3. Ipeglimin attenuates NLRP3 inflammasome activation by restoring mitochondrial functions in macrophages (Corresponding author). *Journal of Pharmacological Sciences* 2024;155:35–43.
4. Associations of updated cardiovascular health metrics, including sleep health, with incident diabetes and cardiovascular events in older adults with prediabetes: A nationwide population-based cohort study (Corresponding author). *Diabetes Res Clin Pract.* 2023;7:203:110820.
5. Characteristics and Clinical Course of Diabetes of the Exocrine Pancreas: A Nationwide Population-Based Cohort Study (Corresponding author). *Diabetes Care* 2022;45(5):1141–1150.

Awards and Honors

Dean's Award for Distinguished Service from Ajou University School of Medicine

Psychiatric disorders: an overlooked nontraditional risk factor in the development of atherosclerotic cardiovascular disease

Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality worldwide. According to the World Health Organization (2022), CVDs were responsible for an estimated 17.9 million deaths, accounting for approximately 32% of global fatalities. Despite significant advancements in both prevention and treatment, CVD continues to represent a major public health concern. Consequently, recent cardiovascular epidemiology research has increasingly focused on identifying emerging and modifiable CVD risk factors within the general population.

Psychiatric disorders, including depression, anxiety,

chronic stress-related conditions and insomnia, are increasingly recognized as nontraditional yet significant contributors to the development and progression of CVD. Despite mounting epidemiological and mechanistic evidence, these factors remain under-addressed in routine cardiovascular risk assessment and prevention strategies.

This lecture will explore major studies and pathophysiological mechanisms linking psychiatric conditions—such as psychosocial stress, mood and anxiety disorders, and insomnia—to the development of CVD.

Symposium 16

Defining the Optimal LDL-C Target for Prevention of Atherosclerotic Cardiovascular and Cerebrovascular Disease

Sep 12(Fri) 13:00–14:30 | Room 4 (5F)

CHAIRPERSONS : Jeong-Taek Woo (Kyung Hee University, Republic of Korea)
Min Kyong Moon (Seoul National University, Republic of Korea)

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|-------------|---|
| 13:00–13:20 | Optimal LDL-C goals for secondary prevention in high-risk patients – a cardiologist’s perspective
Hyun Sung Joh (Seoul National University, Republic of Korea) |
| 13:20–13:40 | Optimal LDL-C level for stroke prevention – insights from a neurologist’s perspective
Wookjin Yang (University of Ulsan, Republic of Korea) |
| 13:40–14:00 | Different LDL-C targets by duration of diabetes: what is the evidence? – an endocrinologist’s perspective
Young-Sang Lyu (Chosun University, Republic of Korea) |
| 14:00–14:30 | Panel Discussion
Kyung-Soo Kim (CHA University, Republic of Korea)
Minwoo Lee (Hallym University, Republic of Korea)
Jung-Kyu Han (Seoul National University, Republic of Korea)
Joon Ho Moon (Seoul National University, Republic of Korea) |

CURRICULUM VITAE**Hyun Sung Joh**

Clinical Assistant Professor, Seoul National University College of Medicine,
Boramae Medical Center, Seoul, Republic of Korea

**Education and Training**

2012.12	Sungkyunkwan University, Korea, M.D., Medicine
2024.08	Sungkyunkwan University, Korea, Master of Clinical Medicine, Internal Medicine

Employment and Position

2012–2013	Samsung Medical Center, Korea, Internship
2013–2017	Samsung Medical Center, Korea, Residency in Internal Medicine
2017–2020	Army Aviation Command, Korean Army, Military Medical Officer, Captain
2020–2022	Samsung Medical Center, Korea, Clinical and Interventional Cardiology Fellowship
2022–2023	Boramae Medical Center, Korea, Medicine Assistant Professor
2023–	Boramae Medical Center, Korea, Clinical Assistant Professor

Important Publications

1. Hyun Sung Joh (co-first author), SH Lee, Jinhwan Jo, Hyun Kuk Kim, WH lim, HL kim, JB Seo, WY Chung, SH Kim, Myung-A Kim, JM Lee, KAMIR Investigators. Intravascular imaging-guided percutaneous coronary intervention in patients with acute myocardial infarction and cardiogenic shock. *Rev Esp Cardiol (Engl Ed)*. 2024 Dec;77(12):995–1007.
2. Hyun Sung Joh (co-first author), Woonchan Kwon, Doosup Shin, SH Lee, Choi KH, TK park, JH Yang, YB song, SH Choi, JY Hahn, HC Gwon, Lee JM. Drug-Coated Balloon Angioplasty in Patients Undergoing Complex Percutaneous Coronary Intervention. *JACC Asia*. 2024 Jun 18;4(7):519–531.
3. Lee JM, Hyun Sung Joh (co-first author), Choi KH, David Hong, TK park, JH Yang, YB song, SH Choi, JY Hahn, HC Gwon, SMART-REWARD Investigators. Safety and Efficacy of Everolimus-Eluting Bioresorbable Vascular Scaffold Versus Second-Generation Drug-Eluting Stents in Real-World Practice. *J Korean Med Sci*. 2023 Feb 06;38(5):e34.
4. Hyun Sung Joh (co-first author), Doosup Shin, Lee JM, Lee SH, Choi KH, Hwang DY, The ILIAS Registry Investigators. Prognostic Impact of Coronary Flow Reserve in Patients With Reduced Left Ventricular Ejection Fraction. *JAHA*. 2022;11:e025841, Jul 2022 doi:10.1161/JAHA.122.025841.

Awards and Honors

2021.10	Young Investigator Award, Korean Society of Cardiology
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Optimal LDL-C goals for secondary prevention in high-risk patients – a cardiologist's perspective

Low-density lipoprotein (LDL) particles are the primary determinants of atherosclerotic cardiovascular risk. In clinical practice, LDL cholesterol (LDL-C) is commonly measured as a surrogate for LDL particle number due to its accessibility and strong correlation. Robust evidence has established a causal relationship between LDL-C and vascular events. Meta-analyses of large randomized trials demonstrate that an absolute LDL-C reduction of 1 mmol/L ($\approx$ 38.9 mg/dL) yields a relative 22% reduction in major cardiovascular events

over 5 years, consistent across baseline risk profiles. However, the absolute risk reduction (ARR) and number needed to treat (NNT) vary according to baseline patient risk. Importantly, very low achieved LDL-C levels (even <20 mg/dL) have not been associated with safety concerns, supporting a “lower is better” strategy in patients with major ASCVD. Further data are needed in patients with non-major ASCVD or without prior ASCVD to define the balance of benefit and cost-effectiveness.

CURRICULUM VITAE

Wookjin Yang

Assistant Professor, Asan Medical Center, Republic of Korea



Education and Training

2013.08	Seoul National University, Korea, Bachelor of Science, Medicine
2021.08	Seoul National University, Korea, Master of Science, Medicine
2023.08	Seoul National University, Korea, Doctor of Philosophy, Medicine

Employment and Position

2013–2014	Seoul National University Hospital, Intern
2014–2018	Seoul National University Hospital, Resident, Neurology
2018–2021	Republic of Korea Army, Military doctor
2021–2023	Seoul National University Hospital, Clinical Fellow, Neurology (Stroke)
2023–2024	Seoul National University Hospital, Assistant Professor
2024–	Asan Medical Center, Assistant Professor

Important Publications

1. 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;26:342–346.
2. Yang W, Jung KH, Kang DW, Lee EJ, Jeong HY, Chung M, Kim Y, Ha J, Kim JM, Lee SH. Characteristics and Clinical Implication of White Matter Lesions in Patients With Adult Moyamoya Disease. *Neurology*. 2023;100:e1912–e1921.
3. Yang W, Kang DW, Kim JM, Jung KH, Lee SH. Neuroimaging features of antiphospholipid antibody-related stroke compared with atrial fibrillation-related stroke. *Sci Rep*. 2022;12:11686.
4. Yang W, Kang DW, Ha SY, Lee SH. Drinking Patterns and Risk of Ischemic Stroke in Middle-Aged Adults: Do Beneficial Drinking Habits Indeed Exist? *Stroke*. 2021;52:164–171.
5. Yang W, Kim CK, Kim DY, Jeong HG, Lee SH. Gamma-glutamyl transferase predicts future stroke: A Korean nationwide study. *Ann Neurol*. 2018;83:375–386.

Awards and Honors

Fellow Research Award, Korean Stroke Society, 2023.

Research Interest

Stroke, Neuroimaging, Clinical trials

Optimal LDL-C level for stroke prevention – insights from a neurologist's perspective

Low-density lipoprotein cholesterol (LDL-C) is a key contributor to atherosclerosis, and treatment guidelines have progressively adopted lower LDL-C targets as evidence has accumulated. Landmark cardiovascular trials, including PROVE IT, IMPROVE-IT, and Proprotein convertase subtilisin/kexin type 9 inhibitor studies, demonstrated benefits with intensive LDL-C reduction, reinforcing the principle that “the lower, the better”. Stroke-specific evidence is consistent with this paradigm. The SPARCL trial established the benefit of high-intensity statins in non-cardioembolic ischemic stroke, while the TST trial showed that targeting LDL-C <70 mg/dL is superior to a target of 90–110 mg/dL in patients with atherosclerotic stroke or transient ischemic attack. More recent studies suggest

that rapid and intensive LDL-C lowering after acute ischemic stroke may reduce early neurological deterioration, prevent early stroke recurrence, and improve functional outcomes. Despite these advances, major knowledge gaps persist. Ischemic stroke is a heterogeneous disease with diverse etiologies, including small vessel occlusion (SVO) and cardioembolism (CE), for which evidence-based LDL-C targets are lacking. Current guidelines focus primarily on large artery atherosclerosis, leaving uncertainty regarding optimal lipid management in other stroke subtypes such as SVO and CE strokes. Ongoing large-scale clinical trials focusing specifically on these populations are expected to address this gap and clarify whether intensive LDL-C lowering confers similar preventive benefits.

CURRICULUM VITAE**Young-Sang Lyu**

Associated Assistant 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
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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

Different LDL-C targets by duration of diabetes: what is the evidence? – an endocrinologist's perspective

The relationship between low-density lipoprotein cholesterol (LDL-C) and cardiovascular disease risk is well established, especially among patients with diabetes, who have significantly higher cardiovascular morbidity and mortality. Intensive LDL-C reduction is generally recommended for diabetes management; however, recent studies indicate that optimal LDL-C targets may vary based on diabetes duration.

From an endocrinological viewpoint, the duration and complication of diabetes and significantly affects underlying pathophysiological mechanisms, including the progression of atherosclerosis, endothelial dysfunction, and inflammation. Patients with a longer

duration of diabetes typically have more extensive vascular damage and higher residual cardiovascular risk, potentially necessitating more aggressive LDL-C lowering strategies compared to those with shorter diabetes duration.

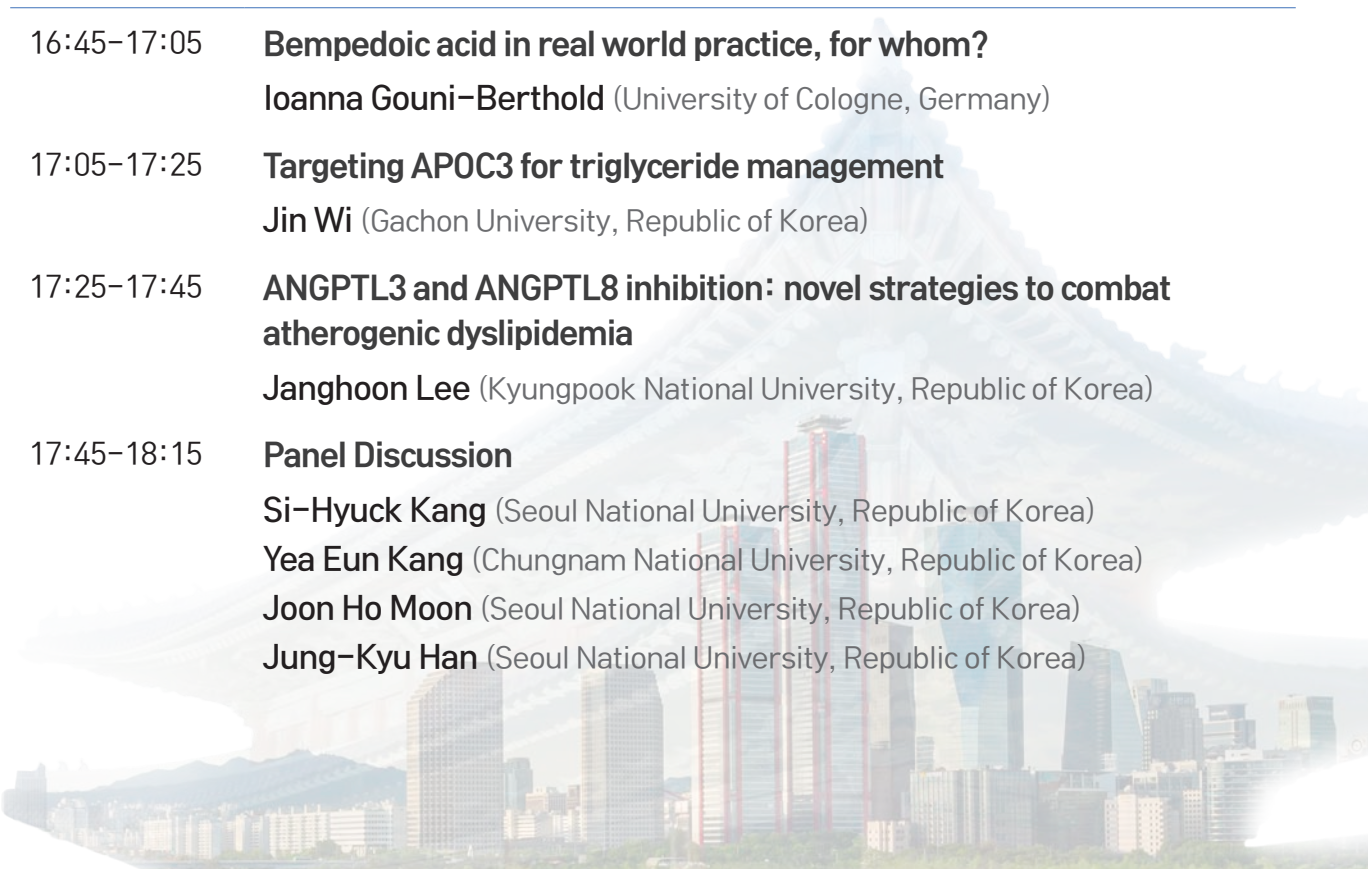
This presentation will review current clinical guidelines and recent major clinical studies evaluating LDL-C levels and cardiovascular outcomes stratified by diabetes duration and complication. It will present evidence supporting individualized LDL-C targets, discuss practical challenges in clinical application, and explore optimal LDL-C targets based on diabetes duration to enhance cardiovascular protection.

Symposium 17

Emerging Drugs for Dyslipidemia Management

Sep 12(Fri) 16:45–18:15 | Room 1 (3F)

CHAIRPERSONS : Ki Chul Sung (Sungkyunkwan University, Republic of Korea)
Hack-Lyoung Kim (Seoul National University, Republic of Korea)

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|-------------|---|
| 16:45–17:05 | Bempedoic acid in real world practice, for whom?
Ioanna Gouni-Berthold (University of Cologne, Germany) |
| 17:05–17:25 | Targeting APOC3 for triglyceride management
Jin Wi (Gachon University, Republic of Korea) |
| 17:25–17:45 | ANGPTL3 and ANGPTL8 inhibition: novel strategies to combat atherogenic dyslipidemia
Janghoon Lee (Kyungpook National University, Republic of Korea) |
| 17:45–18:15 | Panel Discussion
Si-Hyuck Kang (Seoul National University, Republic of Korea)
Yea Eun Kang (Chungnam National University, Republic of Korea)
Joon Ho Moon (Seoul National University, Republic of Korea)
Jung-Kyu Han (Seoul National University, Republic of Korea) |

CURRICULUM VITAE

Ioanna Gouni-Berthold

Professor, Head of the Lipid Clinic and Lipid Research Clinic, Center for Endocrinology, Diabetes and Preventive Medicine, University of Cologne, Germany



Education and Training

1984 Aristotle University, Thessaloniki, Greece, M.D, Medicine

Employment and Position

1985–1986	Boston University, Research Fellow
1986–1989	Franklin Square Hospital Center, Resident
1989–1992	Baylor College of Medicine, Fellow
1992–1993	Harvard Medical School, Fellow
1994–1996	Aristotle University, Attending physician
1996–1999	University of Bonn, Attending physician
1999–2002	University of Bonn, Lecturer
2002–2020	University of Cologne, Professor, Senior Consultant
2020–	University of Cologne, Head Lipid Clinic and Lipid Research Clinic

Important Publications

1. Volanesorsen to prevent acute pancreatitis in hypertriglyceridemia. Alexander VJ, Karwatowska-Prokopczuk E, Prohaska TA, Li L, Geary RS, Gouni-Berthold I, Oral EA, Hegele RA, Stroes ESG, Witztum JL, Tsimikas S. *N Engl J Med*. 2024 Feb 1;390(5):476–477.
2. Association Between Achieved Low-Density Lipoprotein Cholesterol Levels and Long-Term Cardiovascular and Safety Outcomes: An Analysis of FOURIER-OLE. Gaba P, O'Donoghue ML, Park JG, Wiviott SD, Atar D, Kuder JF, Im K, Murphy SA, De Ferrari GM, Gaciong ZA, Toth K, Gouni-Berthold I, Lopez-Miranda J, Schiele F, Mach F, Flores-Arredondo JH, López JAG, Elliott-Davey M, Wang B, Monsalvo ML, Abbasi S, Giugliano RP, Sabatine MS. *Circulation*. 2023 Apr 18;147(16):1192–1203.

Bempedoic acid in real world practice, for whom?

In my talk I will focus on two main topics. First, I will present data from the first ever real-world trial with bempedoic acid examining the efficacy and safety of bempedoic acid in a real-life setting, the MILOS trial (NCT04579367). The European Society of Cardiology (ESC)/European Atherosclerosis Society (EAS) 2019 joint guidelines for the management of dyslipidemia recommend goals of an LDL-C reduction of $\geq 50\%$ from baseline alongside absolute LDL-C goals of $<70\text{mg/dl}$ and $<55\text{mg/dl}$ for patients at high and very high CV risk, respectively. However, despite available lipid-lowering therapies, many patients do not achieve the guideline-recommended LDL-C goals. Bempedoic acid is a first-in-class adenosine triphosphate-citrate lyase inhibitor, which has demonstrated efficacy in reducing both LDL-C levels and the risk of CV events in the CLEAR clinical trial program.

Based on these results, bempedoic acid has been approved by the European Medicines Agency for the treatment of primary hypercholesterolemia and mixed

dyslipidemia, and for the reduction of CV risk by lowering LDL-C levels.

MILOS is an ongoing European, prospective, observational study, evaluating the effectiveness and safety of bempedoic and the bempedoic acid + ezetimibe fixed-dose combination (FDC) in a real-world setting.

Planned enrolment for MILOS is approximately 4250 adults with primary hypercholesterolemia or mixed dyslipidemia in routine clinical practice from approximately 490 sites in Austria, Belgium, Germany, Italy, the Netherlands, Spain, Switzerland and the UK.

In my presentation I will report data from the German cohort of the MILOS trial, the first European cohort that completed the 2-year study.

The second subject I will address is the position of bempedoic acid has in the new ESC update of the 2019 ESC/EAS Dyslipidemia Guidelines which is planned to be presented at the 2025 Congress of the ESC in Madrid just 2 weeks before the ICoLA congress starts.

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

2005-2010	Severance Hospital, Intern & Resident, Internal Medicine
2010-2013	Severance Hospital, Clinical Fellow, Cardiology
2013-2019	Severance Hospital, Assistant Professor, Cardiology
2019-	Gil Medical Center, Professor, Cardiology

Important Publications

1. Dose Optimization of Meropenem in Patients on Veno-Arterial Extracorporeal Membrane Oxygenation in Critically Ill Cardiac Patients: Pharmacokinetic/Pharmacodynamic Modeling. *J Clin Med* 2022;11:6621 (IF 5.098).
2. 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).
3. 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).
4. Association of vitamin D deficiency with profound cardiogenic shock in patients resuscitated from sudden cardiac arrest. *Shock* 2020;53:717-722 (IF 3.533).
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

Targeting APOC3 for triglyceride management

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

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

Important Publications

1. Intravascular modality-guided versus angiography-guided percutaneous coronary intervention in acute myocardial infarction. Kim N, Lee JH, Jang SY, Bae MH, Yang DH, Park HS, Cho Y, Jeong MH, Park JS, Kim HS, Hur SH, Seong IW, Cho MC, Kim CJ, Chae SC; Korea Acute Myocardial Infarction Registry - National Institute of Health Investigators. Catheter Cardiovasc Interv. 2020 Mar 1;95(4):696-703. doi: 10.1002/ccd.28359. Epub 2019 May 27.
2. Usefulness of Calculation of Cardiovascular Risk Factors to Predict Outcomes in Patients With Acute Myocardial Infarction. Kim CY, Lee JH, Jang SY, Bae MH, Yang DH, Park HS, Cho Y, Jeong MH, Park JS, Kim HS, Hur SH, Seong IW, Cho MC, Kim CJ, Chae SC; Korea Acute Myocardial Infarction Registry - National Institute of Health Investigators. Am J Cardiol. 2019 Sep 15;124(6):857-863. doi: 10.1016/j.amjcard.2019.06.010. Epub 2019 Jun 25.

ANGPTL3 and ANGPTL8 inhibition: novel strategies to combat atherogenic dyslipidemia

Background and Rationale: Atherogenic dyslipidemia, characterized by elevated triglycerides, small dense LDL particles, and reduced HDL-C, represents a critical unmet need in cardiovascular risk management. Despite optimal statin therapy, patients with mixed hyperlipidemia remain at elevated risk for atherosclerotic cardiovascular disease due to persistent triglyceride-rich lipoproteins and remnant cholesterol. ANGPTL3 and ANGPTL8 have emerged as compelling therapeutic targets, offering novel mechanisms to address residual cardiovascular risk through their central role in lipid metabolism regulation.

Mechanistic Insights: ANGPTL3 and ANGPTL8 function as master regulators of lipoprotein metabolism by inhibiting lipoprotein lipase and endothelial lipase activities. ANGPTL3 forms a heterotrimeric complex with ANGPTL8 in 2:1 stoichiometry, acting as an atypical unfoldase that catalyzes LPL inactivation through irreversible enzyme unfolding. This ATP-independent mechanism explains why substoichiometric ANGPTL3/ANGPTL8 concentrations effectively control capillary LPL activity, preferentially inhibiting LPL in oxidative tissues during fed states while directing triglyceride-rich nutrients toward adipose storage.

Clinical Development: Multiple pharmacological strategies targeting ANGPTL3/ANGPTL8 have demonstrated clinical efficacy. Evinacumab, an approved monoclonal antibody for homozygous familial hypercholester-

olemia, achieves 47% LDL-C reduction through LDL receptor-independent mechanisms. RNA interference approaches (zodasiran, solbinsiran) provide sustained hepatic ANGPTL3 suppression with convenient quarterly subcutaneous dosing. In ARCHES-2, zodasiran demonstrated dose-dependent reductions in triglycerides (63%), non-HDL cholesterol (36%), and LDL-C (20%) at 24 weeks. Critically, siRNA-based approaches avoid hepatic steatosis associated with antisense oligonucleotides, suggesting superior safety profiles.

Clinical Implications: ANGPTL3/ANGPTL8 inhibition represents a paradigm shift in dyslipidemia management, offering benefits independent of statin mechanisms. These therapies demonstrate broad efficacy across diverse dyslipidemic populations, particularly benefiting patients with elevated triglycerides and metabolic syndrome features. Ongoing cardiovascular outcome trials will establish definitive clinical benefit, while future developments include oral small-molecule inhibitors and selective ANGPTL8 antagonists for optimized lipoprotein profiles.

Conclusion: ANGPTL3/ANGPTL8 inhibition addresses critical gaps in lipid-lowering therapy through innovative mechanisms and convenient dosing regimens. As personalized medicine advances, these agents are poised to transform treatment paradigms and significantly reduce residual cardiovascular risk in high-risk populations.

Symposium 18

Sex and Gender Differences in Cardiometabolic Health: From Biology to Treatment

Sep 12(Fri) 16:45–18:15 | Room 3 (3F)

CHAIRPERSONS : Wook Bum Pyun (Ewha Womans University, Republic of Korea)
Mi-Seung Shin (Gachon University, Republic of Korea)

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- | | |
|-------------|---|
| 16:45–17:05 | Biological and hormonal mechanisms linking sex and cardiometabolic risk
Erin D. Michos (Johns Hopkins University, USA) |
| 17:05–17:25 | Sex and gender differences in risk assessment and biomarkers for cardiovascular disease
Ji Eun Lee (Korea University, Republic of Korea) |
| 17:25–17:45 | Do women and men respond differently to cardiometabolic therapies?
Hyun Ju Yoon (Chonnam National University, Republic of Korea) |
| 17:45–18:15 | Panel Discussion
Hyun-Jin Kim (Hanyang University, Republic of Korea)
Ye Seul Yang (Seoul National University, Republic of Korea)
Hyeonju Jeong (Hanyang University, Republic of Korea) |

CURRICULUM VITAE

Erin D. Michos

Professor, Johns Hopkins University School of Medicine, USA



Education and Training

1992-1996	Northwestern University, B.A., Biology
1996-2000	Northwestern University, M.D., Medicine
2000-2003	Johns Hopkins University, Internal Medicine
2003-2007	Johns Hopkins University, Cardiovascular Medicine
2005-2007	Johns Hopkins University, M.H.S., Epidemiology

Employment and Position

2007-2008	Johns Hopkins University, Instructor of Medicine
2008-2014	Johns Hopkins University, Assistant Professor of Medicine
2014-2024	Johns Hopkins University, Associate Professor of Medicine
2014-Present	Johns Hopkins University, Associate Director of Preventive Cardiology
2019-Present	Johns Hopkins University, Director of Women's Cardiovascular Health
2024-Present	Johns Hopkins University, Professor of Medicine

Important Publications

1. Ebong IA, Appiah D, Mauricio R, Narang N, Honigberg MC, Ilonze OJ, Aggarwal NR, Zanni MV, Mohammed SF, Cho L, Michos ED. Sex Hormones and Heart Failure Risk. JACC Adv. 2025 Apr;4(4):101650. PubMed Central PMCID: PMC11937663.
2. Ebong IA, Wilson M, Racette SB, Appiah D, Schreiner PJ, Allison M, Watson K, Bertoni AG, Michos ED. The Association of Menopausal Age with Sex Hormones and Anthropometric Measures Among Postmenopausal Women in the Multi-Ethnic Study of Atherosclerosis Study. J Womens Health (Larchmt). 2025 Mar;34(3):294-306. PubMed PMID: 39804188.
3. Rajendran A, Minhas AS, Kazzi B, Varma B, Choi E, Thakkar A, Michos ED. Sex-specific differences in cardiovascular risk factors and implications for cardiovascular disease prevention in women. Atherosclerosis. 2023 Nov;384:117269. PubMed Central PMCID: PMC10841060.
4. Michos ED, McEvoy JW, Blumenthal RS. Lipid Management for the Prevention of Atherosclerotic Cardiovascular Disease. N Engl J Med. 2019 Oct 17;381(16):1557-1567. PubMed PMID: 31618541.
5. Zhao D, Guallar E, Ouyang P, Subramanya V, Vaidya D, Ndumele CE, Lima JA, Allison MA, Shah SJ, Bertoni AG, Budoff MJ, Post WS, Michos ED. Endogenous Sex Hormones and Incident Cardiovascular Disease in Post-Menopausal Women. J Am Coll Cardiol. 2018 Jun 5;71(22):2555-2566. PubMed Central PMCID: PMC5986086.

Biological and hormonal mechanisms linking sex and cardiometabolic risk

Cardiovascular disease (CVD) is the major cause of death in women worldwide. Women and men share many risk factors in common, but several are unique to women. Assessing a reproductive history is important for early prevention & treatment of CVD. The learning objectives of this talk are to:

1. Identify reproductive factors that are “risk-enhancers” in the assessment of cardiovascular risk in women related to ovarian dysfunction, adverse

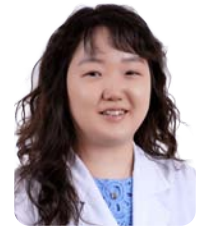
pregnancy outcomes, and early menopause, as well the impact of female-predominant conditions on CV risk including auto-immune disease

2. Discuss the impact of sex hormone levels, particularly elevated androgen levels, on cardiovascular risk in women across the lifespan
3. Review the approach to CVD risk assessment in women, including assessment of subclinical atherosclerosis when appropriate

CURRICULUM VITAE

Ji Eun Lee

Clinical Assistant Professor, Korea University Guro Hospital, Republic of Korea



Education and Training

2016.02	Korea University, Seoul, Korea, M.D, Medicine
2024.08	Korea University, Seoul, Korea, Ph.D., Internal Medicine

Employment and Position

2016-2017	Korea University Guro Hospital, Internship Training
2017-2020	Korea University Guro Hospital, Residency Training (Internal Medicine)
2020-2022	Korea University Guro Hospital, Cardiology Fellowship/ Clinical Instructor
2022-	Korea University Guro Hospital, Clinical Assistant Professor

Important Publications

1. Lee JE, Choi JY, Choi BG, Choi YJ, Park S, Kang DO, Park EJ, Kim JB, Roh SY, Na JO, Choi CU, Kim EJ, Park CG, Jeong MH, Hwang JY, Hur SH, Jeong JO, Oh S, Rha SW. Different diabetogenic effect of statins according to intensity and dose in patients with acute myocardial infarction: a nationwide cohort study. *Sci Rep.* 2024 Aug 21;14(1):19438.
2. Lee JE, Beak MJ, Choi JY. Ventricular Septal Defect Combined with Double-Chambered Right Ventricle and Severe Right Ventricular Outflow Tract Stenosis in a Symptom-Free Septuagenarian. *Eur Heart J.* 2024 Jan 14;45(3):237.
3. Lee JE, Choi JY, Yum YJ, Joo HJ, Kim YH, An HG, Kim EJ. Clinical effectiveness and safety of amlodipine/losartan-based single-pill combination therapy in patients with hypertension: Findings from real-world, multicenter observational databases. *J Clin Hypertens (Greenwich).* 2021 Nov;23(11):1975-1983.

Awards and Honors

1. Hypertension Seoul 2024, Young Investigator Award
2. AHA 2023, Paul Dudley White International Scholar Award
3. 2023 Annual Spring Scientific Conference of the KSC with Affiliated Cardiac Societies, Case competition winner

Research Interest

1. Blood Pressure Measurement/Monitoring
2. Genomic and Precision Medicine Approaches in Cardiovascular Disease
3. Management of Cardiometabolic Risk Factors

Sex and gender differences in risk assessment and biomarkers for cardiovascular disease

Sex and gender play a critical role in cardiovascular disease, yet conventional risk assessment often tends to overlook these differences. This lecture will discuss

sex and gender differences in cardiovascular disease, and how these differences can bring more personalized care and better outcomes for everyone.

CURRICULUM VITAE

Hyun Ju Yoon

Professor, Chonnam National University, Republic of Korea



Education and Training

2001.02	Konyang University, Korea, M.D, Medicine
2007.08	Konyang University, Korea, Ph.D., Internal Medicine

Employment and Position

2001-2006	Konyang Univerdity Hospital, Intern, Resident, Fellowship Training
2007-2012	Chonnam National University Hospital, Assistant-Associated Professor
2013-2014	DUKE Medical Center, Visiting Scholar
2015-2025	Chonnam National University Medical School and Hospital, Professor

Important Publications

1. Prognosis after withdrawal of cardioprotective therapy in patients with improved cancer therapeutics-related cardiac dysfunction. JACC CardioOncology 2025.
2. Sex-specific predictors of long term mortality in elderly patients with ischemic cardiomyopathy. Journal of Clinical medicine 2023.
3. Characteristics and clinical outcomes of cancer patients who developed constrictive physiology after pericardiocentesis. Korean circulation journal 2021.
4. Usefulness of diastolic function score as a predictor of long term prognosis in patients with acute myocardial infarction. Frontiers in cardiovascular medicine 2021.
5. COVOD-19 vaccination-related pericarditis : A Korean National Study. Mayo Clinic Proceedings 2024.

Research Interest

Structural heart disease, Cardiooncology, Cardiac remodeling after Myocardial infarction

Do women and men respond differently to cardiometabolic therapies?

Sex-based differences in the response to cardiometabolic therapies are increasingly recognized as clinically significant, yet they remain underrepresented in research and clinical guidelines. Emerging evidence shows that women and men may respond differently to a range of treatments for conditions such as type 2 diabetes, hypertension, heart failure, and atherosclerotic cardiovascular disease. For instance, glucose-lowering agents like GLP-1 receptor agonists have been shown to provide greater cardiovascular protection in women compared to men, while SGLT-2 inhibitors and DPP-4 inhibitors generally yield comparable outcomes across sexes. However, adverse drug reactions tend to occur more frequently in women, likely due to differences in pharmacokinetics and historical biases in dosing standards derived from male-dominant clinical trials.

Despite comparable disease prevalence, women are often less likely to be prescribed evidence-based therapies such as statins, ACE inhibitors, or beta-blockers, and may face diagnostic delays, particularly in the context of cardiovascular disease. In terms of

non-pharmacologic interventions, men and women also differ in their responses. While men may demonstrate greater absolute gains in exercise capacity through cardiac rehabilitation or high-intensity interval training, women appear to benefit more from lifestyle interventions in terms of relative mortality reduction and long-term cardiometabolic risk modification.

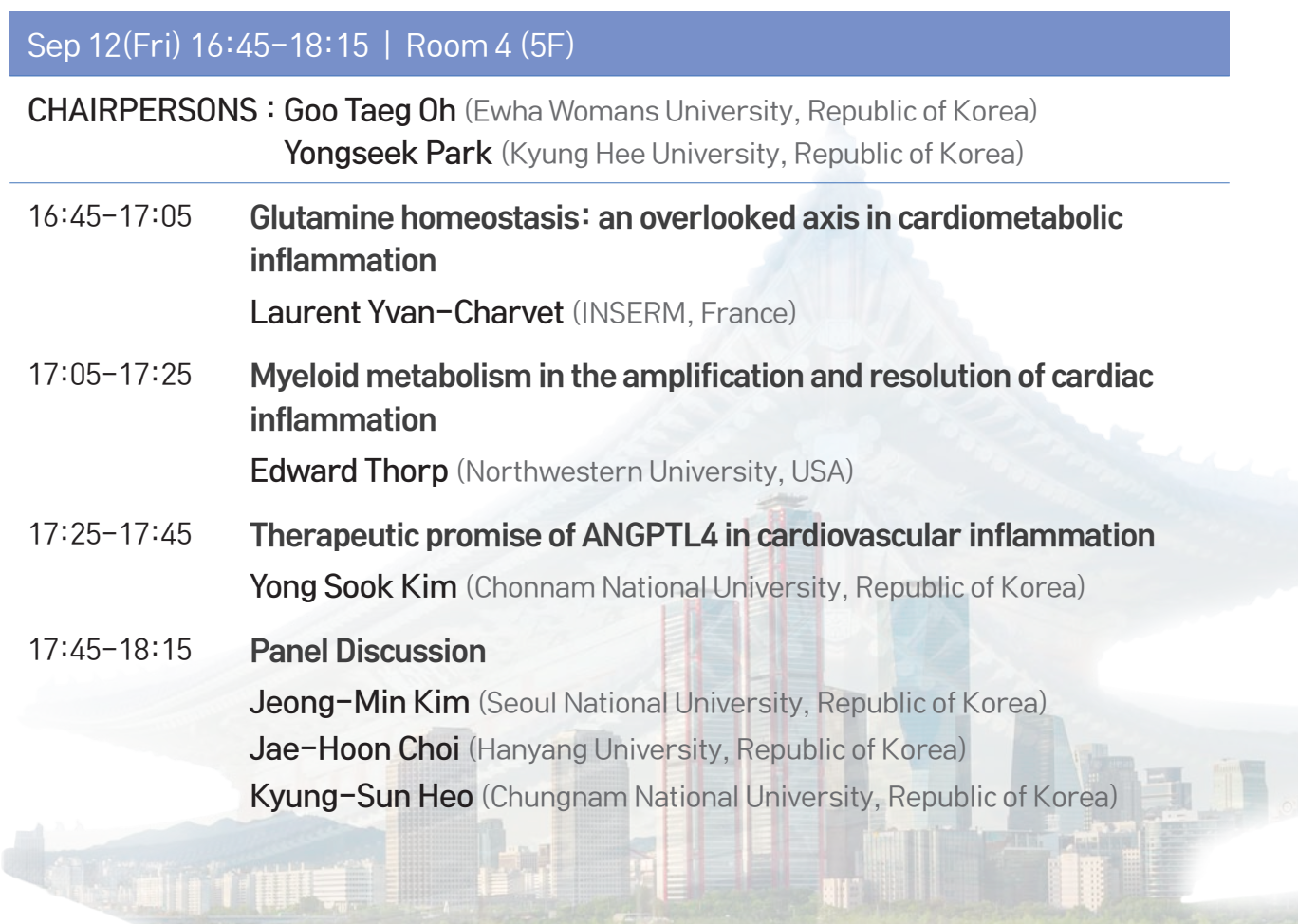
Biological factors such as hormonal status, adipokine profiles, lipid metabolism, and endothelial function contribute to sex-specific disease progression and therapeutic outcomes. Notably, postmenopausal women with diabetes exhibit a more atherogenic risk profile and a higher likelihood of adverse cardiovascular events than men with comparable risk factors. These findings underscore the need for more inclusive research, sex-stratified data analysis, and personalized treatment strategies that account for the unique physiological and clinical characteristics of women and men. Addressing these disparities is essential to advancing equity and improving outcomes in the management of cardiometabolic disease.

Symposium 19

Immunometabolic Programming of Macrophages in Cardiovascular Injury and Repair

Sep 12(Fri) 16:45–18:15 | Room 4 (5F)

CHAIRPERSONS : Goo Taeg Oh (Ewha Womans University, Republic of Korea)
Yongseek Park (Kyung Hee University, Republic of Korea)

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|-------------|---|
| 16:45–17:05 | Glutamine homeostasis: an overlooked axis in cardiometabolic inflammation
Laurent Yvan-Charvet (INSERM, France) |
| 17:05–17:25 | Myeloid metabolism in the amplification and resolution of cardiac inflammation
Edward Thorp (Northwestern University, USA) |
| 17:25–17:45 | Therapeutic promise of ANGPTL4 in cardiovascular inflammation
Yong Sook Kim (Chonnam National University, Republic of Korea) |
| 17:45–18:15 | Panel Discussion
Jeong-Min Kim (Seoul National University, Republic of Korea)
Jae-Hoon Choi (Hanyang University, Republic of Korea)
Kyung-Sun Heo (Chungnam National University, Republic of Korea) |

CURRICULUM VITAE

Laurent Yvan-Charvet

Professor, Inserm U1065, France



Introduction

Laurent Yvan-Charvet is director of research of the laboratory 'Physiopathology of Cardiometabolic Diseases' in Nice. He previously held an affiliation as Director of Innovative Medicine research within Pfizer Cardiovascular and Metabolic Diseases. His laboratory has a longstanding interest in immunometabolism and the therapeutic potential in human diseases. Dr. Yvan-Charvet obtained his Ph.D. in Endocrinology in 2005 from Paris University. He enjoyed postdoctoral training in molecular medicine under the mentorship of Prof. Tall at Columbia University, NY. His research focused on novel antiinflammatory functions of HDL raising therapies (i.e, macrophage and stem cell biology) in the context of cardiometabolic diseases before starting his current research exploring the nodes linking metabolism and inflammation in inflammatory diseases, but also in a constellation of biological processes woven into the immunometabolic fabric. He returned to France in 2013 to set up a laboratory in Nice thanks to a national Atip-Avenir excellence program. He has been promoted to the rank of DR2 in 2018 and DR1 in 2021. The team has adopted a dedicated and integrated approach to identify novel immunometabolic pathways, biomarkers and therapies to fight metaflammation in chronic diseases. Pioneer studies participate to 1) prove the causality of hyperglycemia in metaflammation, 2) identify novel origins of fatty acid-dependent metaflammation, and 3) pinpoint non-canonical glutamine metabolism as a novel and promising immunometabolic pathway at the origin of metaflammation'. This work was supported by a very competitive (ERC)-consolidator Award since 2017 and participated to the creation of a novel University Hospital Center (IHU) aimed at facilitating translational research and the identification of innovative therapeutic strategies to improve diseases linked to altered metabolism (<https://www.oncoage.org/our-teams>). The team has developed tools to interrogate in-depth molecular and cellular mechanisms using cultured primary macrophages and to test relevance and causation in vivo using genetically altered mice, including the Cre-lox conditional knockout system. The laboratory has worked in the immunometabolism field for over 15 years with over 150 invitations to international conferences or institutional seminars and has gained a high level of conceptual and technical expertise in this area (citations>15000)."

Education and Training

2005.10	Paris VI University, France, Ph.D., Endocrinology
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Employment and Position

2013-2018	Inserm U1065, Team leader Atip-Avenir
2018-2021	Inserm U1065, Director of Research (DR2, eq. to 'tenure track' Ass. Professor)
2021-	Inserm U1065, Director of Research (DR1, eq. to 'tenure track' Full Professor)

Glutamine homeostasis: an overlooked axis in cardiometabolic inflammation

Despite intensive interventions targeting blood glucose, lipids, and blood pressure, cardiometabolic diseases and their complications continue to cause 17 million deaths annually and remain a major public health challenge. My research has focused on analyzing the molecular and cellular processes underlying atherosclerotic vascular disease, as well as developing therapeutic approaches targeting metabolic and inflammatory disorders in this context.

Beyond the well-established roles of cholesterol and glucose metabolism in atherosclerosis, our work has progressively highlighted a central role for glutamine metabolism—often referred to as the "primary fuel of the immune system"—in supporting anaplerosis for

hematopoiesis (the production of immune cells) and efferocytosis (the clearance of aging or defective cells).

At the whole-body level, glutamine metabolism is regulated by the liver. In this context, using an integrated approach combining epidemiological data, Mendelian randomization analyses based on human genetics, and preclinical models, we demonstrated the critical role of this metabolic pathway in atherosclerotic vascular disease.

Locally, glutamine metabolism not only controls the effector functions of macrophages—thereby influencing necrotic core expansion—but also regulates the matrix-related functions of stromal cells, contributing to arterial wall remodeling.

CURRICULUM VITAE

Edward Thorp

Professor, Northwestern University, USA



Education and Training

2005 Loyola University Chicago, Ph.D., Virology

Employment and Position

2011 Northwestern, Professor

Important Publications

1. Filipp M, Ge ZD, DeBerge M, Lantz C, Ginton K, Gao P, Smolgovsky S, Dai J, Zhao YY, Yvan-Charvet L, Alcaide P, Weinberg SE, Schiattarella GG, Hill JA, Feinstein MJ, Shah SJ, Thorp EB. Myeloid Fatty Acid Metabolism Activates Neighboring Hematopoietic Stem Cells to Promote Heart Failure With Preserved Ejection Fraction. *Circulation*. 2025 May 20;151(20):1451-1466. doi: 10.1161/CIRCULATIONAHA.124.070248. Epub 2025 Mar 12. PMID: 40071347; PMCID: PMC12088907.
2. Lantz C, Becker A, DeBerge M, Filipp M, Ginton K, Ananthakrishnan A, Urbanczyk J, Cetlin M, Alzamroon A, Abdel-Latif A, Spite M, Ge ZD, Thorp EB. Early-age efferocytosis directs macrophage arachidonic acid metabolism for tissue regeneration. *Immunity*. 2025 Feb 11;58(2):344-361.e7. doi: 10.1016/j.immuni.2024.11.018. Epub 2025 Feb 11. PMID: 39938482; PMCID: PMC11839170.

Awards and Honors

NIH Outstanding Investigator Award

Research Interest

Biomedical Research with a focus on immune mechanisms of inflammation in cardiovascular systems.

Myeloid metabolism in the amplification and resolution of cardiac inflammation

Cardiac Inflammation leading to heart failure is a growing concern with limited treatment options. Complexities in treatment stem from high comorbidity burden, including obesity and hypertension, yet underlying disease and inflammation-promoting mechanisms remain obscure. Our findings indicate elevated peripheral hematopoietic stem cells in patients with cardiometabolic Heart Failure with preserved Ejection Fraction. This inflammatory stem cell signature can be reproduced in animal models in which high fat diet and hypertension also trigger heart failure. Elevated stem cells require intercellular communication with

peripheral macrophages, and this is perpetuated by cardiometabolic stress. Uncoupling of this crosstalk by inhibiting either macrophage lipid uptake, or expression of myeloid intercellular adhesion molecules, ameliorated heart failure and systemic inflammation, respectively. This can be explained by heightened mitochondrial fatty acid metabolism, which in turn remodels adhesion molecule promoters to enhance gene expression. Our findings reveal a new stem cell signature of cardiometabolic heart failure patients, and a new immunometabolic crosstalk axis for future study.

CURRICULUM VITAE

Yong Sook Kim

Research Professor, Chonnam National University Hospital, Republic of Korea



Education and Training

1992.02	Chonnam National University, Korea, BS, Food and Nutrition
1995.02	Chonnam National University, Korea, MS, Cell Signaling Biochemistry
2004.04	Chonnam National University, Korea, PhD, Cell Signaling Biochemistry

Employment and Position

2004-2007	Chonnam National University Hospital, Post-doc
2007-	Chonnam National University Hospital, Research Professor

Important Publications

1. IKK ϵ -deficient macrophages impede cardiac repair after myocardial infarction by enhancing macrophage-myofibroblast transition. *Exp Mol Med*, 2024 Sep;56(9):2052-64.
2. Paintable and Adhesive Hydrogel Cardiac Patch with Sustained Released of ANGPTL4. *Bioactive Materials*, 2024;31:395.
3. A conductive and adhesive hydrogel composed of MXene nanoflakes as a paintable cardiac patch for infarcted heart repair. *ACS Nano* 2023;12:12290.
4. ANGPTL4 stabilizes atherosclerotic plaques and modulates the phenotypic transition of vascular smooth muscle cell through KLF4 downregulation. *Exp Mol Med*, 2023 Feb;55(2):426-442.
5. Antiinflammatory activity of ANGPTL4 facilitates macrophage polarization to induce cardiac repair. *JCI Insight*. 2019;4(16).

Awards and Honors

1. Excellence in Peer Review Award, Korean Society of Cardiology (2024)
2. Paul Dudley White International Scholar Award, Basic Cardiovascular Science, The American Heart Association (2020, 2022)
3. Young Investigator Award, The Korean Society of Cardiology (2009)

Research Interest

Cardiovascular pathogenesis, Inflammation-induced fibrosis, Organoids, Lipoprotein metabolism

Therapeutic promise of ANGPTL4 in cardiovascular inflammation

Anti-inflammatory therapeutic strategies have emerged as promising approaches for cardiovascular disease treatment, given the central role of inflammation in disease progression. However, clinical translation remains challenging. Through our investigation of mesenchymal stem cell-derived anti-inflammatory factors, we discovered that angiopoietin-like 4 (ANGPTL4) represents a critical mediator of cardio-protective effects. We evaluated the therapeutic efficacy of ANGPTL4 in experimental model of atherosclerosis as a potential alternative regeneration therapy.

In atherosclerotic mice, ANGPTL4 treatment promoted plaque stabilization through enhanced fibrotic cap integrity, achieved by preventing the pathological

transdifferentiation of vascular smooth muscle cells into macrophages and inhibiting endothelial-to-mesenchymal transition. Clinical correlation studies demonstrated that elevated serum ANGPTL4 levels were associated with reduced cardiovascular disease risk, establishing a protective relationship in human subjects.

Our data reveal that ANGPTL4 exhibits robust anti-inflammatory activity with significant therapeutic benefits across diverse cardiovascular conditions. These results support the clinical development of ANGPTL4-targeted interventions for cardiovascular disease prevention and treatment, representing a viable pathway for advancing laboratory findings toward therapeutic application in patients.

Symposium 20

Optimized Management of Cardiometabolic Syndrome in the Elderly: Focus on Multimorbidity and Frailty

Sep 13(Sat) 08:50–10:20 | Room 3 (3F)

CHAIRPERSONS : Kwang-il Kim (Seoul National University, Republic of Korea)
Hidenori Arai (National Center for Geriatrics and Gerontology, Japan)

08:50–09:10 **Strategies for managing hyperlipidemia in fragile elderly patients**

Dong-Ho Shin (Yonsei University, Republic of Korea)

09:10–09:30 **Optimizing glucose control in fragile elderly patients with cardiometabolic syndrome**

Soo Jin Yun (Kyung Hee University, Republic of Korea)

09:30–09:50 **How to deal with multimorbidity and frailty for the prevention of cardiovascular disease**

Hidenori Arai (National Center for Geriatrics and Gerontology, Japan)

09:50–10:20 **Panel Discussion**

Jung-Woo Son (Yonsei University, Republic of Korea)

Min Kyung Lee (Hanyang University, Republic of Korea)

Ja Young Jeon (Ajou University, Republic of Korea)

Jung Yeon Choi (Seoul National University, Republic of Korea)

CURRICULUM VITAE**Dong-Ho Shin**

Clinical Professor, Yonsei University College of Medicine, Republic of Korea

**Education and Training**

2001.02	Seoul National University, Korea, M.D, Medicine
2009.02	Seoul National University, Korea, MPH, Epidemiology
2015.08	Yonsei University, Korea, Ph.D., Epidemiology

Employment and Position

2011-2016	Severance Cardiovascular Hospital, Assistant Professor
2016-2018	Duke Clinical Research Institute, Research Scholar
2018-	Severance Hospital, Assistant Professor → Associate Professor → Professor

Important Publications

1. Han, SJ, Shin Dong-Ho, et al. Nationwide survey of internal medicine hospitalists in Korea: motivation and sustainability of a hospitalist career. *Korean J. Intern. Med.* 38, 434-443 (2023).
2. Shin Dong-Ho. The Value of Hospital Medicine. *Korean J Medicine* 97, 28-32 (2022).
3. Shin Dong-Ho, Kang H-J, Jang J-S, et al. The Current Status of Percutaneous Coronary Intervention in Korea: Based on Year 2014 & 2016 Cohort of Korean Percutaneous Coronary Intervention (K-PCI) Registry. *Korean Circ J [Internet]*. 2019;49(12):1136.
4. Shin Dong-Ho, Hong SJ, Mintz GS, et al. Effects of Intravascular Ultrasound-Guided Versus Angiography-Guided New-Generation Drug-Eluting Stent Implantation: Meta-Analysis With Individual Patient-Level Data From 2,345 Randomized Patients. *JACC Cardiovasc Interv* 2016;9(21):2232-9.

Research Interest

Epidemiology, Meta-analysis
Digital therapeutics, Hypertension, Hospital medicine

Strategies for managing hyperlipidemia in fragile elderly patients

This lecture will provide an overview of practical and patient-centered approaches to lipid management in frail older adults. As the elderly population grows, clinicians are increasingly confronted with the challenges of balancing cardiovascular risk reduction against potential harms from intensive lipid-lowering therapy. The session will discuss key considerations

including individualized risk assessment, cautious initiation and adjustment of statins, and the role of non-statin agents. Attention will also be given to tailoring lifestyle interventions to functional capacity and integrating shared decision-making to align treatment with patient goals and quality of life.

CURRICULUM VITAE

Soo Jin Yun

Assistant Professor, Kyung Hee University, Republic of Korea



Education and Training

2025.02	Kyung Hee University, Korea, Ph.D., Internal Medicine
2014.02	Kyung Hee University, Korea, M.D., Medicine
2008.02	Yonsei University, Korea, B.A., B.S., Biology, Chinese & Chinese Literature

Employment and Position

2025-	Kyung Hee University Hospital, Assistant Professor
2021-2025	Kyung Hee University Hospital, Clinical Assistant Professor
2019-2021	Kyung Hee University Hospital, Fellow
2015-2019	Kyung Hee University Hospital, Resident
2014-2015	Kyung Hee University Hospital, Intern

Important Publications

1. Yun, S.J., et al., Effect of Hyperprolactinemia on Bone Metabolism: Focusing on Osteopenia/Osteoporosis. *Int J Mol Sci*, 2024. 25(3): p. 1474.
2. Yun, S.J., et al., Descriptive Epidemiology and Survival Analysis of Acromegaly in Korea. *J Korean Med Sci*, 2021. 36(23): p. e159.
3. Yun, S.J., et al., Current Status of Low-Density Lipoprotein Cholesterol Target Achievement in Patients with Type 2 Diabetes Mellitus in Korea Compared with Recent Guidelines. *Diabetes Metab J*, 2021. 46(3): p. 464-475.
4. Choi, E.M., et al., Oleuropein attenuates the 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)-perturbing effects on pancreatic beta-cells. *J Environ Sci Health A Tox Hazard Subst Environ Eng*, 2021. 56(7): p. 752-761.
5. Park, J.S., et al., Descriptive Epidemiology and Survival Analysis of Prolactinomas and Cushing's Disease in Korea. *Endocrinol Metab (Seoul)*, 2021. 36(3): p. 688-696.

Research Interest

Diabetes, elderly diabetes, and diabetes prevention

Optimizing glucose control in fragile elderly patients with cardiometabolic syndrome

The growing elderly population faces rising burdens of diabetes and cardiometabolic syndrome, with frailty, multimorbidity, and polypharmacy increasing the risks of hypoglycemia, falls, and hospitalization. This lecture highlights individualized glucose management for fragile older adults, emphasizing guideline-based relaxed HbA1c targets to ensure safety and preserve function. Therapeutic strategies include minimizing

sulfonylurea or intensive insulin use, while favoring agents with cardiovascular and renal benefits such as SGLT2 inhibitors and GLP-1 receptor agonists. Lifestyle, nutrition, and resistance exercise remain crucial. A multidisciplinary approach aims not at strict normalization but at safeguarding quality of life and cardiometabolic health.

CURRICULUM VITAE

Hidenori Arai

President, National Center for Geriatrics and Gerontology, Japan



Education and Training

1978–1984	Kyoto University School of Medicine/Kyoto, M.D., Medicine
1984	Japanese Medical License Registration (No. 279757)
1984	Kyoto University Hospital/Kyoto, Resident, Internal Medicine
1987–1991	Kyoto University Graduate School of Medicine/Kyoto, Ph.D., Internal Medicine

Employment and Position

2003–2009	Department of Geriatric Medicine, Kyoto University Graduate School of Medicine, Assistant Professor
2009–2014	Department of Human Health Sciences, Kyoto University Graduate School of Medicine, Professor
2015–2018	National Center for Geriatrics and Gerontology, Deputy Director
2015–2019	National Center for Geriatrics and Gerontology, Director
2019–Present	National Center for Geriatrics and Gerontology, President

Important Publications

1. Sakurai Y, Hori A, Okamura M, Tsuge T, Sato H, Nakashima Y, Endo K, Hayashi S, Yamamoto N, Matsumoto D, Fudeyasu K, Arai H. Digital health interventions for non-older individuals at risk of frailty: A systematic review and meta-analysis. *Digit Health*. 2025;11:20552076251328566.
2. Chen LK, Meng LC, Peng LN, Lee WJ, Zhang S, Nishita Y, Otsuka R, Yamada M, Pan WH, Kamaruzzaman SB, Woo J, Hsiao FY, Arai H. Mapping Normative Muscle Health Metrics Across the Aging Continuum: A Multinational Study Pooling Data From Eight Cohorts in Japan, Malaysia and Taiwan. *J Cachexia Sarcopenia Muscle*. 2025;16(1):e13731.

How to deal with multimorbidity and frailty for the prevention of cardiovascular disease

Cardiovascular disease (CVD) remains a leading cause of global mortality and morbidity, posing a significant barrier to healthy aging. While traditional risk factor management has improved outcomes, the growing prevalence of multimorbidity and frailty, particularly in older adults, presents a complex challenge to CVD prevention. These conditions synergistically interact; for example, diabetes and chronic kidney disease can increase frailty risk, while frailty itself can elevate diabetes risk and, notably, CVD risk independently of multimorbidity.

Frailty, characterized by decreased physiological reserve, necessitates a cautious approach to aggressive CVD risk factor modification. Intense glycemic control, for instance, may be poorly tolerated and harmful in frail individuals, increasing risks like hypoglycemia and falls without significant CVD benefit. Therefore, managing CVD risk in this population shifts towards

preserving functional status and quality of life. This involves prioritizing tailored physical activity, nutritional support, and managing symptoms that impair daily living. Comprehensive geriatric assessment is crucial for identifying specific vulnerabilities and informing personalized care plans.

Ultimately, effective CVD prevention in older adults with multimorbidity and frailty requires moving beyond purely disease-specific targets. A patient-centered approach that balances CVD risk reduction with maintaining function and minimizing treatment burden is essential for healthy aging. This necessitates integrating person-centered care models, shared decision-making, and interdisciplinary team approaches to provide holistic, individualized care. This presentation will explore best practices and innovative strategies for addressing these complex interactions in CVD prevention.

Symposium 21

From Treatment to Prevention: A Paradigm Shift in Obesity

Sep 13(Sat) 08:50–10:20 | Room 4 (5F)

CHAIRPERSONS : Jang-Young Kim (Yonsei University, Republic of Korea)
Cheol-Young Park (Sungkyunkwan University, Republic of Korea)

08:50–09:10 **Anti-obesity effects of bioactive compound from natural products: a mechanism-oriented approach focusing on cellular signaling and metabolic regulation**

Choon Young Kim (Yeungnam University, Republic of Korea)

09:10–09:30 **Semaglutide for obesity treatment and cardiometabolic benefits**

Yun Kyung Cho (University of Ulsan, Republic of Korea)

09:30–09:50 **Tirzepatide a dual GIP GLP-1 receptor agonist in obesity and metabolic care**

Sang-Ho Jo (Hallym University, Republic of Korea)

09:50–10:20 **Panel Discussion**

Bu Kyung Kim (Kosin University, Republic of Korea)

Hyun-Suk Yang (Konkuk University, Republic of Korea)

Hyemoon Chung (Kyung Hee University, Republic of Korea)

CURRICULUM VITAE

Choon Young Kim

Professor, Yeungnam University, Republic of Korea



Education and Training

2007.12	Purdue University, USA, MS, Food and Nutrition
2011.12	Purdue University, USA, Ph.D., Food Science

Employment and Position

2011–2014	Purdue University, Research Fellow
2014–	Yeungnam University, Professor

Important Publications

1. Lee, T-W., Song, Y.-B., Kim, C.Y., Lee, J.H., Lee, B.-H. Regulation of Glucose Uptake Level by Black Corn-Derived Anthocyanins at the Small Intestinal α -Glucosidases and Different Types of Glucose Transporters. *Journal of Agricultural and Food Chemistry* 2025. 73(23), 14523–14532.
2. Aung, T., Kim, C.Y. Kim, M.J. Fabrication and characterization of novel sericin bigels for the co- encapsulation of bioactive ingredients and in vitro gastrointestinal release profile. *Food Hydrocolloids* 2025. 166, 111386.
3. Maykrishan, V. Knnapan, P., Balakarthikeyan, J. Kim, C.Y. Rodent model intervention for prevention and optimal management of sarcopenia: A systematic review on the beneficial effects of nutrients & non-nutrients and exercise to improve skeletal muscle health. *Ageing Research Review* 2024. 102, 102543.
4. Maykrishan, V. Lee, D.-H., Kim, K.-H., Kim, C.Y. Role of corn peptide powder in lipopolysaccharide-induced inflammatory responses in 3T3-L1 adipocytes. *Nutrients* 2024. 16, 1924.
5. Kim, J., Kim, N.-H., Youn, I., Seo, E.K., Kim, C.Y. Effects of *Allium macrostemon* bunge extract on adipose tissue inflammation and hepatic endoplasmic reticulum stress in high-fat diet-fed and bisphenol A-treated C57BL/6N mice. *Foods* 2023. 12, 3777.

Anti-obesity effects of bioactive compound from natural products: a mechanism-oriented approach focusing on cellular signaling and metabolic regulation

Obesity is a global health challenge closely associated with chronic inflammation and metabolic dysregulation in adipose tissue. Increasing interest has emerged in the use of natural bioactive compounds for obesity management, particularly through modulation of cellular signaling pathways. *Allium macrostemon*, traditionally consumed in East Asia, has recently gained attention as a potential anti-obesity agent due to its bioactive phytochemicals. This study investigated the anti-obesity effects of *Allium macrostemon* extract (AME) in a high-fat diet (HFD)-induced obesity model in C57BL/6N mice, along with its cellular mechanisms in 3T3-L1 preadipocytes. Mice fed an HFD and supplemented with AME (200 mg/kg/day) for 9 weeks showed significantly reduced body weight gain, fat mass, and adipocyte hypertrophy compared to HFD-only controls. AME supplementation downregulated key adipogenic and lipogenic genes, including PPAR γ , C/EBP β , and fatty acid synthase (FASN), and

also attenuated adipose tissue inflammation. These effects were supported by reduced crown-like structures and decreased mRNA and protein levels of inflammatory cytokines (e.g., IL-6 and TNF- α) and ER stress markers (e.g., GRP78, CHOP, and phosphorylated JNK). In vitro, the leaf extract of *A. macrostemon* (LAE) significantly inhibited adipocyte differentiation in 3T3-L1 cells, reducing intracellular lipid accumulation by nearly 50% at 100 μ g/mL. LAE suppressed the expression of adipogenic transcription factors, including C/EBP β , C/EBP α , and PPAR γ . The anti-adipogenic effects of LAE may be attributed to its high antioxidant activity and rich polyphenol content, particularly quercetin and kaempferol derivatives. These findings suggest that *Allium macrostemon*, especially its whole-plant and leaf extracts, holds promise as a functional food ingredient for the prevention of obesity via modulation of adipogenesis, inflammation, and ER stress-related signaling pathways.

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. Efficacy and safety of pioglitazone, empagliflozin and glimepiride as third-line agents in patients with type 2 diabetes inadequately controlled with metformin and DPP-4 inhibitors: A multicentre, phase 4 randomized controlled trial. Cho YK, Cho JH, Hong SM, Park JH, Lee BW, Yoo JH, Kim JH, Chun SW, Hwang YC, Song KH, Lee WJ. *Diabetes Obes Metab*. 2025 Aug 14. doi: 10.1111/dom.70030. Online ahead of print.
2. Efficacy and safety of combination therapy using SGLT2 and DPP4 inhibitors to treat type 2 diabetes: An updated systematic review and meta-analysis with focus on an Asian subpopulation. Kim MJ, Cho YK, Kim S, Moon JY, Jung CH, Lee WJ. *Diabetes Obes Metab*. 2025 Sep;27(9):5019–5031. doi: 10.1111/dom.16550. Epub 2025 Jun 24.
3. 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*. 2025 Jul 22. doi: 10.4093/dmj.2024.0693. Online ahead of print.

Semaglutide for obesity treatment and cardiometabolic benefits

Semaglutide, a long-acting glucagon-like peptide-1 receptor agonist (GLP-1 RA), represents a paradigm shift in obesity and cardiometabolic care. Engineered through acylation with a C18 fatty diacid chain, it achieves strong albumin binding, an extended half-life, and stable plasma concentrations, enabling once-weekly subcutaneous administration. By activating the GLP-1 receptor, semaglutide enhances glucose-dependent insulin secretion, suppresses glucagon, delays gastric emptying, and reduces appetite through central and peripheral mechanisms, thereby addressing both energy balance and glycemic regulation.

The efficacy of semaglutide in obesity management has been robustly demonstrated in the STEP clinical trial program. In STEP 1, semaglutide 2.4 mg induced a mean weight reduction of 14.9% at 68 weeks in adults without diabetes, while STEP 2 confirmed meaningful weight loss in patients with type 2 diabetes. The long-term STEP 5 trial further established sustained weight reduction over two years, positioning semaglutide as the first GLP-1 RA approved for chronic weight management.

Beyond weight reduction, semaglutide has shown compelling cardiometabolic benefits. The SELECT trial demonstrated a 20% reduction in major adverse cardiovascular events among overweight and obese adults with established cardiovascular disease but without diabetes, highlighting its disease-modifying potential. In addition, semaglutide has shown promise in metabolic dysfunction-associated steatotic liver disease (MASLD) and heart failure with preserved ejection fraction (HF-pEF), with improvements in hepatic steatosis, biomarkers, exercise tolerance, and quality of life.

Oral semaglutide, developed to improve accessibility and adherence, has also demonstrated significant efficacy in glycemic control, weight reduction, and cardiovascular outcomes, as confirmed in the SOUL trial and the OASIS 1 study.

Collectively, semaglutide has redefined the therapeutic landscape of obesity, extending benefits across multiple cardiometabolic domains, and serving as a benchmark for next-generation incretin-based multi-agonists.

CURRICULUM VITAE

Sang-Ho Jo

Professor, Div. of Cardiology, Dept. of Internal Medicine, College of Medicine,
Hallym University, Hallym University Sacred Heart Hospital, 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

Societies

Academic activities as editor and reviewer:
 Editor of the Journal of Korean Medical Science (JKMS) 2012-2015
 Editor of the of the Korean Circulation Journal (KCJ)
 Editor of the of Journal of the Korean Society of Hypertension
 Editor of the Korean Edition of Circulation. Aug 2008
 Editor of the Korean Edition of Hypertension. July 2007.
 Editor of the Korean Edition of Atherosclerosis, thrombosis, vascular biology (ATVB), March 2009
 Editor of the Korean Edition of European Heart Journal
 Member of Heart Failure Research at Korean Heart Society. May 2008
 Editorial members of Journal of the Korean Society of Hypertension

Tirzepatide a dual GIP GLP-1 receptor agonist in obesity and metabolic care

Tirzepatide is the first dual GIP and GLP-1 receptor agonist, combining complementary incretin pathways to achieve unprecedented weight loss and broad metabolic benefits. In SURMOUNT-1, mean weight reduction approached 20% at 72 weeks, with consistent improvements in glycemia, blood pressure, and lipids. The drug received FDA approval (Zepbound, 2023) for chronic weight management, marking a paradigm shift comparable in efficacy to bariatric surgery.

Beyond weight loss, tirzepatide has shown disease-specific impact. SURMOUNT-OSA demonstrated significant reductions in apnea-hypopnea index and body weight, leading to its approval for obesity-related obstructive

sleep apnea in 2024. Pooled analyses indicate reductions in albuminuria, suggesting renal protective potential.

Cardiovascular outcomes remain under investigation: SURPASS-CVOT confirmed noninferiority versus dulaglutide, with full results anticipated. Until definitive data emerge, the robust weight-centric and cardiometabolic effects already support its integration into cardiometabolic care.

Overall, tirzepatide establishes a new benchmark in pharmacological management of obesity, bridging endocrinology, cardiology, and sleep medicine, and setting the stage for broader risk-reduction strategies in high-risk populations.

Symposium 22

Atherosclerosis beyond Traditional Risks: Emerging Clinical Challenges in Comorbid Conditions

Sep 13(Sat) 14:50–16:20 | Room 3 (3F)

CHAIRPERSONS : Sang Hong Baek (The Catholic University of Korea, Republic of Korea)
Masayuki Yoshida (Institute of Science Tokyo, Japan)

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- | | |
|-------------|---|
| 14:50–15:10 | Atherosclerosis and lipid management in cancer survivors: balancing cardiovascular and oncologic risks
Nuri Lee (Chonnam National University, Republic of Korea) |
| 15:10–15:30 | Metabolic dysfunction–associated steatotic liver disease as a cardiovascular risk equivalent
Won Kim (Seoul National University, Republic of Korea) |
| 15:30–15:50 | Atherosclerosis in chronic inflammatory diseases: focus on rheumatoid arthritis and systemic autoimmunity
Ji-Won Kim (Daegu Catholic University, Republic of Korea) |
| 15:50–16:20 | Panel Discussion
Minjeong Kim (Ewha Womans University, Republic of Korea)
Sungjoon Park (Seoul National University, Republic of Korea)
Sung Kee Ryu (Ewha Womans University, Republic of Korea) |

CURRICULUM VITAE

Nuri Lee

Clinical Assistant Professor, Chonnam National University Hwasun Hospital, Republic of Korea



Education and Training

2016.02	Chonnam National University, Korea, M.D, Medical Science
2020.02	Chonnam National University, Korea, Master's degree, Medical Science
2025.02	Chonnam National University, Korea, Ph.D., Medical Science

Employment and Position

2016–2017	Chonnam National University Hospital, Internship
2017–2020	Chonnam National University Hospital, Resident, Internal Medicine
2020–2022	Chonnam National University Hospital, Clinical Fellowship
2022–	Chonnam National University Hwasun Hospital, Clinical Assistant Professor

Important Publications

1. 2025. Lee N, Lee W, Park H. Pneumopericardium treated with repeated pericardiocentesis: a case report. Eur Heart J Case Rep. 2025;9(2):ytaf031.
2. 2024. Park H, Lee N, Hwang CH, et al. Prognosis After Withdrawal of Cardioprotective Therapy in Patients With Improved Cancer Therapeutics-Related Cardiac Dysfunction. JACC CardioOncol. 2024;6(5):699–710.
3. 2024. Lee N, Kim KH, Park JH, et al. COVID-19 Vaccination-Related Pericarditis: A Korean Nationwide Study. Mayo Clin Proc. 2024;99(10):1577–1588.
4. 2023. Lee N, Bang H, Park H, Shim HJ. Case report: Successful treatment of malignant pericardial effusion with pericardiocentesis, concurrent anti-inflammatory therapy and cancer therapy. Front Cardiovasc Med. 2023;10:1285233.
5. 2022. Lee N, Cho JY, Kim KH, et al. Impact of Cardiac Troponin Elevation on Mortality of Patients with Acute Heart Failure: Insights from the Korea Acute Heart Failure (KorAHF) Registry. J Clin Med. 2022;11(10):2800.

Atherosclerosis and lipid management in cancer survivors:
balancing cardiovascular and oncologic risks

Historically, the diagnosis of cancer shifted medical focus away from long-term health maintenance toward urgent, life-saving interventions. Conditions like atherosclerosis were often deprioritized in favor of addressing the immediate threat of cancer. As a result, cardiovascular care was not seen as highly relevant for most cancer patients. However, with advances in cancer treatment, more patients now achieve remission and live longer, transforming many into long-term survivors. This shift calls for a reassessment of chronic disease management, including cardiovascular risk reduction.

Cholesterol management, particularly through statin use, is widely acknowledged as beneficial. While offering statins to all cancer patients who meet guideline-based criteria seems reasonable, the situation is nuanced. Dyslipidemia management involves ongoing blood monitoring, possible dose changes, and poten-

tial side effects, which may not be appropriate for all patients—especially those with limited life expectancy. Therefore, a blanket approach to lipid-lowering therapy may not be effective or necessary across all cancer subgroups.

To address these complexities, the involvement of specialized cardio-oncology teams is proposed. These multidisciplinary teams would assess both cancer prognosis and cardiovascular risk, tailoring prevention strategies to each individual while respecting patient preferences. Their role would be especially critical at two time points: at cancer diagnosis and during disease control or progression, allowing for dynamic, updated cardiovascular risk assessments. In the absence of robust clinical trial data, such personalized, team-based approaches offer a practical and thoughtful alternative to improve cardiovascular outcomes in cancer patients.

CURRICULUM VITAE

Won Kim

Professor, Seoul National University College of Medicine, Republic of Korea



Education and Training

1997.02	Seoul National University, Korea, M.D, Medicine
2007.02	Seoul National University, Korea, Ph.D, Internal Medicine

Employment and Position

2008–2024	Seoul National University College of Medicine, Professor
2018–2019	Korean Liver Cancer Association, Director of Research Committee
2022–2023	Korean Association for the Study of the Liver (KASL), Director of Research Committee
2024–	Korean Association for the Study of the Liver (KASL), Director of Publication Committee

Important Publications

1. Single-cell eQTL analysis identifies genetic variation underlying metabolic dysfunction-associated steatohepatitis. *Nature Genetics*. 2025.
2. Outcomes of Various Classes of Oral Antidiabetic Drugs on Nonalcoholic Fatty Liver Disease. *JAMA Intern Med*. 2024.
3. Metabolic dysfunction-associated steatotic liver disease increases the risk of incident cardiovascular disease: a nationwide cohort study. *EClinicalMedicine*. 2023.
4. Steatotic liver disease predicts cardiovascular disease and advanced liver fibrosis: A community-dwelling cohort study with 20-year follow-up. *Metabolism*. 2024.
5. Tropifexor for nonalcoholic steatohepatitis: an adaptive, randomized, placebo-controlled phase 2a/b trial. *Nature Medicine*. 2023.

Metabolic dysfunction-associated steatotic liver disease as a cardiovascular risk equivalent

The causal relationship between metabolic dysfunction-associated steatotic liver disease (MASLD) and cardiovascular disease (CVD) remains an area of active scientific debate. Epidemiological studies have consistently demonstrated an increased prevalence of CVD in patients with MASLD; however, determining causality requires careful analysis beyond mere associations. This presentation explores the causal relationship between MASLD and cardiovascular disease (CVD), synthesizing recent evidence from large-scale genetic and epidemiological studies. Key mechanisms linking MASLD to CVD—including insulin resistance, inflammation, oxidative stress, lipotoxicity, endothelial dysfunction, and gut microbiota alterations—are explored in depth. Additionally, genetic insights from Mendelian randomization studies provide perspective on potential causal pathways. Clinical evidence from cohort studies and meta-analyses suggests a gradient relationship between MASLD severity and CVD risk, but confounding factors and reverse causation remain important considerations. Two distinct MASLD subtypes were identified through data-driven clustering: i) liver-specific type and ii) cardiometabolic type. Genetic studies reveal variants

like PNPLA3 rs738409 that increase liver disease risk but may protect against CVD, suggesting a complex causal relationship. Partitioned polygenic risk scores (pPRS) also demonstrate differential associations: i) liver-specific pPRS and ii) systemic pPRS. Transcriptomic and metabolomic analyses support distinct biological profiles for these MASLD subtypes. Long-term follow-up data from the UK Biobank confirm divergent clinical trajectories for the identified MASLD types. These findings challenge the notion of a simple causal relationship between MASLD and CVD, highlighting the disease's heterogeneity and the need for personalized risk assessment and management strategies. The identification of distinct MASLD subtypes with different underlying biology and clinical outcomes suggests potential for tailored therapeutic approaches and more precise prognostication. Ultimately, while compelling data support a significant association between MASLD and increased cardiovascular risk, definitive evidence establishing direct causality remains elusive. Further prospective studies and randomized controlled trials are imperative to clarify this complex relationship and inform targeted clinical interventions.

CURRICULUM VITAE

Ji-Won Kim

Associate Professor, Daegu Catholic University School of Medicine, Republic of Korea



Education and Training

2011.02	Daegu Catholic University, Korea, M.D, Medicine
2020.02	Catholic University of Korea, Korea, Ph.D., Internal Medicine (Rheumatology)

Employment and Position

2011–2016	Daegu Catholic University Medical Center, Intern & Resident
2016–2017	Daegu Catholic University Medical Center, Fellow
2017–2019	Seoul St. Mary's Hospital, Fellow
2019–2025	Daegu Catholic University School of Medicine, Assistant Professor
2025–	Daegu Catholic University School of Medicine, Associate Professor

Important Publications

1. Kim JW, Yoon JS, Park S, Kim H, Lee JS, Choe JY. Risk of cardiovascular disease with high-dose versus low-dose use of non-steroidal anti-inflammatory drugs in ankylosing spondylitis. *Ann Rheum Dis*. 2024;83:1028–1033.
2. Kim JW, Yoon JS, Park S, Kim H, Kim BY, Lee H, Park SH, Kim SK, Choe JY. Risk of cardiovascular disease associated with long-term use of non-steroidal anti-inflammatory drugs in ankylosing spondylitis. *Rheumatology*. 2025;64:261–267.
3. Kim JH, Lee G, Hwang J, Kim JW, Kwon JW, Song YK. Performance of Cardiovascular Risk Prediction Models in Korean Patients With New-Onset Rheumatoid Arthritis: National Cohort Study. *J Am Heart Assoc*. 2023;12:e030604.
4. Song YK, Lee G, Hwang J, Kim JW, Kwon JW. Cardiovascular risk of Janus kinase inhibitors compared with biologic disease-modifying antirheumatic drugs in patients with rheumatoid arthritis without underlying cardiovascular diseases: a nationwide cohort study. *Front Pharmacol*. 2023;14:1165711.
5. Kim JW, Chung SW, Pyo JY, Chang SH, Kim MU, Park CH, Lee JS, Lee JS, Ha YJ, Kang EH, Lee YA, Park YB, Lee EY, Choe JY. Methotrexate, leflunomide and tacrolimus use and the progression of rheumatoid arthritis-associated interstitial lung disease. *Rheumatology (Oxford)*. 2023;62:2377–2385.

Atherosclerosis in chronic inflammatory diseases: focus on rheumatoid arthritis and systemic autoimmunity

Rheumatic diseases, including rheumatoid arthritis (RA) and ankylosing spondylitis (AS), have a higher risk of cardiovascular disease (CVD) than the general population. Management of CVD would be important because it is a main cause of death in rheumatic diseases.

Atherosclerosis is involved in the development of CVD in rheumatic diseases. Endothelial dysfunction and increased carotid intima-media thickness and atherosclerotic plaques are observed more commonly in patients with RA and AS than in the general population. Atherosclerosis is highly associated with traditional cardiovascular (CV) risk factors, but it is also observed in patients without such risk factors. This suggests that traditional CV risk factors alone cannot explain the higher risk of CVD in rheumatic diseases. The actual occurrence of CVD is underestimated when predicting the risk of CVD using the CV risk prediction algorithms in patients with

RA and AS. It is considered that nontraditional factors are involved in development of CVD, and many studies assume that inflammation is one of them.

Studies have found that disease activity, inflammatory markers such as ESR and CRP, and autoantibodies were related to the occurrence of atherosclerosis and CVD in patients with RA and AS, and controlling inflammation might delay the progression of atherosclerosis and reduce the risk of CVD. Conventional, biological, and targeted DMARDs can reduce the risk of CVD by controlling inflammation, although some drugs might have increased CV risk due to having negative effects on traditional CV risk factors or inducing thrombotic events.

Therefore, it is important to control both traditional CV risk factors and inflammation in the management of CVD in rheumatic diseases.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Symposium 23

Publication Committee Session: JLA Award for Outstanding Article

Sep 13(Sat) 14:50–16:20 | Room 4 (5F)

CHAIRPERSONS : Hyuk-Sang Kwon (The Catholic University of Korea, Republic of Korea)
In-Kyung Jeong (Kyung Hee University, Republic of Korea)

- | | |
|-------------|--|
| 14:50–15:05 | Discrepant Effect of High-Density Lipoprotein Cholesterol on the Hematologic Malignancy Risk: A Nationwide Cohort Study
Su Youn Nam (Kyungpook National University, Republic of Korea) |
| 15:05–15:20 | Cholesterol and Cardiovascular Risk in Type 2 Diabetes: The Role of Kidney Function
Mee Kyoung Kim (The Catholic University of Korea, Republic of Korea) |
| 15:20–15:35 | Association Between Lipoprotein (a) Levels and Coronary Artery Disease (CAD) Among Patients With or Without CAD Family History
Hayato Tada (Kanazawa University, Japan) |
| 15:35–15:50 | Investigation of the Influence of Lipoprotein(a) and Oxidized Lipoprotein(a) on Plasminogen Activation and Fibrinolysis
Wen-Liang Song (Brown University Health, USA) |
| 15:50–16:05 | Fatty Acids in Childhood Obesity: A Link Between Nutrition, Metabolic Alterations and Cardiovascular Risk
Belen Davico (University of Buenos Aires, Argentina) |

CURRICULUM VITAE

Su Youn Nam

Professor, Kyungpook National University, School of Medicine, Republic of Korea



Education and Training

1997.02	Kyungpook National University, Korea, M.D, Medicine
2005.08	Kangwon National University, Korea, Ph.D., Pharmacology

Employment and Position

2003-2005	Seoul National University Hospital, Fellow
2005-2015	National Cancer Center, Staff Scientist
2010-2012	Visiting scholar, Experimental therapeutics, MD Anderson Cancer Center (long non-coding RNA, GI cancer)
2015-2025	Kyungpook National University, Professor

Important Publications

1. A population-based cohort study of longitudinal change of high-density lipoprotein cholesterol impact on gastrointestinal cancer risk. Nature communications 2024.
2. Factor Modification in the Association between High-density Lipoprotein Cholesterol and Liver Cancer Risk in a Nationwide Cohort. International journal of epidemiology 2024.
3. Discrepant effect of high-density lipoprotein cholesterol on esophageal and gastric cancer risk in a nationwide cohort. Gastric cancer 2024 May;27(3):451-460.
4. Therapeutic potential of FLANC, a novel primate-specific long non-coding RNA in colorectal cancer. Gut. 2020;69:1818-1831.
5. Demographic and Clinical Factors Associated With Anti-SARS-CoV-2 Antibody Levels After 2 BNT162b2 mRNA Vaccine Doses. JAMA Netw Open. 2022 May 2;5(5):e2212996.

Discrepant Effect of High-Density Lipoprotein Cholesterol on the Hematologic Malignancy Risk: A Nationwide Cohort Study

Objective: Although high-density lipoprotein cholesterol (HDL-C) has been inversely associated with hematologic malignancies, potential modification by smoking has not been explored. We aimed to investigate the modifying roles of smoking and menopausal status in these associations.

Methods: In this population-based cohort study, cancer-free individuals who underwent the Korean National Cancer Screening Program in 2010 were followed until December 2017. HDL-C levels were categorized into eight groups at 10 mg/dL intervals (<30, 30-39, 40-49, 50-59, 60-69, 70-79, 80-89, and ≥90 mg/dL).

Results: Among 4,517,892 participants, 5,887 developed lymphoma, 3,348 developed leukemia, and 12,151 developed other hematologic malignancies. Compared with the reference range of 70-79 mg/dL, the adjusted hazard ratios (aHRs) for the lowest HDL-C

category were 1.83 (95% CI, 1.45-2.31) for lymphoma, 3.14 (95% CI, 2.41-4.08) for leukemia, and 2.34 (95% CI, 2.01-2.72) for other hematologic malignancies. The detrimental effects of low HDL-C were generally consistent across sexes. Low HDL-C was associated with a higher risk of leukemia regardless of smoking status, whereas extremely high HDL-C levels increased the risk of leukemia (aHR, 2.32; 95% CI, 1.18-4.55) only among current smokers. The adverse effect of low HDL-C on lymphoma was significant only among never smokers (aHR, 2.01; 95% CI, 1.51-2.68). For leukemia, the harmful association of low HDL-C was observed exclusively in postmenopausal women (aHR, 2.94; 95% CI, 1.69-5.11).

Conclusion: Low HDL-C levels were associated with increased risks of lymphoma and leukemia, with distinct effect modifications by smoking and menopausal status.

CURRICULUM VITAE

Mee Kyoung Kim

Professor, The Catholic University of Korea, Republic of Korea



Education and Training

2001.02	The Catholic University of Korea, M.D, Medicine
2012.08	The Catholic University of Korea, Ph.D., Internal Medicine

Employment and Position

2009-2016	Yeouido St. Mary's Hospital, Assistant Professor
2016-2019	Yeouido St. Mary's Hospital, Associate Professor
2020-	Eunpyeong St. Mary's Hospital, Professor

Important Publications

1. Han K, Kim B, Lee SH, Kim MK. A nationwide cohort study on diabetes severity and risk of Parkinson disease. NPJ Parkinsons Dis. 2023 Jan 27;9(1):11.
2. Kim MK, Lee KN, Han K, Lee SH. Diabetes Duration, Cholesterol Levels, and Risk of Cardiovascular Diseases in Individuals With Type 2 Diabetes. J Clin Endocrinol Metab. 2024 Nov 18;109(12):e2317-e2323.
3. Kim MK, Kim J, Park SJ, Song YJ, Kwon HS. Impact of caloric restriction on diabetes remission in Korean adults with obesity (CREDO-K study). Diabetes Obes Metab. 2025 Mar;27(3):1609-1613.

Research Interest

Diabetes mellitus, Obesity

Cholesterol and Cardiovascular Risk in Type 2 Diabetes: The Role of Kidney Function

The association between lipid parameters and cardiovascular disease (CVD) risk in patients with type 2 diabetes mellitus (T2DM) may vary according to kidney function, but this relationship has not been fully elucidated. Using the Korean National Health Insurance Service Cohort database, we analyzed 2,343,882 patients with T2DM who underwent health examinations between 2015 and 2016 and were followed until December 2020. Baseline lipid levels and kidney function were evaluated, and participants were categorized by estimated glomerular filtration rate (eGFR) into three groups: ≥ 60 , 30-59, and < 30 mL/min/1.73 m². The group with eGFR ≥ 60 and low-density lipoprotein cholesterol (LDL-C) < 70 mg/dL was used as the reference. In patients with eGFR ≥ 60 , the risk of CVD

significantly increased when LDL-C was ≥ 100 mg/dL. Among those with eGFR 30-59, the risk of CVD was elevated by 43% even at LDL-C < 70 mg/dL and increased progressively with higher LDL-C levels: hazard ratios (95% confidence intervals) were 1.48 (1.43-1.53) for 70-99 mg/dL, 1.54 (1.49-1.60) for 100-129 mg/dL, 1.55 (1.48-1.63) for 130-159 mg/dL, and 1.88 (1.77-2.00) for ≥ 160 mg/dL. In patients with eGFR < 30 , a 3.3-fold higher CVD risk was observed even when LDL-C was < 70 mg/dL. These findings indicate that the LDL-C threshold associated with increased CVD risk differs according to kidney function and suggest that individualized lipid-lowering strategies based on renal function are essential for optimizing cardiovascular risk reduction in patients with T2DM.

CURRICULUM VITAE

Hayato Tada

Assistant Professor, Kanazawa University, Japan



Education and Training

2003.03	Kanazawa University, Japan, M.D, Medicine
2011.06	Kanazawa University, Japan, Ph.D., Cardiovascular Medicine

Employment and Position

2003–2012	Kanazawa University Hospital, Staff
2012–2014	Massachusetts General Hospital, 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.

Association Between Lipoprotein (a) Levels and Coronary Artery Disease (CAD) Among Patients With or Without CAD Family History

Objective: Lipoprotein (a) (Lp[a]), which is a highly heritable trait, is associated with coronary artery disease (CAD). However, the insight into whether the association between Lp(a) and CAD differs according to the family history of CAD remains unclear.

Methods: We investigated clinical data of 4,512 participants who underwent serum Lp(a) level measurement at Kanazawa University Hospital between 2008 and 2016. The association between Lp(a) and CAD according to CAD family history was investigated through logistic regression analyses.

Results: CAD family history and Lp(a) levels were significantly associated with CAD development (odds ratio [OR]: 1.32, 95% confidence interval [CI]: 1.12–1.52, $P < 0.001$, and OR: 1.13, 95% CI: 1.03–1.23, $P < 0.001$

per 10 mg/dL, respectively). In patients without CAD family history, those with Lp(a) levels ≥ 30 mg/dL had higher CAD risk than those with Lp(a) levels < 30 mg/dL (reference) (OR: 1.33, 95% CI: 1.05–1.61, $P < 0.001$). In patients with CAD family history, those who had Lp(a) levels < 30 and ≥ 30 mg/dL were both highly at risk for CAD (OR: 1.24, 95% CI: 1.04–1.44, $P < 0.001$, and OR: 1.68, 95% CI: 1.34–2.02, $P < 0.001$, respectively). Adding CAD family history and Lp(a) information to other conventional risk factors enhanced CAD risk discrimination (C-statistics: 0.744 [0.704–0.784] to 0.768 [0.730–0.806], and 0.791 [0.751–0.831], respectively; $P < 0.05$ for both).

Conclusion: Lp(a) level was associated with CAD development regardless of CAD family history status.

CURRICULUM VITAE

Wen-Liang Song

Director of Lipid Research, Assistant Professor, Brown University, USA



Education and Training

2003.06	Shandong University, MD, Medicine/Cardiology
2009.02	University of Pennsylvania, Postdoc, Biomedical science
2012.08	University of Pennsylvania, MTR, Translational Medicine
2016.06	Yale University Bridgeport Hospital, Resident, Internal Medicine
2019.06	Vanderbilt University Medicine Center, Fellow, Cardiology

Employment and Position

2023.10–	Director of Lipid Research, Assistant Professor, Brown University
2019.12–2023.09	Assistant Professor, Vanderbilt University
2019.07–2019.11	Instructor, Vanderbilt University
2016.07–2019.06	Cardiology Fellow, Vanderbilt University Medicine Center
2013.07–2016.06	Internal Medicine Resident, Yale University Bridgeport Hospital
2012.02–2013.06	Research Assistant Professor, School of Medicine, University of Pennsylvania
2009.02–2012.02	Research Associate, Institute of Translational Medicine and Therapeutics, University of Pennsylvania
2004.02–2009.02	Postdoc Fellow, Department of Pharmacology, University of Pennsylvania

Important Publications

1. Investigation of the Influence of Lipoprotein (a) and Oxidized Lipoprotein (a) on Plasminogen Activation and Fibrinolysis. M Yao, SK Dickeson, K Dhanabalan, S Solomevich, C Dennewitz, ... Journal of Lipid and Atherosclerosis 14 (2), 229.
2. K Dhanabalan, P Yancey, H Li, S Salamevich, J Li, Y Li, J Huang, C Dennewitz, C Wang, H Tao, L Smith, D Gailani, J Du, K Martin, J Hwa, S Davies, M Linton, W Song*. Dysfunctional HDL and Its Impact on Antithrombotic Activity and Platelet Function in Cardiovascular Disease Submitted to Circ. Research.

Investigation of the Influence of Lipoprotein(a) and Oxidized Lipoprotein(a) on Plasminogen Activation and Fibrinolysis

In this study, we investigated the effects of oxidized lipoprotein(a) [Lp(a)] compared with unoxidized Lp(a) on plasminogen activation during fibrinolysis, aiming to elucidate potential atherogenic mechanisms of oxidized Lp(a) with a particular focus on its role in thrombosis. Chromogenic substrate assays were employed to assess plasminogen activation kinetics, with fibrin clots generated by incubating fibrinogen with thrombin and initiating plasminogen activation using tissue plasminogen activator (tPA). Experiments were conducted under both low and high concentrations of Lp(a) or oxidized Lp(a), the latter prepared via chemical oxidation of isolated samples, to evaluate their respective influences on plasmin generation. At low concentrations, Lp(a) enhanced plasminogen activation and fibrinolysis, consistent with its physiological

role; however, at higher concentrations, oxidized Lp(a) exerted a marked inhibitory effect, causing an earlier plateau in plasmin generation and reducing overall plasmin levels relative to unoxidized Lp(a). These inhibitory effects likely result from the structural similarity of oxidized Lp(a) to plasminogen, combined with its increased oxidized phospholipid content, which competes with plasminogen for fibrin binding sites and interferes with interactions between fibrin fragments and tPA, collectively impairing fibrinolysis. Taken together, these findings indicate that while physiological levels of Lp(a) may facilitate fibrinolysis, oxidation transforms Lp(a) into a potent inhibitor of plasminogen activation, underscoring its atherogenic potential and its contribution to thrombotic cardiovascular risk.

CURRICULUM VITAE

Belen Davico

Teaching Assistant and PhD Student, Laboratory of Lipids and Atherosclerosis, School of Pharmacy and Biochemistry, University of Buenos Aires and National Scientific and Technical Research Council (CONICET), Argentina



Education and Training

2021.12 University of Buenos Aires, Biochemistry, Health Science

Employment and Position

2021– National Scientific and Technical Research Council, PhD Fellow
2022– University of Buenos Aires, Teaching Assistant

Important Publications

1. Davico B, Martin M, Condori A, Lozano Chiappe E, Gaete L, Tetzlaff WF, Yanez A, Osta V, Sáez MS, Bava A, Godoy MF, Palenque P, Ballerini MG, Trifone L, Gómez Rosso L, Feliu MS, Brites F. Fatty acids in childhood obesity: A link between nutrition, metabolic alterations and cardiovascular risk. *J Lipid Atheroscler*. 2025;14:e15. doi: 10.12997/jla.2025.14.e15 Please list up to 5 of your most important papers, in order of recency.
2. Davico B, Martín M, Lozano Chiappe E, Esparza Iraola R, Marchesini M, Pérez M, Gómez Rosso L, Boero L, Giunta G, Brites F. Differential Functions of High-density Lipoproteins in Response to SARS-CoV-2 Infection. *Revista Argentina de Cardiología*. 2024;92:126–132. doi: 10.7775/rac.es.v92.i2.2075.
3. Davico B, Lozano Chiappe E, Gaete L, Gómez Rosso L, Sáez MS, Tetzlaff W, Palenque P, Osta V, Yáñez A, Boero L, Trifone L, Martin M, Brites F. Insulin resistance in childhood obesity: The role of dietary habits and their association with novel markers of cardiovascular disease. *Mediterranean Journal of Nutrition and Metabolism* 2024;18(1):27–36. doi:10.1177/1973798X241304946.

Fatty Acids in Childhood Obesity: A Link Between Nutrition, Metabolic Alterations and Cardiovascular Risk

Childhood obesity represents a growing public health challenge, significantly increasing the risk of developing metabolic and cardiovascular diseases. In this presentation, we aim to explore how dietary patterns impact on lipid and glucose metabolism, vascular inflammation, and antioxidant capacity in children and adolescents with obesity. Our study highlights the role of fatty acid composition as a potential mediator linking dietary habits to cardiometabolic alterations associated with childhood obesity.

In our study, children with obesity showed a clear pattern of unhealthy food intake, characterized by higher consumption of bakery products and lower intake of cereals. This was associated with a distinct plasma fatty acid profile compared to that of a normal weight control group, marked by increased levels of saturated and pro-inflammatory fatty acids such as myristic and palmitoleic acids, and decreased levels of linoleic, EPA, and other anti-inflammatory species. These alterations were accompanied by a more atherogenic lipid profile, signs of impaired glucose metabolism, increased Lp-PLA2 activity (a marker of vascular inflammation), and reduced PON1 activity,

reflecting attenuated antioxidant function.

Several significant associations emerged, emphasizing the impact of dietary choices and the mediator role of fatty acids on the development of obesity and related metabolic alterations. Moreover, the positive correlation between myristic acid and Lp-PLA2, and the inverse correlation between EPA and hs-CRP, suggest that specific fatty acids may modulate inflammatory pathways. Importantly, mediation analysis confirmed that both fatty acids and cardiometabolic markers partially explained the relationship between diet and the presence of obesity.

Taken together, our findings reveal a complex network of interactions linking dietary habits to functional lipid alterations and early markers of cardiometabolic dysfunction in pediatric obesity. These results reinforce the importance of improving nutritional patterns during childhood, not only to manage weight but also to preserve lipoprotein function and reduce long-term cardiovascular risk. The potential for specific fatty acids to act as biomarkers or therapeutic targets calls for further investigation.

Symposium 24

Dyslipidemia Management in Special Populations

Sep 13(Sat) 16:20–17:50 | Room 1 (3F)

CHAIRPERSONS : Yong-Jae Kim (Kim's Neurology Clinic, Republic of Korea)
Young Joon Hong (Chonnam National University, Republic of Korea)

16:20–16:40 **Managing dyslipidemia in women before and after menopause**

Mi-Na Kim (Korea University, Republic of Korea)

16:40–17:00 **Managing dyslipidemia in patients with liver disease**

Byung Sik Kim (Hanyang University, Republic of Korea)

17:00–17:20 **HIV, chronic inflammation, and dyslipidemia**

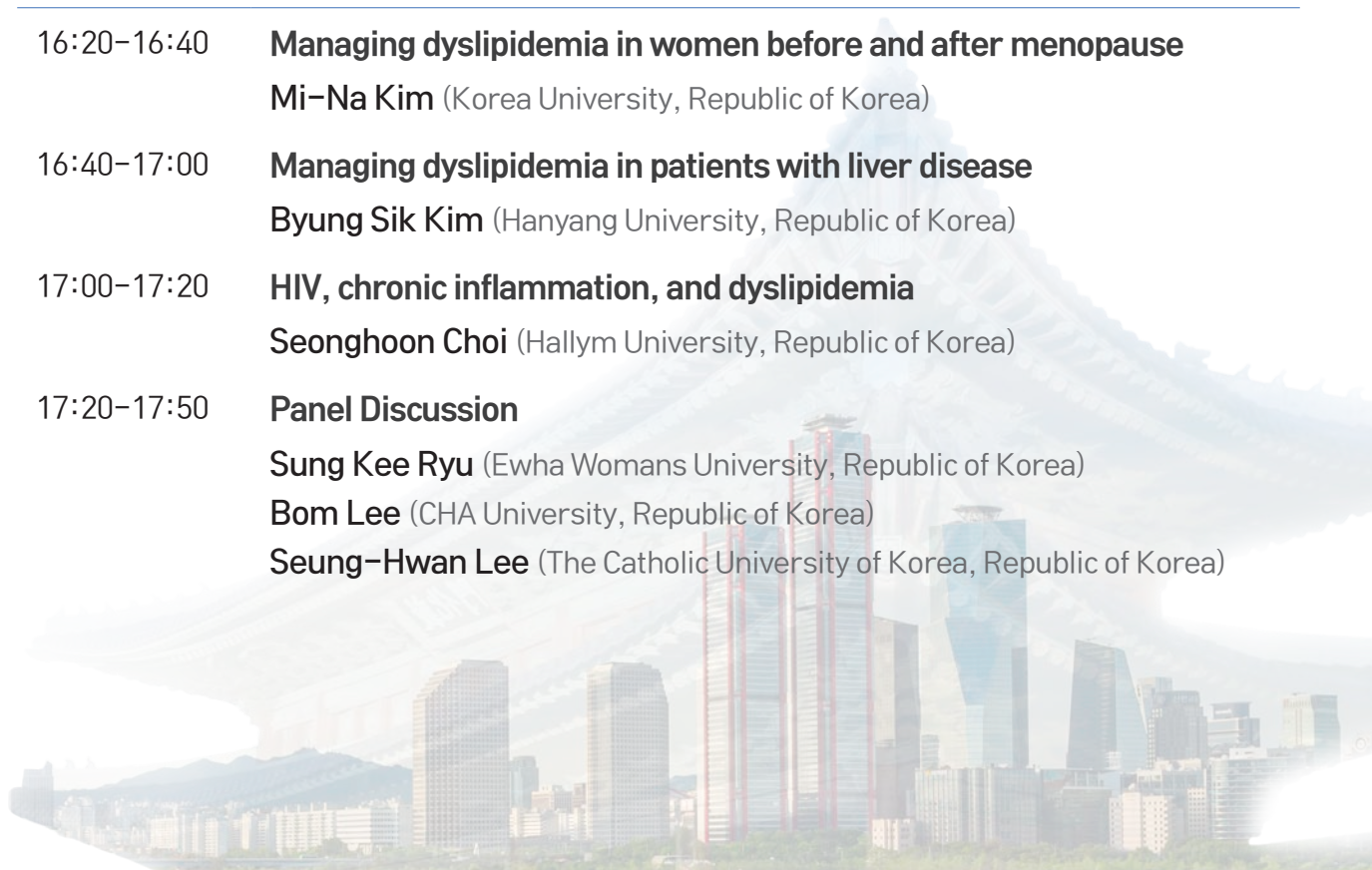
Seonghoon Choi (Hallym University, Republic of Korea)

17:20–17:50 **Panel Discussion**

Sung Kee Ryu (Ewha Womans University, Republic of Korea)

Bom Lee (CHA University, Republic of Korea)

Seung-Hwan Lee (The Catholic University of Korea, Republic of Korea)



CURRICULUM VITAE**Mi-Na Kim**

Professor, Korea University, Republic of Korea

**Education and Training**

2006.02	Korea University, Korea, M.D, Medicine
2016.03	Korea University, Korea, Ph.D., Internal Medicine

Important Publications

1. Kim SR, Cho DH, Kim JH, Park SM, Kim MN. Oxidative Stress Biomarkers Predict Myocardial Dysfunction in a Chemotherapy-Induced Rat Model. *Diagnostics (Basel)*. 2025 Mar 12;15(6):705. doi: 10.3390/diagnostics15060705.
2. Kim MN, Lee YS, Park Y, Jung A, So H, Park J, Park JJ, Choi DJ, Kim SR, Park SM. Deep learning for predicting rehospitalization in acute heart failure: Model foundation and external validation. *ESC Heart Fail*. 2024 Jul 9. doi: 10.1002/ehf2.14918.
3. Kim SR, 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*. 2023 Dec 19. doi: 10.1007/s00392-023-02358-2.
4. Kim MN, Kim SR, Kim HD, Cho DH, Jung SP, Park KH, Park SM. Serial changes of layer-specific myocardial function according to chemotherapy regimen in patients with breast cancer. *Eur Heart J Open*. 2022 Feb18;2(4):oeac008. doi:10.1093/ehjopen/oeac008. eCollection 2022 Jul.
5. Kim MN, Park SM. Current Status of Pharmacologic and Nonpharmacologic Therapy in Heart Failure with Preserved Ejection Fraction *Heart Fail Clin*. 2021 Jul;17(3):463–482.

Managing dyslipidemia in women before and after menopause

CURRICULUM VITAE

Byung Sik Kim

Assistant Professor, Hanyang University Guri Hospital, Republic of Korea



Education and Training

2009.03	Hanyang University, Korea, M.D, Medicine
2024.03	Hanyang University, Korea, Ph.D., Internal Medicine

Employment and Position

2021–2023	Hanyang University Guri Hospital, Clinical Assistant Professor
2024–	Hanyang University Guri Hospital, Assistant Professor

Important Publications

1. Kim BS, Kim H-J, Jeon SW, Kim KH, Kim DW, Shin J-H. Comparing non-alcoholic fatty liver disease indices in predicting the prevalence and incidence of metabolic syndrome in middle-aged adults. *Heliyon*. 2025; 11(7): e43073. doi.org/10.1016/j.heliyon.2025.e43073.
2. Kim BS, Kim H-J, Kim H, Lee J, Shin J-H, Sung K-C. Longitudinal Changes in Cardiovascular-Kidney-Metabolic Syndrome Stages and Their Impact on Outcomes: A Nationwide Cohort Study. *Journal of Clinical Medicine*. 2025; 14(11):3888. doi.org/10.3390/jcm14113888.
3. Kim HJ, Kim BS, Kim H, Lee J, Shin JH, Sung KC. Impact of blood pressure and medication adherence on clinical outcomes in patients with hypertension. *Front Med (Lausanne)*. 2025;12:1564791. Published 2025 Apr 28. doi.org/10.3389/fmed.2025.1564791.
4. Kim BS, Lim YH, Shin J, Shin JH. Association of systolic blood pressure target and variability with long-term clinical outcomes in patients undergoing percutaneous coronary intervention. *Clin Hypertens*. 2025;31:e13. doi.org/10.5646/ch.2025.31.e13.

Research Interest

Cardiovascular disease
Metabolic disease

Managing dyslipidemia in patients with liver disease

Dyslipidemia is highly prevalent in patients with liver disease and contributes substantially to cardiovascular risk. Given the liver’s central role in lipid metabolism, management in this population is both important and challenging. Although concerns about hepatotoxicity have limited the use of lipid-lowering therapy, current evidence supports their overall safety and efficacy in most liver diseases.

Statins remain the cornerstone of therapy. Mild elevations in aminotransferases occur in up to 3% of patients, but clinically significant drug-induced liver injury is rare. Available data indicate that statins can be used safely across most liver diseases, including compensated cirrhosis. Caution is warranted in patients with decompensated cirrhosis or in those with a history of drug-induced liver injury.

CURRICULUM VITAE

Seonghoon Choi

Professor, Hallym University, Republic of Korea



Education and Training

1997.02 Yonsei University, Korea, M.D, Medicine

Employment and Position

2001–2004 Severance Hospital, Internal Medicine, Resident
 2004–2005 Severance Hospital, Cardiology, Fellowship
 2006– Hallym University Kangnam Sacred Heart Hospital, Professor

Research Interest

Resistant hypercholesterolemia
 Atherosclerosis

HIV, chronic inflammation, and dyslipidemia

HIV infection is a complex chronic condition that, even with effective treatment, is associated with long-term health issues. The interplay between HIV, chronic inflammation, and dyslipidemia is a key factor in the increased risk of cardiovascular disease (CVD) and other non-AIDS comorbidities in people living with HIV (PLHIV).

Chronic inflammation is a hallmark of HIV infection. Even with successful antiretroviral therapy (ART) that suppresses the virus to undetectable levels, a state of low-grade inflammation often persists. This is a significant driver of long-term health complications. Mechanisms of Inflammation: The persistent inflammation in PLHIV is driven by several factors: Ongoing viral replication: While ART suppresses HIV in the bloodstream, the virus can persist in various "reservoirs" in the body, such as lymph nodes and the gastrointestinal tract. This low-level replication continues to activate the immune system. Microbial translocation: HIV infection damages the gut lining, which normally acts as a barrier to bacteria and their products. This damage allows bacterial products, such as lipopolysaccharide (LPS), to leak into the bloodstream, triggering a systemic inflammatory response. Immune cell dysfunction: HIV infection leads to a state of chronic immune activation and dysfunction. There is an imbalance between pro- and anti-inflammatory responses, and immune cells like T cells and monocytes remain in a hyper-activated state. This leads to the sustained production of pro-inflammatory cytokines like IL-6 and TNF- α .

Chronic inflammation is closely linked to dyslipidemia, an abnormal level of lipids (fats) in the blood. In the context of HIV, this relationship is two-way. Inflammation's Impact on Lipids: The inflammatory state in PLHIV contributes to dyslipidemia. Inflammatory cytokines can disrupt lipid metabolism, leading to: Increased levels of triglycerides (hypertriglyceridemia). Decreased levels of high-density lipoprotein cholesterol (HDL-c), often called "good" cholesterol. Lipids' Impact on Inflammation: Dyslipidemia, in turn, can exacerbate inflammation. Altered lipid profiles contribute to the formation and progression of atherosclerotic plaques, which are a key feature of CVD. These plaques are themselves sites of chronic inflammation. Dyslipidemia in PLHIV is a complex issue with two main contributing factors: the virus itself and

antiretroviral therapy. HIV-Associated Dyslipidemia (without ART): In untreated HIV infection, individuals often have a characteristic lipid profile: Lower levels of HDL-c and LDL-c. The exact mechanisms for these changes are not fully understood but may involve the virus "hijacking" host cell lipid rafts for replication and altering lipid transport. ART-Associated Dyslipidemia: While ART has transformed HIV into a manageable chronic condition, some drug classes can have metabolic side effects, including dyslipidemia. Protease Inhibitors (PIs): PIs, particularly older generations, were strongly associated with dyslipidemia, characterized by increased total cholesterol, LDL-c, and triglycerides. Nucleoside Reverse Transcriptase Inhibitors (NRTIs): Some NRTIs, such as stavudine and zidovudine, have also been linked to dyslipidemia and lipodystrophy (abnormal fat distribution). Newer ARTs: Newer classes of drugs, such as integrase inhibitors, generally have a more favorable metabolic profile. However, a "return to health" phenomenon can occur, where lipid levels (including cholesterol and triglycerides) increase after viral suppression is achieved.

Management and Clinical Implications

The combination of HIV, chronic inflammation, and dyslipidemia significantly increases the risk of cardiovascular disease in PLHIV. This risk is often higher than in the general population, even when traditional risk factors like smoking or hypertension are controlled. Risk Assessment: It is recommended that PLHIV have their 10-year CVD risk estimated annually using specific tools, such as the SCORE2, to account for their heightened risk. Lifestyle Modifications: A cornerstone of management is encouraging healthy lifestyle changes, including smoking cessation, a balanced diet, regular physical activity, and weight management. Antiretroviral Therapy: Choosing ART regimens with a favorable lipid profile is crucial. Switching to a different ART class may be considered if dyslipidemia is a significant issue. Lipid-Lowering Medications: Statins are the primary treatment for dyslipidemia in PLHIV, following the same guidelines as the general population. However, it is essential to consider potential drug-drug interactions between statins and certain ARTs (especially PIs). Pravastatin and atorvastatin are commonly used due to a lower risk of interaction, while simvastatin and lovastatin are often contraindicated.

Symposium 25

Lipid-immune Interactions Shaping Vascular Repair and Disease

Sep 13(Sat) 16:20–17:50 | Room 3 (3F)

CHAIRPERSONS : Chi Dae Kim (Pusan National University, Republic of Korea)
Young Mi Park (Ewha Womans University, Republic of Korea)

16:20–16:40 **IKK ϵ -deficient macrophages impede cardiac repair after myocardial infarction by enhancing the macrophage-myofibroblast transition**
Youngkeun Ahn (Chonnam National University, Republic of Korea)

16:40–17:00 **Sensing ceramides by CYSLTR2 and P2RY6 to aggravate atherosclerosis**
Wei Kong (Peking University, China)

17:00–17:20 **Targeting the USP35-FASN axis and choline metabolism in cancer: a lipidomics perspective**
Hyunbeom Lee (Korea Institute of Science and Technology (KIST), Republic of Korea)

17:20–17:50 **Panel Discussion**
Jun Namkung (Yonsei University, Republic of Korea)
Taesik Park (Gachon University, Republic of Korea)
Jae-Han Jeon (Kyungpook National University, Republic of Korea)
Su Myung Jung (Sungkyunkwan University, Republic of Korea)

CURRICULUM VITAE

Youngkeun Ahn

Professor, Chonnam National University, Republic of Korea



Education and Training

- 1989.02 Chonnam National University, Korea, MD, Medicine
 1998.02 Chonnam National University, Korea, PhD, Cardiology

Employment and Position

- 1998- Clinical Instructor, Assistant, Associate, Full Professor of Department of Cardiology, Chonnam National University Hospital
 2000-2002 Research Fellowship, Cardiovascular Research Center, Department of Cardiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA
 2007- Course Director of Gwangju-Boston Joint Cardiology Symposium
 2006-2013 Director of Cardiac Catheterization Laboratory, Chonnam National University Hospital
 2006-2013 Director, Department of Cardiology, Chonnam National University Hospital
 2010- Course Director of Gwangju International Interventional Cardiology Symposium
 2012-2015 Director, Regional Cardiocerebrovascular Center
 2014-2016 Vice-Director, Clinical Departments, Chonnam National University Hospital
 2012- Director of Scientific Committee, Secretary General, Neutral Director, The Korean Society of Thrombosis and Hemostasis
 2014-2016 Director of Information and Publicity, Korean Society of Interventional Cardiology
 2010-2016 Director of Education, Foreign Cooperation Committee, Korean Society of Lipidology and Atherosclerosis
 2014- Board Member, Korean Society of Stem Cell Research
 2014- Member (Medical Sciences) of the Korean Academy of Science and Technology
 2016-2017 Director of Planning & Coordination, Chonnam National University Hospital
 2017-2018 Director of Research Committee, The Korean Society of Cardiology
 2018- Chairman of Korean Working Group of Basic Cardiovascular Sciences, The Korean Society of Cardiology
 2018- Member of the National Academy of Medicine of Korea
 2018- Director of Scientific, Korean Society of Interventional Cardiology
 2018- Chairman of The Korean Society of Thrombosis and Hemostasis
 2018- International Associate Editor in Circulation Reports
 2019- Director of Publishing Committee, The Korean Society of Cardiology
 2022- President of The Korean Society of Thrombosis and Hemostasis
 2020-2024 President & CEO, Chonnam National University Hospital
 2021-2024 President of Gwangju Jeonnam Hospital Association
 2024- President of Korean Society of Interventional Cardiology

IKK ϵ -deficient macrophages impede cardiac repair after myocardial infarction by enhancing the macrophage-myofibroblast transition

The regulatory role of the inhibitor of NF- κ B kinase ϵ (IKK ϵ) in postmyocardial infarction (MI) inflammation remains uncertain. Using an MI mouse model, we examined the cardiac outcomes of IKK ϵ knockout (KO) mice and wild-type mice. We employed single-cell RNA sequencing (scRNA-seq) and phosphorylated protein array techniques to profile cardiac macrophages. IKK ϵ KO mice exhibited compromised survival, heightened inflammation, pronounced cardiac fibrosis, and a reduced ejection fraction. A distinct cardiac macrophage subset in IKK ϵ KO mice exhibited increased fibrotic marker expression and decreased phosphorylated p38 (p-p38) levels, indicating an enhanced macrophage-myofibroblast transition (MMT)

post-MI. While cardiac inflammation is crucial for initiating compensatory pathways, the timely resolution of inflammation was impaired in the IKK ϵ KO group, while the MMT in macrophages accelerated post-MI, leading to cardiac failure. Additionally, our study highlighted the potential of 5-azacytidine (5-Aza), known for its anti-inflammatory and cardioprotective effects, in restoring p-p38 levels in stimulated macrophages. The administration of 5-Aza significantly reduced the MMT in cardiac macrophages from the IKK ϵ KO group. These findings underscore the regulation of the inflammatory response and macrophage transition by the IKK ϵ -p38 axis, indicating that the MMT is a promising therapeutic target for ischemic heart disease.

CURRICULUM VITAE

Wei Kong

Professor, Peking University, China



Education and Training

1995.07	Huazhong University of Science & Technology, China, M.D, Medicine
2000.07	Huazhong University of Science & Technology, China, Ph.D., Pathophysiology

Employment and Position

2005–2010	Peking University, Associate Professor
2010–Present	Peking University, Professor
2016–Present	Peking University, Chair, Department of Physiology and Pathophysiology
2023–Present	Peking University, Dean, School of Basic Medical Science

Important Publications

1. Zhang S, Lin H, Wang J, Rui J, Wang T, Cai Z, Huang S, Gao Y, Ma T, Fan R, Dai R, Li Z, Jia Y, Chen Q, He H, Tan J, Zhu S, Gu R, Dong Z, Li M, Xie E, Fu Y, Zheng J*, Jiang C*, Sun J*, Kong W*. Sensing ceramides by CYSLTR2 and P2RY6 to aggravate atherosclerosis. *Nature*. 2025 Mar 6.
2. Lin H, Ma C, Cai K, Guo L, Wang X, Lv L, Zhang C, Lin J, Zhang D, Ye C, Wang T, Huang S, Han J, Zhang Z, Gao J, Zhang M, Pu Z, Li F, Guo Y, Zhou X, Qin C, Yi F, Yu X*, Kong W*, Jiang C*, Sun JP*. Metabolic signaling of ceramides through the FPR2 receptor inhibits adipocyte thermogenesis. *Science*. 2025:eado4188.
3. Zhang L, Zhou J, Kong W*. Extracellular matrix in vascular homeostasis and disease. *Nat Rev Cardiol*. 2025 Jan.
4. Yang X, Xu C, Yao F, Ding Q, Liu H, Luo C, Wang D, Huang J, Li Z, Shen Y, Yang W, Li Z, Yu F, Fu Y, Wang L, Ma Q, Zhu J, Xu F, Cong X, Kong W*. Targeting endothelial tight junctions to predict and protect thoracic aortic aneurysm and dissection. *Eur Heart J*. 2023 Apr 7;44(14):1248–1261.
5. Ma Z, Mao C, Chen X, Yang S, Qiu Z, Yu B, Jia Y, Wu C, Wang Y, Wang Y, Gu R, Yu F, Yin Y, Wang X, Xu Q, Liu C, Liao Y, Zheng J*, Fu Y*, Kong W*. Peptide Vaccine Against ADAMTS-7 Ameliorates Atherosclerosis and Post-injury Neointima Hyperplasia. *Circulation*. 2023 Feb 28;147(9):728–742.

Sensing ceramides by CYSLTR2 and P2RY6 to aggravate atherosclerosis

Atherosclerosis is the primary cause of cardiovascular diseases such as coronary artery disease, myocardial infarction, and stroke. Current pharmacological strategies mainly focus on lowering cholesterol, yet over 60% of patients continue to experience residual risk, indicating the existence of other crucial pathogenic factors beyond lipids. Recent evidence has shown that increased levels of circulating long-chain ceramides predict atherosclerotic cardiovascular disease independently of cholesterol. Although targeting ceramide signalling may provide therapeutic benefits beyond the treatment of hypercholesterolaemia, the underlying mechanism by which circulating ceramides aggravate atherosclerotic cardiovascular disease remains elusive. In our latest work, we examined whether circulating long-chain ceramides activated membrane G-protein-coupled receptors to exacerbate atherosclerosis. We performed a systematic screen that combined G-protein-signalling quantification, bioinformatic analysis of G-protein-coupled receptor expression and functional examination of NLRP3 inflammasome activation. The results suggested that CYSLTR2 and P2RY6 were potential endogenous re-

ceptors of C16:0 ceramide-induced inflammasome activation in both endothelial cells and macrophages. Inhibition of CYSLTR2 and P2RY6 genetically or pharmacologically alleviated ceramide-induced atherosclerosis aggravation. Moreover, increased ceramide levels correlated with the severity of coronary artery disease in patients with varying degrees of renal impairment. Notably, CYSLTR2 and P2RY6 deficiency mitigated chronic-kidney-disease-aggravated atherosclerosis in mice without affecting cholesterol or ceramide levels. Structural analyses of ceramide-CYSLTR2-Gq complexes revealed that both C16:0 and C20:0 ceramides bound in an inclined channel-like ligand-binding pocket on CYSLTR2. We further revealed an unconventional mechanism underlying ceramide-induced CYSLTR2 activation and the CYSLTR2-Gq interface. Overall, our study provided structural and molecular mechanisms of how long-chain ceramides initiated transmembrane Gq and inflammasome signalling through direct binding to CYSLTR2 and P2RY6 receptors. Therefore, blocking these signals may provide a new therapeutic potential to treat atherosclerosis-related diseases.

CURRICULUM VITAE

Hyunbeom Lee

Senior Researcher, Korea Institute of Science and Technology, Republic of Korea



Education and Training

2008.05 Illinois Institute of Technology, USA, B.S, Chemistry
 2014.08 Northwestern University, USA, Ph.D., Chemistry

Employment and Position

2014–2016 Korea Institute of Science and Technology, Researcher
 2016–Current Korea Institute of Science and Technology, Senior Researcher
 2019–Current Hanyang University, Adjunct Professor

Important Publications

1. Kong, M., Hong D. H., Paudel, S., Lee, H.* "Metabolomics and miRNA profiling reveals feature of gallbladder cancer-derived biliary extracellular vesicles", *Biochem. Biophys. Res. Comm.*, 2024, 705, 149724.
2. Park, BS., Jeon, H., Kim, Y. et al. "Polyamine and EIF5A hypusination downstream of c-Myc confers targeted therapy resistance in BRAF mutant melanoma", *Mol. Cancer* 2024, 23, 136.
3. Ju Y. H., Bhalla M., Hyeon S.J., Lee H., H. Ryu, C. J. Lee, "Astrocytic urea cycle detoxifies A β -derived ammonia while impairing memory in Alzheimer's disease", *Cell Metabolism*, 2022, 34, 1104.
4. Yang, J. S., Yoon, N., Kong, M., Jung, B.H., Lee, H.* Park J.* "USP14 regulates cancer cell growth in a fatty acid synthase-independent manner", *Int. J. Mol. Sci.* 2021, 22(24), 13437.
5. Oh, J.E., Jung, B.H., Park, J., Kang, S., Lee H.* Deciphering Fatty Acid Synthase Inhibition-Triggered Metabolic Flexibility in Prostate Cancer Cells through Untargeted Metabolomics. *Cells*. 2020, 9(11):2447.

Research Interest

- Clinical Metabolomics and Lipidomics for Drug development and Mechanism studies.
- Bioinformatics and Biodata analysis for Predicting Drug Response/Toxicity.

Targeting the USP35-FASN axis and choline metabolism in cancer: a lipidomics perspective

Fatty acid synthase (FASN) is a central enzyme in de novo lipogenesis and is markedly upregulated in various cancers, supplying the lipids necessary for membrane synthesis, energy storage, and signaling to support tumor growth. Despite the promise of FASN inhibition as a therapeutic strategy, its clinical utility has been limited by the remarkable metabolic adaptability of cancer cells.

To elucidate these adaptive responses, we examined the metabolic effects of three distinct FASN inhibitors in prostate cancer cells. Lipidomics and metabolomics profiling revealed that, even with reduced palmitate synthesis, cancer cells compensated by upregulating choline kinase- α (CK α)—a critical enzyme in the Kennedy pathway for phosphatidylcholine (PC) synthesis—thereby increasing fatty acid and phospholipid levels. Furthermore, we discovered that lipid droplets became an alternative source of diacylglycerol (DAG) for phospholipid production, effectively bypassing the dependency on de novo fatty acid synthesis. These

results indicate that combined inhibition of FASN and CK α may be necessary to fully suppress the compensatory lipid synthesis in cancer cells.

In parallel, studies in colorectal cancer (CRC) models identified USP35 as a novel deubiquitinating enzyme that stabilizes FASN by blocking its ubiquitin-mediated degradation. Silencing USP35 led to diminished FASN expression, reduced triglyceride and lipid droplet accumulation, and increased uptake of polyunsaturated fatty acids (PUFAs), which resulted in heightened lipid peroxidation and ferroptosis. Lipidomic analyses further confirmed that the USP35-FASN axis is pivotal for maintaining lipid homeostasis and promoting cell survival in CRC.

Collectively, our findings demonstrate that cancer cells sustain lipid synthesis through both post-translational regulation of FASN and metabolic rerouting via CK α . Targeting these adaptive mechanisms with combination therapies may significantly enhance the effectiveness of FASN inhibition in cancer treatment.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

2025 KSoLA Awards for Scientific Excellence & Young Investigator

Sep 13(Sat) 14:50–15:25 | Room 1 (3F)

CHAIRPERSONS : Ki Hoon Han (University of Ulsan, Republic of Korea)
Sang-Hyun Kim (Seoul National University, Republic of Korea)

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|-------------|--|
| 14:50–14:55 | 2025 KSoLA Award Ceremony for Scientific Excellence |
| 14:55–15:00 | 2025 KSoLA Award Ceremony for Young Investigator |
| 15:00–15:25 | Triglyceride metabolism, hypertriglyceridemia, and its therapeutics
Nam Hoon Kim (Korea University, Republic of Korea) |
| 15:20–15:25 | Q&A |



CURRICULUM VITAE

Nam Hoon Kim

Professor, Korea University, Republic of Korea



Education and Training

2004	Korea University, Korea, M.D, Medicine
2015	Korea University, Korea, Ph.D., Internal Medicine

Employment and Position

2016-2018	Korea University Anam Hospital, Assistant Professor
2018-2023	Korea University Anam Hospital, Associate Professor
2023-	Korea University Anam Hospital, Professor

Important Publications

1. Kim NH, Kim JY, Choi J, Kim SG. Associations of omega-3 fatty acids vs. fenofibrate with adverse cardiovascular outcomes in people with metabolic syndrome: propensity matched cohort study. *Eur Heart J Cardiovasc Pharmacother.* 2024 Feb 23;10(2):118-127.
2. Kim JY, Kim NH. New Therapeutic Approaches to the Treatment of Dyslipidemia 1: ApoC-III and ANGPTL3. *J Lipid Atheroscler.* 2023 Jan;12(1):23-36.
3. Kim JY, Choi J, Kim SG, Kim NH. Comparison of on-Statin Lipid and Lipoprotein Levels for the Prediction of First Cardiovascular Event in Type 2 Diabetes Mellitus. *Diabetes Metab J.* 2023 Nov;47(6):837-845.
4. Kim JY, Choi J, Kim SG, Kim NH. Relative contributions of statin intensity, achieved low-density lipoprotein cholesterol level, and statin therapy duration to cardiovascular risk reduction in patients with type 2 diabetes: population based cohort study. *Cardiovasc Diabetol.* 2022 Feb 22;21(1):28.
5. Kim NH, Han KH, Choi J, Lee J, Kim SG. Use of fenofibrate on cardiovascular outcomes in statin users with metabolic syndrome: propensity matched cohort study. *BMJ.* 2019 Sep 27;366:l5125.

Triglyceride metabolism, hypertriglyceridemia, and its therapeutics

Triglycerides are the primary storage form of energy, and hypertriglyceridemia reflects a phenotype of systemic energy excess. This state is closely linked to obesity, insulin resistance, and type 2 diabetes mellitus, forming a pathophysiologic axis that predisposes individuals to both microvascular and macrovascular complications. Elevated triglycerides and remnant lipoproteins not only represent disordered energy metabolism but also contribute directly to atherogenesis, thereby driving residual cardiovascular risk despite adequate low-density lipoprotein cholesterol (LDL-C) control.

Our research has addressed this continuum from epidemiologic, clinical, and therapeutic perspectives. In large-scale cohort analyses, we showed that fenofibrate use in statin-treated patients with metabolic syndrome reduced cardiovascular risk, reinforcing the importance of triglyceride-lowering as a complement to statins. Furthermore, comparative analyses between fenofibrate and omega-3 fatty acids revealed differential associations with adverse outcomes, suggesting that energy-related lipid phenotypes may require distinct therapeutic strategies. In type 2 diabetes populations, we demonstrated that triglyceride-rich lipopro-

tein fractions, when measured on background statin therapy, improve prediction of first cardiovascular events, while the balance of statin intensity, LDL-C achievement, and treatment duration clarifies optimal pathways for risk reduction.

Extending beyond established therapies, our review of apolipoprotein C-III (APOC3) and angiopoietin-like protein 3 (ANGPTL3) highlighted their roles as critical modulators of triglyceride metabolism, remnant clearance, and energy partitioning. Targeting these regulators represents an innovative therapeutic frontier for patients in whom energy overload and lipid dysregulation drive persistent risk.

In summary, hypertriglyceridemia may be understood not merely as a biomarker but as an expression of energy excess that links obesity and insulin resistance to diabetic complications and cardiovascular disease. Continued research into both conventional agents such as fenofibrate and omega-3 fatty acids, as well as novel therapies targeting APOC3 or ANGPTL3, will be essential to determine how best to translate these metabolic insights into improved cardiovascular outcomes.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Joint Symposia



ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

JAS-TSLA-KSoLA Joint Symposium

Familial Hypercholesterolemia (FH) in the Era of Precision Medicine

Sep 11(Thu) 16:20–17:50 | Room 2 (3F)

CHAIRPERSONS : Donghoon Choi (Yonsei University, Republic of Korea)
Woo Je Lee (University of Ulsan, Republic of Korea)

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|-------------|--|
| 16:20–16:40 | Early detection of FH: role of genetic screening and cascade testing
Hayato Tada (Kanazawa University, Japan) |
| 16:40–17:00 | Cardiovascular risk in FH: how early is early enough for intervention
Po-Sheng Chen (National Cheng Kung University, Taiwan) |
| 17:00–17:20 | Beyond statins: advances in FH management
Chan Joo Lee (Yonsei University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Hoyoun Won (Chung-Ang University, Republic of Korea)
Dae Young Cheon (Hallym University, Republic of Korea)
Seong Huan Choi (Inha University, Republic of Korea)
Hun Jee Choe (Hallym University, Republic of Korea) |

CURRICULUM VITAE

Hayato Tada

Assistant Professor, Kanazawa University, Japan



Education and Training

2003.03	Kanazawa University, Japan, M.D, Medicine
2011.06	Kanazawa University, Japan, Ph.D., Cardiovascular Medicine

Employment and Position

2003–2012	Kanazawa University Hospital, Staff
2012–2014	Massachusetts General Hospital, 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

Early detection of FH: role of genetic screening and cascade testing

Familial Hypercholesterolemia (FH) in general has been associated with elevated cardiovascular risk. However, there are still substantial differences in the degree of their cardiovascular risk among patients with FH. First of all, we need to identify homozygous FH (HoFH), since their cardiovascular risk would be the highest, and we have special options, such as lomitapide, and evinacumab that can be introduced in patients with HoFH. In addition, we can risk stratify patients with FH via their genotype. We have shown that

patients with FH caused by protein truncating variant (PTV) exhibit worse prognosis compared with those cause by missense variant. Furthermore, we learned a lot that early diagnosis of FH could lead to their better prognosis, motivating us to screen them at their young age. We aimed to establish cascade screening program in Japan at the age of 9 or 10 years, and do far, it worked very well. I would like to provide a variety of topics for early detection of FH, role of genetic screening and cascade testing.

CURRICULUM VITAE

Po-Sheng Chen

Associate Professor, National Cheng Kung University Hospital, Taiwan



Education and Training

1999.06	School of Medicine, National Cheng Kung University, Taiwan, M.D, Medicine
2020.06	Institute of Clinical Medicine, National Cheng Kung University, Taiwan, Ph.D, Basic Research

Employment and Position

2001–2004	Department of Internal Medicine, National Cheng Kung University Hospital, Resident
2004–2006	Division of Cardiology, Department of Internal Medicine, National Cheng Kung University Hospital, Fellow
2006–2007	Division of Critical Medicine, Department of Internal Medicine, National Cheng Kung University Hospital, Fellow
2007–	Division of Critical Medicine, Department of Internal Medicine, National Cheng Kung University Hospital, Attending Physician

Important Publications

1. Chen PS, Lin JL, Lin HW, Lin SH, Li YH. Risk of hemorrhagic stroke among patients treated with high-intensity statins versus statin-ezetimibe: a population based study. *Tohoku Journal of Experimental Medicine* 2024;263:105–113.
2. Chen PS, Feng WH, Tsai TH, Hong YK, Lee AS, Chang KC, Chung HC, Liu YW, Hsieh CC, Fang YH, Yang PJ, Luo CY, Liu PY, Cheng TL, Li YH. Tumor endothelial marker 1 is upregulated in heart after cardiac injury and participates in cardiac remodeling. *Sci Rep* 2022;12:10532.
3. Chen PS, Lin SH, Lee CH, Lin HW, Li YH. Efficacy and safety of high-intensity statins in patients with acute myocardial infarction: an Asian perspective. *Can J Cardiol* 2020;36:886–892.
4. Chen PS, Wang KC, Chao TH, Chung HC, Tseng SY, Luo CY, Shi GY, Wu HL, Li YH. Recombinant thrombomodulin exerts anti-autophagic action in endothelial cells and provides anti-atherosclerosis effect in apolipoprotein E deficient mice. *Sci Rep* 2017;7(1): 3284. doi: 10.1038/s41598-017-03443-z.
5. Chen PS, Cheng CL, Chang YC, Kao Yang YH, Yeh PS, Li YH. Early statin therapy in patients with acute intracerebral hemorrhage without prior statin use. *Eur J Neurol* 2015;22:773–780.

Cardiovascular risk in FH: how early is early enough for intervention

Familiar Hypercholesterolemia (FH) is an autosomal hereditary disease characterized by abnormal LDL metabolism. Patients with FH have extremely high low density lipoprotein (LDL) and may have premature atherosclerotic disease (ASCVD), especially coronary artery disease (CAD) and peripheral artery disease (PAD), which contribute to acute cardiovascular events or death in early life. Most patients with FH are suspected by clinical features and laboratory lipid profile and confirmed by genetic test. To date, FH is still underdiagnosed in real world practice. Because of more than 10 times risk increase in the development of ASCVD in untreated FH patients, modern consensus or guidelines in managements of FH recommend reduce LDL using efficacious lipid-lowering agents as well as strict lifestyle modifications in adult patients

having diagnosis of FH. Recent studies also demonstrated that effective lipid lowering strategies slowed down the progression of atherosclerosis and decreased acute events of ASCVD. On the other hand, there was less consensus addressing the managements of dyslipidemia in children with FH. Recent studies had demonstrated the long-term safety of statin therapy in children with FH. Additionally, less progression of carotid intima-media thickness was found in FH patients receiving long-term statin since childhood. However, whether initiating lipid lowering therapy as early as childhood is associated with better clinical outcome in FH patients is still inconclusive. Further studies are needed to verify clinical benefits of lipid lowering agents in children with FH.

CURRICULUM VITAE**Chan Joo Lee**

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

**Education and Training**

2005.02	Yonsei University, Korea, M.D, Medicine
2010.02	Yonsei University, Korea, M.S, Internal Medicine
2015.02	Yonsei University, Korea, Ph.D, Cell Biology and Pharmacology

Employment and Position

2005–2006	Severance Hospital, Intern
2006–2010	Severance Hospital, Resident (Internal Medicine)
2010–2015	Yonsei University, Teaching Assistant (Molecular Biology)
2015–2016	Severance Hospital, Fellow (Cardiology)
2017–2018	Severance Hospital, Clinical Assistant Professor (Health Promotion)
2019–2019	Severance Hospital, Clinical Assistant Professor (Cardiology)
2020–2024	Severance Hospital, Clinical Associate Professor (Cardiology)
2025–	Severance Hospital, Clinical Professor (Cardiology)

Important Publications

1. MicroRNA-1912 regulates cholesterol homeostasis by targeting PCSK9. *Mol Ther Nucleic Acids*. 2025 Jun 9;36(3):102589. (1st author)
2. Spironolactone vs Amiloride for Resistant Hypertension: A Randomized Clinical Trial. *JAMA*. 2025 Jun 17;333(23):2073–2082. (1st author)
3. Cardiovascular Risk and Treatment Outcomes in Severe Hypercholesterolemia: A Nationwide Cohort Study. *J Am Heart Assoc*. 2022 May 3;11(9):e024379. (1st author)
4. Blood Pressure Levels and Risks of Dementia: a Nationwide Study of 4.5 Million People. *Hypertension*. 2022 Jan;79(1):218–229. (1st author)
5. Deep-learning-based cardiovascular risk stratification using coronary artery calcium scores predicted from retinal photographs. *Lancet Digit Health*. 2021 May;3(5):e306–e316. (1st author)

Research Interest

Lipid metabolism; Heart failure; Basic research regarding PCSK9

Beyond statins: advances in FH management

Familial hypercholesterolemia (FH) is a common genetic disorder characterized by lifelong elevation of low-density lipoprotein cholesterol (LDL-C) and markedly increased risk of premature atherosclerotic cardiovascular disease. Statins remain the cornerstone of treatment; however, many patients with FH fail to achieve recommended LDL-C targets despite maximally tolerated statin therapy. This gap underscores the urgent need for therapeutic strategies that go beyond statins.

Recent advances have introduced several novel lipid-lowering agents with distinct mechanisms of action. Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors, including monoclonal antibodies and small interfering RNA (siRNA) therapies, have demonstrated robust LDL-C reductions of up to 60% and improved cardiovascular outcomes. Bempedoic acid, an ATP citrate lyase inhibitor, provides additional LDL-C lowering with favorable tolerability and is particularly useful

in statin-intolerant patients. Emerging therapies such as angiopoietin-like protein 3 (ANGPTL3) inhibitors, antisense oligonucleotides targeting apolipoprotein(a), and gene-editing approaches hold promise for addressing residual cardiovascular risk in FH patients.

This lecture will provide an overview of the evolving therapeutic landscape of FH, focusing on clinical trial evidence, real-world data, and practical considerations in the application of non-statin therapies. Special attention will be given to treatment strategies for heterozygous and homozygous FH, cost-effectiveness issues, and the integration of these therapies into current clinical practice.

By moving beyond statins, we are entering a new era in the management of FH, where precision medicine and innovative pharmacologic strategies offer the potential to close the treatment gap and substantially reduce the burden of premature cardiovascular disease.

AAS-KSoLA Joint Symposium

Comprehensive Strategies for the Prevention of Atherosclerosis: Current Insights and Future Directions

Sep 12(Fri) 08:30–10:00 | Room 2 (3F)

CHAIRPERSONS : Jae Bum Kim (Seoul National University, Republic of Korea)
Hyeong Kyu Park (Soonchunhyang University, Republic of Korea)

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|-------------|--|
| 08:30–08:50 | Metabolic disease promotes deoxyceramide accumulation in immune cells altering cytokine production
Andrew Murphy (Baker Heart and Diabetes Institute, Australia) |
| 08:50–09:10 | The role of digital health technology in managing atherosclerosis
Hojin Choi (Hanyang University, Republic of Korea) |
| 09:10–09:30 | Impact of lipid-lowering therapy on aortic aneurysm progression and cardiovascular outcomes
Gyu Chul Oh (The Catholic University of Korea, Republic of Korea) |
| 09:30–10:00 | Panel Discussion
Ung Kim (Yeungnam University, Republic of Korea)
Da Young Lee (Korea University, Republic of Korea)
Jong-Young Lee (Sungkyunkwan University, Republic of Korea) |

CURRICULUM VITAE

Andrew Murphy

Professor, Baker Heart and Diabetes Institute, Australia

**Education and Training**

2003	Queensland University of Technology, Australia, BSc, Biotechnology
2008	Monash University, Australia, Ph.D, Cell Biology

Employment and Position

2009–2013	Columbia University, Postdoctoral Fellow
2013–Current	Baker Heart and Diabetes Institute, Laboratory Head
2019–Current	Baker Heart and Diabetes Institute, Program Head

Important Publications

1. PK Morgan, G Pernes, K Huynh, C Giles, S Paul, AAT Smith, NA Mellett, A Liang, T van Buuren-Milne, C Bertuzzo Veiga, TJC Collins, Y Xu, MKS Lee, TM De Silva, PJ Meikle, GI Lancaster and AJ Murphy. A lipid atlas of human and mouse immune cells provides insights into ferroptosis susceptibility. *Nat Cell Biology*. 2024.
2. G Sreejit, A Abdel-Latif, B Athmanathan, A Dhyani, SK Noothi, GA Quaife-Ryan, A Al-Sharea, G Pernes, D Dragoljevic, H Lal, Kate Schroder, BY Hanaoka, C Raman, MB Grant, JE Hudson, SS Smyth, ER Porrello, AJ Murphy*, PR Nagareddy*. Neutrophil-Derived S100A8/A9 Amplify Granulopoiesis After Myocardial Infarction. *Circulation*. 2020;141:1080–1094. *Joint senior authors.
3. D Dragoljevic, MJ Kraakman, PR Nagareddy, D Ngo, W Shihata, HL Kammoun, A Whillas, MKS Lee, A Al-Sharea, G Pernes, MC Flynn, GI Lancaster, MA Febbraio, J Chin-Dusting, BY Hanaoka, IP Wicks, AJ Murphy. Defective cholesterol metabolism in haematopoietic stem cells promotes monocyte-driven atherosclerosis in rheumatoid arthritis. *European Heart Journal*. 2018;14:39(23):2158–2167.
4. Nagareddy PR, Kraakman M, Masters SL, Stirzaker RA, Gorman DJ, Grant RW, Dragoljevic D, Hong ES, Abdel-Latif A, Smyth SS, Choi SH, Korner J, Bornfeldt KE, Fisher EA, Dixit VD, Tall AR, Goldberg IJ and Murphy AJ. Adipose tissue macrophages promote myelopoiesis and monocytosis in obesity. *Cell Metab*. 2014;19:821–35.
5. Murphy AJ, Bijl N, Yvan-Charvet L, Welch CB, Bhagwat N, Reheman A, Wang Y, Shaw JA, Levine RL, Ni H, Tall AR and Wang N. Cholesterol efflux in megakaryocyte progenitors suppresses platelet production and thrombocytosis. *Nature Medicine*. 2013;19:586–94.

Metabolic disease promotes deoxyceramide accumulation in immune cells altering cytokine production

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder and a major risk factor for accelerated cardiovascular disease. Moreover, T2DM has been shown to profoundly affect immune cell function. Emerging evidence suggests that lipid metabolism in immune cells is altered in T2DM, contributing to systemic inflammation and immune dysregulation. Here, we employed high-resolution lipidomics to characterize the immune cell lipidome in individuals with T2DM, focusing on sphingolipid metabolism. Compared to healthy participants, immune cells isolated from individuals with T2DM displayed significant remodelling of the sphingolipidome, marked by increased abundance of deoxyceramides, a non-canonical ceramide species synthesized via the serine palmitoyltransferase (SPT) enzyme. Studies in mouse

models demonstrate that obesity (insulin resistance) and hypercholesterolemia, two major risk factors for T2DM, revealed similar lipidomic changes, suggesting a shared pathway of sphingolipid dysregulation in metabolic disorders. Functional assays in macrophages demonstrated that deoxyceramide accumulation influences the production of inflammatory cytokines from immune cells. These findings establish a mechanistic link between sphingolipid dysregulation and immune dysfunction in T2DM, with deoxyceramides emerging as key drivers of inflammation. Our study provides new insights into how metabolic stress reshapes the immune cell lipidome, emphasizing the potential of targeting deoxyceramide metabolism as a therapeutic strategy to mitigate inflammation and immune dysregulation in T2DM and related conditions.

CURRICULUM VITAE

Hojin Choi

Professor, Hanyang University Guri Hospital, Republic of Korea



Education and Training

2002.02	Hanyang University, Korea, M.D, Medicine
2012.02	Hanyang University, Korea, Ph.D., Neurology

Employment and Position

2010–2012	Hanyang University Guri Hospital, Fellowship
2013–2016	Hanyang University Guri Hospital, Clinical professor
2017–2022	Hanyang University Guri Hospital, Associate Professor
2023–	Hanyang University Guri Hospital, Professor

Important Publications

1. Choi H(Correspondence), Oh J, Kim CK, Ryu H, Ryu Y. Residents need competence not confidence: A retrospective evaluation of the new competency education program for Korean neurology residents. PLoS One. 2023 Oct 5;18(10):e0290503.
2. Kim SY, Park J, Choi H, Loeser M, Ryu H, Seo K. Digital Marker for Early Screening of Mild Cognitive Impairment Through Hand and Eye Movement Analysis in Virtual Reality Using Machine Learning: First Validation Study. J Med Internet Res. 2023 Oct 20;25:e48093.
3. Sung W, Kwon HS, Park Y, Kim SH, Park S, Kang DR, Choi H(Correspondence). Gout and the Prevalence of Dementia: A Nationwide Population-Based Study. J Alzheimers Dis. 2023;96(1):343–349.
4. Kim SE, Shin C, Yim J, Seo K, Ryu H, Choi H, Park J, Min BK. Resting-state electroencephalographic characteristics related to mild cognitive impairments. Front Psychiatry. 2023 Sep 13;14:1231861.
5. Kwon HS, Park Y, Kim JH, Kim SH, Jun JB, Park S, Kang DR, Choi H(Correspondence). Prevalence of motor neuron diseases in gout patients: a nationwide population-based cohort study. Neurol Sci. 2023 Feb;44(2):593–600.

Awards and Honors

2011	Commendation for meritocracy in the medical service sector of the Seoul Dementia Management Project Evaluation Committee
2018	Best paper award, Korean Dementia Association
2019	Commendation from the Minister of Health and Welfare for the dementia policy

The role of digital health technology in managing atherosclerosis

Digital health technologies are increasingly being applied in the management of atherosclerosis, with tools such as AI-based imaging analysis, data-driven platforms, and wearable monitoring devices being developed. However, despite technological advances, adoption in real-world clinical practice remains limited. This is due to multiple challenges, including poor interoperability between systems, concerns over data privacy, low digital literacy—particularly among older adults—and insufficient insurance coverage or clinical evidence.

Above all, many clinicians remain cautious because AI-generated predictions often do not lead to actionable medical interventions. In medicine, prediction

without intervention is meaningless. “We treat patients, not probabilities.” AI and digital tools should not replace physicians’ decisions but rather support them, strengthening human-centered care instead of automating it blindly.

To address these issues, it is essential not only to improve healthcare professionals’ understanding of digital health technologies but also to ensure that these tools meet certain standards of reliability that make them trustworthy to clinicians. Greater efforts are needed to enhance both technological literacy and clinical understanding in order to bridge the gap between innovation and meaningful application in patient care.

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

Impact of lipid-lowering therapy on aortic aneurysm progression and cardiovascular outcomes

Patients with aortic aneurysm carry substantial cardiovascular risk, and their long-term outcomes are driven not only by rupture but also by systemic atherosclerotic disease. Lipid-lowering therapy, particularly statins, has therefore become a cornerstone of care.

Evidence from observational cohorts and meta-analyses suggests that statin therapy is associated with slower aneurysm expansion, lower rupture risk, and reduced perioperative mortality, although randomized controlled trials targeting aneurysm growth endpoints remain absent. After endovascular or open repair, statin users consistently demonstrate improved long-term survival, with high-intensity therapy offering greater protection compared with low-intensity or no therapy.

Beyond statins, additional strategies are being explored. Ezetimibe has shown reductions in inflammatory mediators within the aortic wall, while genetic

and preclinical studies implicate PCSK9 as a relevant therapeutic target. In animal models, PCSK9 inhibition attenuates aneurysm formation, providing a rationale for evaluating advanced LDL-C-lowering therapies, such as PCSK9 inhibitors, in this population.

Contemporary cardiovascular guidelines recommend aggressive LDL-C lowering for patients with aneurysmal disease, with goals of <55 mg/dL and $\geq 50\%$ reduction from baseline in very-high-risk individuals. For interventional cardiologists and endocrinologists, the clinical implication is clear: comprehensive lipid management should be considered standard of care in all patients with aortic aneurysm, irrespective of whether they undergo repair. While the direct impact on aneurysm growth remains uncertain, the survival and cardiovascular benefits of lipid-lowering therapy are well established, supporting its central role in optimizing outcomes.

VAS-KSoLA Joint Symposium

Lipid-Lowering Strategies: Targets and Therapies in Clinical Practice

Sep 12(Fri) 16:45–18:15 | Room 2 (3F)

CHAIRPERSONS : Ki Hoon Han (University of Ulsan, Republic of Korea)
Eun-Jung Rhee (Sungkyunkwan University, Republic of Korea)

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- | | |
|-------------|---|
| 16:45–17:05 | Triglyceride and HDL-C focus in Asian populations: rethinking priorities?
Jaehoon Chung (Dongguk University, Republic of Korea) |
| 17:05–17:25 | Combined lipid-lowering therapy: when statins alone are not enough
Chang Hee Jung (University of Ulsan, Republic of Korea) |
| 17:25–17:45 | Ethnic differences in managing dyslipidemia: tailoring treatment strategies for Asian and Western populations
Tien Hoang Anh (Hue University of Medicine and Pharmacy, Vietnam) |
| 17:45–18:15 | Panel Discussion
Hyun-Suk Yang (Konkuk University, Republic of Korea)
Jong-Young Lee (Sungkyunkwan University, Republic of Korea)
Jung Rae Cho (Hallym University, Republic of Korea) |

CURRICULUM VITAE**Jaehoon Chung**

Professor, Dongguk University, Republic of Korea

**Education and Training**

2010.02 Yeung-nam University, Korea, M.D, Medicine

Employment and Position

2017-2024 National Medical Center, Clinical Doctor
2024-Present Dongguk University Hospital, Assistant 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>.

Research Interest

Atherosclerosis, Arterial stiffness, Lipid, Hypertension, Metabolic syndrome, Coronary artery disease

Triglyceride and HDL-C focus in Asian populations: rethinking priorities?

Asian populations, particularly South Asians and East Asians, exhibit a distinct lipid phenotype characterized by higher triglycerides and lower HDL-C, often in the presence of relatively normal or only mildly elevated LDL-C.

This dyslipidemic profile is associated with greater

cardiovascular risk, especially coronary heart disease, as opposed to stroke or peripheral arterial disease.

In this presentation, we will discuss the clinical role of triglycerides and HDL-C in addition to LDL-C in Asians.

CURRICULUM VITAE

Chang Hee Jung

Professor, Asan Medical Center, University of Ulsan College of Medicine, Republic of Korea



Education and Training

1996.02	Korea University, Korea, M.D, Medicine
2010.02	Ulsan University, Korea, M.S., Internal Medicine
2012.02	Ulsan University, Korea, Ph.D., Internal Medicine

Employment and Position

2003–2007	Asan Medical Center, Resident
2010–2012	Asan Medical Center, Fellow (Endocrinology)
2012–2016	Asan Medical Center, Clinical Assistant Professor
2016–2020	University of Ulsan College of Medicine, Associate Professor
2018–2020	University of Virginia, VA, USA, Visiting Scholar
2020–	University of Ulsan College of Medicine, Professor

Important Publications

1. 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* 2025, in press.
2. Kim MJ, Cho YK, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH: Association between estimated glucose disposal rate and subclinical coronary atherosclerosis. *Nutr Metab Cardiovasc Dis* 2025, 35:103686.
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 incident pancreatic cancer: A nationwide population-based cohort study. *Diabetes Metab* 2025, 51:101605.
4. Kim MJ, Cho YK, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH: Association between metabolic dysfunction-associated steatotic liver disease and myosteatosis measured by computed tomography. *J Cachexia Sarcopenia Muscle* 2024, 15:1942–52.
5. Kim HS, Cho YK, Kim MJ, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH: Association between atherogenic dyslipidemia and muscle quality defined by myosteatosis. *Front Endocrinol (Lausanne)* 2024, 15:1327522.

Combined lipid-lowering therapy: when statins alone are not enough

Despite the proven efficacy of statins in reducing low-density lipoprotein cholesterol (LDL-C) and preventing atherosclerotic cardiovascular disease (ASCVD), a substantial proportion of patients fail to achieve recommended lipid targets with statin monotherapy. This residual risk may result from statin intolerance, insufficient LDL-C lowering, or the presence of atherogenic dyslipidemia not fully addressed by statins alone. In such cases, combined lipid-lowering therapy provides a more comprehensive approach to cardiovascular risk reduction.

Ezetimibe, when added to statins, can lower LDL-C by an additional 15–20% and has demonstrated incremental cardiovascular benefit in large outcome trials. Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors achieve profound LDL-C reductions of 50–60% and are particularly effective in patients with familial hypercholesterolemia or very high-risk ASCVD. Bempedoic acid, a novel ATP citrate lyase inhibitor,

offers an oral non-statin alternative with complementary efficacy. Beyond LDL-C, addressing triglyceride-rich lipoproteins with fibrates or icosapent ethyl may provide added benefit, especially in patients with type 2 diabetes or metabolic syndrome. Combination approaches tailored to individual risk profiles can therefore optimize lipid control and reduce residual cardiovascular events.

This lecture will review the rationale, evidence, and practical considerations for combination lipid-lowering strategies, including efficacy, safety, and guideline-based recommendations. Emphasis will be placed on identifying patient populations who derive the greatest benefit, strategies for overcoming therapeutic inertia, and future perspectives in precision lipid management. By moving beyond statin monotherapy, clinicians can better address the multifactorial nature of dyslipidemia and further reduce the global burden of cardiovascular disease.

CURRICULUM VITAE

Tien Hoang Anh

Associate Professor, Vice President of Cardiovascular Center of Hue University of Medicine and Pharmacy Hospital, Vietnam



Education and Training

2002	Hue University of Medicine and Pharmacy, Vietnam, M.D, General Medicine
2011	Hue University of Medicine and Pharmacy, Vietnam, Ph.D, Cardiology
2024	Hue University of Medicine and Pharmacy, Vietnam, Assoc. Prof, Cardiology

Employment and Position

2023-Now	Vietnam Atherosclerosis Society, Vice President
2022-Now	Vietnam Heart Failure, Vice President
2019-Now	Hue University of Medicine and Pharmacy, Vietnam, Deputy Head of Cardiovascular Centre, Head of Cardiology Department

Important Publications

1. Prognosis value of heart rate variability measured by Camera HRV application in patients after acute myocardial infarction. Indian Heart Journal 2024 DOI: <https://doi.org/10.1016/j.ihj.2024.07.008>. 2024.
2. Double-Kissing Crush Versus Provisional Stenting for Coronary Bifurcations: A 1-Year Follow-up Study in Vietnam. Journal of the Society for Cardiovascular Angiography & Interventions. Online ISSN: 2772-9303 <https://doi.org/10.1016/j.jscai.2024.102500>. 2025.
3. Alterations in Aortic Elasticity Indices among Type 2 Diabetes Patients in a Low and Middle Income Country Using M-Mode Echocardiography: A Cross-Sectional Comparative Study. PLOS ONE Online ISSN: 1549-1676 <https://doi.org/10.1371/journal.pone.0305799>. 2025.

Ethnic differences in managing dyslipidemia:
tailoring treatment strategies for Asian and Western populations

Background: Dyslipidemia is a major modifiable risk factor for atherosclerotic cardiovascular disease (ASCVD) globally. However, emerging evidence highlights important ethnic differences in lipid profiles, cardiovascular risk patterns, and pharmacological responses between Asian and Western populations. These disparities necessitate ethnically tailored strategies for optimal management.

Objectives: This review aims to compare the characteristics and treatment responses of dyslipidemia between Asian and Western populations and to propose individualized therapeutic approaches based on ethnic-specific considerations.

Methods: We analyzed current literature, including clinical trials, epidemiological studies, and major guidelines, with a focus on variations in LDL-C levels, lipoprotein(a), triglyceride-rich particles, and statin metabolism across ethnic groups. The efficacy and safety profiles of lipid-lowering therapies—including statins, ezetimibe, PCSK9 inhibitors, and novel agents—were evaluated in both populations.

Results: Asians generally exhibit higher prevalence of hypertriglyceridemia, lower HDL-C levels,

and increased statin sensitivity compared to Western populations. Genetic polymorphisms affecting lipid metabolism (e.g., SLCO1B1 variants) and a heightened response to standard statin doses underscore the need for lower initiation doses in Asian patients. Moreover, region-specific guidelines such as the Japan Atherosclerosis Society (JAS), China Cholesterol Education Program (CCEP), and Korean Society of Lipid and Atherosclerosis (KSoLA) provide risk stratification frameworks more suitable for their populations compared to ACC/AHA or ESC recommendations.

Conclusion: Recognizing and addressing ethnic differences in dyslipidemia is essential to improve cardiovascular outcomes. Future guideline development and clinical trial designs must incorporate ethnic-specific data to support precision medicine approaches in lipid management.

Keywords: Dyslipidemia, ethnicity, Asian populations, Western populations, statins, precision medicine, cardiovascular risk, lipid-lowering therapy.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

CSATVB-KSoLA Joint Symposium

Long-term Exposure to LDL-cholesterol and Early Cardiovascular Risk Prevention

Sep 13(Sat) 08:50–10:20 | Room 2 (3F)

CHAIRPERSONS : In-Ho Chae (Seoul National University, Republic of Korea)
Jeong Hyeon Noh (Inje University, Republic of Korea)

08:50–09:10 **Cumulative LDL-cholesterol burden and cardiovascular risk: the unsuspected role of LDL transcytosis**

Warren Lee (Unity Health Toronto and University of Toronto, Canada)

09:10–09:30 **Dyslipidemia in youth: why early intervention matters for lifelong cardiovascular health**

Aram Yang (Sungkyunkwan University, Republic of Korea)

09:30–09:50 **Beyond LDL-C: role of ApoB and non-HDL cholesterol in early prevention**

Jae Hyung Park (Korea University, Republic of Korea)

09:50–10:20 **Panel Discussion**

Yeoree Yang (The Catholic University of Korea, Republic of Korea)

Seung Hun Lee (Chonnam National University, Republic of Korea)

Sang Yup Lim (Korea University, Republic of Korea)

Jung Rae Cho (Hallym University, Republic of Korea)

CURRICULUM VITAE

Warren Lee

Professor of Medicine, University of Toronto, Canada



Education and Training

1997	University of Toronto, Canada, M.D, Medicine
2006	University of Toronto, Ph.D., Cell Biology
2007	Weill Cornell Medicine, USA, Post-doctoral fellow, Microbiology/Immunology

Employment and Position

2007–Present	Unity Health Toronto, Attending physician, Medical-Surgical Intensive Care Unit
2007–2017	University of Toronto, Assistant Professor, Medicine
2017–2025	University of Toronto, Associate Professor, Medicine
2025–	University of Toronto, Professor, Medicine, Biochemistry, Laboratory Medicine and Pathobiology

Important Publications

1. Jang E, Ghaffari S, Henry A, Wen G, Cheng WH, Lyu Y, Ho TWW, Wei K, Wang C, Romanoski CE, Wang Y, Cybulsky M, Lee WL. Lipid Raft Proteomics Identify Endothelial Myosin-9 (MYH9) as a Regulator of Low-Density Lipoprotein Transcytosis and Atherosclerosis. *Proceedings of the National Academy of Sciences* (revision requested) 2025.
2. Jang E, Ho TWW, Brumell JH, Lefebvre F, Wang C, Lee WL. IL-1 β Induces LDL Transcytosis by a Novel Pathway Involving LDLR and Rab27a. *Arterioscler Thromb Vasc Biol.* 2024 Sep;44(9):2053–2068.
3. Ghaffari S, Jang E, Naderinabi F, Sanwal R, Khosraviani N, Wang C, Steinberg BE, Goldenberg NM, Ikeda J, Lee WL. Endothelial HMGB1 Is a Critical Regulator of LDL Transcytosis via an SREBP2-SR-BI Axis. *Arterioscler Thromb Vasc Biol.* 2021 Jan;41(1):200–216.
4. Ghaffari S, Naderi Nabi F, Sugiyama MG, Lee WL. Estrogen Inhibits LDL (Low-Density Lipoprotein) Transcytosis by Human Coronary Artery Endothelial Cells via GPER (G-Protein-Coupled Estrogen Receptor) and SR-BI (Scavenger Receptor Class B Type 1). *Arterioscler Thromb Vasc Biol.* 2018 Oct;38(10):2283–2294.
5. Armstrong S, Sugiyama M, Fung YY, Gao Y, Levy A, Wang C, Azizi P, Roufaiel M, Neculai D, Bolz SS, Seidah N, Cybulsky M, Heit B, Lee WL. A novel assay uncovers an unexpected role for SR-BI in LDL transcytosis. *Cardiovascular Research.* 2015;108(2): 268–77. The first description of a receptor capable of mediating LDL transcytosis in the systemic circulation.

Cumulative LDL-cholesterol burden and cardiovascular risk: the unsuspected role of LDL transcytosis

Accumulation of LDL-derived cholesterol under the arterial endothelium triggers an inflammatory reaction that culminates in luminal narrowing and eventually an unstable arterial plaque. However, how the LDL gets under the endothelium is poorly understood.

Autopsy studies on young individuals dying of non-cardiac causes reveal a healthy, continuous endothelial layer overlying cholesterol deposits and the average LDL particle is too large to pass through intact cell-cell junctions; thus, LDL is likely to cross the endothelium by a form of vesicular traffic known as transcytosis. It is now apparent that transcytosis of LDL into the vessel intima over many decades is the initiating cause of atherosclerosis.

Despite its initial description based on electron microscopy decades ago, there is surprisingly little liter-

ature on LDL transcytosis. This is attributable to technical difficulties in distinguishing paracellular versus transcellular leakage in cultured endothelial cells.

In my lecture, I will discuss advances in the study of LDL transcytosis that have permitted rapid elucidation of its molecular regulation by both physiologic and pathologic stimuli. This includes the identification of receptors that carry LDL across the endothelium and the observation that inhibition of LDL transcytosis by genetic or pharmacologic means prevents atherosclerosis in animal models, implying that it is a veritable therapeutic target.

Understanding how endothelial transcytosis is regulated is likely to have broad-reaching pathologic and therapeutic implications.

CURRICULUM VITAE

Aram Yang

Associate Professor, Department of Pediatrics, Kangbuk Samsung Hospital,
Sungkyunkwan University School of Medicine, Republic of Korea



Education and Training

2011.11	Chungnam National University, Korea, M.D, Medicine
2016.06	Sungkyunkwan University, Korea, M.M.Sc., Medicine
2023.02	Sungkyunkwan University, Korea, Ph.D, Medicine
2012.03–2016.02	Samsung Medical Center, Residency, Dept. of Pediatrics
2016.03–2018.02	Clinical fellow, Samsung Medical Center, Dept. of Pediatric Endocrinology, Genetics, and Metabolism
2018.03–2019.02	Clinical instructor, Inha University Hospital, Dept. of Pediatric Endocrinology, Genetics, and Metabolism
2019.03–2019.08	Assistant Professor, Inha University Hospital, Dept. of Pediatric Endocrinology, Genetics, and Metabolism
2019.09–2021.02	Clinical Assistant Professor, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Dept. of Pediatric Endocrinology, Genetics, and Metabolism
2021.03–2025.02	Assistant Professor, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Dept. of Pediatric Endocrinology, Genetics, and Metabolism
2025.03–	Associate Professor, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Dept. of Pediatric Endocrinology, Genetics, and Metabolism

Employment and Position

Currently, deputy secretary and member of the Medical Guidelines Committee and Public Relations Committee of the Korean Society of Pediatric Endocrinology
Member of the Pediatric and Adolescent Committee of the Korean Society of Obesity
Asia Pacific Endocrine Society Fellow School Committee Member
ENDO, APPE, ESPE regular member
Certified Clinical Geneticist

Dyslipidemia in youth: why early intervention matters for lifelong cardiovascular health

The prevalence of dyslipidemia in children and adolescents is steadily increasing, largely driven by rising rates of obesity and lifestyle-related risk factors. Recognizing this trend, the 2022 Korean pediatric dyslipidemia guidelines were established, emphasizing age-appropriate screening, diagnostic thresholds, and treatment strategies tailored for the pediatric population.

Unlike in adults, pharmacologic treatment options for dyslipidemia in youth are limited due to stricter regulatory approvals and safety concerns. As a result, the decision to initiate medication often requires careful risk stratification, typically reserved for patients with familial hypercholesterolemia (FH) or severe, persistent dyslipidemia.

However, mounting evidence suggests that dyslipid-

emia beginning in childhood—particularly when left uncontrolled—can lead to significantly worse cardiovascular outcomes than adult-onset cases. Longitudinal cohort studies have shown strong associations between persistent elevations in non-HDL cholesterol from childhood to adulthood and increased incidence of cardiovascular events.

This presentation will provide an overview of pediatric dyslipidemia with a focus on current epidemiologic trends, key components of the 2022 Korean guidelines, limitations in pediatric lipid-lowering therapy, and emerging evidence linking early lipid abnormalities to future cardiovascular risk. Together, these insights support the importance of timely intervention and long-term management strategies to prevent premature atherosclerotic disease.

CURRICULUM VITAE**Jae Hyoung Park**

Clinical Professor, Korea University Anam Hospital, Republic of Korea

**Education and Training**

1999.02	Korea University, Korea, M.D, Medicine
2012.02	Korea University, Korea, Ph.D., Internal Medicine

Employment and Position

2007-2008	Asan Medical Center, Fellow
2008-2009	Korea University Guro Hospital, Clinical Assistant Professor
2009-	Korea University Anam Hospital, Clinical Professor

Important Publications

1. Jae Hyoung Park, YM Ro, SY Suh, YH Kim, JO Na, SH Shin, MY Park, JS Park, SH Kim, JW Kim, SJ Hong, HN Park, DS Lim, YH Kim, WJ Sim, DJ Oh : Carotid Artery Intima-Media Thickness in Patients with Hypertension with Left Ventricular Hypertrophy. J Kor Soc Echo 11 : 94-101, 2003
2. Jeong HS, Hong SJ, Cho SA, et al. Comparison of Ticagrelor Versus Prasugrel for Inflammation, Vascular Function, and Circulating Endothelial Progenitor Cells in Diabetic Patients With Non-ST-Segment Elevation Acute Coronary Syndrome Requiring Coronary Stenting: A Prospective, Randomized, Crossover Trial. JACC Cardiovasc Interv. 2017;10(16):1646-1658. doi:10.1016/j.jcin.2017.05.064
3. Cho SA, Joo HJ, Cho JY, et al. Visceral Fat Area and Serum Adiponectin Level Predict the Development of Metabolic Syndrome in a Community-Based Asymptomatic Population. PLoS One. 2017;12(1):e0169289. Published 2017 Jan 3. doi:10.1371/journal.pone.0169289
4. Joo HJ, Ahn SG, Park JH, et al. Effects of genetic variants on platelet reactivity and one-year clinical outcomes after percutaneous coronary intervention: A prospective multicentre registry study. Sci Rep. 2018;8(1):1229. Published 2018 Jan 19. doi:10.1038/s41598-017-18134-y
5. 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

Research Interest

CVD prevention & treatments

Beyond LDL-C: role of ApoB and non-HDL cholesterol in early prevention

KoSFoST–KSoLA Joint Symposium

Role of Medicinal Foods in Atherosclerosis and Cardiometabolic Disease

Sep 13(Sat) 14:50–16:20 | Room 2 (3F)

CHAIRPERSONS : **Hyojee Joung** (Seoul National University, Republic of Korea)
Min-Jeong Shin (Korea University, Republic of Korea)

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- | | |
|-------------|---|
| 14:50–15:10 | Novel health functional products to prevent post-MI pathologic remodeling
Hun-Jun Park (The Catholic University of Korea, Republic of Korea) |
| 15:10–15:30 | Ultrasonicated <i>Lespedeza cuneata</i> extract prevents TNF-α-induced early atherosclerosis in vitro and in vivo
Sung Keun Jung (Kyungpook National University, Republic of Korea) |
| 15:30–15:50 | Regulation of mitochondrial metabolic reprogramming during aging
Jiyun Ahn (Korea Food Research Institute, Republic of Korea) |
| 15:50–16:20 | Panel Discussion
Hyunju Kang (Keimyung University, Republic of Korea)
Bohkyung Kim (Pusan National University, Republic of Korea)
Dahyun Park (Korea University, Republic of Korea)
Hyunjung Lim (Kyung Hee University, Republic of Korea) |

CURRICULUM VITAE

Hun-Jun Park

Professor, The Catholic University of Korea, Republic of Korea



Education and Training

1997.02	The Catholic University of Korea, Korea, M.D, Medicine
2012.02	The Catholic University of Korea, Korea, Ph.D., Internal Medicine

Employment and Position

2009-2014	Seoul St. Mary's Hospital, Assistant Professor
2014-2019	Seoul St. Mary's Hospital, Associate Professor
2019-2024	Seoul St. Mary's Hospital, Professor
2024-2025	Uijeonbu St. Mary's Hospital, Professor

Important Publications

1. Spatiotemporal control of neutrophil fate to tune inflammation and repair for myocardial infarction therapy. Nat Commun. 2024;15:8481, Role: Corresponding author
2. Versatile human cardiac tissues engineered with perfusable heart extracellular microenvironment for biomedical applications. Nat Commun. 2024;15:2564. Role: Corresponding author
3. Daily oral administration of probiotics engineered to constantly secrete short-chain fatty acids effectively prevents myocardial injury from subsequent ischaemic heart disease. Cardiovasc Res. 2024 25:120:1737-1751. Role: Corresponding author
4. Hyaluronic acid stimulation of stem cells for cardiac repair: a cell-free strategy for myocardial infarct. J Nanobiotechnology. 2024;22:149. Role: Corresponding author
5. Robust and customizable spheroid culture system for regenerative medicine. Biofabrication. 2024 2:16 Role: Corresponding author

Research Interest

- Cardiology, Interventional
- Acute Myocardial Infarction, Heart failure
- Cardiovascular regenerative medicine

Novel health functional products to prevent post-MI pathologic remodeling

Myocardial infarction (MI) remains a leading cause of heart failure due to adverse cardiac remodeling that follows the ischemic injury. Pathologic remodeling involves ventricular dilation, fibrosis, and contractile dysfunction, ultimately impairing cardiac performance and increasing mortality. Current therapeutic strategies primarily focus on symptom management and prevention of further ischemic events, with limited options to directly target or reverse remodeling processes. In this context, novel health functional products derived from natural compounds, bioactive

peptides, and nutraceuticals have garnered increasing interest for their potential cardioprotective and anti-remodeling effects. This review introduces novel health functional products designed to prevent or mitigate post-MI pathologic remodeling. We discuss the underlying mechanisms, including anti-inflammatory, antioxidant, and antifibrotic pathways, supported by preclinical evidence. The integration of these novel interventions into current treatment paradigms holds promise for improving long-term outcomes in MI patients and reducing the burden of heart failure.

CURRICULUM VITAE

Sung Keun Jung

Professor, Kyungpook National University, Republic of Korea



Education and Training

2009.02 Seoul National University, Korea, Ph.D., Functional Food

Employment and Position

2012–2018 Korean Food Research Institute, Senior Researcher
 2020– Applied Biological Chemistry, Editor
 2024– J. of Microbiology and Biotechnology, Editor
 2024– KFDA, Health Functional Foods Committee Member

Important Publications

1. Kim MJ, Choi HJ, Kang HY, Kim HS, Jung CH, Jung SK. CREB1 controls mitochondrial dysfunction in 1-nitropyrene-mediated apoptosis of human bronchial cells and lung injury. *Sci Total Environ*. 2025 Mar 10;968:178877.
2. Kim S, Jang SH, Kim MJ, Lee JJ, Kim KM, Kim YH, Lee JH, Jung SK. Hybrid nutraceutical of 2-ketoglutaric acid in improving inflammatory bowel disease: Role of prebiotics and TAK1 inhibitor. *Biomed Pharmacother*. 2024 Feb;171:116126.
3. Jang JH, So BR, Yeo HJ, Kang HJ, Kim MJ, Lee JJ, Jung SK, Jung YH. Preparation of cellulose microfibril (CMF) from *Gelidium amansii* and feasibility of CMF as a cosmetic ingredient. *Carbohydr Polym*. 2021 Apr 1;257:117569. (Corresponding author)
4. So BR, Yeo HJ, Lee JJ, Jung YH, Jung SK. Cellulose nanocrystal preparation from *Gelidium amansii* and analysis of its anti-inflammatory effect on the skin in vitro and in vivo. *Carbohydr Polym*. 2021 Feb 15;254:117315.
5. Lee J, Hyon JY, Min JY, Huh YH, Kim HJ, Lee H, Yun SH, Choi CW, Jeong Ha S, Park J, Chung YH, Jeong HG, Ha SK, Jung SK, Kim Y, Han EH. Mitochondrial carnitine palmitoyltransferase 2 is involved in N ϵ -(carboxymethyl)-lysine-mediated diabetic nephropathy. *Pharmacol Res*. 2020 Feb;152:104600. (Corresponding author)

Research Interest

Development of health functional food materials for respiratory and gut health
 Gut and skin and lung axis
 Development of prebiotics by using upcycling technologies

Ultrasonicated *Lespedeza cuneata* extract prevents TNF- α -induced early atherosclerosis in vitro and in vivo

This study evaluated the use of ultrasonication to extract *Lespedeza cuneata* as a potential nutraceutical for preventing vascular inflammation. Ultrasonicated *L. cuneata* extract (ULCE) was prepared using 20% ethanol and 2 h of ultrasonication at room temperature, and its effects were investigated using relevant in vitro and in vivo models. ULCE suppressed tumor necrosis factor- α (TNF- α)-induced adhesion capacity, vascular cell adhesion protein 1 (VCAM-1) expression, and nuclear factor kappa-B (NF- κ B) activity in human umbilical vein endothelial cells (HUVECs). ULCE also suppressed TNF- α -induced NF- κ B signaling pathways and p65 translocation from the cytosol to the nucleus,

as well as the mRNA expression of IL-1 β , IL-6, and TNF- α in HUVECs. Oral administration of ULCE suppressed TNF- α -induced monocyte infiltration into the intima and VCAM-1 expression, as well as the IL-1 β , IL-6, TNF- α , and monocyte chemoattractant protein-1 (MCP-1) mRNA expression in the main artery in mice. Among the compounds identified in the hydrolyzed ULCE, quercetin exhibited the strongest inhibitory effect against TNF- α -induced cell adhesion capacity. These results demonstrate that ULCE contains potent preventive factors against early atherosclerosis, which act by suppressing the NF- κ B and VCAM-1 signaling axis

CURRICULUM VITAE

Jiyun Ahn

Head, Aging Research Group
 Chief Major Professor, Food Biotechnology
 Korea Food Research Institute, UST-KFRI School, Republic of Korea



Education and Training

1999.02	Konkuk University, Korea, D.V.M., Veterinary Medicine
2001.02	Konkuk University, Korea, M.S., Veterinary Medicine
2006.02	Konkuk University, Korea, Ph.D., Veterinary Medicine

Employment and Position

2001-	KFRI, Principal Researcher
2008-	UST, Professor
2023-	UST, Chief Major Professor
2025-	KFRI, Head, Aging Research Group

Important Publications

1. Norharmane prevents muscle aging via activation of SKN-1/NRF2 stress response pathways. Nirmala FS, Lee H, Cho Y, Um MY, Seo HD, Jung CH, Hahm JH, Ahn J. Redox Biol. 2025 80:103512.
2. Exercise-induced signaling activation by Chrysanthemum zawadskii and its active compound, linarin, ameliorates age-related sarcopenia through Sestrin 1 regulation. Nirmala FS, Lee H, Kim YI, Hahm JH, Seo HD, Kim M, Jung CH, Ahn J, Phytomedicine. 2024 129:155695.
3. Nutritional approaches targeting mitochondria for the prevention of sarcopenia. Hahm JH, Nirmala FS, Ha TY, Ahn J, Nutr Rev. 2023 82(5):676–694.
4. MiR-141-3p promotes mitochondrial dysfunction in ovariectomy-induced sarcopenia via targeting Fkbp5 and Fibin. Lee H, Kim YI, Nirmala FS, Kim JS, Seo HD, Ha TY, Jang YJ, Jung CH, Ahn J. Aging (Albany NY). 2021 13(4):4881–4894.
5. Mitochondrial dysfunction in skeletal muscle contributes to the development of acute insulin resistance in mice. Lee H, Ha TY, Jung CH, Nirmala FS, Park SY, Huh YH, Ahn J. J Cachexia Sarcopenia Muscle. 2021 12(6):1925–1939.

Awards and Honors

1. Minister of Agriculture, Food and Rural Affairs Award, 2023
2. Governor of Jeollabuk-do Commendation, 2023
3. Minister of Education, Science and Technology Award, 2009

Research Interest

Nutritional modulation of muscle aging

Regulation of mitochondrial metabolic reprogramming during aging

Aging disrupts mitochondrial homeostasis and drives a metabolic shift from oxidative phosphorylation to glycolysis, contributing to sarcopenia and systemic metabolic decline. In this talk, I will present recent findings demonstrating that blocking this glycolytic switch restores mitochondrial respiration, improves

muscle function, and delays aging-related decline. By using natural phytochemicals as examples, I will also discuss the potential of targeting metabolic reprogramming as a promising strategy not only for muscle aging but also for broader cardiometabolic health.

EAS-KSoLA Joint Symposium

The Impact of Remnant Cholesterol on Various Disease Pathologies and Clinical Outcomes

Sep 13(Sat) 16:20–17:50 | Room 2 (3F)

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

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|-------------|--|
| 16:20–16:40 | Remnant cholesterol as a key contributor to atherosclerotic cardiovascular disease (ASCVD): mechanisms and clinical implications
Alberto Zambon (University of Padua, Italy) |
| 16:40–16:55 | The role of remnant cholesterol in chronic kidney disease (CKD)
Soo Yeon Jang (Korea University, Republic of Korea) |
| 16:55–17:10 | Association between remnant cholesterol and dementia: potential mechanisms and clinical perspectives
Han Na Jung (Hallym University, Republic of Korea) |
| 17:10–17:30 | Remnant cholesterol in women's cardiometabolic health
Meral Kayikcioglu (Ege University, Türkiye) |
| 17:30–17:50 | Panel Discussion
Mi-Hyang Jung (The Catholic University of Korea, Republic of Korea)
Eun Jeong Cho (Chung-Ang University, Republic of Korea)
Ji Hye Huh (Hallym University, Republic of Korea) |

CURRICULUM VITAE

Alberto Zambon

University of Padua, Italy



Expertise

- MD: Internal Medicine, Endocrinology and Metabolism
- PhD: Atherosclerosis, Lipid and lipoprotein metabolism, Lipid-lowering therapy and cardiovascular disease prevention, Atherosclerotic plaque pathophysiology

Work Experience

Present	University of Padova, Dept. of Medicine, Via Giustiniani 2, 35128 Padova - Italy Physician - Academic teaching Hospital Associate Professor of Medicine Inpatient clinic: Internal medicine ward; Outpatient: lipid clinic; Tutor for post-doctoral fellows Endocrinology and Metabolism, Teaching activity: General Medicine and Endocrinology-Metabolism courses for Medical School Students
1999.07-2000.06	Division of Metabolism Endocrinology and Nutrition, University of Washington, 1959 N.E. Pacific St., Seattle, WA, USA Physician - Academic teaching Hospital Acting Assistant Professor of Medicine Research on lipid metabolism; Teaching classes: Endo-Metabolism Course, Medical School
2000.07-2010.12	Division of Metabolism Endocrinology and Nutrition, University of Washington, 1959 N.E. Pacific St., Seattle, WA, USA Physician - Academic teaching Hospital Affiliate Assistant Professor of Medicine Research on lipid metabolism and atherosclerosis, and genetic forms of lipid disorders

Remnant cholesterol as a key contributor to atherosclerotic cardiovascular disease (ASCVD): mechanisms and clinical implications

The critical and causal role of cholesterol-rich apolipoprotein B (apoB)-containing lipoproteins in atherogenesis has been clearly identified over the past decades. LDL is considered the archetypal atherogenic lipoprotein. As the main carrier of cholesterol in the blood, it is for most people the principal vehicle delivering cholesterol into the artery wall and thereby initiating and progressing lesion formation. However, mounting evidence has established that triglyceride-rich lipoproteins (TRLs) and their remnants are also causal factors in atherosclerotic cardiovascular disease (ASCVD), and their contribution to pathological processes appears statistically independent of, and additional to, that of LDL. The strongest evidence for this comes from studies in which variants in genes known to alter circulating triglyceride (TG), and hence, TRL concentrations are associated with coronary heart disease (CHD) outcomes. Recent advances in human genetics, together with epidemiologic and clinical trial results, indicate that the “residual” ASCVD risk is in part caused by elevated levels of TRLs and their

remnants. TRL/remnant particles are likely more atherogenic than LDL on a per particle (per apoB) basis. The greater atherogenicity of TRL/remnants appeared not to be explained fully by their higher cholesterol content (the risk per unit cholesterol was higher in TRL/remnants than in LDL), and this raised the question as to what other features of these particles might have pathogenic actions such as proinflammatory effects. Population studies have demonstrated links between plasma TG, chronic inflammation, and CHD; TG levels are related strongly to CRP and to blood leucocyte count. Remnant cholesterol lowering is associated with part of the cardiovascular risk reduction in statin, ezetimibe, and PCSK9 inhibitor trials. Finally, recent evidence strongly suggests that the lipid-related risk reduction of CAD is most accurately reflected by the total count of apoB containing lipoproteins and is largely unaffected by major particle type (VLDL, remnants, IDL/LDL), count, or size. Future guidelines should consider Apo B as primary target for CAD risk reduction.

CURRICULUM VITAE

Soo Yeon Jang

Clinical Assistant Professor, Korea University, Republic of Korea



Education and Training

2018.02	Korea University, Korea, M.D, Medicine
2025.08	Korea University, Korea, Ph.D., Internal Medicine

Employment and Position

2018–2019	Korea University Medical Center, Intern
2020–2023	Korea University Guro Hospital, Resident
2023–2025	Korea University Guro Hospital, Fellow
2025–	Korea University Guro Hospital, Clinical Assistant Professor

Important Publications

1. Jang SY, Choi KM. Impact of Adipose Tissue and Lipids on Skeletal Muscle in Sarcopenia. *J Cachexia Sarcopenia Muscle*. 2025;16(4):e70000.
2. Jang SY, Hwang SY, Jang A, Kim KJ, Yu JH, Kim NH, Yoo HJ, Kim NH, Baik SH, Choi KM. Association of remnant cholesterol with sarcopenia in Korean adults: a nationwide population-based study using data from the KNHANES. *Front Endocrinol (Lausanne)*. 2024 Aug 23;15:1391733.
3. Jang SY, Choi KM. Bidirectional crosstalk between bone and muscle: the role of RANKL pathway in osteosarcopenia. *J Endocrinol*. 2024 Jul 18;262(3):e240093.
4. Jang SY, Kang M, Song E, Jang A, Choi KM, Baik SH, Yoo HJ. Remnant cholesterol is an independent risk factor for the incidence of chronic kidney disease in newly-diagnosed type 2 diabetes: A nationwide population-based study. *Diabetes Res Clin Pract*. 2024 Apr;210:111639.

Research Interest

Diabetes, Sarcopenia, Dyslipidemia

The role of remnant cholesterol in chronic kidney disease (CKD)

Chronic kidney disease (CKD) is becoming a more significant health concern in the context of aging population and the rising incidence of type 2 diabetes and hypertension. Despite its progressive feature, CKD is often asymptomatic until the very advanced stage, making the early detection and intervention challenging. Pharmacological interventions for CKD remain limited, therefore it is necessary to explore novel therapeutic targets for preventing development and progression of the disease.

Dyslipidemia shares a close pathophysiological relationship with CKD and may contribute adversely to both its onset and progression. Although previous studies have suggested that managing traditional lipid profiles, such as low-density lipoprotein cholesterol (LDL-C) and triglycerides (TGs), may improve CKD outcomes, the effectiveness of these conventional lipid-lowering interven-

tions in altering CKD progression remains underexplored.

Remnant cholesterol (remnant-C), which represents the cholesterol contents of triglyceride-rich lipoproteins, has garnered considerable attention as a contributor to residual cardiovascular risk. Remnant-C has greater permeability across the arterial wall and a longer retention time in the circulation compared to LDL-C. In the kidney, which is highly vascularized, such accumulation of remnant-C may promote endothelial dysfunction, inflammation, and oxidative stress, ultimately accelerating renal injury. Mounting evidence showed that elevated levels of remnant-C are associated with an increased risk of development and adverse course of CKD independent of traditional risk factors.

This talk will address the effects of remnant-C on kidney and its potential as a novel preventive and therapeutic target of CKD.

CURRICULUM VITAE

Han Na Jung

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



Education and Training

2011.03	Korea University, Korea, M.D, Medicine
2019.03	Ulsan University, Korea, M.S, Internal Medicine
2018.03	Asan Medical Center, Korea, Residency, Internal Medicine
2021.03	Asan Medical Center, Korea, Clinical Fellow, Endocrinology and Metabolism
2023.03	Hallym University Sacred Heart Hospital, Korea, Clinical Fellow, Endocrinology and Metabolism

Employment and Position

2024-	Endocrinology and Metabolism, Hallym University Sacred Heart Hospital, Korea, Clinical Assistant Professor
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Important Publications

1. Jung HN, Huh JH, Roh E, Kim BJ, Lee M, Kim JK, Kim JH, Han B, Han KD, Kang JG, Lee SJ, Ihm SH. Risk of pancreatic cancer according to glycemic status in nonalcoholic fatty liver disease: a nationwide cohort study. *Sci Rep*. 2025 Jul 2;15(1):23308.
2. Heo JH, Jung HN (co-first), Roh E, Han KD, Kang JG, Lee SJ, Ihm SH. Association of remnant cholesterol with risk of dementia: a nationwide population-based cohort study in South Korea. *Lancet Healthy Longev*. 2024 Jul 25:S2666-7568(24)00112-0.
3. Jung HN (co-first), Huh JH, Roh E, Han KD, Kang JG, Lee SJ, Ihm SH. High remnant-cholesterol levels increase the risk for end-stage renal disease: a nationwide, population-based, cohort study. *Lipids Health Dis*. 2024 Jun 4;23(1):165.
4. Jung HN, Kim SO, Jung CH, Lee WJ, Kim MJ, Cho YK. Preserved Muscle Strength Despite Muscle Mass Loss After Bariatric Metabolic Surgery: a Systematic Review and Meta-analysis. *Obes Surg*. 2023 Nov;33(11):3422-3430.
5. Jung HN (co-first), Jung CH, Hwang YC. Sarcopenia in youth. *Metabolism*. 2023 Jul;144:155557.

Association between remnant cholesterol and dementia: potential mechanisms and clinical perspectives

Remnant cholesterol, defined as the cholesterol content of triglyceride-rich lipoproteins (including VLDL, IDL, and chylomicron remnants), has emerged as a potent residual atherogenic factor beyond low-density lipoprotein cholesterol (LDL-C) reduction. Although remnant cholesterol has been shown to independently increase cardiovascular disease and mortality risks beyond LDL-C concentrations, few studies have examined the association between remnant cholesterol and dementia. Recent Mendelian randomization studies, however, have suggested a possible link between elevated remnant cholesterol and cognitive decline. Considering evidence that dyslipidemia plays an increasingly important role in the pathogenesis of chronic age-related diseases as well as cognitive impairment, it is imperative to determine whether remnant cho-

lesterol contributes to the development of dementia. Mechanistically, remnant cholesterol may promote vascular dementia primarily through atherosclerosis, which subsequently leads to cerebral hypoperfusion, aberrant vascular morphogenesis, and oxidative stress. Additionally, alterations in cerebral lipid metabolism, possibly mediated through apoE-related pathways, could also be involved in neurodegenerative processes. In this lecture, I will first provide a concise overview of remnant cholesterol biology, and then highlight our nationwide cohort findings demonstrating independent associations of remnant cholesterol with all-cause dementia, Alzheimer's disease, and vascular dementia in Korean adults—emphasizing clinical implications of remnant cholesterol for dementia risk prediction.

CURRICULUM VITAE

Meral Kayikcioglu

Professor of Cardiology, Ege University, Izmir, Türkiye



Education and Training

1995.5	Ege University Medical School of Medicine, Izmir, Türkiye, M.D., Medicine
1999.7	Ege University Medical School of Medicine, Izmir, Türkiye, M.D., Cardiology
2019-Ongoing	Ege University Medical School of Medicine, Izmir, Türkiye, PhD, Doctoral Program (in progress), Molecular Biology

Employment and Position

1999–2004	Ege University Medical School, Cardiology Consultant
2004–2011	Ege University Medical School, Associate Professor of Cardiology
2011–	Ege University Medical School, Professor of Cardiology

Important Publications

1. Roeters van Lennep JE, Öörni K, Binder CJ, Kronenberg F, Mallat Z, Raggi P, Benn M, Afzal S, Holven KB, Kayikcioglu M, Laufs U, Liberopoulos E, Ray KK, Nordestgaard BG, Boren J. The essential role of sex and gender in atherosclerosis research: A statement from the Editorial Board of Atherosclerosis and Executive Committee of the European Atherosclerosis Society. *Atherosclerosis*. 2025;407:120403. doi: 10.1016/j.atherosclerosis.2025.120403.
2. Yurtseven E, Ural D, Gursoy E, Cunedioglu BO, Guler OU, Baysal K, Aytekin S, Aytekin V, Kayikcioglu M. Is There a Need for Sex-Tailored Lipoprotein(a) Cut-Off Values for Coronary Artery Disease Risk Stratification? *Clin Cardiol*. 2024;47:e70012. doi: 10.1002/clc.70012.

Remnant cholesterol in women's cardiometabolic health

Remnant cholesterol (RC)—the cholesterol content of triglyceride-rich lipoproteins including very-low-density lipoproteins (VLDL), intermediate-density lipoproteins, and chylomicron remnants—has emerged as a significant atherogenic factor beyond LDL-C, particularly in women's cardiometabolic risk. While RC is increasingly recognized as a causal contributor to atherosclerotic cardiovascular disease (ASCVD), its sex-specific impact remains underexplored.

In cardiometabolic diseases, a key but underrecognized shift occurs: the cholesterol content of triglyceride-rich lipoproteins (RC) increases substantially, rendering LDL-C alone insufficient to reflect the total atherogenic cholesterol burden. Women—especially postmenopausal—often exhibit higher plasma triglyceride and RC levels in the presence of metabolic syndrome, obesity, type 2 diabetes, or polycystic ovary syndrome. Obstructive sleep apnea (OSA) is another critical but underrecognized risk factor that exacerbates dyslipidemia by increasing RC in women. In regression analyses, the prevalence of hypercholesterolemia increases with age, while non-HDL and LDL cholesterol levels rise significantly with both OSA

severity and age. Even after excluding patients with confounding obesity and diabetes, women with OSA exhibit an impaired lipid profile, including elevated LDL-C, non-HDL-C, and triglycerides. A significant association between dyslipidemia and OSA severity has been observed in women, but not in men, highlighting a sex-specific vulnerability.

RC contributes to atherosclerosis through its interplay with systemic inflammation, endothelial dysfunction, and insulin resistance, which are central to the cardiometabolic continuum in women. These findings underscore the inadequacy of relying solely on LDL-C for risk assessment in women. Measuring non-HDL-C and calculated RC (total cholesterol minus HDL-C minus LDL-C) may offer better insights. Therapeutic strategies combining LDL-C-lowering with RC-targeted approaches—such as fibrates, omega-3 fatty acids, and novel agents like ANGPTL3 inhibitors—may prove especially beneficial for high-risk women.

In conclusion, RC is a critical but underrecognized determinant of women's cardiometabolic health. Addressing it demands a broader, sex-specific approach to lipid assessment and therapy.

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

MSA-KSoLA Joint Symposium

Recent Evidence in Non-invasive Cardiovascular Risk Assessment Techniques

Sep 13(Sat) 16:20–17:50 | Room 4 (5F)

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

Sang-Hyun Lhm (The Catholic University of Korea, Republic of Korea)

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|-------------|--|
| 16:20–16:40 | Recent updates on coronary artery calcium scoring
Nor Ashikin Md Sari (Universiti Malaya, Malaysia) |
| 16:40–17:00 | The role of carotid ultrasonography for the primary prevention of cardiovascular disease
Jun Hwa Hong (Eulji University, Republic of Korea) |
| 17:00–17:20 | The usefulness of brachial-ankle PWV in the risk prediction for Asians
Hack-Lyoung Kim (Seoul National University, Republic of Korea) |
| 17:20–17:50 | Panel Discussion
Jong Shin Woo (Kyung Hee University, Republic of Korea)
Hyun Sung Joh (Seoul National University, Republic of Korea)
Hyo-In Choi (Sungkyunkwan 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.jeimp.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

Recent updates on coronary artery calcium scoring

Coronary artery calcium (CAC) scoring has proven to be a useful marker in the evaluation of patients' cardiovascular risk beyond the traditional risk factors. Major clinical practice guidelines have adopted CAC in their recommendations. This presentation reviews the similarities and differences in the recommenda-

tions for CAC among these guidelines. It also looks into several studies including on-going prospective trials that attempt to answer if a management strategy that is based on CAC will result in better cardiovascular outcomes when compared to the conventional management and therapeutic strategies.

CURRICULUM VITAE

Jun Hwa Hong

Associate Professor, Daejeon Eulji Medical Center, Eulji University, Republic of Korea



Education and Training

2024.02	Eulji University, Korea, M.D, Medicine
2015.02	Eulji University, Korea, Ph.D, Internal Medicine

Employment and Position

2013-2014	Chungnam National University Hospital, Daejeon, Korea, Clinical Fellow in Internal Medicine
2014-2016	Kyungpook National University Hospital, Daegu, Korea, Clinical Assistant Professor in Internal Medicine
2016-2022	Eulji University Hospital, Daejeon, Korea, Assistant Professor in Internal Medicine
2022-Present	Eulji University Hospital, Daejeon, Korea, Associate Professor in Internal Medicine

Important Publications

1. Efficacy and safety of High dose Pioglitazone as Add-on Therapy in Patients with Type 2 Diabetes Mellitus Inadequately Controlled with Dapagliflozin and Metformin: Double-Blind, Randomized, Placebo-Controlled Trial. *Diabetes Metab J.* 2025 Accepted.
2. Efficacy and safety of a fixed-dose combination of dapagliflozin and linagliptin (AJU-A51) in patients with type 2 diabetes mellitus: A multicentre, randomized, double-blind, parallel-group, placebo-controlled phase III study. *Diabetes Obes Metab.* 2025 Jan;27(1):81-91. doi: 10.1111/dom.15985. Epub 2024 Oct 7.
3. Pioglitazone as Add-on Therapy in Patients with Type 2 Diabetes Mellitus Inadequately Controlled with Dapagliflozin and Metformin: Double-Blind, Randomized, Placebo-Controlled Trial. *Diabetes Metab J.* 2024 Sep;48(5):937-948. doi: 10.4093/dmj.2023.0314. Epub 2024 Feb 2.
4. Efficacy and Safety of Pioglitazone Add-on in Patients with Type 2 Diabetes Mellitus Inadequately Controlled with Metformin and Dapagliflozin: A Multicenter, Randomized, Double-blind, and Placebo-controlled Study. *Clin Ther.* 2024 Sep;46(9):662-669. doi: 10.1016/j.clinthera.2024.06.023. Epub 2024 Jul 26.
5. Real-World Effectiveness and Safety of a Single-Pill Combination of Olmesartan/Amlodipine/Hydrochlorothiazide in Korean Patients with Hypertension and Cardiovascular Risk Factors. *Adv Ther.* 2023 Nov;40(11):4817-4835. doi: 10.1007/s12325-023-02632-9. Epub 2023 Aug 31.

Awards and Honors

2018, 2019, 2020	Marquis Who's Who 2018 & 2019 & 2020 Selected
2017	SICEM Best Presentation Award
2015	SICEM Best Oral Presentation Award

Research Interest

Dyslipidemia, Obesity, Diabetes

The role of carotid ultrasonography for the primary prevention of cardiovascular disease

Subclinical carotid plaque and intima media thickness (IMT) are established indicators of early-stage atherosclerosis. They can be easily, inexpensively, and noninvasively assessed by ultrasound imaging. Both of them, carotid plaque in particular, show additive value beyond traditional risk factors in predicting CVD risk. In addition, traditional CVD risk factors could only explain a minor proportion (19.5%) of the carotid plaque burden. It is thus speculated that multiple non-traditional risk factors could promote subclinical carotid

atherosclerosis, and further increase the CVD risk. Risk assessment based on traditional risk factors could perform differently among those with and without subclinical carotid atherosclerosis. Therefore, where available, subclinical carotid atherosclerosis could be one important factor to consider to evaluate CVD risk. In this session, we will introduce the role of carotid ultrasonography for the primary prevention of cardiovascular disease.

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, Korea, M.D, Medicine
2011.08	Seoul National University, 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. Impact of three-month treatment with pitavastatin on arterial stiffness in patients with hypercholesterolemia: a prospective observational study. *Hellenic J Cardiol* 2024 Oct 18:S1109-9666(24)00214-8.
2. The Associations of Arterial Stiffness and Central Hemodynamics with Carotid Atherosclerosis in Patients at a High Coronary Risk: A Cross-Sectional Study. *Pulse (Basel)*. 2024 Dec 5;13(1):11–21.
3. The association between arterial stiffness and socioeconomic status: a cross-sectional study using estimated pulse wave velocity. *Clin Hypertens* 2024 Oct 1;30(1):26.
4. Arterial Stiffness and Heart Failure With Preserved Ejection Fraction. *J Korean Med Sci* 2024 Jun 17;39(23):e195.
5. Prognostic value of combining atherogenic index of plasma and brachial-ankle pulse wave velocity for major adverse cardiovascular events in subjects without cardiovascular disease. *Cardiometab Syndr J*. 2024 Sep;4(2):95–103.

The usefulness of brachial-ankle PWV in the risk prediction for Asians

Brachial-ankle pulse wave velocity (baPWV) is a widely used and convenient measure of arterial stiffness that has garnered increasing attention as a predictor of cardiovascular risk, particularly in Asian populations. Unlike carotid-femoral PWV, which is considered the gold standard but requires specialized equipment and technical expertise, baPWV can be measured noninvasively and reproducibly in outpatient settings, making it a practical tool for large-scale population screening and clinical practice. Recent cohort studies in East Asian populations have demonstrated that elevated baPWV is independently associated with a higher risk of major adverse cardiovascular events (MACE), including myocardial infarction, stroke, and cardiovascular mortality. Furthermore, baPWV has shown incremental predictive value when added to traditional risk models, such as the Framingham Risk Score or ACC/AHA 10-year risk score, enhancing the identification of high-risk individuals who may

benefit from early intervention. Importantly, the prognostic significance of baPWV appears to be particularly relevant in Asian populations, who often present with different cardiovascular risk profiles compared to Western populations, such as a higher prevalence of stroke and greater sensitivity to blood pressure-related vascular damage. In this context, baPWV serves not only as a surrogate marker of vascular aging but also as a culturally and regionally appropriate risk stratification tool. This lecture will review the pathophysiological basis of baPWV, summarize key evidence from recent Asian epidemiological studies, and discuss its potential integration into routine clinical care and public health strategies. Practical issues such as cut-off values, reproducibility, and interpretation across sex and age groups will also be addressed. Overall, baPWV represents a promising, noninvasive biomarker that could support personalized cardiovascular prevention strategies, particularly in Asian contexts.

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Satellite Symposia



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Breakfast Symposium 1

Sep 12(Fri) 07:30-08:00 | Room 1 (3F)

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

07:30-07:50 **The cardio-renal-metabolic vicious cycle: choosing the suitable statin for complex risk profiles**

Youngwoo Jang (Gachon University, Republic of Korea)

07:50-08:00 **Panel Discussion**

So Hee Kwon (Soonchunhyang University, Republic of Korea)

Woohyeun Kim (Hanyang University, Republic of Korea)



CURRICULUM VITAE

Youngwoo Jang

Gachon University, Republic of Korea



Education & Career

2008–2012	School of Medicine, Gachon University, Doctor of Medicine
2017–2018	Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
2018–2019	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Fellow
2019–2021	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
2022–	Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

Research Interests

Cardiovascular intervention, Atherosclerosis, Acute myocardial infarction, Angina and heart failure, Pulmonary hypertension, Atrial fibrillation

Important Publications

1. Jang Y, Park SD, Lee JP, et al. One-month dual antiplatelet therapy followed by prasugrel monotherapy at a reduced dose: the 4D-ACS randomised trial. *EuroIntervention* 2025.
2. Kang SH, Pack KY, Kim JH, Jang Y (corresponding author). The effect of sarpogrelate compared to aspirin in high- or very-high-risk diabetes for primary prevention. *Sci Rep.* 2025 Jan 29;15(1):3616. doi: 10.1038/s41598-025-87868-x.
3. Kim S, Jang Y, Inflammation in Atherosclerotic Cardiovascular Diseases: Biomarkers to Therapeutics in Clinical Settings. *J Cardiovasc Interv.* 2024 Oct;3(4):199–215.
4. Jang Y, Han SH, Sohn IS, Oh PC, Koh KK. Lipoprotein(a) and Cardiovascular Diseases — Revisited —. *Circ J* 2020; 84: 867 – 874 doi: 10.1253/circj.CJ-20-0051.
5. Jang Y, Kim M; Oh, PC, Suh SY, Lee K, Kang WC, and Han SH, Long-term Clinical Outcomes and Its Predictors Between 1- and 2-stent Strategy in Coronary Bifurcation Lesions: A Baseline Clinical and Lesion Characteristic Matched Analysis, *Circ J.* 2022 Aug 25;86(9):1365–1375. doi: 10.1253/circj.CJ-22-0163.2.
6. Jang Y, Lee HH, Lee H, Kim HC and Chung WJ. Epidemiology of PAH in Korea: An Analysis of the National Health Insurance Data, 2002–2018. *Korean Circ J.* 2023;53:313–327.
7. Jang Y, Kim B-G, Kwon S, Seo J, Kim HK, Chang H-J, et al. (2020) Prevalence and clinical features of bone morphogenetic protein receptor type 2 mutation in Korean idiopathic pulmonary arterial hypertension patients: The PILGRIM explorative cohort. *PLoS ONE* 15(9): e0238698. Sept. 2020. <https://doi.org/10.1371/journal.pone.0238698>.
8. Jang Y, Scherer PE, Kim JY, Lim S, and Koh KK. Adiponectin and cardiometabolic trait and mortality: where do we go? *Cardiovasc Res.* 2021 Jun 12;cvab199. doi: 10.1093/cvr/cvab199.
9. Jang Y, Yu JW, and Kang WC et al. Real-World Long-Term Clinical Outcomes of Ultrathin Strut Biodegradable Polymer Drug-Eluting Stents in Korean ST-Segment-Elevation Myocardial Infarction (STEMI) Patients with or without Acute Heart Failure Undergoing Primary Percutaneous Coronary Intervention. *J Clin. Med.* 2021, 10(24), 5898.
10. Oh S, Jang Y (co-first author), Chae S, Hwang D, Byun K, and Chung WJ. Comparative Analysis on the Anti-inflammatory/immune Effect of Mesenchymal Stem Cell Therapy for the Treatment of Pulmonary Arterial Hypertension. *Sci Rep.* 2021 Jan 21;11(1):2012. PMID: 33479312 PMCID: PMC7820276 DOI: 10.1038/s41598-021-81244-1.

**The cardio-renal-metabolic vicious cycle:
choosing the suitable statin for complex risk profiles**

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 2

Sep 12(Fri) 07:30-08:00 | Room 2 (3F)

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

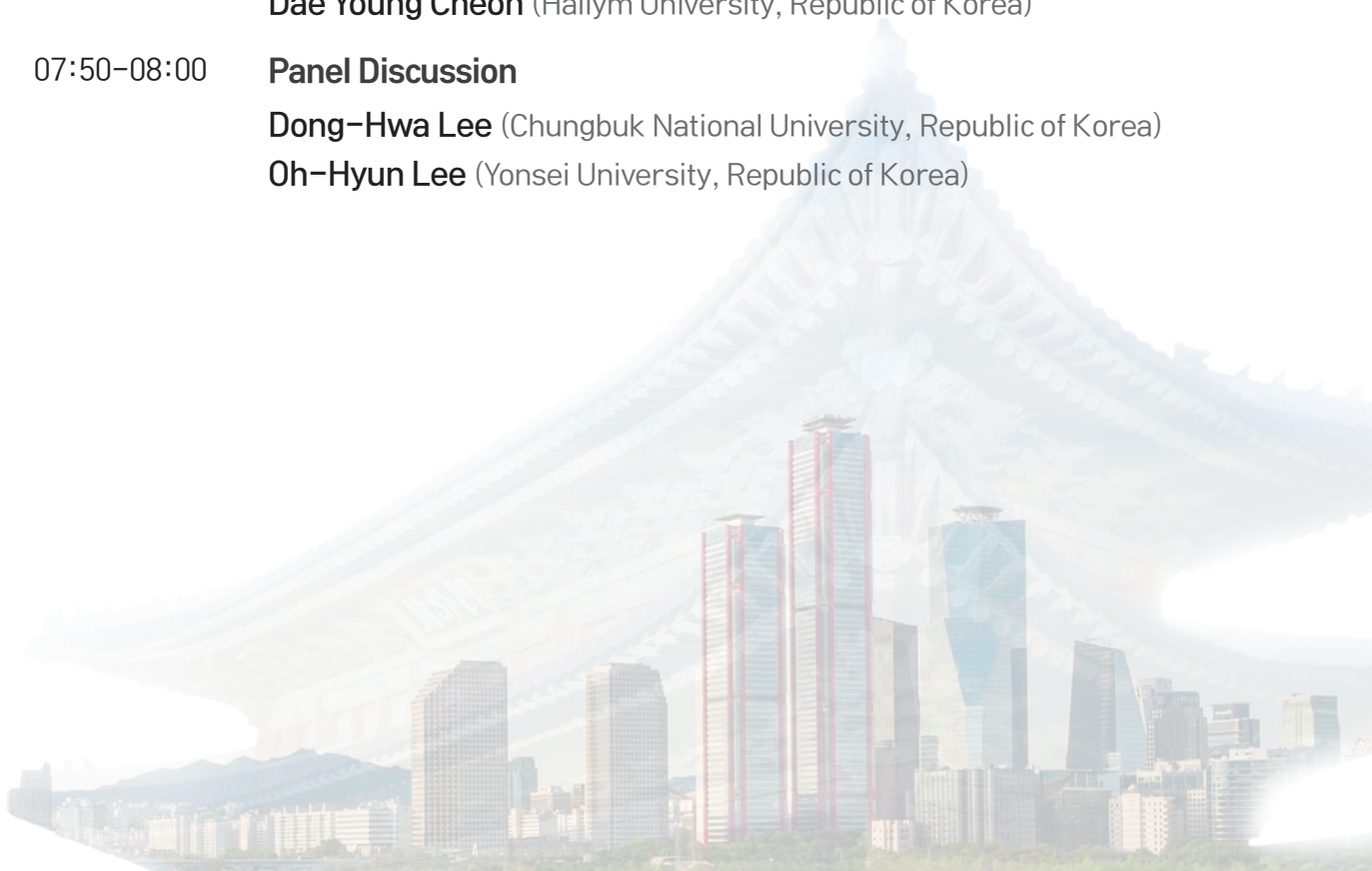
07:30-07:50 **Lowest is the best: reduction of LDL-C level and CV risk in ASCVD patients**

Dae Young Cheon (Hallym University, Republic of Korea)

07:50-08:00 **Panel Discussion**

Dong-Hwa Lee (Chungbuk National University, Republic of Korea)

Oh-Hyun Lee (Yonsei University, Republic of Korea)



CURRICULUM VITAE

Dae Young Cheon

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



Education and Training

2012.02	Hallym University, Korea, M.D, Medicine
2005.08	Korea National Open University, Korea, M.S, Bioinformatics & Statistics

Employment and Position

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

Important Publications

1. Association between Metabolic Syndrome and Young-Onset Dementia: A Nationwide Population-Based Study. Neurology, 2025.4.
2. Diabetes status, duration, and risk of dementia among ischemic stroke patients, Alzheimer's Research & Therapy, 2025.3.
3. Korea Hypertension Fact Sheet 2024: nationwide population-based analysis with a focus on young adults. Clinical Hypertension, 2025.01.
4. Depression and Risk of Stroke and Mortality after Percutaneous Coronary Intervention: A Nationwide Population Study, Journal of Internal Medicine, 2024.09.
5. Associations between migraine and major cardiovascular events in type 2 diabetes mellitus. Cardiovasc Diabetol, 2022.12.

Awards and Honors

2025	Asia-Pacific Cardiometabolic Syndrome Congress (APCMS), Distinguished Young Investigator Award
2024	Korean Society of Hypertension, Young Investigator Award

Lowest is the best: reduction of LDL-C level and CV risk in ASCVD patients

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 3

Sep 12(Fri) 07:30-08:00 | Room 3 (3F)

CHAIRPERSON : Do-Sun Lim (Korea University, Republic of Korea)

07:30-07:50 **Upfront lipid-lowering combination therapy in very high-risk patients: not a choice, a necessity**

Kang-Un Choi (Yeungnam University, Republic of Korea)

07:50-08:00 **Panel Discussion**

Keehwan Lee (University of Ulsan, Republic of Korea)

A Ram Hong (Chonnam National University, Republic of Korea)



CURRICULUM VITAE**Kang-Un Choi**

Professor, Yeungnam University, Republic of Korea

**Education and Training**

2011.02	Yeungnam University, Korea, M.D, Medicine
2019.08	Yeungnam University, Korea, Ph.D., Internal Medicine

Employment and Position

2018-2019	Yonsei University Severance Hospital, Clinical Research Assistant Professor
2019-2021	Dongguk University, Assistant Professor
2021-	Yeungnam University, Assistant Professor

Research Interest

Cardiac Critical care
Advanced Heart failure

**Upfront lipid-lowering combination therapy in
very high-risk patients: not a choice, a necessity**

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

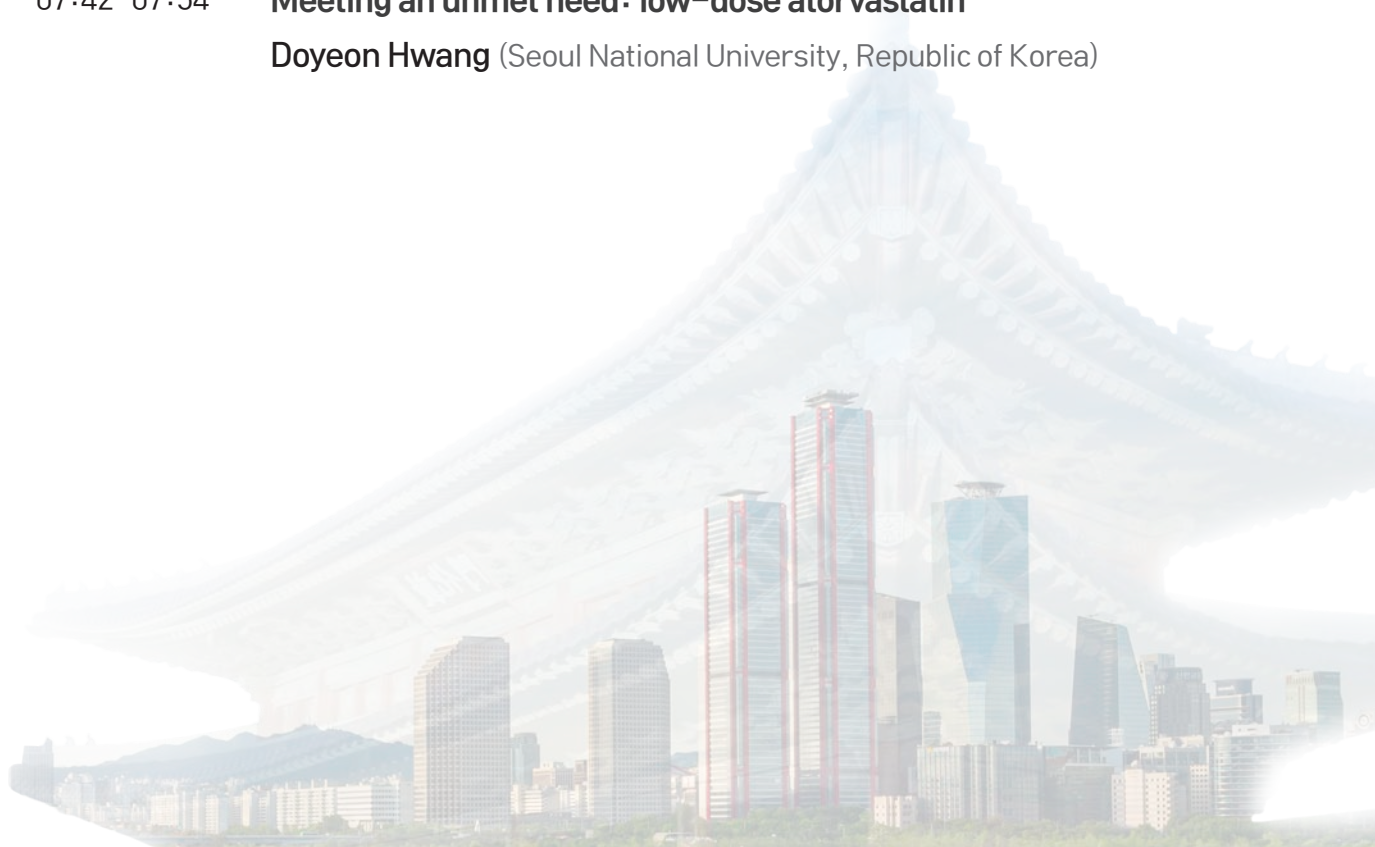
Breakfast Symposium 4

Sep 12(Fri) 07:30-07:54 | Room 4 (5F)

CHAIRPERSON : Myung A Kim (Seoul National University, Republic of Korea)

07:30-07:42 **New concepts of dyslipidemia medication: the lower, the smaller**
Wonjin Kim (CHA University, Republic of Korea)

07:42-07:54 **Meeting an unmet need: low-dose atorvastatin**
Doyeon Hwang (Seoul National University, Republic of Korea)



CURRICULUM VITAE**Wonjin Kim**

Associate Professor, CHA University, Republic of Korea

**Education and Training**

2008.02	Wonju College of Medicine, Yonsei University, M.D, Medicine
2023.02	Yonsei University, Korea, Ph.D., Internal Medicine

Employment and Position

2009-2013	Severance Hospital, Resident
2015-2025	CHA Gangnam Medical Center, Assistant Professor
2025-Current	CHA Gangnam Medical Center, Associate Professor

Important Publications

1. W Kim, SK Park, YL Kim. Fetal Abdominal Overgrowth is Already Present at 20-24 Gestational Weeks Prior to Diagnosis of Gestational Diabetes Mellitus. Scientific Reports. 2021, 11:23821.
2. JH Jang, W Kim, JS Moon, E Roh, JG Kang, SJ Lee, SH I, JH Huh. Association between Sleep Duration and Incident Diabetes Mellitus in Healthy Subjects: A 14-Year Longitudinal Cohort Study. Journal of Clinical Medicine. 2023, 12, 2899.
3. HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, KH Chun, SH Han, KK Koh. Metabolic Syndrome Fact Sheet 2024: Executive Report. Cardiometab Syndr J. 2024;4:e14.
4. KH Chun, HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, SH Han, KK Koh. Sex-Specific Impact of the COVID-19 Outbreak on the Incidence of Metabolic Syndrome: A Comparative Study of 2018-2019 and 2020-2021. Korean J Intern Med 2025 Mar;40(2):262-274. doi: 10.3904/kjim.2024.288. Epub 2025 Mar 1.
5. W Kim, MK Seo, YJ Kim, SH Choi, CR Ku, S Kim, EJ Lee, JS Yoon. Role of the suppressor of cytokine signaling-3 in the pathogenesis of Graves' orbitopathy. Front. Endocrinol., 04 March 2025. <https://doi.org/10.3389/fendo.2025.1527275>.

New concepts of dyslipidemia medication: the lower, the smaller

CURRICULUM VITAE

Doyeon Hwang

Assistant Professor, Seoul National University Hospital, Republic of Korea



Education and Training

Mar. 2005-Feb.2012 Korea University College of Medicine, Korea, M.D, Medicine
 Mar. 2015-Feb.2017 Seoul National University, Seoul, Master of Science, Clinical Medical Sciences
 Mar. 2020- Seoul National University, Seoul, PhD., Internal Medicine

Employment and Position

Mar. 2012-Feb. 2013 Seoul National University Hospital, Internship
 Mar. 2013-Feb. 2017 Seoul National University Hospital, Residency in Internal Medicine
 Apr. 2017-Apr. 2020 Republic of Korea Army, Army Doctor, Captain
 May 2020-Feb. 2022 Seoul National University Hospital, Clinical Fellow
 Mar. 2022- Seoul National University Hospital, Assistant Professor

Important Publications

1. Doyeon Hwang MD^{1†}, Haneol J Kim BS^{2†}, Seung-Pyo Lee MD PhD^{1†}, Seonhee Lim PhD^{2†}, Bon-Kwon Koo MD PhD¹, Yong-Jin Kim MD PhD¹, Woong Kook PhD², Daniele Andreini MD PhD³, Mouaz H. Al-Mallah MD⁴, Matthew J. Budoff MD⁵, Filippo Cademartiri MD PhD⁶, Kavitha Chinnaiyan MD⁷, Jung Hyun Choi MD PhD⁸, Edoardo Conte MD³, Hugo Marques MD PhD⁹, Pedro de Araújo Gonçalves MD PhD⁹, Ilan Gottlieb MD PhD¹⁰, Martin Hadamitzky MD¹¹, Jonathon A. Leipsic MD¹², Erica Maffei MD¹³, Gianluca Pontone MD PhD³, Gilbert L. Raff MD⁷, Sanghoon Shin MD¹⁴, Byoung Kwon Lee MD PhD¹⁵, Eun Ju Chun MD PhD¹⁶, Ji Min Sung PhD^{17,18}, Sang-Eun Lee MD PhD^{14,18}, Daniel S. Berman MD¹⁹, Fay Y Lin MD²⁰, Renu Virmani MD²¹, Habib Samady MD²², Peter H. Stone MD²³, Jagat Narula MD PhD²⁴, Jeroen J. Bax MD PhD²⁵, Leslee J. Shaw PhD²⁰, James K. Min MD²⁰, Hyuk-Jae Chang MD PhD^{17,18}. Topological Data Analysis of Coronary Plaques Demonstrates the Natural History of Coronary Atherosclerosis. JACC Cardiovasc Imaging. 2021 Jul;14(7):1410-1421.
2. Doyeon Hwang, MD^{1*}, Young-Hyo Lim, MD^{2*}, Kyung Woo Park, MD¹, Kook Jin Chun, MD³, Jung-Kyu Han, MD¹, Han-Mo Yang, MD¹, Hyun-Jae Kang, MD¹, Bon-Kwon Koo, MD¹, Jeehoon Kang, MD¹, Yun-Kyeong Cho, MD⁴, Soon Jun Hong, MD⁵, Sanghyun Kim, MD⁶, Sang-Ho Jo, MD⁷, Yong Hoon Kim, MD⁸, Won Kim, MD⁹, Sung Yun Lee, MD¹⁰, Young Dae Kim, MD¹¹, Seok Kyu Oh, MD¹², Jung Hee Lee, MD¹³, Hyo-Soo Kim, MD¹, on behalf of the HOST-RP-ACS investigators, Prasugrel Dose De-escalation Therapy After Complex Percutaneous Coronary Intervention in Acute Coronary Syndrome: A Post Hoc Analysis from the HOST-REDUCE-POLYTECH-ACS trial. JAMA Cardiol. 2022 Mar 9:e220052. doi: 10.1001/jamacardio.2022.0052.
3. Jung-Kyu Han, Doyeon Hwang, Seokhun Yang, Sang-Hyeon Park, Jeehoon Kang, Han-Mo Yang, Kyung Woo Park, Hyun-Jae Kang, Bon-Kwon Koo, Seung-Ho Hur, Weon Kim, Seok Yeon Kim, Sang-Hyun Park, Seung Hwan Han, Sang-Hyun Kim, Sanghoon Shin, Yong Hoon Kim, Kyungil Park, Namho Lee, Seung Jin Lee, Jin Won Kim, Hyo-Soo Kim, Comparison of 3- to 6-Month Versus 12-Month Dual Antiplatelet Therapy After Coronary Intervention Using the Contemporary Drug-Eluting Stents With Ultrathin Struts: The HOST-IDEA Randomized Clinical Trial. Circulation. 2023 May 2;147(18):1358-1368.

Meeting an unmet need: low-dose atorvastatin

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 5

Sep 13(Sat) 07:50–08:20 | Room 1 (3F)

CHAIRPERSON : Jeong Euy Park (Drs Park & Kim Heart and Lung International Clinic, Republic of Korea)

07:50–08:10 **Benefits of atorvastatin / ezetimibe combination in diabetic dyslipidemia**

Yun Kyung Cho (University of Ulsan, Republic of Korea)

08:10–08:20 **Panel Discussion**

In Tae Moon (Eulji University, Republic of Korea)

Eunshil Hong (National Medical Center, 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. Efficacy and safety of pioglitazone, empagliflozin and glimepiride as third-line agents in patients with type 2 diabetes inadequately controlled with metformin and DPP-4 inhibitors: A multicentre, phase 4 randomized controlled trial. Cho YK, Cho JH, Hong SM, Park JH, Lee BW, Yoo JH, Kim JH, Chun SW, Hwang YC, Song KH, Lee WJ. Diabetes Obes Metab. 2025 Aug 14. doi: 10.1111/dom.70030. Online ahead of print.
2. Efficacy and safety of combination therapy using SGLT2 and DPP4 inhibitors to treat type 2 diabetes: An up-dated systematic review and meta-analysis with focus on an Asian subpopulation. Kim MJ, Cho YK, Kim S, Moon JY, Jung CH, Lee WJ. Diabetes Obes Metab. 2025 Sep;27(9):5019-5031. doi: 10.1111/dom.16550. Epub 2025 Jun 24.
3. 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. 2025 Jul 22. doi: 10.4093/dmj.2024.0693. Online ahead of print.

Benefits of atorvastatin / ezetimibe combination in diabetic dyslipidemia

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The 14th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 6

Sep 13(Sat) 07:50–08:35 | Room 2 (3F)

CHAIRPERSONS : Soon Jun Hong (Korea University, Republic of Korea)
Ade Meidian Ambari (Universitas Indonesia, Indonesia)

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- | | |
|-------------|---|
| 07:50–08:05 | Minimizing LDL–C variability via sustained, long–term efficacy
Youngwoo Jang (Gachon University, Republic of Korea) |
| 08:05–08:20 | The lower the better: Rosuvastatin’s next option for dyslipidemia management
Jina Choi (Seoul National University, Republic of Korea) |
| 08:20–08:25 | Panel Discussion
Bambang Widyanoro (Universitas Indonesia, Indonesia) |
| 08:25–08:35 | MOU Ceremony – The Indonesian Heart Association (IHA) & The Korean Society of Lipid and Atherosclerosis (KSoLA) |



CURRICULUM VITAE

Youngwoo Jang

Gachon University, Republic of Korea



Education & Career

2008-2012	School of Medicine, Gachon University, Doctor of Medicine
2017-2018	Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
2018-2019	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Fellow
2019-2021	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
2022-	Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

Research Interests

Cardiovascular intervention, Atherosclerosis, Acute myocardial infarction, Angina and heart failure, Pulmonary hypertension, Atrial fibrillation

Important Publications

1. Jang Y, Park SD, Lee JP, et al. One-month dual antiplatelet therapy followed by prasugrel monotherapy at a reduced dose: the 4D-ACS randomised trial. *EuroIntervention* 2025.
2. Kang SH, Pack KY, Kim JH, Jang Y (corresponding author). The effect of sarpogrelate compared to aspirin in high- or very-high-risk diabetes for primary prevention. *Sci Rep.* 2025 Jan 29;15(1):3616. doi: 10.1038/s41598-025-87868-x.
3. Kim S, Jang Y, Inflammation in Atherosclerotic Cardiovascular Diseases: Biomarkers to Therapeutics in Clinical Settings. *J Cardiovasc Interv.* 2024 Oct;3(4):199-215.
4. Jang Y, Han SH, Sohn IS, Oh PC, Koh KK. Lipoprotein(a) and Cardiovascular Diseases — Revisited —. *Circ J* 2020; 84: 867 – 874 doi: 10.1253/circj.CJ-20-0051.
5. Jang Y, Kim M; Oh, PC, Suh SY, Lee K, Kang WC, and Han SH, Long-term Clinical Outcomes and Its Predictors Between 1- and 2-stent Strategy in Coronary Bifurcation Lesions: A Baseline Clinical and Lesion Characteristic Matched Analysis, *Circ J.* 2022 Aug 25;86(9):1365-1375. doi: 10.1253/circj.CJ-22-0163.2.
6. Jang Y, Lee HH, Lee H, Kim HC and Chung WJ. Epidemiology of PAH in Korea: An Analysis of the National Health Insurance Data, 2002-2018. *Korean Circ J.* 2023;53:313-327.
7. Jang Y, Kim B-G, Kwon S, Seo J, Kim HK, Chang H-J, et al. (2020) Prevalence and clinical features of bone morphogenetic protein receptor type 2 mutation in Korean idiopathic pulmonary arterial hypertension patients: The PILGRIM explorative cohort. *PLoS ONE* 15(9): e0238698. Sept. 2020. <https://doi.org/10.1371/journal.pone.0238698>.
8. Jang Y, Scherer PE, Kim JY, Lim S, and Koh KK. Adiponectin and cardiometabolic trait and mortality: where do we go? *Cardiovasc Res.* 2021 Jun 12;cvab199. doi: 10.1093/cvr/cvab199.
9. Jang Y, Yu JW, and Kang WC et al. Real-World Long-Term Clinical Outcomes of Ultrathin Strut Biodegradable Polymer Drug-Eluting Stents in Korean ST-Segment-Elevation Myocardial Infarction (STEMI) Patients with or without Acute Heart Failure Undergoing Primary Percutaneous Coronary Intervention. *J Clin. Med.* 2021, 10(24), 5898.
10. Oh S, Jang Y (co-first author), Chae S, Hwang D, Byun K, and Chung WJ. Comparative Analysis on the Anti-inflammatory/immune Effect of Mesenchymal Stem Cell Therapy for the Treatment of Pulmonary Arterial Hypertension. *Sci Rep.* 2021 Jan 21;11(1):2012. PMID: 33479312 PMCID: PMC7820276 DOI: 10.1038/s41598-021-81244-1.

Minimizing LDL-C variability via sustained, long-term efficacy

CURRICULUM VITAE

Jina Choi

Seoul National University, Republic of Korea



Education

2008-2015	B.S., Food and Nutrition, Yonsei University, Seoul, Korea
2015-2019	M.D., Medicine, CHA University School of Medicine, Korea, Graduated with second class honor
2021-Present	Ph.D., Medicine, Seoul National University College of Medicine, Korea

Academic Honors and Awards

2011	6 th place, 2011 Valley Forensics League #4, Informative speaking, USA
2011	2 nd place, JV Nationals Speech Tournament 2011, Informative speaking, USA
2012	Highest Academic Honors, Yonsei University
2012-2014	Academic Scholarship, Yonsei University
2015-2017	Academic Scholarship, CHA University, School of Medicine
2015	Best Presentation Award, Invited Young Scientists English Oral Presentation, The Korean Nutrition Society International Conference
2016	Young Investigator Award, The Korean Society of Cancer Prevention Osaek Symposium
2017	Finalist, The American Society for Nutrition's Emerging Leaders in Nutrition Poster Competition, USA
2019	President's Award, CHA University (Graduation with second class honor)
2021	Best Presentation Award, Western Pacific Regional Conference 2021 of the MWIA
2022	Outstanding Resident Award, Department of Internal Medicine, Seoul National University Bundang Hospital
2023	Outstanding Participant Award, MD-PhD/Medical Scientist Training Program, Korea Health Industry Development Institute (KHIDI) and the Ministry of Health & Welfare
2023	Best Abstract Award, KHRS Annual Scientific Session 2023
2023	Best Case Presentation Award, The 67 th Annual Scientific Meeting of the KSC
2024	Boryung Fellowship Academic Award 2024, Korean Medical Women's Association
2025	Excellence in Fellowship Abstract Award, KHRS Annual Scientific Session 2025

Research Experience

2011-2013	Research Assistant, Laboratory of Functional Food, Yonsei University
2014-2019	Research Assistant, Laboratory of Healthy Aging, CHA University
2018.01-02	Won lab, Institute for Diabetes, Obesity and Metabolism, Perelman School of Medicine, University of Pennsylvania, USA
2020-2022	Completion of MD-PhD/Medical Scientist Training Program supported by the Korea Health Industry Development Institute (KHIDI) and the Ministry of Health & Welfare, Republic of Korea

The lower the better: Rosuvastatin's next option for dyslipidemia management

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

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The 14th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 7

Sep 13(Sat) 07:50–08:20 | Room 3 (3F)

CHAIRPERSON : Kyong Soo Park (Konkuk University, Republic of Korea)

07:50–08:10 **Safety and efficacy of moderate–intensity statin with ezetimibe in elderly patient with ASCVD**

Jung–Joon Cha (Korea University, Republic of Korea)

08:10–08:20 **Panel Discussion**

Shin–young Woo (Konyang University, Republic of Korea)

Kyu–Sun Lee (Eulji University, Republic of Korea)



CURRICULUM VITAE**Jung-Joon Cha**

Clinical Associate Professor, Korea University Anam Hospital, Republic of Korea

**Education and Training**

2009.02	Yonsei University Wonju College of Medicine, Korea, M.D, Medicine
2018.02	Gwangju Institute of Science and Technology, Korea, Ph.D., Biomedical Science & Engineering

Employment and Position

2020-2024	Korea University Anam Hospital, Korea Univ. College of Medicine, Clinical Assistant Professor
2024-Present	Korea University Anam Hospital, Korea Univ. College of Medicine, Clinical Associate Professor

Important Publications

1. Cha JJ, Bae S, 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. Cha JJ, Kim JY, Kim H, et al. Long-term Clinical Outcomes and Prognostic Factors After Endovascular Treatment in Patients With Chronic Limb Threatening Ischemia. *Korean Circ J*. 2022;52:429-440.
3. Cha JJ, Hong SJ, Lim S, et al. The Use of Coronary Imaging for Predicting Future Cardiovascular Events. *Pulse (Basel)*. 2024;12:34-39.
4. Cha JJ, Hong SJ, Kim JH, et al. Effect of rosuvastatin 20 mg versus rosuvastatin 5 mg plus ezetimibe on statin side-effects in elderly patients with ASCVD: SaveSAMS trial. *Am Heart J*. 2023;261:45-50.
5. Lee SJ, Cha JJ, et al. Moderate-Intensity Statin With Ezetimibe vs High-Intensity Statin Monotherapy in Very High-Risk ASCVD Patients: Post Hoc RACING Trial. *JAMA Cardiol*. 2023. doi:10.1001/jamacardio.2023.2222.

Awards and Honors

- Winner of Young Investigator Competition - Korean Society of Cardiology (KSC), 2018
- Best Abstract Award - KOVAS 2024
- Best Presenter - Korean Society of Interventional Cardiology (KSIC), 2025

Research Interest

- Clinical outcomes and risk stratification in coronary artery disease and acute myocardial infarction
- Precision medicine in interventional cardiology, including pharmacogenomics-guided therapy
- Advanced coronary imaging (IVUS/OCT) and machine learning-based diagnostic tools
- Endovascular therapies in peripheral artery disease
- Translational research in endothelial dysfunction, vascular aging, and atherosclerosis

**Safety and efficacy of moderate-intensity statin
with ezetimibe in elderly patient with ASCVD**

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Breakfast Symposium 8

Sep 13(Sat) 07:50–08:26 | Room 4 (5F)

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

- 07:50–08:02 **New approaches to treating hypertriglyceridemia in patients with renal impairment**
Young–Sang Lyu (Chosun University, Republic of Korea)
- 08:02–08:14 **Profile of Pravastatin and its effects in transplant recipients**
Jae Yoon Park (Dongguk University, Republic of Korea)
- 08:14–08:26 **Comparative effectiveness of lercanidipine and amlodipine on MACE in hypertensive patients**
Hyung Joon Joo (Korea University, Republic of Korea)



CURRICULUM VITAE

Young-Sang Lyu

Associated Assistant 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
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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 approaches to treating hypertriglyceridemia
in patients with renal impairment

CURRICULUM VITAE

Jae Yoon Park

Division of Nephrology, Dongguk University Ilsan Hospital, Republic of Korea



Educational Background and Degrees

Mar 2000-Feb 2006	M.D., College of Medicine, Chung-Ang University, Seoul, Korea
Mar 2009-Feb 2011	M.S., Graduate School, Chung-Ang University, Seoul, Korea
Sep 2014-Feb 2021	Ph.D., Graduate School of Medicine, Seoul National University, Seoul, Korea

Training Background

Mar 2006-Feb 2007	Internship, Seoul National University Hospital
Mar 2007-Feb 2011	Residency in Internal Medicine, Seoul National University Hospital
May 2014-Feb 2016	Fellowship in Nephrology, Seoul National University Hospital

Professional Experience

Mar 2011-Feb 2012	Medical Officer, UN Command Security Battalion-Joint Security Area
Mar 2012-Apr 2014	Chief of Internal Medicine, Armed Forces Goyang Hospital
Mar 2016-Feb 2022	Assistant Professor, Division of Nephrology, Dongguk University College of Medicine
Mar 2022-Present	Associate Professor, Division of Nephrology, Dongguk University College of Medicine
Mar 2022-Present	Head, Division of Nephrology, Dongguk University Ilsan Hospital
Jan 2022-Present	Director, Research Institute of Chronic Disease and Environmental Medicine, Dongguk University College of Medicine
Mar 2025-Present	Director, Clinical Trial Center, Dongguk University Ilsan Hospital

Memberships

- Active Member, Korean Society of Nephrology
- Active Member, American Society of Nephrology
- Active Member, Korean Society of Organ Transplantation
- Active Member, The Transplantation Society

Licensure

Feb 2006	Medical Doctor, Ministry of Health and Welfare, South Korea (License No.: 88693)
Feb 2011	Board Certified in Internal Medicine, South Korea (License No.: 12699)

Profile of Pravastatin and its effects in transplant recipients

CURRICULUM VITAE

Hyung Joon Joo

Professor, Korea University College of Medicine,
Korea University Anam Hospital, Republic of Korea



Education and Training

2003	Korea University College of Medicine, Korea, M.D., Medicine
2011	KAIST, Korea, Ph.D., Medical Science

Employment and Position

2003-2008	Korea University Guro Hospital, Resident (Internal Medicine)
2011-2013	KAIST Natural Science Research Institute, Post-Doctoral Researcher
2013-Present	Korea University Anam Hospital, Division of Cardiology, Clinical Fellow~(Present) Professor
2021-Present	Korea University, College of Medicine, Department of Medical Informatics, Adjunct Professor
2019-Present	Korea University Medical Center, Information Management Office, Director
2021-2024	Tilder Inc., CEO

Important Publications

1. Comparative Effectiveness of Lercanidipine and Amlodipine on Major Adverse Cardiovascular Events in Hypertensive Patients. Am J Hypertens. 2024.
2. Cardiovascular outcomes in Parkinson's disease patients from a retrospective cohort study. Sci Rep. 2024.
3. Association of the protective effect of telmisartan on hearing loss among patients with hypertension. Front Neurol. 2024.
4. Three-year cardiovascular outcomes of telmisartan in patients with hypertension: an electronic health record-based cohort study. Am J Hypertens. 2024.
5. Impact of cholesterol variability as a potential residual cardiovascular risk factor in the elderly. CardioMetabolic Syndrome Journal. 2024.

Research Interest

Cardiovascular disease, Electronic Health Records (EHR), Personal Health Records (PHR), Deep Learning, Artificial Intelligence, Natural Language Processing.

Comparative effectiveness of lercanidipine and amlodipine on MACE in hypertensive patients

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 1 (K)

Sep 12(Fri) 12:00–12:30 | Room 1 (3F)

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

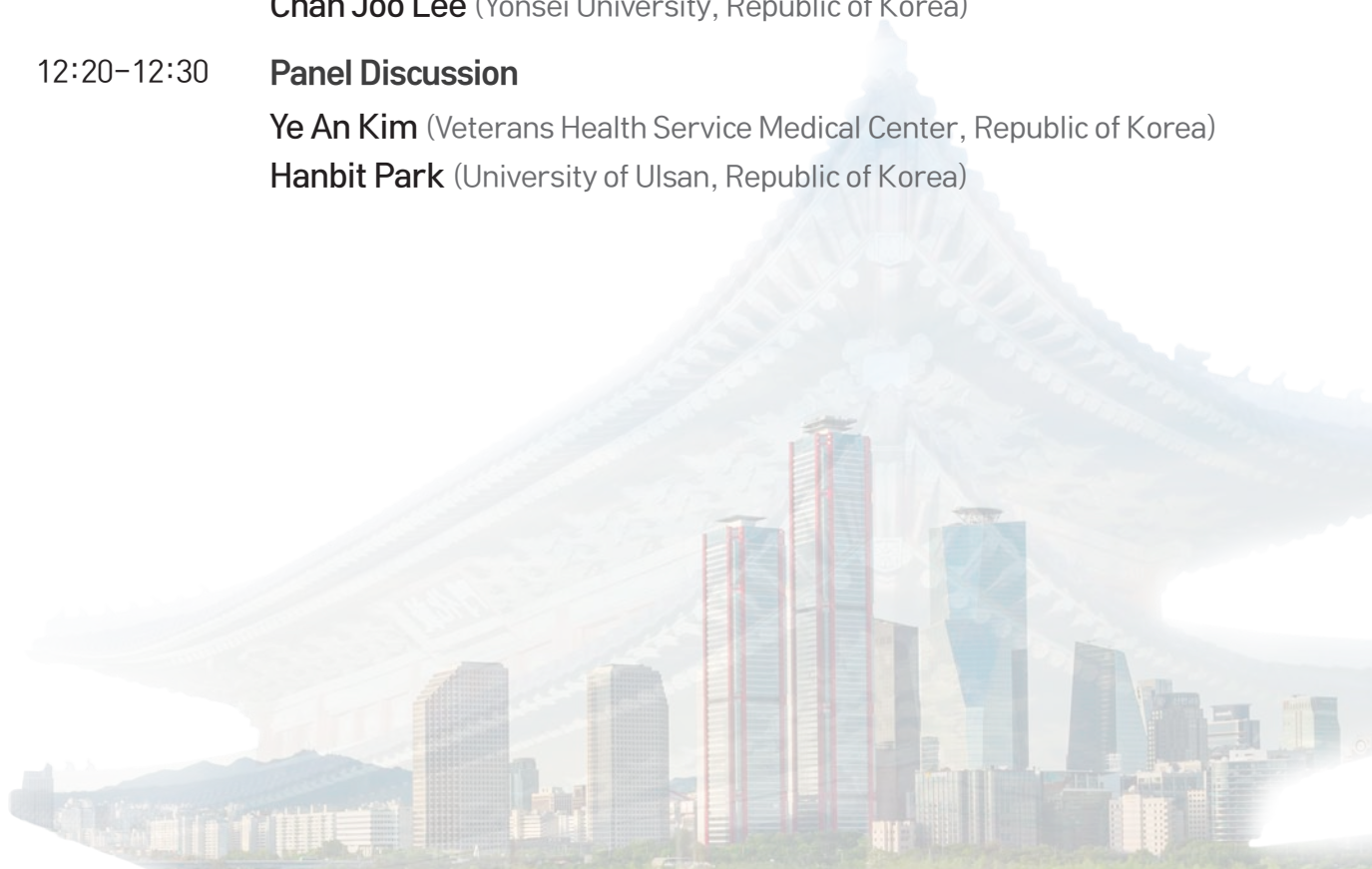
12:00–12:20 **Integrated management of hypertension and dyslipidemia for cardiovascular disease prevention**

Chan Joo Lee (Yonsei University, Republic of Korea)

12:20–12:30 **Panel Discussion**

Ye An Kim (Veterans Health Service Medical Center, Republic of Korea)

Hanbit Park (University of Ulsan, Republic of Korea)



CURRICULUM VITAE**Chan Joo Lee**

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

**Education and Training**

2005.02	Yonsei University, Korea, M.D, Medicine
2010.02	Yonsei University, Korea, M.S, Internal Medicine
2015.02	Yonsei University, Korea, Ph.D, Cell Biology and Pharmacology

Employment and Position

2005–2006	Severance Hospital, Intern
2006–2010	Severance Hospital, Resident (Internal Medicine)
2010–2015	Yonsei University, Teaching Assistant (Molecular Biology)
2015–2016	Severance Hospital, Fellow (Cardiology)
2017–2018	Severance Hospital, Clinical Assistant Professor (Health Promotion)
2019–2019	Severance Hospital, Clinical Assistant Professor (Cardiology)
2020–2024	Severance Hospital, Clinical Associate Professor (Cardiology)
2025–	Severance Hospital, Clinical Professor (Cardiology)

Important Publications

1. MicroRNA-1912 regulates cholesterol homeostasis by targeting PCSK9. *Mol Ther Nucleic Acids*. 2025 Jun 9;36(3):102589. (1st author)
2. Spironolactone vs Amiloride for Resistant Hypertension: A Randomized Clinical Trial. *JAMA*. 2025 Jun 17;333(23):2073–2082. (1st author)
3. Cardiovascular Risk and Treatment Outcomes in Severe Hypercholesterolemia: A Nationwide Cohort Study. *J Am Heart Assoc*. 2022 May 3;11(9):e024379. (1st author)
4. Blood Pressure Levels and Risks of Dementia: a Nationwide Study of 4.5 Million People. *Hypertension*. 2022 Jan;79(1):218–229. (1st author)
5. Deep-learning-based cardiovascular risk stratification using coronary artery calcium scores predicted from retinal photographs. *Lancet Digit Health*. 2021 May;3(5):e306–e316. (1st author)

Research Interest

Lipid metabolism; Heart failure; Basic research regarding PCSK9

**Integrated management of hypertension and
dyslipidemia for cardiovascular disease prevention**

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 2

Sep 12(Fri) 12:00–12:30 | Room 2 (3F)

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

12:00–12:20 **A new “standard” in dyslipidemia treatment**

Jeehoon Kang (Seoul National University, Republic of Korea)

12:20–12:30 **Panel Discussion**

Soo Kyoung Kim (Gyeongsang National University, Republic of Korea)

Subin Lim (Ewha Womans University, Republic of Korea)



CURRICULUM VITAE

Jeehoon Kang

Seoul National University, Republic of Korea



Education

1996–1999	Deajon Science High School, Daejeon, Korea
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

Awards and Recognitions

2012	Best Speaker at the 63th Korean Internal Medicine Conference
2013	Best Medical Officer, 2013
2016	Best Speaker at the 12th KSIC International Conference
2017	Best Speaker at the 13th KSIC International Conference
2019	Young investigator Award, TCTAP 2019
2021	Doosan Yonkang Academic award
2022	Hamchoon Academic award

License and Membership

2008	Korean Medical License
2013	Board Certification in Internal Medicine
2017	Board Certification in Cardiovascular Medicine
2020	Board Certification in Critical Care Medicine

Publications

1. Lee H, Kang J*. Nuances of pleural effusion after left ventricular assist devices implantation: insights from therapeutic drainage and preoperative predictors. *Acute Crit Care*. 2024 Feb;39(1):192–193
2. Yang S, Kang J*, Hwang D, Zhang J, Jiang J, Hu X, Hahn JY, Nam CW, Doh JH, Lee BK, Kim W, Huang J, Jiang F, Zhou H, Chen P, Tang L, Jiang W, Chen X, He W, Ahn SG, Yoon MH, Kim U, Lee JM, Ki YJ, Shin ES, Kim HS, Tahk SJ, Wang J, Koo BK. Physiology- or Imaging-Guided Strategies for Intermediate Coronary Stenosis. *JAMA Netw Open*. 2024 Jan 2;7(1):e2350036.

A new “standard” in dyslipidemia treatment

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 3

Sep 12(Fri) 12:00–12:30 | Room 3 (3F)

CHAIRPERSON : Chee Jeong Kim (Chung-Ang University, Republic of Korea)

12:00–12:20 **Recent trends and clinical evidence for optimizing lipid management**

Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)

12:20–12:30 **Panel Discussion**

Sang Ah Lee (Jeju National University, Republic of Korea)

Sungsoo Cho (Yonsei 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

ISHLT, International Travelling Scholarship Award (2018)
Asian Pacific Society of Hypertension Young Investigator Award (2016)
Korean Society of Hypertension, The Best Young Investigator Award (2013)

Research Interest

Heart Failure, Cardiac Amyloidosis, Cardio-Oncology
Heart Transplantation, Left Ventricular Assist Device (LVAD)
Immune Aging (Immunosenescence), Transplantation Immunology

Recent trends and clinical evidence for optimizing lipid management

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 4

Sep 12(Fri) 12:00–12:30 | Room 4 (5F)

CHAIRPERSON : Shung Chull Chae (Kyungpook National University, Republic of Korea)

12:00–12:20 **Closing the LDL–C gap in ASCVD: the role of siRNA therapy**

Jung Ho Heo (Kosin University, Republic of Korea)

12:20–12:30 **Panel Discussion**

Youngsook Kim (Soonchunhyang University, Republic of Korea)

Sungjoon Park (Seoul National University, Republic of Korea)



CURRICULUM VITAE

Jung Ho Heo

Professor, Kosin university Gospel Hospital, Republic of Korea



Education

1989-1995	Kyung-Pook National University of medicine Medical (License # 56412)
2000-2002	A master's degree of internal medicine at Kyung-Pook National University
2003-2011	A Ph.D degree at Kyung-Pook National University of medicine

Training

1995-1996	Internship training at Kyung-Pook National University hospital
1996-1999	Served as Military medical officer at Korean navy
1999-2003	Residency training at Internal medicine of Kyung-Pook University hospital
2003-2004	Fellowship training in Cardiology at Kyung-Pook national University
2004.04-2004.05	Short-term Training Program for Young Interventionist at Asan Medical Center (6 weeks) - Lenox Hill Heart & Vascular Institute, New York (2 weeks)
2010.10-2012.04	Research fellow in Thoraxcenter, Erasmus University Netherlands
2005.03-	Positions Held & Faculty Appointment
2007-2012	Instructor at cardiology of Department of Cardiology, Kosin University Hospital
2012-2018	Assistant Professor at Department of Cardiology, Kosin University Hospital
2018	Associate professor Department of Cardiology, Kosin University Hospital
	Professor at Department of Cardiology, Kosin University Hospital

Important Publications

1. Anatomical Snuffbox Versus Dorsum of the Hand for Optimal Access Site in Distal Radial Access: Insight From the KODRA Registry. Korean Circ J. 2025 Jul 21. doi: 10.4070/kcj.2025.0107.
2. Optical coherence tomography-guided versus angiography-guided percutaneous coronary intervention for patients with complex lesions (OCCUPI): an investigator-initiated, multicentre, randomised, open-label, superiority trial in South Korea. Lancet. 2024 Sep 14;404(10457):1029-1039. doi: 10.1016/S0140-6736(24)01454-5. Epub 2024 Sep 2.
3. Distal Radial Access for Coronary Procedures in a Large Prospective Multicenter Registry: The KODRA Trial. JACC Cardiovasc Interv. 2024 Feb 12;17(3):329-340. doi: 10.1016/j.jcin.2023.11.021.
4. Combination Moderate-Intensity Statin and Ezetimibe Therapy for Elderly Patients With Atherosclerosis. J Am Coll Cardiol. 2023 Apr 11;81(14):1339-1349. doi: 10.1016/j.jacc.2023.02.007.
5. Long-term efficacy and safety of moderate-intensity statin with ezetimibe combination therapy versus high-intensity statin monotherapy in patients with atherosclerotic cardiovascular disease (RACING): a randomised, open-label, non-inferiority trial. Lancet. 2022 Jul 30;400(10349):380-390. doi: 10.1016/S0140-6736(22)00916-3. Epub 2022 Jul 18.

Research Interest

Imaging of coronary atherosclerosis
OCT and IVUS in clinical practice
Drug effect of lipid lowering agents

Closing the LDL-C gap in ASCVD: the role of siRNA therapy

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 5 (K)

Sep 13(Sat) 12:20–12:50 | Room 1 (3F)

CHAIRPERSON : Kwang-Won Kim (Gachon University, Republic of Korea)

12:20–12:40 **Cutting edge care of pitavastatin with ezetimibe combination therapy**

Soo Lim (Seoul National University, Republic of Korea)

12:40–12:50 **Panel Discussion**

Ji Yoon Kim (Sungkyunkwan University, Republic of Korea)

Hyun Sung Joh (Seoul National University, Republic of Korea)



CURRICULUM VITAE**Soo Lim**

Professor, Seoul National University, Republic of Korea

**Education and Training**

1996.02 Seoul National University, Korea, M.D, Medicine
2005.08 Seoul National University Graduate School, Korea, Ph.D., Internal Medicine

Employment and Position

2005-Present Seoul National University Bundang Hospital, Professor

Important Publications

1. Lee YH, Lim S* (co-corresponding), Davies MJ*. Cardiometabolic and Renal Benefits of SGLT2 Inhibitors: Mechanisms and Clinical Implications. *Nature Reviews Endocrinology* [Accepted].
2. Lim S (first and corresponding author), Buranapin S, Bao X, Quiroga M, Park KH, Kang JH, Rinnov AR, Suwanagool A. Once-weekly semaglutide 2.4 mg in an Asian population with obesity, defined locally as BMI ≥ 25 kg/m² (STEP 11). *Lancet Diabetes Endocrinol* [Accepted].
3. Neeland IJ, Lim S* (corresponding author), Tchernof A, Gastaldelli A, Rangaswami J, Ndumele CE, Powell-Wiley TM, Després JP*. Metabolic syndrome. *Nature Reviews Disease Primers*. 2024 Oct 17;10(1):77. PMID: 39420195.
4. Ahmad E*, Lim S* (co-first author), Lamprey R, Webb DR, Davies MJ. Type 2 diabetes. *Lancet* 2022 Nov 19;400(10365):1803-1820. PMID: 36332637.
5. Kadowaki T*, Isendahl J, Khalid U, Lee SY, Nishida T, Ogawa W, Tobe K, Yamauchi T, Lim S* (co-corresponding author). Effect of once-weekly subcutaneous semaglutide in adults with overweight or obesity, with or without type 2 diabetes, in an East Asian population. *Lancet Diabetes Endocrinol* 2022 Mar;10(3):193-206. PMID: 35131037.

Awards and Honors

- The Moonsuk Academic Award from the Korean Society for the Study of Obesity, 2023
- The Bulgok Creative Research Award from Seoul National University Bundang Hospital, 2022, 2023

Research Interest

Diabetes mellitus, dyslipidemia, obesity, fatty liver, and metabolic syndrome

Cutting edge care of pitavastatin with ezetimibe combination therapy

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 6

Sep 13(Sat) 12:20–12:50 | Room 2 (3F)

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

12:20–12:40 **Clinical efficacy of ultra-low-dose triple combination therapy (Amlodipine/Losartan/Chlorthalidone) in hypertension management**

Kyu-Yong Ko (Inje University, Republic of Korea)

12:40–12:50 **Panel Discussion**

Yu Ji Kim (Jeonbuk National University, Republic of Korea)

Ki Hong Choi (Sungkyunkwan University, Republic of Korea)



CURRICULUM VITAE

Kyu-Yong Ko

Assistant Professor, Inje University, Republic of Korea



Education and Training

2009.02	Inha University, Korea, M.D, Medicine
2023.08	Inha University, Korea, M.S., Internal Medicine

Employment and Position

2017-2018	Inha University Hospital, Fellowship in Cardiology
2018-2020	Gwangmyeong Sungae Hospital, Clinical Director, Cardiology
2021-2022	Severance Hospital, Clinical Assistant Professor, Cardiology
2022-2023	Severance Hospital, Assistant Professor, Cardiology
2023-	Ilsan Paik Hospital, Assistant Professor, Cardiology

Important Publications

1. Ko KY, Cho I, et al. Identification of Distinct Subgroups in Moderately Severe Rheumatic Mitral Stenosis Using Data-Driven Phenotyping of Longitudinal Hemodynamic Progression. *J Am Heart Assoc.* 2022;11:e026375. <https://doi.org/10.1161/JAHA.121.026375>.
2. Ko KY, Jang JH, et al. Impact of right atrial enlargement on clinical outcome in patients with atrial fibrillation. *Front Cardiovasc Med.* 2022;9:989012. <https://doi.org/10.3389/fcvm.2022.989012>.
3. Uhm JS, Ko KY, et al. Effects of RF Catheter Ablation for PVCs from the RVOT on RV Function. *J Cardiovasc Electrophysiol.* <https://doi.org/10.1111/jce.15741>.
4. Ko KY, et al. Novel technique of sutureless pulmonic valve replacement for quadricuspid pulmonic valve with huge pulmonary artery aneurysm. *Eur Heart J.* 2022;43(33):3183.
5. Ko KY, et al. Optimizing Percutaneous Mitral Valvuloplasty for Rheumatic Mitral Stenosis - Clinical Significance of Changes in Mitral Valve Area -. *Circ J.* 2024. <https://doi.org/10.1253/circj.CJ-23-0552>.

Research Interest

My primary research interests lie in the management of hypertension and dyslipidemia, the clinical application of echocardiography, and diagnostic and therapeutic strategies in heart failure.

**Clinical efficacy of ultra-low-dose triple combination therapy
(Amlodipine/Losartan/Chlorthalidone) in hypertension management**

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Luncheon Symposium 7

Sep 13(Sat) 12:20–12:50 | Room 3 (3F)

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

12:20–12:40 **Reversing atherosclerosis – 20 years of data for CRESTOR**

Kausik Kumar Ray (Imperial Centre for Cardiovascular Disease Prevention, UK)

12:40–12:50 **Panel Discussion**

Seokhun Yang (Seoul National University, Republic of Korea)

Kyong Hye Joung (Chungnam National University, Republic of Korea)



CURRICULUM VITAE

Kausik Kumar Ray

Professor, Imperial Centre for Cardiovascular Disease Prevention, UK



Degrees and Qualifications Held

2023	FMEDSci
2015	FRCP (Ed)
2013	FAHA
2011	FRCP (Lon)
2008	FESC
2008	FACC
2007	MPhil (epidemiology), University of Cambridge
2004	MD, University of Sheffield
1994	MRCP, UK
1991	MBChB, University of Birmingham (Medical School)

Education

University	
1990	Elective, Anaesthetics
1988	Intercalated BSc, University of Birmingham
1985–1991	Medical School, University of Birmingham
School	
1985	'A' Levels • 3 Grade A • 1 Grade B 'S' Level • 1 Grade 1
1983	'O' Levels • 6 Grade A • 3 Grade B
1978–1985	Queen Mary's Grammar School, Walsall

Prizes and Distinctions

1990	Recommendation for Elective Report
1985	J.F. Crump Chemistry Prize
1984	Michael Bollen Chemistry Prize

Reversing atherosclerosis - 20 years of data for CRESTOR

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Oral Presentations



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Oral Presentation 1

Sep 11(Thu) 14:40–16:10 | Room 1 (3F)

CHAIRPERSONS : **Hyun Kook** (Chonnam National University, Republic of Korea)
Kyung Woo Park (Seoul National University, Republic of Korea)



OP1-1

ApoA1 and HDL selectively protect the heart during doxorubicin chemotherapy via hepatic SR-B1

Jeong-Ah Yoo^{1,2*}, Bernardo Trigatti^{1,2}

¹Department of Biochemistry and Biomedical Sciences, McMaster University, Hamilton, Ontario, Canada.

²Thrombosis and Atherosclerosis Research Institute, Hamilton Health Sciences and McMaster University, Hamilton, Ontario, Canada

Objectives: Doxorubicin (DOX) is a chemotherapy drug but can cause life-threatening cardiotoxicity. Our lab found that over-expression or injection of Apolipoprotein (Apo) A1, the major component of high-density lipoprotein (HDL), protects non-tumor bearing mice from DOX-induced cardiotoxicity. This protection depends on an HDL receptor, SR-B1, expressed in a variety of tissues including the liver and cardiomyocytes. Given the similar mechanisms by which DOX induces toxicity in tumor cells and cardiomyocytes, it is critical to test the specificity of any potential cardioprotective agent. We aimed to determine if ApoA1 or HDL-mediated protection against DOX-cytotoxicity was selective for the heart versus breast tumor cells, and to explore the requirement for SR-B1 in the liver versus cardiomyocytes.

Methods: In vitro, neonatal mouse cardiomyocytes and 4T1 breast cancer cells were treated with DOX±HDL or ApoA1, and apoptosis evaluated using TUNEL staining. In vivo, BALB/c mice bearing breast tumors received DOX±HDL or ApoA1 for 4 weeks. Tumor growth, cardiac apoptosis and cardiomyocyte atrophy were evaluated after treatment. To investigate the requirement for SR-B1 expression in hepatocytes versus cardiomyocytes, liver- and cardiomyocyte- specific SR-B1 knockout mice and control mice were treated for 5 weeks with DOX±ApoA1, and cardiotoxicity were assessed.

Results: In vitro, treatment with HDL but not ApoA1 protected cardiomyocytes against DOX-induced apoptosis and atrophy but did not protect 4T1 tumor cells against DOX-induced apoptosis. In vivo, treatment of tumor bearing BALB/c mice with either ApoA1 or HDL protected them against DOX-induced cardiotoxicity, without affecting tumor suppression. Liver-specific SR-B1 knockout abolished ApoA1 -mediated cardioprotection, while cardiomyocyte-specific SR-B1 knockout had no effect.

Conclusions: HDL-based therapies may reduce DOX-induced cardiotoxicity without impacting the effectiveness of DOX-mediated chemotherapy, and this selective cardioprotective effect appears to be dependent on hepatic expression of the HDL receptor, SR-B1.

Keywords: HDL, SR-B1, Doxorubicin, Chemotherapy, Cancer, Cardiotoxicity

OP1-2

Lipid profiling of plasma extracellular vesicles from adiponectin deficient mice reveals pro-oxidative and pro-inflammatory lipid signatures driving metabolic dysfunction

Sungji Cho^{1*}, Hyekyoung Sung¹, Khang Nguyen¹, Yubin Lei¹, Eddie Tam¹, Yihan Luo², Evgeniy V. Petrotchenko³, Kacey J. Prentice², Dylan Burger^{4,5}, Gary Sweeney¹

¹Department of Biology, York University, Canada, ²Department of Physiology, University of Toronto, Canada,

³Segal Cancer Proteomics Centre, Lady Davis Institute for Medical Research, Jewish General Hospital, McGill University, Canada,

⁴Kidney Research Centre, Inflammation and Chronic Disease Program, Ottawa Hospital Research Institute, Canada,

⁵Department of Cellular and Molecular Medicine and School of Pharmaceutical Sciences, University of Ottawa, Canada

Objectives: Extracellular vesicles (EV) are critical mediators of intercellular communications, carrying bioactive cargo that reflects and influences metabolic health. This study investigated the lipidomic profile of plasma EV from adiponectin-knockout (KO) versus WT mice to identify a lipid signature associated with metabolic disease. We hypothesized that adiponectin deficiency alters EV lipid composition, thereby modulating their bioactivity in recipient cells.

Methods: Plasma EV were isolated using size exclusion chromatography, followed by comprehensive characterizations via nanoparticle tracking analysis, Cryo-EM and Western blotting. Lipidomic profiling was via LC-MS/MS with multiple reaction monitoring (LC-MRM-MS). Functional assessments of EV were conducted in RAW 264.7 macrophages, L6 myoblasts, and INS-1 pancreatic β -cells.

Results: When compared to WT, the KO EV were found to be enriched in sphingolipids and phospholipids, particularly those containing polyunsaturated fatty acids (PUFAs). We tested the uptake and functional effects of these EV in metabolically relevant target cell types. KO EV exhibited enhanced uptake by RAW 264.7 macrophages, leading to increased reactive oxygen species (ROS) production and activation of NF- κ B and IRF pathways. In L6 skeletal muscle cells, WT EV increased ATP production, while KO EV failed to elicit this effect. Additionally, KO EVs impaired glucose-stimulated insulin secretion (GSIS) in INS-1 pancreatic beta cells. These findings reveal a distinct lipid signature in EV from KO mice that drive oxidative stress, inflammation, and metabolic dysfunction.

Conclusions: Our study provides mechanistic insight into the role of EV lipid composition in metabolic disease pathophysiology, highlighting adiponectin's critical role in EV-mediated communication. These insights may facilitate biomarker discovery and inform therapeutic strategies for metabolic diseases.

Keywords: Adiponectin, Lipidomic, Extracellular vesicles, Oxidative stress, Inflammation, Insulin secretion

OP1-4

Atherosclerosis associated with HIV - is it a result of treatment or inflammation

Nuriyat Efendieva^{*}, Alexey Sozykine, Oleg Shevchenko

Cardiology, Pirogov Russian National Research Medical University, Russian Federation

Objectives: Chronic infection by HIV evolves with a vascular inflammatory action causing endothelial dysfunction. The action of the virus as well as the side effects of antiretroviral drugs contributes to the progression of cardiovascular diseases. The study aimed to characterise the changes of the structure of the coronary wall and the thickening of the intima by Optical Coherence Tomography in HIV-infected patients with or without symptoms of coronary heart disease.

Methods: Fifty-two HIV-infected individuals had a mean age of 49.8 ± 11.4 years. There were 75% men, diabetes 30.8%, hypertension 30.8%, smokers 34.62% and 7.7% with cholesterol levels ≥ 99 mg/dl. Control group included 120 non-HIV-infected controls with coronary heart disease. All the participants from HIV-group receive ART, 100% of participants had plasma HIV RNA < 20 copies/ml and 78.85% of them have symptoms of coronary artery disease.

Results: The average diffuse homogeneous thickening of the intima in patients with HIV was 0.67 ± 0.24 mm, and 0.34 ± 0.18 mm in control group, with normal values not exceeding 0.05 mm. There was impaired three-layered structure of coronary wall in 90.4% (47 of 52) HIV-infected participants and in 60% of control group, atherosclerotic plaque had only 34.62% of HIV group. All HIV-infected patients receive ART more than 5 years.

Conclusions: OCT demonstrated that the inflammatory process resulting from HIV-infection or ART may be relevant in the changes of coronary arteries in HIV-positive patients. The changes are predominantly represented by thickening of the intima, impaired three-layered structure of arterial wall and accelerating atherosclerosis.

Keywords: Optical coherence tomography, HIV-infection, Intimal hyperplasia

OP1-5

Anti-atherosclerotic effect of thymol extract via regulating inflammation and oxidative stress and EGFR/PI3K/Akt/GSK-3 β pathway in rats

Manvendra Singh^{2*}, Niti Singh³, Deepika Singh¹

¹Maa Gange Research Foundation, Kashi, India, ²Computer Sciences, HMFA-MIET, India, ³Basic Sciences, CSVTU, Bhilai, India

Objectives: The accumulation of lipids, particularly low-density lipoprotein cholesterol (LDL-C), and macrophages, in addition to the malfunctioning of endothelial and vascular smooth muscle cells, is the primary characteristic of atherosclerosis, a chronic inflammatory process. The present study aims to scrutinize the Anti-atherosclerotic effect of thymol extract via regulating inflammation and oxidative stress and EGFR/PI3K/Akt/GSK-3 β pathway in rats

Methods: The development of a rat model of atherosclerosis by high-fat diet in conjunction with vitamin D3. The effectiveness of thymol extract (Thy-ex) in preventing atherosclerosis was investigated from several different perspectives, including blood lipid regulation, anti-oxidative stress, anti-inflammatory response, and vascular protection function.

Results: Thy-ex decreased the levels of total cholesterol, total fat, and LDL-C, as well as ALT and AST, while simultaneously increasing the level of HDL-C and antioxidant benefits by significantly increasing blood SOD and GSH-Px and decreasing MDA levels in rats. It also reduced the levels of pro-inflammatory factors (IL-1 β , IL-6, TNF- α , and CRP) and enhanced levels of the anti-inflammatory factor IL-10. It also elevated level of NO, eNOS, and 6-keto-PGF1 α and lowered the ET-1 and TXB2 levels. Thy-ex dramatically decreased the mRNA and protein levels of EGFR, p-PI3K, p-AKT, GSK-3 β , Bax, and Caspase-3 and reduced the Bax/Bcl-2 ratio. By inhibiting the activation of NF- κ B and MAPK pathways, thy-ex lower the expression of proinflammatory factors such as COX-2, iNOS, IL-1 β , IL-6, and TNF- α . It also diminishes the expression of intercellular adhesion molecule-1 and vascular cell adhesion molecule-1. and alleviated HFD-enhanced hepatic steatosis, which was accomplished by downregulating gene expression for lipid biosynthesis (SREBP2, HMGCR, and FASN) and lipid uptake (LDLR), as well as by upregulating CYP7A1 expression.

Conclusions: it appears that Thy-ex has the ability to reduce the risk of atherosclerosis by regulating inflammation, hyperlipidaemia, and lipid metabolism and these effects may be associated with the inhibition of the EGFR/PI3K/AKT/GSK-3 β signalling pathway

Keywords: Signalling pathway

OP1-6

PCSK9 deficiency promotes ANP-mediated protection against post-infarction cardiac inflammation

NaHyeon Yoon*, GooTaeg Oh

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

Objectives: Myocardial infarction (MI) triggers cardiomyocyte apoptosis, followed by compensatory left ventricular hypertrophy to compensate for cell loss. The protein convertase subtilisin/kexin type 9 (PCSK9) has been identified as directly impacting cardiomyocyte viability and function by altering tissue structure and reducing cardiac function. ANP, predominantly produced by atrial cardiomyocytes, is upregulated in response to cardiac stress. However, the regulatory relationship between PCSK9 and ANP remains unexplored. This study investigates whether PCSK9 modulates ANP expression in the setting of MI.

Methods: To investigate the effects of PCSK9 deletion on heart function and inflammation following MI, 9-11-week-old PCSK9^{+/+} and PCSK9^{-/-} male mice were subjected to MI by occluding the LAD branch of the coronary artery. Heart function after MI was analyzed using echocardiography, Trichrome staining, qPCR, western blotting, and immunohistochemistry. Additionally, the level of ANP, which increases after MI, was measured by ELISA, and the effect of ANP on macrophages was evaluated using bone marrow-derived macrophages.

Results: PCSK9-deficient mice exhibited enhanced cardiac function, increased collagen synthesis, and reduced infarct size within the affected region. Furthermore, PCSK9-deficient mice demonstrated a significant survival advantage after acute myocardial infarction. These beneficial effects could potentially be attributed to the preserved contractility of cardiomyocytes in PCSK9-deficient hearts, leading to elevated expression of the cardiac hormone ANP. ANP functions by inhibiting inflammation mediated by macrophages, thereby alleviating macrophage-induced cardiomyocyte cell death. The upregulation of ANP in PCSK9-deficient hearts appears to contribute to the improved cardiac function and reduced inflammation observed after MI.

Conclusions: This investigation provides evidence that elevated cardiac ANP levels in ischemic hearts lacking PCSK9 function serve as a suppressor of inflammatory processes. The anti-inflammatory properties of ANP can contribute to the maintenance of cardiac function in PCSK9-deficient hearts.

Keywords: MI, PCSK9, ANP

OP1-7

Targeting LGI3 attenuates atherosclerosis by modulating inflammatory responses and plaque development

Jing Jin*, Do Hee Yu, Goo Taeg Oh

Department of 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 Glioma Inactivated 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-1 β , 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- κ B 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-1 β , and IL-6. Pharmacological inhibition of LGI3 with P34 suppressed the expression of these cytokines and attenuated NF- κ B 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- κ B-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, LGI3, Macrophage, Inflammation

OP1-8

Proteomic profiling of inflammatory and non-lipid determinants of plaque instability in myocardial infarction

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Objectives: Atherosclerosis is a chronic vascular condition marked by disrupted lipid regulation and sustained inflammation within large and medium-sized arteries. Myocardial infarction (MI) typically results from thrombotic events following rupture or destabilization of atherosclerotic plaques. Although conventional risk factors such as hypertension and dyslipidemia are well recognized, recurrent events and mortality post-MI remain substantial. This study aimed to identify potential contributors to plaque instability by analyzing markers of inflammation, extracellular matrix (ECM) remodeling, thrombosis, and lipid metabolism in North Indian patients with first-time and recurrent myocardial infarction (RMI).

Methods: We enrolled 139 patients diagnosed with MI within 24 hours of symptom onset and classified them as either first-time or recurrent MI cases. Serum concentrations of inflammatory (NF- κ B, hs-CRP, TNF- α , IFN- γ , IL-6), endothelial adhesion (VCAM-1), ECM remodeling (MMP-9, stromelysin, TIMP-1, MCP-1, PAPP-A), thrombosis-related (vWF, D-dimer, PLA2), and lipid-associated (PON-1, Apo-B, Apo-A1, ox-LDL, anti-oxidized LDL antibodies) markers were measured using ELISA. Risk stratification was performed using multivariate logistic regression analysis.

Results: The results revealed that average age was 58.4 ± 23.3 years in the first-time MI group and 53.9 ± 22.9 years in the RMI group. Patients with recurrent MI exhibited significantly elevated levels of TNF- α , VCAM-1, MMP-9, PAPP-A, and the antioxidant enzyme PON-1 ($p < 0.05$). Conversely, IL-6 and D-dimer were notably higher in first-time MI patients. Proteomic data at a 95% confidence interval enabled the classification of RMI risk at a 0.5 cut-off.

Conclusions: The findings highlighted that non-lipid biomarkers provide valuable insight into the mechanisms underlying plaque vulnerability. Inflammatory mediators, thrombosis markers, ECM remodeling enzymes, and oxidative stress regulators such as PON-1 may serve as effective indicators for identifying patients at risk of recurrent myocardial infarction.

Keywords: Myocardial infarction, Plaque instability, Inflammatory markers, Extracellular matrix remodeling

OP1-9

The role of NLRP3 inflammasome activation in atherosclerosis: a systematic review and meta-analysis

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Objectives: To determine the expression of NLRP3 and downstream inflammatory markers in atherosclerosis, based on evidence from both human tissue research and experimental studies.

Methods: PubMed, Scopus, Embase and Web of Science database searches were conducted systematically up to February 2025. The studies were eligible if they reported NLRP3 or IL-1 β expression in atherosclerotic tissue versus healthy vascular tissue in human patients or in applicable murine models. Expression levels were quantified using standardized mean differences (SMD), and odds ratios were used for intervention outcomes of NLRP3 or IL-1 β inhibition.

Results: Eighteen studies were included, seven human and eleven animal studies. In human tissues ($n=223$ plaque samples), the expression of NLRP3 was significantly greater in atherosclerotic plaques than in normal arteries, with a combined SMD of 1.42 (95% CI: 0.94-1.89). In atherosclerosis models in mice (ApoE-/- and LDLr-/- mice), NLRP3 and IL-1 β were greater in aortic tissue, with an SMD of 1.87 (95% CI: 1.33-2.41). In experimental testing of IL-1 β inhibition, treatment reduced plaque burden by 34%, inferring a therapeutic effect.

Conclusions: NLRP3 inflammasome activation is consistently associated with atherosclerotic plaque development and progression across both human and preclinical models.

Keywords: NLRP3 inflammasome, Atherosclerosis, Interleukin-1 β , Vascular inflammation

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Oral Presentation 2

Sep 12(Fri) 13:00–14:30 | Room 1 (3F)

CHAIRPERSONS : Ju Han Kim (Chonnam National University, Republic of Korea)

Ung Kim (Yeungnam University, Republic of Korea)



OP2-1

Protective effect of biofabricated curcumin silver nanoparticles against atherosclerosis in rodent model via modulating EGFR/PI3K/Akt/GSK-3 β signaling pathway

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Objectives: Atherosclerosis (AS) is a chronic inflammatory cardiovascular disease pathophysiologically hallmarked by the accumulation of lipid plaques inside the arteries, causing a significant health risk to human beings. Curcumin, i.e., diarylheptanoid, is a natural curcuminoid obtained from *Curcuma longa* rhizomes. Low bioavailability of curcumin restricts its clinical use, therefore, to overcome this problem, silver nanoparticles of curcumin were synthesized, characterized, and explored against ApoE^{-/-} mice model of AS.

Methods: Silver nanoparticles of curcumin (CrAgN) were synthesized with silver nitrate solution (0.1N) by co-precipitation method. Synthesis of CrAgN was confirmed with various characterization techniques, i. e., UV, FTIR, FESEM, etc. AS was induced in mice with high-fat emulsion supplemented with vitamin D3 and lipopolysaccharide and then administered with biosynthesized CrAgN for a consecutive 90 days. The anti-AS effect of CrAgN was evaluated by estimating various parameters, including blood lipid regulation, extent of atherosclerotic lesions in the aortas, anti-oxidative stress, anti-inflammatory response, oxidative stress status, along with the effect on vascular protection.

Results: Characterization techniques results indicated the formation of the aggregated type of nanoparticles with 100-150 nm size range with surface plasmon resonance at 420 nm. Pharmacodynamic study exhibited a significantly reduced ($p < 0.01$) level of total cholesterol (TC), triglycerides (TG), and LDL-C as well as ALT and AST, while HDL-C ($p < 0.01$) level was increased. CrAgN decreased the coronary artery wall thickness and improved atherosclerotic lesions in the aorta. A statistically significant increase in SOD, GPx enzymatic activity was observed in CrAgN-treated mice. mRNA and protein expression of EGFR, p-PI3K, p-AKT, GSK-3 β , Bax, Caspase-3 as well as Bax/Bcl-2 ratio were also regulated ($p < 0.01$) by CrAgN.

Conclusions: CrAgN has the potential to mitigate high-fat emulsion, vitamin D and LPS-induced AS in ApoE^{-/-} mice by exhibiting hypolipidemic, anti-inflammatory, anti-oxidative, anti-thrombotic and vascular endothelium protective effects via inhibiting EGFR/PI3K/AKT/GSK-3 β signaling pathway.

Keywords: Silver nanoparticles

OP2-2

Extracellular vesicles produced by IL4-polarized macrophages drive atherosclerosis stabilization by reprogramming Ly-6Chi monocytes & enhancing efferocytosis

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Objectives: Examine transcriptional responses sensitive to THP1-IL4-EVs among circulating Ly-6Chi monocytes that could contribute to their ability to drive the resolution of inflammation and atherosclerosis.

Methods: THP-1 macrophages were stimulated with IL-4 and EVs purified from conditioned medium using ultracentrifugation. LDLR-deficient mice fed HFD were infused triweekly with 1010 THP1-IL4-EVs for six weeks. Circulating Ly-6Chi monocytes were isolated and RNA was examined using NanoString Autoimmune and Metabolic gene panels. Plasma cytokines were quantified with V-PLEX assays. Atherosclerosis was assessed by detecting myeloid cell subsets in aortae and in ORO-positive lesions. The aortic root was stained with Sirius Red for collagen deposition, TMR red for DNA strand breaks, MOMA2 for macrophages, and ACTA2 for fibrous cap. Efferocytosis was examined both in vitro and ex vivo by exposing THP1-IL4-EV-treated primary macrophages with CFSE-labeled Jurkat cells.

Results: THP1-IL4-EVs caused transcriptional reprogramming among Ly-6Chi monocytes, resulting in reduced signaling pathways controlled by MAPK, PI3K, TLR, and inflammasomes. We identified reduced levels of Casp-1, Nlrp1a, and Aim2, genes recognized to program monocytes to drive atherosclerosis. THP1-IL4-EVs upregulated metabolic pathways involved in arginine metabolism, IDH1/2 activity, redox stress control, nucleotide salvage, mitochondrial respiration, and FAO. In contrast, they reduced pathways involved in glucose transport, hypoxia, KEAP-NRF2 pathway, and tryptophan metabolism. Control of these pathways in Ly-6Chi monocytes contributed to reduced levels of IL-1b, IL-6, IFNg, and TNFa in plasma, CD45+ aortic myeloid cells, and ORO-positive aortic lesions. This also led to plaque stabilization as noted by increased collagen deposition and a thicker fibrous cap, reduced necrotic core volumes, and elevated expression of MertK in lesion macrophages, supporting improvements in efferocytosis. In vitro studies using primary macrophages treated with THP1-IL4-EVs supported their capacity to enhance efferocytosis.

Conclusions: THP1-IL4-EVs communicate transcriptional reprogramming to Ly-6Chi monocytes, suppressing their propensity to accelerate atherosclerosis while benefitting their ability to contribute to atheroma remodeling and stabilization.

Keywords: Transcriptional reprogramming, microRNA, Atherosclerosis stabilization, Inflammation control, Ly-6Chi monocytes, Extracellular vesicles

OP2-3

Alteration of lipid gene modulation and gut microbiota by gallic acid loaded liposomes in atherosclerosis in apolipoprotein-E deficient mice

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Objectives: The primary independent risk factors for atherosclerosis are thought to be faulty lipid metabolism and obesity-induced visceral fat deposition. Atherosclerosis is regarded as a chronic inflammatory illness. The purpose of this study was to determine can Gallic acid loaded liposomes (GA-LPS) prevent atherosclerosis in rodent with the disease by modifying their gut flora and lipid metabolism.

Methods: All the apolipoprotein-E deficient mice were received high cholesterol diet for 12 weeks and received varies dosage of GA-LPS. After the completion of the study, the serum was used to measure the biochemical parameter and faces were also collected. 16S rRNA gene sequencing was used to detect the gut microbiota. RT-qPCR and western blotting was used to measure the genes which was related to lipid metabolism. Histopathology study were also performed.

Results: Pretreatment with GA-LPS modulates the level of TC, TG, LDL-C and HDL-C. It also reduced the level of LPO, ROS, and catalase, SOD, GPx and GSH in HCD mice. Administration of GA-LPS altered the composition of the gut microbiota. After the administration of the GA-LPS, it enhanced the relative abundance of the Muribaculaceae and Ruminococcaceae and reduced the Erysipelotrichaceae., it significantly impacted the gene metabolism related to the gene. It also reduced the level of gene metabolism (LXR- α and SREBP1) along with intracellular adhesion molecule-1, the chemokine (C-X3-C-motif) ligand 1, E-selectin and the adhesion molecules vascular cell adhesion molecule-1, and; down-regulation of high mobility group box-, tumor necrosis factor- α , interleukin-6, and cathepsin levels in in the aortic sinuses of mice.

Conclusions: Potentially preventing atherosclerosis is demonstrated by GA-LPS, and this impact is intimately linked to gut microbiota and lipid metabolism. This research could lead to new understandings of GA-LPS effects on atherosclerosis and a foundation for its potentially beneficial clinical application.

Keywords: Gallic acid, Mice

OP2-4

Candidates of STAT3-mediated miRNAs in LPS-induced mouse endothelium

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Objectives: Endothelial cell (EC) dysfunction plays a critical role in the development of cardiovascular diseases (CVDs), triggered by inflammatory stimuli such as lipopolysaccharide (LPS). Signal transducer and activator of transcription 3 (STAT3) is known to mediate vascular inflammation and remodeling and has recently been linked to EC-cardiomyocyte communication. While microRNAs (miRNAs) have been recognized as regulators of EC function, the interaction between STAT3 signaling and miRNA regulation in this context remains poorly understood.

Methods: To investigate this, we analyzed miRNA expression profiles in aortic ECs from mice treated under four conditions: control, LPS, Stattic (a STAT3 inhibitor), and LPS combined with Stattic.

Results: Extracted RNA from mouse aortic EC was subjected to small RNA sequencing, revealing 91 differentially expressed miRNAs linked to 28 biological processes. Cross-referencing with known endothelial miRNA datasets identified 8 novel miRNAs with 4 upregulated (miR-28a-5p, miR-411-3p, miR-501-3p, miR-676-3p) and 4 downregulated (miR-184-3p, miR-382-3p, miR-298-5p, miR-541-5p). Promoter analysis (5 kb upstream and 1 kb downstream of the transcription start sites) using miRGen v3, TransmiR v3.0, and JASPAR2024 revealed shared transcriptional regulators including KLF14, KLF16, SP1, and SP2. Target prediction through five databases (DIANA micro-T, miRmap, TargetScan, PicTar, and miRDB) highlighted shared downstream genes, including Luzp1 and Zbtb20, both highly expressed in cardiomyocytes. Although STAT3 was not initially predicted as a direct regulator or target, JASPAR2024 analysis revealed potential STAT3 binding sites in the promoters of several identified miRNAs, suggesting potential regulatory role.

Conclusions: These findings suggest that STAT3 may modulate a subset of inflammation-responsive miRNAs in aortic ECs, potentially affecting cardiomyocyte behavior via EC-cardiomyocyte crosstalk. Ongoing studies aim to validate these interactions and clarify STAT3-miRNA signaling in EC dysfunction and CVD progression.

Keywords: STAT3, EC dysfunction

OP2-5

KAI1 on perivascular cells: a key switch regulator of angiogenesis

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Objectives: This study explores the role of KAI1 (CD82), predominantly expressed in perivascular cells (PVCs), in suppressing angiogenesis. While its anti-metastatic effects in cancer are well-established, its regulatory function in vascular homeostasis has remained unclear. Here, we examine the mechanisms by which KAI1 inhibits angiogenic signaling and assess its therapeutic potential for controlling pathological angiogenesis.

Methods: Using in vivo models and molecular analyses, we investigated KAI1 expression patterns in PVCs, particularly pericytes (PCs), where it undergoes lipid raft localization via zDHHC4-mediated palmitoylation. We evaluated its role in inducing Leukemia Inhibitory Factor (LIF) through the Src/p53 pathway, as well as its direct interaction with angiogenic factors. Functional assays were performed to examine the impact of KAI1 on vascular quiescence, including gene expression profiling and angiogenesis suppression in tumor models.

Results: KAI1 was found to be highly expressed in pericytes, localizing to lipid rafts via palmitoylation, where it stabilized anti-angiogenic signaling. It induced LIF expression, suppressing angiogenic gene expression in both pericytes and endothelial cells. Additionally, KAI1 interacted directly with angiogenic factors, further inhibiting VEGFR-mediated signaling. Reduced KAI1 levels were detected in pathological conditions such as cancer, emphasizing its role in modulating the angiogenic niche.

Conclusions: KAI1 in pericytes serves as a critical switch regulator of angiogenesis by integrating lipid raft-mediated localization and Src/p53-driven LIF induction. This study highlights its therapeutic potential as an anti-angiogenic target, offering new avenues for cancer treatment and vascular disease management.

Keywords: Angiogenesis, CD82, Pericyte, Palmitoylation

OP2-6

Distribution of LDL-cholesterol and its association with cardiovascular outcomes in young adults under 40 years: a nationwide cohort study

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Objectives: We examined the distribution of low-density lipoprotein-cholesterol (LDL-C) levels and compared the hazards of incident myocardial infarction (MI), stroke, and their composite according to the LDL-C concentration among young adults.

Methods: This was a nationwide, population-based cohort study. From the Korean National Health Insurance Service database (2002-2022), adults aged 20-39 years without prior MI or stroke who underwent at least one health examination between 2009 and 2012 (N=6,458,006) were included. The hazards of MI, stroke, and their composite were evaluated according to LDL-C levels.

Results: During a median 12.6 years, 47,107 MI, 28,536 stroke, and 73,897 composite events occurred. The hazard of outcomes showed a J-shaped association with LDL-C levels, with the lowest hazard observed at LDL-C levels around 90-110 mg/dL. Above this range, the hazard increased progressively with higher LDL-C levels. When analyzed by the LDL-C categories, LDL-C levels ≥ 130 mg/dL were associated with significantly increased hazards of outcomes, with the highest risks observed among individuals with LDL-C ≥ 160 mg/dL. The increased hazards in the higher LDL-C categories were more prominent in individuals with abdominal obesity and current smokers for all three outcomes, and in those with higher BMI category, low HDL-C, and hypertriglyceridemia for MI and composite outcome.

Conclusions: Among young adults aged 20-39 years, cardiovascular risk demonstrated a J-shaped association with LDL-C levels. Beyond around 90-110 mg/dL, risks increased progressively at higher LDL-C levels, particularly in the presence of other cardiometabolic risk factors. These findings highlight the importance of lipid monitoring and tailored risk assessment even in young adults, especially those with cardiometabolic risk factors.

Keywords: Low-density lipoprotein-cholesterol, Myocardial infarction, Stroke, Young adult

OP2-7

Lipoprotein(a) variability and risk of major adverse cardiovascular event: insights from a real-world multicenter cohort

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Objectives: Lipoprotein(a) [Lp(a)] is a genetically determined lipoprotein and an independent risk factor for atherosclerotic cardiovascular disease. Although Lp(a) levels are generally stable over time, recent evidence suggests the presence of intra-individual variability. However, the clinical significance of this variability remains unclear. This study aimed to examine the association between Lp(a) variability and cardiovascular outcomes.

Methods: This multicenter retrospective cohort study analyzed data from three tertiary hospitals in South Korea using the OMOP-CDM framework. 13,914 patients with repeated Lp(a) measurements at least 90 days apart between 2019 and 2025 were included. Lp(a) variability was defined as both absolute (>10 mg/dL) and relative (>25%) change. The primary outcome was major adverse cardiovascular events (MACE), including cardiovascular death, new-onset myocardial infarction, stroke, coronary revascularization, and heart failure hospitalization.

Results: During a median follow-up of 378 days, 2,156 patients (15.5%) exhibited high Lp(a) variability, despite strong intra-individual correlation (Spearman's $\rho=0.92$). Compared to those with low variability, these patients were older and had a higher prevalence of hypertension, diabetes mellitus, chronic kidney disease, and prior stroke. High Lp(a) variability was associated with a significantly greater incidence of MACE (6.8% vs. 4.2%, $p<0.001$), and this association remained significant after adjustment for baseline characteristics and laboratory values (adjusted HR 1.57, 95% CI 1.22-2.03, $p=0.001$). Kaplan-Meier curves demonstrated higher cumulative MACE incidence in the high variability group over three years (Figure). In event-specific analyses, high variability was significantly associated with increased risk of new-onset myocardial infarction ($p=0.017$), heart failure hospitalization ($p<0.001$), and percutaneous coronary intervention ($p<0.001$), while the rates of stroke, cardiovascular death, and coronary artery bypass graft did not differ significantly.

Conclusions: In this large, multicenter cohort, intra-individual variability in Lp(a) levels was independently associated with increased risk of adverse cardiovascular outcomes. These findings suggest that temporal changes

Keywords: Lipoprotein(a), Variability, Major adverse cardiovascular event

OP2-8

Discontinuation and non-publication of atherosclerosis clinical studies: a cross-sectional analysis

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Objectives: The frequent discontinuation and non-publication of clinical studies in atherosclerosis represent a significant challenge and research waste. This study examines characteristics associated with study termination and non-dissemination to inform preventive strategies.

Methods: We searched ClinicalTrials.gov for all clinical studies related to atherosclerosis until May 2025. To ensure accurate publication status ascertainment, we excluded studies completed after May 2022 and ongoing trials, as they may still be undergoing peer review. Publication status was verified through NCT number searches and protocol matching. We performed multivariable binomial logistic regression to analyze associations between trial characteristics (including enrollment size, study location, funding source, study design, and intervention type) and two primary outcomes: discontinuation and non-publication.

Results: A total of 1,901 eligible registered clinical studies were included in the analysis, with 1,605 (84.4%) completed and 970 (51%) remaining unpublished. Binomial logistic regression identified small sample sizes (

Conclusions: Our study confirms significant non-dissemination bias in atherosclerosis research, with 51% of completed studies remaining unpublished. This represents both research waste and an ethical concern, as participants incur risks without advancing medical knowledge.

Keywords: Atherosclerosis, Clinical studies

Association between dietary antioxidant intake and cardiovascular diseases risk factors in Singapore individuals with eczema

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Objectives: Dietary antioxidants possess antioxidants and anti-inflammatory properties, which may contribute to the prevention of cardiovascular diseases (CVD). However, evidence remains limited in populations with chronic inflammatory conditions such as eczema. Therefore, this study aimed to investigate the association between dietary antioxidant intake and CVD risk factors in Singapore individuals with eczema.

Methods: This is a secondary analysis from a single-blinded, parallel design of a 12-week randomized controlled trial with 40 Singapore individuals with eczema. Dietary and biochemical assessments were collected at pre- and post-intervention (80 total observations). Dietary intake was assessed using 3-day food records, and total and individual antioxidant intakes were categorized into quartiles (Q1-Q4). Multiple linear regression models evaluated associations between antioxidant intake and CVD risk factors, including anthropometric measurements, blood pressure and lipid-lipoprotein profile, Framingham Risk Score (FRS), with adjustments for age, sex, BMI, energy, protein, fat, and carbohydrate intake.

Results: No differences in the levels of CVD risk factors among quartiles of antioxidant intake and no significant associations were also found between total antioxidant intake and CVD risk factors. However, higher β -carotene intake (Q4) was significantly associated with higher HDL and lower triglyceride (TG) levels ($p < 0.05$) as compared to lower intake (Q1). Higher lutein/zeaxanthin intake (Q4) was also linked to lower TG level ($p = 0.038$), with a significant trend across quartiles ($p = 0.025$).

Conclusions: Among individuals with eczema, overall total dietary antioxidant intake was not associated with CVD risk factors however, specific antioxidants like β -carotene and lutein/zeaxanthin may exert selective beneficial effects, particularly on lipid-lipoprotein regulation. This finding warrants further research into their potential role in CVD risk management among individuals with eczema.

Keywords: Cardiovascular disease, Dietary Antioxidant, Eczema

ICoLA 2025

The 14th International Congress on Lipid & Atherosclerosis

Oral Presentation 3

Sep 13(Sat) 08:50–10:20 | Room 1 (3F)

CHAIRPERSONS : Dae Jung Kim (Ajou University, Republic of Korea)

Soo Lim (Seoul National University, Republic of Korea)



OP3-1

Statin use and site-specific cancer risk: a nationwide cohort study using Korean health insurance data

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Objectives: Emerging evidence suggests statins, widely used for dyslipidemia and cardiovascular disease, may also exert anticancer effects by modulating the mevalonate pathway and tumor microenvironment. However, population-based evidence in Koreans remains limited. This study aimed to evaluate the association between statin use and cancer risk using Korean nationwide health data.

Methods: We conducted a cohort study using the Korean National Health Insurance Service database. Individuals undergoing general health screening in 2009–2010 were followed through 2019. Statin users were defined as those prescribed statins for ≥ 6 months; non-users had no statin exposure. Incident cancers were identified using ICD-10 codes. Multi-variable Cox proportional hazards models were used to estimate adjusted hazard ratios (HRs).

Results: Among 236,118 participants (3,998 statin users and 232,120 non-users), the cumulative incidence of all cancers was slightly higher in statin users than in non-users (10.80% vs. 8.90%). In adjusted models, statin use was associated with a modest but statistically non-significant increase in overall cancer risk (adjusted HR, 1.05). Site-specific analyses showed that statin use was associated with a slightly reduced risk of liver cancer (adjusted HR, 0.85) and pancreatic cancer (adjusted HR, 0.89). Notably, the cumulative incidence of thyroid cancer was substantially higher in statin users compared to non-users (1.65% vs. 1.04%), and statin use was significantly associated with an increased risk of thyroid cancer (adjusted HR, 1.58, $p < .001$).

Conclusions: In this large Korean cohort, statin use was associated with a significantly higher risk of thyroid cancer, despite suggesting a reduced risk of liver and pancreatic cancers. These findings suggest that statins may exert organ-specific effects on carcinogenesis, underscoring the need for further investigation into the biological mechanisms underlying these divergent associations.

Keywords: Statins, Cancer prevention, Thyroid cancer

OP3-2

Association between circulating trimethylamine N-oxide levels and carotid perivascular fat density in acute ischemic stroke patients

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Objectives: Trimethylamine N-oxide (TMAO), a gut microbiota-derived metabolite, has been associated with systemic inflammation and atherosclerosis. Perivascular fat density on computed tomography angiography (CTA) reflects local vascular inflammation and may contribute to stroke pathogenesis. We investigated the association between circulating TMAO levels and carotid perivascular fat density in patients with acute ischemic stroke.

Methods: We included 233 patients with acute ischemic stroke who presented within 7 days of symptom onset. All patients underwent CTA and plasma TMAO measurement during the acute phase. Perivascular fat density was quantified in Hounsfield Units (HU) using standardized regions-of-interest (ROIs) adjacent to the internal carotid artery (ICA) on the side of the infarction. Multivariable linear regression was used to evaluate the association between TMAO levels and perivascular fat density, adjusting for conventional vascular risk factors.

Results: Higher circulating TMAO levels were significantly associated with increased perivascular fat density (i.e., less negative HU values), indicative of greater pericarotid inflammation ($p < 0.01$). This association remained robust after adjustment for age, sex, hypertension, diabetes mellitus, LDL cholesterol, and statin use. The relationship was more pronounced in patients with large-artery atherosclerosis subtype compared to other stroke mechanisms.

Conclusions: In patients with acute ischemic stroke, elevated TMAO levels are significantly associated with increased carotid perivascular fat density, suggesting a potential mechanistic link between gut-derived metabolites and local vascular inflammation. These findings support the role of TMAO as a biomarker reflecting vulnerable atherosclerotic plaque and stroke risk.

Keywords: Carotid stenosis, TMAO

OP3-3

The relationship of inflammatory markers with the development of coronary artery calcification: a multicenter, longitudinal cohort study

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Objectives: Inflammation is associated with atherosclerotic cardiovascular disease. There exists limited evidence supporting a relationship between inflammation and coronary artery calcification (CAC).

Methods: CAC was assessed by cardiac computed tomography (CT) using the CAC score in a multicenter cohort of asymptomatic individuals. The relationships between leukocyte count and high-sensitivity C-reactive protein (hsCRP) with baseline CAC prevalence and severity, and longitudinal incidence and progression, were analyzed using multivariable regression. Mediation analysis was used to analyze the indirect effect of leukocyte count on all-cause mortality through CAC.

Results: A total of 53,557 individuals (age 52.3±9.5 years, 71.6% male) were selected for baseline analysis, and higher leukocyte count was associated with higher CAC prevalence (p<0.001) and CAC severity (p=0.048). In longitudinal analysis of 7,608 individuals with follow-up CT scans (20,584 scans, median follow-up 3.5 years), leukocyte counts also showed an association with higher probability of incident CAC (adjusted OR per 109/L, 1.129, 95% CI 1.033-1.234, p=0.003) and faster CAC score progression (β -coefficient per 109/L increase, 1.13, 95% CI 0.45-1.91, p=0.003). A J-shaped relation between leukocyte counts and CAC development was noted. However, no relationship with CAC was observed for hsCRP. Leukocyte counts were associated with increased all-cause mortality (adjusted HR 1.157, 95% CI 1.089-1.229, p<0.001) during a median follow-up of 5.5 years, and 4.43% (95% CI, 1.60-7.23, p=0.002) of this risk was attributable to CAC severity.

Conclusions: Higher leukocyte counts are associated with faster CAC development, which mediates mortality risk.

Keywords: Atherosclerosis, Inflammation, Leukocyte count

OP3-4

Saffron as a natural statin alternative: in silico modulation of MMP-9 and TIMP-1 for plaque stability

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Objectives: Matrix metalloproteinase-9 (MMP-9) and its endogenous inhibitor, tissue inhibitor of metalloproteinase-1 (TIMP-1), regulate extracellular matrix remodeling and play pivotal roles in atherosclerotic plaque stability. Excessive MMP-9 activity destabilizes plaques, increasing cardiovascular risk. While statins improve plaque stability, some patients experience intolerance. Saffron (*Crocus sativus* L.), rich in bioactive compounds such as crocin, crocetin, and safranal, has shown cardiovascular benefits. This study compares the molecular interactions of saffron compounds with MMP-9 and TIMP-1 against simvastatin to evaluate saffron's potential as a natural alternative.

Methods: Crystal structures of human MMP-9 and TIMP-1 were retrieved from the Protein Data Bank and prepared using UCSF ChimeraX. Ligand structures (crocin, crocetin, safranal, simvastatin) were downloaded from PubChem and energy-minimized in Avogadro. Molecular docking was conducted using AutoDock Vina via the CB-Dock2 platform. Binding affinities were compared, and key ligand-receptor interactions were visualized and analyzed using BIOVIA Discovery Studio, focusing on hydrogen bonding, hydrophobic interactions, van der Waals forces, and π - π stacking.

Results: Crocin demonstrated the strongest binding affinity to both TIMP-1 (-9.0 kcal/mol) and MMP-9 (-8.7 kcal/mol). In the TIMP-1 complex, crocin formed four hydrogen bonds with ASN314, GLY197, TYR52, and ARG95, and interacted with ARG233 via van der Waals and hydrophobic forces. In the MMP-9 complex, crocin established hydrogen bonds with ARG424, ARG426, and GLY197, alongside non-covalent interactions with GLU402 and TYR423. Crocetin showed moderate affinities (-6.6 and -7.0 kcal/mol) with mainly hydrophobic contacts and no hydrogen bonding. Safranal exhibited weaker affinities (-5.4 and -6.1 kcal/mol) and limited interactions. Simvastatin had the lowest binding (-5.3 and -6.0 kcal/mol), forming only hydrophobic and π - π interactions.

Conclusions: Crocin demonstrates superior binding to MMP-9 and TIMP-1 compared to simvastatin, suggesting its potential as a natural dual-action agent for stabilizing atherosclerotic plaques. These in silico findings warrant further investigation into saffron-derived compounds as therapeutic alternatives, particularly for statin-intolerant patients.

Keywords: Plaque stability, Saffron, Statin, Molecular docking, Tissue inhibitor matrix metalloproteinase-1, Matrix metalloproteinase-9

OP3-5

Comparative outcomes of moderate-intensity statin with ezetimibe vs high-intensity statin therapy: a retrospective observational cohort study

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Objectives: The optimal strategy for lipid-lowering therapy in patients at high cardiovascular risk remains controversial. This study compared the effectiveness of moderate-intensity statin therapy combined with ezetimibe versus high-intensity statin monotherapy.

Methods: We conducted a retrospective observational cohort study at a large tertiary university hospital. The study included patients prescribed either moderate-intensity statin with ezetimibe or high-intensity statin between January 1, 2018, and December 31, 2020. Propensity score matching was performed to balance clinical characteristics between the groups. The primary endpoint was an ischemic outcome, defined as a composite of cardiovascular death, non-fatal myocardial infarction, and ischemic stroke. Secondary outcomes included cardiovascular death, myocardial infarction, ischemic stroke, revascularization, and all-cause mortality.

Results: Before matching, 1,380 patients were prescribed moderate-intensity statin-ezetimibe combination therapy, and 1,105 patients were prescribed high-intensity statin therapy. After matching, 971 patients were included in each group. Over a median follow-up of 3.8 years, the primary endpoint occurred in 1.7% of patients in the moderate-intensity statin with ezetimibe group and in 2.1% of patients in the high-intensity statin group. The incidence of major adverse cardiovascular events did not differ significantly between the two groups (hazard ratio 0.82, 95% confidence interval 0.41-1.61, $p=0.558$). No statistically significant differences were observed in secondary outcomes, including cardiovascular death, myocardial infarction, ischemic stroke, revascularization, and all-cause mortality.

Conclusions: In this retrospective cohort study, moderate-intensity statin combined with ezetimibe was as effective as high-intensity statin monotherapy in preventing cardiovascular events.

Keywords: Ezetimibe

OP3-6

Effectiveness and safety of very low-dose rosuvastatin-ezetimibe therapy in dyslipidemia: a multicenter prospective observational study

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Objectives: Dyslipidemia is a key modifiable risk factor for atherosclerotic cardiovascular disease. However, achieving recommended low-density lipoprotein cholesterol (LDL-C) target levels is challenging owing to dose-dependent adverse effects and limited tolerability of high-dose statins. This study evaluated the real-world efficacy and safety of combining very low-dose rosuvastatin (2.5 mg) with ezetimibe (10 mg) in adult patients with dyslipidemia across different cardiovascular risk strata.

Methods: This multicenter prospective study in South Korea comprised 2,388 patients. Participants were stratified into low-, moderate-, or high-risk groups based on the 2019 European Society of Cardiology and European Atherosclerosis Society guidelines. Lipid profiles and safety outcomes were assessed at baseline and after 12 weeks. The primary and secondary outcomes were LDL-C and non-high-density lipoprotein cholesterol (non-HDL-C) target level achievements, respectively, and adverse events were monitored.

Results: After 12 weeks, LDL-C target levels were achieved by 82.6% of low-risk (<116 mg/dL), 73.9% of moderate-risk (<100 mg/dL), and 50.4% of high-risk (<70 mg/dL) patients. Non-HDL-C target level achievement followed a similar trend. Combination therapy with ezetimibe and low-dose statin resulted in significant LDL-C reductions, compared with statins alone. Adverse events were infrequent (0.6%), and only 0.2% of patients discontinued treatment owing to medication-related concerns.

Conclusions: Very low-dose rosuvastatin-ezetimibe combination therapy significantly lowered LDL-C levels and improved lipid profiles across various risk groups, demonstrating a favorable safety profile. These findings support its use as an effective, well-tolerated option for managing dyslipidemia.

Keywords: Dyslipidemia, Very low-dose rosuvastatin

OP3-7

Clinical outcomes of high intensity lipid lowering agents for patients after complex PCI

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Objectives: The impact of high-intensity lipid lowering therapy (HILLT) on clinical outcomes following complex percutaneous coronary interventions (PCI), has not been elucidated.

Methods: Out of three multicenter prospective registries HOST-Synergy, HOST-Alpine, and HOST-Onyx, encompassing patients who underwent PCI with drug-eluting stents (DES) between 2015 and 2020 in South Korea, subjects registered from Seoul National University Hospital were enrolled (n=1,750). Subjects who underwent complex PCI, which was defined by any of the following: ≥ 3 stents implanted, ≥ 3 lesions treated, total stent length ≥ 60 mm, left main PCI, bifurcation PCI, or heavy calcification, were included in the complex PCI group (n=691). The HILLT group was defined as those prescribed either high-intensity statins with or without ezetimibe, or moderate-intensity statins combined with ezetimibe. The primary endpoint was 3-year major adverse cardio-cerebrovascular events (MACCE), a composite of cardiac death, myocardial infarct, and stroke. The secondary endpoints were 3-year all-cause death and target vessel failure (TVF), defined as a composite of cardiac death, target vessel MI (TVMI), and target vessel revascularization (TVR). Baseline characteristics were balanced using propensity score matching (PSM) method.

Results: The HILLT group was younger (median 66 vs. 69 years) and heavier, had a lower prevalence of chronic kidney disease and diabetes, and higher baseline low density lipoprotein (LDL) level. Baseline characteristics were well balanced after PSM. Overall, patients receiving HILLT exhibited a lower risk of MACCE (adjusted HR 0.508; 95% CI: 0.260-0.994; p-value=0.048). Subgroup analysis revealed that this benefit was particularly significant among patients undergoing complex PCI (adjusted HR 0.292; 95% CI: 0.107-0.797; p-value=0.017) (Figure 1A), whereas no significant difference in MACCE incidence was observed in the noncomplex PCI group (adjusted HR 0.896; 95% CI: 0.346-2.322; p-value=0.822) (Figure 1B).

Conclusions: HILLT was significantly associated with better clinical outcomes, especially after complex PCI, in the contemporary DES era.

Keywords: High intensity lipid lowering therapy, Complex PCI

OP3-8

Effect of omega-3 fatty acid supplementation on liver function and lipid profile in tobacco Users: a randomized placebo-controlled trial

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Background: Tobacco consumption is a significant public health challenge, contributing to hepatic dysfunction and dyslipidemia through mechanisms involving oxidative stress and systemic inflammation. Omega-3 fatty acids, particularly α -linolenic acid from flaxseed oil, exhibit anti-inflammatory and lipid-modulating properties. **Objective:** This study aimed to evaluate the effects of omega-3 fatty acid supplementation on liver function tests (LFTs) and lipid profile parameters in individuals with habitual tobacco use.

Methods: A randomized, placebo-controlled, parallel-group trial was conducted at King George's Medical University. A total of 102 tobacco users were enrolled from the outpatient department and allocated to either the omega-3 group (n=53) receiving 10 mL/day of food-grade flaxseed oil or the placebo group (n=49) receiving placebo for six months. LFTs and lipid profiles were assessed at baseline and post-intervention. Written informed consent was obtained from all participants.

Results: The mean age of participants was 47.62 ± 10.21 years, with no significant differences in baseline anthropometric or biochemical parameters between groups. After six months, the omega-3 group exhibited a significant reduction in body mass index (BMI; p=0.0087), direct bilirubin (p=0.0216), and alanine aminotransferase (ALT; p=0.0217). In terms of lipid profile, triglycerides (p=0.0031), low-density lipoprotein cholesterol (LDL-C; p=0.0291), and very-low-density lipoprotein cholesterol (VLDL-C; p=0.0055) were significantly reduced. The placebo group showed no statistically significant changes in any measured parameters.

Conclusions: Omega-3 fatty acid supplementation via flaxseed oil may serve as a beneficial adjunct in managing liver dysfunction and dyslipidemia among tobacco users. These findings suggest its potential role in mitigating tobacco-related metabolic disturbances and support further investigation in larger cohorts.

Keywords: Tobacco, BMI, LFT

Metabolomic patterns of dietary protein intake and their link to cardiometabolic risk: a systematic review and meta-analysis

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Objectives: Metabolomics is increasingly used to provide insights into how dietary protein intake influences cardiometabolic health. This review identified metabolomic patterns linked to protein quantity and source (animal versus plant) and their associations with cardiometabolic risk factors (CRF).

Methods: Four electronic databases (CINAHL, Cochrane, Embase, PubMed) were searched and randomised controlled trials of adults (≥ 18 y) reporting metabolomic outcomes in response to different quantities or sources of dietary protein intakes were included. Data of quantity and source of protein intake, metabolite changes in biological fluids (i.e., blood, urine, feces), and CRF outcomes were extracted. Data were analysed qualitatively and quantitatively by vote-counting and volcano plots respectively. Pathway analyses were also conducted using the KEGG database, and correlations between metabolite fold changes and CRF were analysed.

Results: Fifty-nine studies (N=59 qualitative; N=46 quantitative) were included in the current analyses. Higher total protein intake upregulated most circulating amino acids including leucine, isoleucine, and tyrosine, while glycine, glucose, and citric acid were downregulated. Three metabolic pathways were most affected: arginine biosynthesis ($\uparrow$), starch/sucrose metabolism ($\downarrow$), and one-carbon folate metabolism (folate cycle $\downarrow$; methionine cycle $\uparrow$). Animal protein intake upregulated circulating trimethylamine-N-oxide and branch-chained amino acids, while plant protein consumption increased glycine and glutamine. Correlation analyses revealed that body mass index was positively associated with sarcosine, lactic acid, and citric acid, and negatively with creatinine and beta-alanine. Fasting blood glucose was positively correlated with serine, methionine, and aspartic acid, and negatively with proline, sarcosine, and lysine. Blood triglycerides were positively linked to serine, lysine, and beta-alanine, while total and low-density lipoprotein cholesterol were positively associated with sarcosine and proline, and negatively with beta-alanine and serine.

Conclusions: Distinct metabolomic patterns differentiated different dietary protein quantity and source. Several metabolites were also significantly associated with CRF outcomes, highlighting potential pathways that may mediate diet-health relationships and support source-specific protein recommendations.

Keywords: Cardiometabolic risk factors, Metabolomics, Plant protein, Animal protein, Dietary protein

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Moderated Poster Presentation 1

Sep 12(Fri) 14:40–15:40 | Studio 5 (6F)

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



MPP-01

Hepatic lipase silencing promotes cholesterol excretion with activation of hepatobiliary sterol transport

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Objectives: Although hepatic lipase contributes to catabolism of lipoproteins including high-density lipoprotein (HDL), its effect on cholesterol excretion has not been fully understood. We aimed to investigate the effect of hepatic lipase on cholesterol excretion and associated biological pathways.

Methods: Plasmid with shRNA (5ug) against LIPC was intraperitoneally (IP) injected to C57BL/7 mice every 3 days x 8 times and these mice were compared with controls (n=6/group). Blood lipid levels and in vivo reverse cholesterol transport were compared between the two mice groups. Genes and proteins potentially contributing to cholesterol and bile acid excretion were compared.

Results: Blood lipid levels including triglyceride and HDL-cholesterol were not significantly different between the two groups. Radio-activities of total sterol from the feces were higher (mean difference 314%; p=0.004) (Figure A) in the silencing group. Expressions of SR-B1, Cyp7A1, and LXR-a were not different (Figures B-D) between the mice groups, whereas Abcg8 expression was higher (mean difference 67%; p=0.037) (Figure E) in the silencing group.

Conclusions: Hepatic lipase inhibition promoted cholesterol excretion with activation of LXR-independent sterol transport. Effects of inhibition on hepatic cholesterol uptake and bile acid pathway were uncertain in the current study.

Keywords: Lipid metabolism, LIPC protein, Liver, Atherosclerosis

MPP-02

Cholesterol regulating factor X(CRFX) deficiency exacerbates hypercholesterolemia-induced liver injury by disrupting bile acid synthesis and promoting hepatic cholesterol accumulation

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Objectives: Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) is closely associated with hepatic cholesterol dysregulation. Cholesterol Regulating Factor X(CRFX), a liver-enriched transcription factor, is known to govern the lipid, glucose, and bile acid metabolism. However, the precise role and the underlying mechanism of CRFX in hepatic damages induced by hypercholesterolemia remained unclear. This study aimed to investigate the role of CRFX to hepatic cholesterol homeostasis and liver injury under hypercholesterolemic conditions.

Methods: To assess the role of CRFX, we generated Ldlr^{-/-} CRFX^{-/-} mice following 20weeks of Western diet. Histological analysis, lipid profiling, and transcriptional analysis of liver samples were performed to evaluate hepatic injury and metabolic changes.

Results: CRFX deficiency exacerbated hepatic injury in hypercholesterolemic mice, as evidenced by increased damage and inflammation in liver samples. The levels of hepatic free cholesterol and cholesterol esters were significantly elevated in Ldlr^{-/-} CRFX^{-/-} mice, while hepatic triglyceride levels remained unchanged. Lipogenesis and cholesterol biosynthesis pathways were not transcriptionally altered. Notably, total bile acids were significantly reduced in hypercholesterolemic Ldlr^{-/-} CRFX^{-/-} mice. Moreover, the genes related with bile acid biosynthesis were transcriptionally downregulated, but not in bile acid transports.

Conclusions: CRFX deficiency in hypercholesterolemic Ldlr^{-/-} mice led to hepatic cholesterol accumulation and impaired bile acid biosynthesis, triggering inflammation and liver damage. These findings suggest that targeting CRFX inactivation may be a novel therapeutic strategy against cholesterol-induced hepatic injury.

Keywords: Bile acid, MASLD, Cholesterol metabolism

MPP-03

The role of cereblon in mitochondrial function and metabolic reprogramming in the aging heart

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Objectives: To investigate the role of cereblon (CRBN), a negative regulator of AMP-activated protein kinase (AMPK), in cardiac aging, and to elucidate how CRBN dysregulation drives metabolic toxicity and mitochondrial dysfunction in the aging heart.

Methods: Age-associated CRBN expression was analyzed using in-silico evaluation of GEO datasets from mouse and human hearts. Wild-type (WT), whole-body CRBN knockout (wKO), and cardiomyocyte-specific CRBN knockout (cKO) mice were utilized in this study. Cardiac function was assessed through echocardiography. Measurement of CK-MB level, a cardiac injury marker, histopathological analysis, determine the metabolic profiling, mitochondrial structure and function were evaluated by oxygen consumption rate assays and transmission electron microscopy. In addition, protein expression was analyzed using Western blot.

Results: CRBN expression was significantly upregulated with age in both mouse and human hearts. Among the three genotypes, only cKO mice showed premature death, elevated CK-MB levels, reduced cardiac function, and pronounced hypertrophy and fibrosis. Despite increased AMPK phosphorylation in both wKO and cKO hearts, metabolic toxicity—including lipid accumulation, decreased oxygen consumption, downregulation of CD36, and aberrant GLUT1 upregulation—was observed exclusively in cKO mice. Mitochondrial damage was confirmed via electron microscopy in cKO hearts.

Conclusions: Cardiomyocyte-specific CRBN deletion leads to severe metabolic toxicity and mitochondrial dysfunction during aging, despite AMPK activation. These findings establish CRBN as a key regulator of metabolic homeostasis in the aging heart and suggest its dysregulation as a novel mechanism contributing to cardiac vulnerability in aging.

Keywords: Aging heart, Cereblon

MPP-04

Quail egg yolks has an effects on body weight, lipid profiles and liver tissue injury of rats (*rattus norvegicus*)

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Objectives: One of the foodstuffs that has high cholesterol levels is quail egg yolks. Quail egg yolks contain cholesterol higher than the yolks of other birds. This research aims to determine the effect of quail egg yolks on body weight, lipid profiles, and liver tissue injury of rats.

Methods: This study used a quasi-experimental method with a pre- and post-test control group design. This research was conducted in the laboratory of physiology, Universitas Islam Indonesia (UII), for 4 weeks. This research used 20 rats, male, Wistar strain, aged 2-3 months, with 200-300 grams. Rats were divided into two groups; one group was given the quail egg yolks (G1), and the other group was not given the quail egg yolks (G2). The quail egg yolks were given to the G1 rats with a dosage of 5 ml/200 grBW. Lipid profile (TC, LDL, and HDL) and body weight (BW) were measured before and after intervention. oxidative stress (MDA and SOD) of liver tissue were measured after intervention. All data were expressed as mean $\pm$ SD and considered significant at $p < 0.05$.

Results: The mean of lipid profile for G1 after intervention (mg/dL) was 212,78 $\pm$ 6,79 (TC), 76,12 $\pm$ 3,53 (LDL), 25,57 $\pm$ 1,61 (HDL). The mean of lipid profile for G2 after intervention (mg/dL) was 70,10 $\pm$ 2,68 (TC), 26,76 $\pm$ 1,81 (LDL), and 63,66 $\pm$ 2,18 (HDL). The mean of BW after intervention (grams) was 236,12 $\pm$ 34,74 (G1) and 193,12 $\pm$ 18,11 (G2). The mean MDA levels of liver tissue after intervention (nmol/gr) were 14.8 $\pm$ 0.17 (G1) and 3.2 $\pm$ 0.12 (G2). The Mean of SOD activity of liver tissue after intervention (%) was 31.43 $\pm$ 2.52 (G1)

Conclusions: Quail egg yolks affect lipid profiles, body weight, and liver tissue injury in rats. Quail egg yolks can be one of the risk factors for liver disease.

Keywords: Lipid, Liver

MPP-05

Lipoprotein(a) and long-term cardiovascular events in patients with coronary vasospasm

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Objectives: To assess the association between lipoprotein(a) [Lp(a)] levels and the risk of major adverse cardiovascular events (MACE) and recurrent angina in patients with coronary artery spasm undergoing spasm provocation testing.

Methods: We retrospectively analyzed 2,977 patients who underwent coronary angiography with spasm provocation testing between 2004 and 2021. Patients were stratified by Lp(a) level (<30 mg/dL vs ≥30 mg/dL), and a continuous analysis per 10 mg/dL increment was also performed. The primary endpoint was major adverse cardiovascular events (MACE). A secondary outcome was recurrent angina. Associations were assessed using Kaplan-Meier survival analysis, Cox proportional hazards, and logistic regression, adjusting for age, sex, hypertension, diabetes, smoking, statin use, and angiographic spasm (≥70%).

Results: Among 2,977 patients (mean age 56.9±12.5 years, 52.8% female), 20.6% had Lp(a) ≥30 mg/dL. Coronary spasm was present in 58.7% with low Lp(a) and 57.6% with high Lp(a) (p=0.637). Over a median follow-up of 6,365 days (~17.4 years), MACE occurred in 96 (4.1%) and 27 (4.4%) patients with low and high Lp(a), respectively. Kaplan-Meier analysis showed no difference in MACE-free survival (log-rank p=0.619). In Cox regression, Lp(a) ≥30 mg/dL was not associated with MACE (adjusted HR 1.11, 95% CI 0.73-1.71, p=0.622); neither was Lp(a) per 10 mg/dL (HR 1.01, 95% CI 0.93-1.09, p=0.910). In logistic regression for recurrent angina, Lp(a) ≥30 mg/dL was not significant (OR 1.18, 95% CI 0.94-1.49, p=0.160); nor was Lp(a) per 10 mg/dL (OR 1.02, 95% CI 0.96-1.08, p=0.328).

Conclusions: In this large, long-term spasm registry, Lp(a) was not independently associated with MACE or recurrent angina, whether analyzed as a dichotomous or continuous variable. These findings suggest Lp(a) may have limited prognostic utility in vasospastic angina populations.

Keywords: Lipoprotein(a), Coronary artery spasm, Vasospastic angina, Major adverse cardiovascular events

MPP-06

Exploring novel lncRNA regulators of macrophage mediated inflammation in atherosclerosis

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Objectives: Chronic inflammation is a hallmark of atherogenesis, and macrophages play a pivotal role in initiating and sustaining vascular inflammation. Long non-coding RNAs (lncRNAs) are emerging as key modulators of immune responses, yet their contribution to macrophage-driven inflammation in atherosclerosis remains incompletely understood. This study aimed to identify and functionally characterize novel lncRNAs that regulate macrophage inflammatory responses during early atherogenic events.

Methods: Human monocyte-derived macrophages were stimulated with oxidized low-density lipoprotein (ox-LDL; 50 µg/mL) for 24 hours to mimic the proatherogenic environment. Total RNA was extracted and subjected to high-throughput RNA sequencing (RNA-seq) to identify differentially expressed lncRNAs (fold change >2, p<0.05). Among the top upregulated lncRNAs, lnc-MIR4435-2HG-Δ was selected for further analysis. Gene silencing was performed using specific siRNA, and the impact on inflammatory cytokine expression (TNF-α and IL-6) was evaluated using quantitative PCR and ELISA. NF-κB activation was assessed by immunofluorescence staining and Western blot analysis for p65 nuclear translocation. Rescue experiments were conducted by reintroducing the lncRNA to validate its functional role.

Results: RNA-seq identified 57 significantly upregulated lncRNAs following ox-LDL exposure. Knockdown of lnc-MIR4435-2HG-Δ resulted in a marked reduction in TNF-α (-45%, p<0.01) and IL-6 (-38%, p<0.05) expression. This suppression was accompanied by diminished nuclear translocation of NF-κB p65, suggesting a direct link to pro-inflammatory signaling. Control lncRNA knockdown showed no significant effects. Reintroduction of lnc-MIR4435-2HG-Δ restored cytokine levels and NF-κB activation, confirming its regulatory function.

Conclusions: lnc-MIR4435-2HG-Δ is identified as a novel pro-inflammatory regulator in macrophages under ox-LDL-induced stress, acting through the NF-κB pathway. These findings suggest that targeting this lncRNA could represent a promising therapeutic strategy to mitigate early vascular inflammation and prevent atherosclerotic progression.

Keywords: lncRNAs, Atherosclerosis

MPP-07

Effects of omega-3 fatty acid supplementation on chylomicron remnant concentrations and vascular inflammatory biomarkers: a systematic review

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Objectives: Elevated chylomicron remnant concentrations and vascular inflammation are significant contributors to residual cardiovascular risk. This systematic review aims to comprehensively evaluate the effects of oral omega-3 fatty acid (omega-3 FA) supplementation on chylomicron remnant concentrations and vascular inflammatory biomarker levels in cardiovascular disease.

Methods: An extensive literature search was conducted on PubMed and Scopus databases for articles published in the last 10 years. Inclusion criteria were explicitly defined based on the PICOS framework (Population: adults ≥18 years with dyslipidemia/high cardiovascular risk/cardiovascular disease; Intervention: oral omega-3 FA supplementation (EPA, DHA, or combination); Comparator: placebo/no intervention/standard therapy; Outcome: primary: chylomicron remnant concentrations (including postprandial triglycerides, ApoB-48, TRL-P), secondary: vascular inflammatory biomarkers (hs-CRP, IL-6, TNF- α , VCAM-1, ICAM-1); Study Design: Randomized Controlled Trials (RCTs)). Journal selection followed PRISMA guidelines, followed by data extraction and synthesis.

Results: Six randomized controlled trials (RCTs) involving a total of 975 participants met the inclusion criteria. Significant reductions in postprandial triglycerides were observed in EPA groups, omega-3 ethyl ester groups, and omega-3 CA groups. Significant changes in ApoB-48 were noted in omega-3 ethyl ester groups. Significant decreases in TRL-P (triglyceride-rich lipoprotein particles) were found in both EPA-rich and DHA-rich fish oil groups. Generally, oral omega-3 FA supplementation consistently demonstrated beneficial effects in reducing chylomicron remnant concentrations or related markers. Overall, significant changes were observed in hs-CRP and IL-6. Reported side effects were generally mild and gastrointestinal-related, or no serious adverse events directly linked to the intervention.

Conclusions: Oral omega-3 fatty acid supplementation can effectively lower chylomicron remnant concentrations and shows potential for reducing vascular inflammatory biomarkers in adults with dyslipidemia or high cardiovascular risk. These findings support the crucial role of omega-3 FA in residual cardiovascular risk management.

Keywords: Omega-3 fatty acids, Chylomicron remnant, Vascular inflammation

MPP-08

Cardioprotective effects of β -Lapachone through alleviation of lipotoxicity and fibrosis in type 2 diabetic mice

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Objectives: Diabetic cardiomyopathy (DCM) is a major contributor to heart failure and mortality in patients with type 2 diabetes mellitus. This study aimed to evaluate the therapeutic potential of β -lapachone (B-Lap), a natural quinone compound known to enhance antioxidant defense, in mitigating DCM by reducing cardiac fibrosis and lipotoxicity in a mouse model of type 2 diabetes.

Methods: Male C57BL/6 mice (7 weeks old) were rendered diabetic via a high-fat diet (HFD) for 10 weeks, combined with low-dose streptozotocin (40 mg/kg/day intraperitoneally for 5 days). Diabetic mice were randomized into groups and treated with B-Lap at 20 mg/kg/day (DM+HK20) or 80 mg/kg/day (DM+HK80), or with metformin (200 mg/kg/day, DM+Met) as a reference drug. Non-diabetic mice (WT and WT+HK80) served as controls. Metabolic parameters, cardiac function (echocardiography), histological fibrosis (Masson's staining), CD36 expression, myocardial triglyceride content, and mitochondrial fatty acid oxidation were evaluated.

Results: B-Lap significantly improved metabolic indices including fasting glucose, HbA1c, and insulin resistance compared to untreated diabetic mice. Cardiac function was restored, with reductions in heart weight and improved ejection fraction. B-Lap markedly reduced collagen accumulation in cardiac tissue and suppressed CD36 expression, leading to decreased lipid uptake and intracellular triglyceride storage. Enhanced mitochondrial fatty acid oxidation was also observed, especially at the 80 mg/kg dose, which showed comparable or superior effects to metformin across most endpoints.

Conclusions: β -Lapachone effectively attenuates diabetic cardiomyopathy in type 2 diabetic mice by alleviating cardiac fibrosis and lipotoxicity. These findings support its potential as a novel therapeutic agent for the management of diabetic heart disease, warranting further investigation in translational and clinical contexts.

Keywords: B-lapachone, Diabetic cardiomyopathy, Fibrosis

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Moderated Poster Presentation 2

Sep 13(Sat) 13:30–14:30 | Studio 5 (6F)

CHAIRPERSON : Jong-Chan Youn (The Catholic University of Korea, Republic of Korea)



MPP-09

Association between COVID-19 and cardiovascular disease through small extracellular vesicles

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Objectives: Cardiovascular diseases represent one of the leading causes of mortality worldwide, with their pathogenesis typically initiated through endothelial dysfunction and inflammatory processes. Emerging evidence indicates that coronavirus disease 2019 (COVID-19) may exacerbate cardiovascular complications. This study aims to investigate the mechanistic link between COVID-19 and CVDs, focusing on small extracellular vesicles (sEVs) derived from lung epithelial cells and their microRNA (miRNA) cargo.

Methods: To mimic COVID-19 infection, recombinant SARS-CoV-2 spike protein receptor binding domain (RBD) was applied to human lung epithelial cells (Calu-3). sEVs were isolated from conditioned media of both RBD-treated (RBD-sEVs) and untreated control (X-sEVs) Calu-3 cells. Hyperlipidemic-conditioned human umbilical vein endothelial cells (HUVECs) were incubated with the isolated sEVs to assess monocyte recruitment and endothelial response. Small RNA sequencing was performed to profile sEV-associated miRNAs.

Results: There were no morphological differences observed between X-sEVs and RBD-sEVs. However, RBD-sEVs significantly enhanced monocyte recruitment and infiltration in HUVECs under hyperlipidemic conditions. miRNA sequencing revealed a marked upregulation of miR-548b-5p in RBD-sEVs compared to X-sEVs. Bioinformatic analysis identified *I κ B α* as a direct target of miR-548b-5p, implicating the NF- κ B signaling pathway. Functional assays confirmed that miR-548b-5p suppressed *I κ B α* expression at both transcriptional and translational levels, thereby activating the NF- κ B/eNOS signaling axis.

Conclusions: COVID-19 infection alters the molecular composition of lung epithelial-derived sEVs, notably upregulating miR-548b-5p. This miRNA promotes endothelial dysfunction and inflammation via NF- κ B signaling by targeting *I κ B α* . These findings suggest that sEV-carried miR-548b-5p may serve as a mechanistic link between COVID-19 and CVD pathogenesis, offering potential biomarkers or therapeutic targets.

Keywords: COVID-19, sEV, Endothelial cells, miRNA, NF- κ B/eNOS signaling

MPP-10

Trained immunity (intolerant responses) in patients with atherosclerosis and other non-infectious diseases

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Objectives: The aim of the study was to assess the incidence of trained immunity (intolerant response) in non-infectious diseases. Trained immunity as a useful tool has been widely studied in infections. However, trained immunity has been poorly studied in non-infectious diseases. The objective of our study was to identify trained immunity in macrophages of patients with various chronic inflammatory non-infectious diseases.

Methods: The study involved patients with ischemic heart disease (coronary atherosclerosis), colorectal cancer, type 2 diabetes, rheumatoid arthritis, systemic lupus erythematosus, Parkinson's disease, obesity, chronic obstructive pulmonary disease, and diverticular disease of the intestine. Monocytes were isolated from the patients' blood and cultured. On the 6th day of cultivation, monocyte-derived macrophages were stimulated with bacterial lipopolysaccharide. After 24 hours, cytokines CCL2, IL8, IL6, and IL1b were determined in the culture medium, and the cells were stimulated a second time. Cytokine secretion was determined again 24 hours after repeated stimulation. If cytokine secretion after the second stimulation was not lower than after the first, the cell response was considered intolerant.

Results: Intolerant response was detected in patients with non-infectious diseases in 51% of cases, while in patients of the control group only in 9%. We assume that, unlike infections, intolerant response (trained immunity) plays not a positive but a negative role. It is certain that intolerant response will contribute to the chronification of the inflammatory reaction and thereby create conditions for the transition of the overall protective reaction in the form of acute inflammation to a chronic form dangerous to health.

Conclusions: Thus, intolerant response is quite common in patients with non-infectious diseases. This is apparently the main cause of chronification of inflammation. Understanding the mechanism of formation of intolerant response should contribute to the development of effective approaches to diagnosis, prevention and therapy of chronic inflammatory diseases. This work was financially supported by the Russian Science Foundation (Grant number 25-15-00064).

Keywords: Immunity, Macrophages

MPP-11

LDLR-independent role of PCSK9 in post-myocardial infarction inflammatory response

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Objectives: Myocardial infarction (MI) is a leading cause of cardiovascular morbidity and mortality, primarily resulting from ischemic injury and subsequent inflammation. Proprotein convertase subtilisin/kexin type 9 (PCSK9) regulates plasma cholesterol levels by promoting lysosomal degradation of the low-density lipoprotein receptor (LDLR). While elevated PCSK9 levels correlate with an increased risk of MI, the extent to which PCSK9 contributes to post-MI pathophysiology independently of LDLR remains poorly understood. Therefore, this study aimed to delineate the LDLR-independent role of PCSK9 in the context of MI.

Methods: To investigate the role of PCSK9 in the absence of LDLR, we induced myocardial infarction (MI) in 9- to 11-week-old male LDLR^{-/-} and LDLR^{-/-} PCSK9^{-/-} mice via permanent ligation of the left anterior descending (LAD) coronary artery. Sham-operated mice served as controls. We assessed adverse cardiac remodeling by echocardiography and quantitative PCR, and analyzed immune cell dynamics using flow cytometry. To explore LDLR-independent mechanisms of PCSK9 activation, we polarized bone marrow-derived macrophages in vitro using lipopolysaccharide (LPS) and interferon- γ (IFN- γ).

Results: Compared to sham-operated mice, MI significantly reduced survival and impaired cardiac function. Post-MI, LDLR^{-/-} mice exhibited markedly elevated CCL2 expression and enhanced granulocyte (predominantly neutrophil) infiltration at the infarct zone relative to LDLR^{-/-} PCSK9^{-/-} mice. These findings suggest that PCSK9 exacerbates post-MI inflammation through LDLR-independent pathways, likely by promoting CCL2-mediated neutrophil recruitment.

Conclusions: PCSK9 exacerbates post-MI inflammation by promoting CCL2 expression and neutrophil infiltration through LDLR-independent mechanisms. Beyond its role in LDL lowering, these findings highlight PCSK9 as a potential therapeutic target for modulating inflammatory responses following myocardial infarction.

Keywords: MI, PCSK9, LDLR

MPP-12

A potential rejuvenation factor C improves metabolic dysfunctions and mitigates atherosclerosis in Ldlr KO mice

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Objectives: Atherosclerosis, an age-related disease with metabolic dysfunction and chronic inflammation, continues to be a leading cause of mortality from cardiovascular disease. In order to mitigate the progression of atherosclerosis, recent developments in regenerative medicine have concentrated on identifying extrinsic anti-aging agents that exhibit immunomodulation and metabolic balance. A potential rejuvenation factor C (PRFC refer as an alias), a member of the IL-6 cytokine family, utilizes gp130 in its receptor complex to mediate anti-inflammatory and metabolic regulatory effects. Given its anti-inflammatory and rejuvenating properties, we hypothesized that overexpression of PRFC might prevent metabolic disorders and atherosclerosis.

Methods: To evaluate this hypothesis, LDL receptor knockout mice were intravenously injected at 8 weeks of age with AAV-Ctrl or AAV-Prfc and fed a western diet for 20 weeks and sacrificed. Mice were placed in metabolic cages and allowed to acclimate for 24 hours before energy expenditure and activity were measured. Oxygen consumption (VO₂), carbon dioxide production (VCO₂), and physical activity were automatically measured for 2 days using a computer-controlled indirect calorimetry system.

Results: The results revealed that PRFC overexpression led to notable improvements in both metabolic and vascular function. DEXA and indirect calorimetry analyses revealed that mice overexpressing PRFC had lower body weight gain, reduced fat mass, and increased energy expenditure, despite consuming the same amount of food as the control group. Moreover, these mice exhibited significantly fewer atherosclerotic plaques in their aortic arch and aorta compared to the control mice. At the molecular level, PRFC overexpression reduced the expression of genes associated with the senescence-associated secretory phenotype, such as Mmp13 and Spp1. It also decreased pro-inflammatory biomarkers in the plasma.

Conclusions: These findings suggest that PRFC acts as a novel extrinsic anti-aging factor, capable of modulating immune responses and maintaining metabolic homeostasis, and a promising candidate for developing regenerative therapies targeting age-related diseases, particularly atherosclerosis.

Keywords: Atherosclerosis, Rejuvenation, Inflammation, SASP

MPP-13

Potential rejuvenation factor C overexpression as a novel anti-senescence therapeutic target for atherosclerosis prevention

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Objectives: Atherosclerosis serves as a primary contributing factor to cardiovascular disease, which remains the leading cause of mortality worldwide. Although cellular senescence is recognized as a significant contributor to atherosclerosis, the molecular mechanisms linking senescence to disease progression remain incompletely understood. This study hypothesized that overexpression of potential rejuvenation factor C (PRFC), which exhibits anti-inflammatory and metabolic regulatory effects, could prevent atherosclerosis development.

Methods: Based on this hypothesis, 8-week-old LDL receptor knockout mice were intravenously injected with AAV-Ctrl or AAV-Prfc and fed a Western diet for 20 weeks and sacrifice. QuantSeq 3' mRNA-Seq was performed on three tissues (BCA, liver, eWAT), followed by differentially expressed genes (DEG) analysis and gene set enrichment analysis (GSEA) to identify biological functions regulated by PRFC overexpression.

Results: DEG and GSEA revealed that senescence markers Mmp13 and Spp1 were commonly downregulated across all three tissues. Furthermore, PRFC overexpression was significantly associated with immune regulation, metabolic control, and cell survival pathways.

Conclusions: The anti-senescence properties of PRFC overexpression were strongly associated with attenuated atherosclerosis progression. These findings propose PRFC as a promising therapeutic candidate for cardiovascular disease intervention.

Keywords: Atherosclerosis, Rejuvenation, Senescence

MPP-14

Transcriptomic and network-based analysis reveals inflammatory and matrix-degrading pathways in atherosclerotic plaque instability

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Objectives: Atherosclerosis is a progressive vascular disease characterized by lipid-rich plaque accumulation within arterial walls. The transition from stable to unstable plaques significantly increases the risk of acute cardiovascular events, such as myocardial infarction and stroke. Plaque rupture is a major contributor to these events. Identifying molecular characteristics of unstable plaques is crucial for risk stratification and the development of targeted therapies. This study aims to identify differentially expressed genes (DEGs) and key biological pathways associated with plaque instability through transcriptomic and bioinformatic analyses.

Methods: Gene expression data from human carotid plaques (RNA-Seq dataset from GEO) were analyzed. Plaques were classified as stable or unstable based on morphology. DEGs were identified using a threshold of log2FC >1 and adjusted p-value <0.05. Protein-protein interaction networks were constructed using Cytoscape. Functional enrichment and Gene Ontology analyses were performed using DAVID, Enrichr, and clusterProfiler in R.

Results: Unstable plaques showed upregulation of genes related to inflammation and matrix degradation, including CCL19, MMP1, MMP7, MMP8, MMP9, PLAUR, ITGAM, and CD163, whereas stable plaques were enriched in structural and anti-inflammatory genes such as EFEMP1, GAS6, and DUSP26. GO enrichment revealed key processes including inflammatory response, extracellular matrix disassembly, mononuclear cell migration, and MAPK signaling regulation. Hub genes such as MMP9, MMP1, PLAUR, CD163, and ITGAM orchestrated crosstalk between immune activation and matrix remodeling.

Conclusions: Unstable atherosclerotic plaques are characterized by enhanced inflammation and matrix degradation mediated through MAPK/ERK signaling and immune cell recruitment. These findings provide insight into molecular mechanisms of plaque vulnerability and offer potential targets for predictive biomarkers and therapeutic intervention.

Keywords: Atherosclerosis, Differentially expressed genes, Inflammation, Plaque instability, Transcriptomics

MPP-15

Short variant of mitochondrial calcium uniporter forms plasma-membrane channels in human platelets

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Objectives: MCU is a pore-forming subunit of mitochondrial Ca^{2+} (mt Ca^{2+}) uniporter complex (mtCUC) located at the inner mitochondrial membrane, serving as the major mt Ca^{2+} influx pathway in various cell types, including platelets. Human MCU gene produces the canonical full-length transcript (renamed as MCU-L) and a shorter transcript (termed MCU-S). MCU-S mRNA is highly expressed in human platelets. However, the functional relevance of MCU-S in human platelets has not been fully investigated. We hypothesize that the MCU-S can form Ca^{2+} -permeable channels outside of mitochondria, such as the plasma membrane (PM) in the platelets.

Methods: Human platelets from adult healthy donors and human megakaryoblastic leukemia cell line were used for bio-mechanical, cell biological, and physiological assays. Ca^{2+} permeability via MCU-S was assessed by Ca^{2+} imaging using confocal microscopy.

Results: Expression of MCU protein and several other mtCUC components in the PM was confirmed by cell-surface protein biotinylation in human platelets. Platelets exhibited Ru360-sensitive Ca^{2+} permeabilization via PM after switching the extracellular Ca^{2+} concentration from 0 to 2 mM, even under the inhibition of the store-operated Ca^{2+} entry (SOCE). MEG-01 cells overexpressing MCU-S (but not the cells expressing GFP as a control or dominant-negative pore-forming mutant of MCU-S, termed MCU-S-DN) significantly facilitated the actin cytoskeleton reorganization in response to thrombin stimulation, which is critical for the activation of aggregation/coagulation pathways in platelets.

Conclusions: Short MCU variant can form a Ca^{2+} -permeable channel at the PM of platelets. Elucidating the role of MCU variants in platelets may provide novel insights into the molecular basis of Ca^{2+} -dependent activation of aggregation/coagulation pathways in platelets.

Keywords: CCDC109A, Calcium ion, Thrombin, Ion channel

MPP-16

CBL-b E3 ligase-mediated neddylation and activation of PARP-1 induce vascular calcification

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Objectives: Vascular calcification (VC) refers to the accumulation of mineral deposits on the walls of arteries and veins, and it is closely associated with increased mortality in cardiovascular disease patients, particularly among high-risk patients with diabetes and chronic kidney disease (CKD). Neuronal precursor cell-expressed developmentally downregulated protein 8 (NEDD8) is a ubiquitin like protein that plays a pivotal role in various cellular functions, primarily through its conjugation to target proteins and subsequent relay of biological signals. However, the role of NEDDylation in VC has not been investigated.

Methods: In our study, we observed that MLN4924, an inhibitor of the NEDD8-activating E1 enzyme, effectively impedes the progression of VC. LC-MS/MS analysis revealed that poly (ADP-ribose) polymerase 1 (PARP-1) is subjected to NEDD8 conjugation, leading to an increase in PARP-1 activity during VC.

Results: We subsequently revealed that PARP-1 NEDDylation is mediated by the E3 ligase CBL proto-oncogene B (CBL-b) and is reversed by NEDD8-specific protease 1 (NEDP-1) during VC. Furthermore, the CBL-b C373 peptide effectively mitigated the inactive form of the E3 ligase activity of CBL-b, ultimately preventing VC.

Conclusions: These findings provide compelling evidence that the NEDD8-dependent activation of PARP-1 represents a novel mechanism underlying vascular calcification and suggests a promising new therapeutic target for VC.

Keywords: Vascular calcification, PARP-1, NEDD8

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Moderated Poster Presentation 3

Sep 12(Fri) 14:40–15:40 | Studio 6 (6F)

CHAIRPERSON : Jaehoon Chung (Dongguk University, Republic of Korea)



MPP-17

Adherence to statins: a cornerstone for preventing atherosclerotic cardiovascular disease and mortality

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Objectives: Adherence to lipid-lowering therapy (LLT), especially statins, is essential for preventing ASCVD events, with good adherence defined as taking $\geq 75\%$ of prescribed doses. However, a comprehensive quantification of its impact has been lacking. Our meta-analysis aimed to evaluate how consistent statin adherence influences ASCVD risk and mortality.

Methods: We conducted a systematic review of PubMed through early 2025, identifying randomized trials, prospective cohort, and case-control studies evaluating LLT adherence and ASCVD outcomes. A random-effects meta-analysis was used to assess the relationship between adherence levels and ASCVD risk and mortality.

Results: 66 articles representing 3,345,718 people were included. Statins were assessed as LLT in every study. The risk for all-cause mortality (RR:0.65; 95%CI: 0.56-0.76; I²=98%), any cardiovascular (CV) event (RR:0.76; 95%CI: 0.72-0.80; I²=90%), myocardial infarction (MI) (RR:0.70; 95%CI: 0.62-0.80; I²=90%), and stroke (RR:0.68; 95%CI: 0.58-0.79; I²=81%) was lowered by 35% when statin adherence was good compared to poor. Statin adherence decreased the risk for any CV incident by 32% for persons without established ASCVD (RR:0.68; 95%CI: 0.46-1.00; I²=97%). Statin adherence decreased the risk of any CV incident by 25% (RR:0.75; 95%CI: 0.64-0.88; I²=64%), MI by 51% (RR:0.49; 95%CI: 0.32-0.73; I²=80%), and stroke by 50% (RR:0.50; 95%CI: 0.26-0.97; I²=87%) for people with established ASCVD. The mortality risk increased by 90% when statins were stopped (RR:1.90; 95%CI: 1.33-2.71; I²=87%). The risk for any CV incident was reduced by 32% even for good adherence compared to moderate adherence (40-79%) (RR:0.68; 95%CI: 0.60-0.76; I²=94%). The primary findings were not affected by sensitivity analyses that included studies that did not meet the conventional ($\geq 80\%$) threshold.

Conclusions: Our study confirms that adherence to statin therapy significantly reduces ASCVD risk and mortality, emphasizing the need for targeted interventions to improve patient compliance.

Keywords: Atherosclerotic cardiovascular disease (ASCVD), Lipid-lowering therapy (LLT), Statin adherence, Cardiovascular mortality, Preventive cardiology, Cardiovascular risk reduction

MPP-18

PCSK7 drives post-infarction inflammation via TNF- α /JNK activation in cardiac macrophages

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Objectives: Myocardial infarction (MI), a major manifestation of coronary artery disease (CAD), elicits a robust inflammatory response that contributes to increased cell death and adverse ventricular remodeling. Despite current therapeutic options, MI remains a leading cause of heart failure, underscoring the need to better understand immune mechanisms driving its progression. Proprotein convertase subtilisin/kexin type 7 (PCSK7), expressed in various tissues and immune cells, has been implicated in cardiovascular diseases. However, its role in cardiac immune cells remains largely unexplored. This study aimed to elucidate the function of PCSK7 in the inflammatory response following MI and provide new insights into its pathological relevance.

Methods: To investigate the role of PCSK7 in cardiac inflammation and remodeling, we induced MI in *Pcsk7*^{-/-} mice via surgical ligation of the left anterior descending (LAD) coronary artery. We measured plasma lipid profiles to assess the systemic impact of PCSK7 deficiency and performed flow cytometry to analyze immune cell populations and macrophage lipid uptake in the heart. Immunofluorescence staining and quantitative PCR (qPCR) were used to evaluate cardiac remodeling during post-MI recovery. Additionally, we investigated intracellular signaling pathways involved in the inflammatory response.

Results: PCSK7 deficiency led to reduced circulating cholesterol levels, which may contribute to smaller infarct size and improved cardiac function. Conversely, PCSK7 promoted macrophage activation and lipid uptake at the ischemic site, exacerbating cardiac injury. Mechanistically, we found that intracellular PCSK7 activates the TNF- α /JNK signaling pathway in macrophages, enhancing the inflammatory response following MI.

Conclusions: PCSK7 plays a dual role in cardiovascular disease by modulating both lipid metabolism and immune responses. Targeting PCSK7 in macrophages may offer a promising therapeutic strategy to mitigate post-MI inflammation and improve outcomes in patients with CAD.

Keywords: Myocardial infarction, Proprotein convertase subtilisin/kexin type 7, Cardiac macrophages, Tumor necrosis factor- α

MPP-19

Corticosterone mitigates stroke-induced injury via upregulation of antioxidant Prdx1

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Objectives: Ischemic stroke leads to excessive generation of reactive oxygen species (ROS), contributing to neuroinflammation and cellular damage. Microglia, the resident immune cells of the brain, are particularly vulnerable to oxidative stress. Peroxiredoxins (Prdxs), a family of antioxidant enzymes, play a critical role in limiting ROS-mediated damage; Prdx deficiency exacerbates stroke-induced injury. Corticosterone, a glucocorticoid hormone released via the hypothalamic-pituitary-adrenal (HPA) axis in response to stress, is known for its immunosuppressive and anti-inflammatory properties. Dysregulation of this axis has been linked to increased susceptibility to neurodegenerative conditions. This study explores how corticosterone modulates microglial responses in a Prdx-deficient mouse model of stroke.

Methods: To compare stroke severity in the absence of antioxidant enzymes, we induced transient middle cerebral artery occlusion (tMCAO) in wild-type (WT), Prdx1 knockout (Prdx1KO), and Prdx2 knockout (Prdx2KO) mice. To explore potential mechanisms underlying observed differences, we quantified hypothalamic-pituitary-adrenal (HPA) axis hormones using ELISA. Additionally, to assess the effect of elevated corticosterone under oxidative stress conditions, we treated BV2 mouse microglial cells with corticosterone and subjected them to oxygen-glucose deprivation (OGD), an in vitro model of ischemic stroke.

Results: Prdx1 deficiency resulted in significantly larger infarct volumes compared to WT mice, as assessed by TTC staining, whereas Prdx2 deficiency showed no notable difference. Neurological deficits were also more pronounced in Prdx1KO mice. Interestingly, corticosterone levels were significantly elevated in Prdx2KO mice compared to Prdx1KO mice and remained high into the subacute phase, during which post-stroke inflammation is prominent. In vitro, corticosterone treatment attenuated microglial inflammatory responses and upregulated Prdx1 expression. Furthermore, crystal violet staining revealed that corticosterone enhanced microglial cell viability under oxidative stress conditions.

Conclusions: During ischemic stroke, microglia are subjected to ROS-induced oxidative stress, leading to inflammation and cell death. Elevated corticosterone levels—particularly in the absence of Prdx2—appear to alleviate inflammation and upregulate Prdx1 expression, suggesting a protective role.

Keywords: Stroke, Microglia

MPP-20

Pharmacological assessment of low-dose oral docetaxel for atheroprotection

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Objectives: Our identification of desmocollin 1 (DSC1) as a novel negative regulator of high-density lipoprotein (HDL) biogenesis was followed by the discovery that docetaxel (DTX), an FDA-approved chemotherapy drug, inhibits DSC1 in this pathway. Due to poor oral bioavailability, chemotherapy doses of DTX (1.9–5.1 μ M) are typically administered intravenously. Our findings indicate that approximately 1,000-fold lower, low-nanomolar concentrations are sufficient to promote HDL biogenesis and reduce atherosclerosis without causing cytotoxicity. Based on these results, we investigated the feasibility of developing DTX as a low-dose oral therapy for atherosclerosis.

Methods: Subcutaneous implantation of osmotic pumps in ApoE^{-/-} mice enabled stable delivery of low-nanomolar, atheroprotective doses of DTX. To evaluate whether comparable blood concentrations could be achieved orally, we examined the pharmacokinetics and safety profile of oral DTX administration in ApoE^{-/-} mice.

Results: Continuous DTX delivery via osmotic pumps maintained blood concentrations between 2.7 and 4.3 nM, reducing circulating atherogenic lipids—including triglycerides, low-density lipoprotein cholesterol, and total cholesterol—while increasing the HDL cholesterol/total cholesterol ratio. These effects were associated with a significant reduction in atherosclerotic lesion formation. Among oral formulations tested, DTX at 6 mg/kg in dimethyl sulfoxide/polyethylene glycol 300/polysorbate 80/water achieved a maximum plasma concentration of 6.7 nM and a half-life of 21.4 hours. Plasma concentration-time data were fitted to a three-compartment model and used to simulate DTX exposure under various dosing scenarios. Simulations predicted that twice-daily dosing at 6 mg/kg would sustain plasma concentrations within the 1.5 to 8.2 nM range, encompassing the atheroprotective levels achieved with pump-based delivery (2.7–4.3 nM). This oral regimen produced no signs of hematologic toxicity or histopathological abnormalities in major organs, including the stomach, intestines, liver, and kidneys.

Conclusions: These findings suggest that the low-nanomolar potency of DTX in reducing atherosclerosis may overcome its poor oral bioavailability, supporting its development as a low-dose oral therapy for atherosclerosis.

Keywords: Desmocollin 1, Docetaxel, Cholesterol, High-density lipoprotein, Atherosclerosis

MPP-21

The Interplay between oxidative stress and cholesterol in non-alcoholic steatohepatitis (NASH): a key to understanding disease pathogenesis

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Objectives: Non-Alcoholic Steatohepatitis (NASH) is a progressive form of NAFLD characterized by inflammation and fibrosis. Oxidative stress and lipid metabolism alterations are thought to play a crucial role in NASH pathogenesis. This study aimed to investigate the relationship between oxidative stress markers and cholesterol metabolism in NASH patients with type 2 diabetes mellitus (T2DM).

Methods: In this case-control study, 150 participants were enrolled and classified into two groups: NASH with T2DM (n=100) and healthy controls (n=50). Levels of oxidative parameters, including superoxide dismutase (SOD), catalase, and malondialdehyde (MDA), were assessed using chemiluminescence and qRT-PCR and lipid profiles (e.g., total cholesterol, LDL-C, HDL-C) were measured. Data analysis was performed using GraphPad Prism 10, with significance set at $P < 0.05$.

Results: The study demonstrated a significant decrease in SOD2 enzyme activity in NASH with T2DM patients (1.008 ± 0.957 U/ml) compared to controls (4.029 ± 3.20 U/ml, $P = 0.0014$). Catalase activity was also significantly reduced in NASH with T2DM patients (32.2 ± 6.36 U/ml) compared to controls (41.2 ± 8.70 U/ml, $P < 0.0001$). Plasma MDA levels were significantly increased in NASH patients with T2DM. Gene expression analysis revealed that catalase was significantly down-regulated by 1.94-fold ($P = 0.0045$) and SOD2 by 1.02-fold ($P = 0.46$) in NASH with T2DM patients compared to healthy controls. We found a positive correlation between oxidative stress markers and cholesterol levels, particularly LDL-C, which was significantly elevated in cases compared to controls.

Conclusions: Our study highlights the complex interplay between oxidative stress and cholesterol in NASH. The findings suggest that oxidative stress may contribute to the development of dyslipidemia in NASH patients, and that targeting oxidative stress may be a potential therapeutic strategy for managing NASH.

Keywords: NASH, Oxidative stress, Cholesterol, Lipid metabolism

MPP-22

Moringa oleifera root extracts reduces the apoptotic activity in pancreatic Langerhans cells of wistar rats induced metabolic syndrome

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Objectives: Overnutrition and lifestyle changes in modern society increase the prevalence of metabolic syndrome. Metabolic syndrome causes death of langerhans cells through apoptosis. Moringa oleifera extracts have been reported to reduce apoptotic activity. This study aims to examine the effect of moringa root extract on caspase-3 as indicator of langerhans cells apoptosis.

Methods: Subjects were divided into five groups. G1 (negative control) was given standard feed, meanwhile G2 (positive control), G3, G4, and G5 were given high-fat feed and Streptozotocin-Nicotinamide injection. Ethanolic extracts of moringa root were given to treatment group G3, G4, and G5 with doses of 150 mg/kg, 250 mg/kg, and 350 mg/kg. Observation of caspase-3 expression was carried out after intervention. Data analysis aims to test the differences in caspase-3 expression using Kruskal-Wallis test and post hoc Mann-Whitney test. The relationship between doses and caspase-3 expression was assessed by a linear regression test.

Results: There is a significant difference ($p < 0.05$) in caspase-3 expression between G2 (positive control) and G1 (negative control), also between G2 (positive control) and G5 (treatment group at 350 mg/kg doses). There is no significant difference between G2 and G3, G2 and G4, and between G1 and G5. A significant decrease of caspase-3 expression in langerhans cells was shown by administering moringa root extract at a dose of 350 mg/kg ($p = 0.01$). The linear regression test showed a negative regression relationship between dose of moringa oleifera root extract and caspase-3 expression at langerhans cells ($R^2 = 0.44$).

Conclusions: Moringa root extracts decreased the caspase-3 expression in langerhans cells. Increasing the dose of moringa root extracts reduces the level of caspase-3 expression at langerhans cells. This study's findings indicate that Moringa root extract could serve as a promising new alternative therapy to help reduce the apoptotic activity in pancreatic langerhans cells.

Keywords: Caspase-3, Pancreas, Metabolic syndrome, Moringa root extract

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Moderated Poster Presentation 4

Sep 13(Sat) 13:30–14:30 | Studio 6 (6F)

CHAIRPERSON : Jun Hwa Hong (Eulji University, Republic of Korea)



MPP-26

Different effect of ACE-I vs ARB on TNF- α , IL-1 β , and IL-6 level in type-4 cardio-renal syndrome secondary to diabetic kidney disease

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Objectives: To analyze the difference of Angiotensin Converting Enzyme Inhibitors (ACE-I) compared to Angiotensin Receptor Blockers (ARB) in affecting inflammatory cytokines level including TNF- α , IL-1 β , and IL-6 in patients with Type 4 Cardiorenal Syndrome (CRS) caused by Diabetic Kidney Disease (DKD).

Methods: It was a prospective cohort study with total sampling technique. There were 60 newly diagnosed Type 4 CRS patients (fasting blood glucose >126 mg/dl; eGFR <0.05).

Results: The effect of ACE-I and ARB from pre- to post-intervention within each group, analyzed using the Wilcoxon signed-rank test, was significant in both groups for TNF- α ($p < 0.000$ and $p = 0.002$), IL-1 β ($p = 0.011$ and $p = 0.008$), and IL-6 ($p = 0.006$ and $p = 0.032$). The post-intervention differences between the two groups, assessed using the Mann-Whitney test, were significant for TNF- α ($p = 0.021$) and IL-6 ($p = 0.009$), but not for IL-1 β ($p = 0.064$).

Conclusions: Both ACE-I and ARB significantly reduce TNF- α , IL-1 β , and IL-6 levels in patients with Type 4 CRS secondary to DKD, while ACE-I is significantly more effective than ARB in reducing TNF- α and IL-6 levels.

Keywords: Type 4 cardiorenal syndrome, Angiotensin converting enzyme inhibitors, Angiotensin receptor blockers

MPP-27

Factors associated with vertebral metabolic activity and its implications for cardiovascular disease

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Objectives: Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality, and vertebral metabolic activity measured by fluorodeoxyglucose positron emission tomography (FDG-PET) is increasingly recognized as an indicator of bone marrow activity representing inflammation, and hematopoiesis associated with CVD. However, its broader physiological determinants and precise relationship to cardiovascular risk are not fully understood. We aimed to clarify the interplay among vertebral metabolic activity, systemic inflammation, hematopoiesis, and body composition, and refine its role as an informative marker of cardiovascular risk.

Methods: This retrospective study analyzed data from 252 healthy Asian adults who underwent 18F-FDG PET/CT and dual-energy X-ray absorptiometry (DXA) between 2016 and 2022. Associations between vertebral metabolic activity, demographic characteristics, body composition and anthropometric indicators, laboratory data and cardiovascular risk scores (SCORE2 Asia-Pacific, WHO 10-year CVD risk, and Framingham Risk Score) were assessed using correlation, linear regression, and path analysis.

Results: In correlation analysis, higher vertebral metabolic activity was positively associated with skeletal muscle mass, spine bone mineral density (BMD), platelet count, neutrophil-to-lymphocyte ratio (NLR), and negatively associated with age and cardiovascular risk scores. These relationships were distinct from those observed with other FDG-PET-derived markers such as spleen and amygdala activity. Multivariable linear regression analysis identified younger age, female sex, higher skeletal muscle mass, spine BMD, and elevated inflammatory profiles as independent predictors of increased vertebral metabolic activity. Path analysis revealed complex relationships, including direct positive effects of skeletal muscle mass, NLR, and spine BMD on vertebral metabolic activity, and direct and indirect negative effects of age, and direct positive and indirect negative effects of female sex.

Conclusions: Factors related to vertebral metabolic activity are diverse including aging, sex, hematopoiesis and musculoskeletal status. Further studies across diverse populations are warranted to confirm these findings and clarify its clinical applicability in cardiovascular risk evaluation.

Keywords: Atherosclerosis, FDG-PET, Vertebral metabolic activity, Metabolic activity, Cardiovascular disease risk

MPP-28

Cardiovascular health by life's essential 8 and chronic kidney disease: Korea national health and nutrition examination survey 2019-2021

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Objectives: To investigate the association between cardiovascular health (CVH), as defined by Life's Essential 8 (LE8), and chronic kidney disease (CKD), and to examine whether this association persists across CKD indicators and stages.

Methods: We analyzed data from 12,264 adults in the Korea National Health and Nutrition Examination Survey (VIII, 2019-2021). LE8 scores (range, 0-100), calculated from 8 components, were analyzed as both continuous and categorical variables: low (0-<50), moderate (50-<80), and high CVH (80-100). CKD was defined as an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m² or albuminuria (urine albumin-to-creatinine ratio ≥30 mg/g). Multivariable logistic regression and restricted cubic spline models were used to examine associations between LE8 scores and CKD, with analyses stratified by CKD stages.

Results: Overall, 13.3% of participants were classified as having low CVH, 75.4% as moderate CVH, and 11.3% as high CVH. Compared to low CVH, the odds of CKD were lower in moderate CVH (odds ratio [OR], 0.39; 95% confidence interval [CI], 0.33-0.46) and high CVH (OR, 0.22; 95% CI, 0.15-0.33). Each 10-point higher CVH score was associated with 33% lower odds of CKD (OR, 0.67; 95% CI, 0.63-0.71). After stratifying decreased eGFR and albuminuria by G and A stages, higher CVH was consistently associated with lower odds of CKD, even for early stages.

Conclusions: Higher LE8 scores were inversely associated with CKD and its indicators, including early-stage CKD. These findings underscore the potential value of the LE8 framework in early intervention and prevention for CKD.

Keywords: Cardiovascular health, Life's essential 8, Chronic kidney disease, Primordial prevention, Risk factor

MPP-29

Repeated non-invasive limb ischemic preconditioning protects against myocardial ischemia-reperfusion injury in type 1 diabetic rats via KLK12 signaling activation

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Objectives: Diabetes mellitus exacerbates myocardial ischemia-reperfusion injury (MIRI). While repeated non-invasive limb ischemic preconditioning (rNLIP) shows therapeutic potential, its protective mechanisms in diabetes remain largely unknown. This study aimed to investigate whether rNLIP confers cardioprotection against MIRI in type 1 diabetes and to elucidate its underlying molecular mechanisms.

Methods: Type 1 diabetes was induced in rats using streptozotocin. rNLIP was performed on the left hindlimb (4 cycles of 5-min ischemia/5-min reperfusion per session) for 3 consecutive days. Twenty-four hours after the last rNLIP session, MIRI was induced by left anterior descending coronary artery ligation (30-min ischemia followed by 2-h reperfusion). Myocardial infarct size (assessed by TTC/Evans Blue staining), apoptosis (Western blot), and inflammation (RT-qPCR) were evaluated. RNA-seq was conducted on myocardial tissue to identify differentially expressed genes and potential mechanisms through GO and KEGG analysis. In vitro, H9C2 cardiomyocytes under high glucose conditions underwent simulated rNLIP (sRIPC) followed by 6-h hypoxia/12-h reoxygenation (H/R) injury. siRNA technology was employed to knockdown a key target gene identified by RNA-seq, and its functional role was assessed by measuring cell viability, injury markers, oxidative stress, and target expression.

Results: rNLIP treatment significantly reduced myocardial infarct size and attenuated apoptosis following MIRI in diabetic rats. RNA-seq analysis identified Kallikrein-related peptidase 12 (KLK12), a serine protease with emerging roles in cardiovascular regulation, as a key upregulated gene associated with rNLIP protection. This KLK12 upregulation was confirmed at both mRNA and protein levels. Functional validation in vitro demonstrated that KLK12 knockdown using siRNA abolished the protective effect of sRIPC against H/R-induced injury in high glucose-treated H9C2 cells, exacerbating cellular damage, oxidative stress, and apoptotic pathway activation.

Conclusions: These findings provide new insights into the molecular basis of rNLIP-mediated cardioprotection in diabetes and highlight KLK12 as a potential therapeutic target for mitigating diabetic MIRI.

Keywords: Myocardial ischemia-reperfusion injury, KLK12, rNLIP

MPP-30

Novel mechanism of KAI1 in angiogenesis inhibition: expression in PVCs and VEGF

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Objectives: This study investigates a novel anti-angiogenic mechanism mediated by KAI1 (CD82), which is predominantly expressed in perivascular cells (PVCs). Beyond its role in transcriptional regulation via LIF induction, KAI1 directly sequesters key pro-angiogenic ligands—VEGF-A and PDGF-BB—thereby hindering receptor-mediated signaling. A 20-mer peptide derived from KAI1's large extracellular loop (LEL) was also evaluated for its potential as a minimal, targeted therapeutic agent in VEGF-driven vascular disorders.

Methods: We employed biochemical assays including surface plasmon resonance and co-immunoprecipitation to confirm that KAI1 binds VEGF-A and PDGF-BB in a lipid raft-dependent manner—a process reliant on palmitoylation. Sequence mapping of KAI1 LEL revealed that amino acids 166-185 are critical for ligand binding. A 20-mer peptide mimicking this region was synthesized, and its binding specificity was validated by comparison to a mutant peptide with alanine substitutions at predicted key residues. The in vivo efficacy of these peptides was assessed using a retinal neovascularization model in neonatal mice with oxygen-induced retinopathy.

Results: Our data show that KAI1 directly binds and sequesters VEGF-A and PDGF-BB, preventing their interaction with VEGFR2 and PDGFR β , thereby reducing downstream phosphorylation and angiogenic signaling in endothelial cells. Importantly, the 20-mer peptide maintained this ligand-quenching function. In vivo, treatment with the wild-type peptide significantly reduced pathological neovascularization, whereas the mutant peptide exhibited no such effect, underscoring the sequence specificity of the interaction.

Conclusions: KAI1 in PVCs acts as a dual-function angiogenesis inhibitor by both promoting anti-angiogenic transcriptional regulation and exerting direct extracellular sequestration of angiogenic ligands. The efficacy of the LEL-derived peptide highlights its therapeutic potential for VEGF/PDGF-mediated conditions, offering a compact and targeted strategy to combat pathological angiogenesis.

Keywords: CD82, Angiogenesis, Pericyte

MPP-31

Hidden cardiovascular risks in cancer survivors: atherosclerosis-related mortality trends with implications for lipid-centric surveillance (population based study)

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Objectives: Cancer survival has improved globally, yet a growing number of survivors face an underrecognized risk: atherosclerotic cardiovascular disease (ASCVD). Inflammation, endothelial dysfunction, and dyslipidemia—central to both atherosclerosis and tumor biology—create a bidirectional risk pathway. We analyzed national data to quantify atherosclerosis-related mortality across cancer types and highlight disparities that warrant lipid-centered surveillance.

Methods: We used the SEER 18 database to identify patients aged ≥ 18 years diagnosed with solid and hematologic malignancies between 2000 and 2020. The primary outcome was death attributed to ASCVD (ischemic heart disease, cerebrovascular disease, peripheral arterial disease), stratified by cancer type, age, sex, and race. Time-dependent mortality trends were compared with U.S. age-adjusted cardiovascular mortality rates. Cox models adjusted for treatment type (radiation, chemotherapy) and cancer stage were used to assess predictors of ASCVD death.

Results: Out of 2.4 million cancer patients, 186,320 (7.7%) died from ASCVD, making it the second leading cause of death after cancer itself. Breast (SMR: 1.35) and lymphoma patients (SMR: 1.68) exhibited the highest relative risk, particularly women under 55. Radiation to the chest and anthracycline-based chemotherapy increased ischemic mortality by 42% (HR: 1.42; 95% CI: 1.34-1.51). African American patients had 1.5-fold higher ASCVD mortality vs. Whites (p

Conclusions: Atherosclerosis is an emerging long-term threat among cancer survivors, shaped by treatment exposures, biological overlap, and systemic inflammation. This calls for urgent cardio-oncology-lipidology collaboration to develop survivor-specific ASCVD screening and statin-based prevention protocols. Lipid profiling should be integrated into long-term oncology follow-up, especially for young, female, and minority patients with high-risk cancer histories.

Keywords: Atherosclerosis, Cancer, Cardio-oncology

MPP-32

Machine-learning-assessed abdominal aortic calcification and the brain: associations with brain imaging markers and incident dementia in the UK Biobank

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Objectives: Abdominal aortic calcification (AAC), a marker of systemic atherosclerosis, has been associated with an increased risk of dementia in older women. We examined whether machine-learning assessed AAC-24 scores (ML-AAC24) from lateral spine DXA images, using our previously validated algorithm, is associated with neuroimaging markers and incident dementia in the UK Biobank Imaging Study.

Methods: Among 49,679 participants (mean age 64.6±7.8 years; 51.4% women) ML-AAC24 was graded as low (<2), moderate (2-5) or high (6+). Outcomes included MRI-derived neuroimaging (total brain volume [TBV], total hippocampal volume [THV], white-matter-hyperintensity [WMH] volume) and incident all-cause dementia. Linear and Cox regression models were applied for cross-sectional imaging and prospective dementia, respectively, adjusting for demographic, prevalent disease, lifestyle, and genetic factors.

Results: Compared with low ML-AAC24, moderate and high ML-AAC24 were associated with significantly smaller covariate-adjusted TBV (moderate: -3,242 mm³; high: -6,037 mm³; both p<0.001) and THV (moderate: -31.5 mm³, p=0.010; high: -101.1 mm³, p<0.001). WMH burden progressively increased across ML-AAC24 grades (adjusted exp β: moderate 1.09, high 1.20; both p<0.001). Over a median follow-up of 4.1 years, moderate (adjusted HR 1.94, 95%CI 1.30-2.88) and high ML-AAC24 (HR 1.85, 95%CI 1.07-3.18) were independently associated with incident dementia (130 events), compared to low ML-AAC24. All the associations remained statistically significant in sensitivity analyses excluding individuals with prevalent nervous system neoplasms, neurological and psychiatric disorders, traumatic brain injury, stroke, or ASCVD. Results were also robust to additional adjustments for statin use and antihypertensive medications.

Conclusions: Middle-aged and older adults with moderate to high ML-AAC24 exhibit smaller brain volumes, greater WMH burden, and nearly double the risk of incident dementia. ML-AAC24 scoring on bone density images could serve as an inexpensive biomarker for cognitive health, enabling earlier targeted dementia risk reduction and prevention strategies.

Keywords: Vascular calcification, Cardiovascular diseases, Brain, Cognition, Dementia, Machine learning

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Moderated Poster Presentation 5

Sep 12(Fri) 14:40–15:40 | Studio 7 (6F)

CHAIRPERSON : Hyun-Jin Kim (Hanyang University, Republic of Korea)



MPP-33

Global trends of cardiovascular burden and its metabolic risk factors in adolescent and young adult in Southeast Asia: insights from the global burden of disease study

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Objectives: Cardiovascular disease (CVD) remains the major issue and is the main cause of death in the global space. As half of the world's population is living in Asia and the young population tends to have a bad lifestyle, there is a growing concern among young adults for CVD occurrences. This study aimed to investigate global trends of CVD in adolescents and young adults from 1990 to 2021 in the Asia region.

Methods: We obtained the data from the Global Burden Disease (GBD) Study 2021. We extracted and evaluated data of adolescent and young adult Asian population, including, prevalence rates, mortality, years-life death (YLDs) rate, and mortality rate between 1990 and 2021. We then estimated and assessed attributable risk factors of CVD. Counts and rates per 100,000 population along with a 95% uncertainty interval (95% UI) were reported and compared.

Results: The prevalence of cardiovascular diseases in adolescents and young adults increased from 10 million to 11 million between 1990 and 2021. In 2021, the top three countries have highest prevalence, including Iran (1799 cases per 100,000), Mongolia (1,689 cases per 100,000), and Turkmenistan (1,668 cases per 100,000). The YLD's rate decreased from 80.58 (UI 56.93-109.94) to 73.84 (UI 51.8-103.79) between 1990 and 2021. The mortality rates increased from 5.7% (UI 5.36%-6.10%) to 6.82% (UI 6.44%-7.18%). High blood pressure is the highest risk factor in 1990, followed by high body mass index. Meanwhile, high body mass index became the highest risk factor in 2021, followed by high blood pressure.

Conclusions: The global trends of CVD increased in adolescents and young adults along with the increase of mortality rates. Timely and effective prevention strategies addressing young population are needed to reduce the CVD burden and achieve better health outcomes.

Keywords: Cardiovascular disease, Global burden disease, Metabolic risk factors

MPP-34

Disease burden of cardiovascular disease and metabolic risk factors among elderly in Asian population: a systematic analysis from the global burden of disease study 2021

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Objectives: Cardiovascular disease (CVD) is a major health crisis for Asia's aging population, causing high and rising rates of death and disability among the elderly, especially in less developed countries. In 2019, there were 28,058 CVD cases, 1,196 deaths, and 22,858 disability-adjusted life years (DALY) rate per 100,000 elderly individuals in Asia. This study aimed to evaluate the burden of CVD among the elderly population in Asia.

Methods: The Global Burden of Disease (GBD) Study 2021 tools were used to evaluate the CVD burden of the elderly population in Asia. A systematic analysis was performed to estimate the outcomes, including the prevalence, mortality, years of life lived with disability (YLD), and DALYs rate among older people. The 95% uncertainty intervals (UI) were calculated for final estimates of the outcomes.

Results: The prevalence rate of CVD per 100,000 population among the elderly in Asia was 28,058 (95% UI 25,582-30,482). The mortality rates were 1,196 and DALYs rates were 22,858 per 100,000 population. Meanwhile, YLD rates were 1,451 (95% UI 1,050-1,874). In elderly, several risk factors were found, with the highest prevalence, including high blood pressure, high LDL, high fasting plasma glucose, kidney dysfunction, and high body mass index, respectively. By sex group, the male group was found to have a higher mortality risk with 891 deaths, compared with the female group, with a total of 774.9 deaths per 100,000 population.

Conclusions: CVD remains a major health burden for elderly Asians, with high rates of prevalence, mortality, and disability. Major risk factors include high blood pressure, pollution, high LDL cholesterol, high blood sugar, kidney dysfunction, smoking, and high BMI. Targeted interventions and public health strategies are needed to address modifiable risk factors and reduce the burden of CVD among Asia's aging population.

Keywords: Cardiovascular disease, Risk factors, Elderly

MPP-35

Burden of systemic inflammation and associated health outcomes in adults with atherosclerotic cardiovascular disease managed in routine care

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Objectives: The burden and outcomes of inflammation in people with atherosclerotic cardiovascular disease (ASCVD) are poorly defined, particularly beyond the controlled settings of trials and research cohorts.

Methods: We conducted a longitudinal observational study of adults with ASCVD undergoing C-reactive-protein (CRP) testing in routine healthcare in Stockholm, Sweden. After excluding CRP tests associated with acute illness and patients with medications/conditions that bias CRP interpretation, the systemic inflammation of participants was defined over a 3-month ascertainment window. Baseline determinants of CRP ≥ 2 mg/L were explored with logistic regression, and baseline CRP categories were compared via Poisson and Cox regression for subsequent healthcare resource utilization and occurrence of major adverse cardiovascular events (MACE), heart failure hospitalization, and all-cause death.

Results: After applying inclusion/exclusion criteria, we identified 84,399 adults with ASCVD with a mean age of 71 years and of which 54% were men. In total, 60% had CRP ≥ 2 mg/L. At baseline, female sex, older age, lower kidney function, albuminuria, diabetes, hypertension, and recent anemia, were associated with CRP ≥ 2 mg/L. Conversely, the use of RASi, antiplatelets, and lipid-lowering therapy were associated with lower odds. Over a median follow-up of 6.4 years and compared to people with CRP < 2 mg/L, those with CRP ≥ 2 mg/L had a higher rate of hospitalizations, days spent in hospital, outpatient consultations, and dispensed medications during follow-up ($P < 0.05$ for all). They also had a higher rate of MACE [adjusted hazard ratio (HR), 1.30; 95% CI, 1.27-1.33], heart failure hospitalization [1.24; 1.20-1.39], and all-cause death [1.35; 1.20-1.30]. Results were consistent across subgroups and more granular CRP categories, and robust to the exclusion of extreme CRP values or early events.

Conclusions: Two in three adults with ASCVD have systemic inflammation. A CRP ≥ 2 mg/L is associated with excess healthcare resource utilization as well as increased rates of MACE, heart failure, and death.

Keywords: CRP ≥ 2 mg/L

MPP-36

Insulin resistance assessed by the triglyceride-glucose index and left-ventricular function in non-diabetic ST-elevation myocardial infarction patients: an echocardiographic study from the UK Biobank

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Objectives: The triglyceride glucose (TyG) index is a simple surrogate for insulin resistance (IR) and has been linked to adverse cardiovascular outcomes. We investigated the relationship between TyG defined IR and echocardiographic left ventricular (LV) parameters, as well as its association with mortality, in non diabetic patients presenting with acute ST segment elevation myocardial infarction (STEMI).

Methods: We analyzed 23,780 STEMI cases from the UK Biobank (mean age $\pm$ SD, 60.8 $\pm$ 12.0 years; 29.4 % women). After excluding individuals with diabetes, 19,901 (84.3%) participants remained; 962 of them had complete echocardiographic data. Linear regression models adjusted for age, sex, and BMI assessed associations between TyG index and LV ejection fraction (LVEF), LV end diastolic volume (LVEDV), LV end systolic volume (LVESV), stroke volume (SV), cardiac output (CO), and cardiac index (CI). Cox proportional hazards models evaluated all cause mortality.

Results: In the echocardiography subgroup (n=962), the mean $\pm$ SD TyG index was 8.83 $\pm$ 0.54. TyG was inversely associated with LVEF ($\beta = -1.25\%$ per unit; $p = 0.014$) and positively with LVEDV ($\beta = 5.12$ mL; $p = 0.008$), LVESV ($\beta = 4.48$ mL; $p = 0.011$), SV ($\beta = 0.63$ mL; $p = 0.032$), CO ($\beta = 0.12$ L $\cdot$ min⁻¹; $p = 0.021$), and CI ($\beta = 0.06$ L $\cdot$ min⁻¹ $\cdot$ m⁻²; $p = 0.039$). TyG correlated weakly with post MI survival days (Spearman $r = 0.03$, $p = 0.010$) but was not independently associated with all cause mortality in fully adjusted models (HR=1.04, 95% CI 0.96-1.13; $p = 0.34$).

Conclusions: Among non diabetic STEMI patients, higher TyG index values are linked to adverse LV remodeling but do not predict mortality. The TyG index may therefore complement echocardiographic evaluation of cardiac function without serving as an independent survival marker.

Keywords: Atherosclerosis, LV parameters, Insulin resistance

MPP-37

Comparative outcomes of drug-coated balloon versus uncoated balloon angioplasty in the treatment of femoropopliteal artery disease

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Objectives: Femoropopliteal artery disease represents a significant subset of peripheral arterial disease, often managed via endovascular interventions. Drug-coated balloons (DCBs) have been developed to enhance the durability of percutaneous transluminal angioplasty (PTA) by reducing neointimal hyperplasia. This systematic review evaluates and compares the clinical efficacy and safety profiles of DCBs versus plain balloon angioplasty (PBA) in the treatment of femoropopliteal lesions, based on data from randomized controlled trials (RCTs).

Methods: A systematic literature search was conducted in PubMed, Embase, and the Cochrane Central Register of Controlled Trials from inception to [insert date]. The search strategy included combinations of the following keywords and MeSH terms: "drug-coated balloon," "paclitaxel-coated balloon," "plain balloon angioplasty," "percutaneous transluminal angioplasty," "femoropopliteal artery," and "randomized controlled trial." Only RCTs comparing DCB and PBA in patients with symptomatic femoropopliteal artery disease were included. Primary outcomes were primary vessel patency and target lesion revascularization (TLR).

Results: A total of 122 studies published between 2000 and 2024 were identified, of which 109 studies were excluded based on eligibility criteria. Three RCTs met the inclusion criteria and were included in the analysis, comprising a total of 726 patients (DCBs: n=467; PBA: n=259). Across most studies, DCB angioplasty demonstrated significantly higher primary patency rates and reduced TLR at 6 to 24 months follow-up compared to PBA.

Conclusions: DCB angioplasty is associated with superior mid-term patency and reduced need for reintervention compared to PBA in the management of femoropopliteal artery disease, with no observed increase in mortality or limb loss. Further large-scale, long-term RCTs are warranted to validate these findings and establish optimal patient selection criteria.

Keywords: Drug-coated balloon, Plain balloon angioplasty

MPP-38

Predictors of significant high-sensitivity C-reactive protein re-reduction after use of rosuvastatin/amlodipine and atorvastatin/amlodipine

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Objectives: There are no clear predictors of high-sensitivity C-reactive protein (hsCRP) reductions following antihypertensives and statin. Also, there are no clear data on the effect of BMI on hsCRP changes following antihypertensives and statins. Therefore, we sought to identify predictors of significant hsCRP reduction after rosuvastatin (RSV)/amlodipine (AML) and atorvastatin (ATV)/AML.

Methods: We included 237 patients from 21 institutions in Korea. Patients were randomly assigned to 1 of 3 treatment groups: RSV 10 mg/ AML 5 mg, RSV 20 mg/AML 5 mg, or ATV 20 mg /AML 5 mg. Multivariate logistic regression analysis was performed to evaluate the predictors for the hsCRP responder (hsCRP reduction $\geq 40\%$ after 8 weeks). We also compared baseline hsCRP and their changes after 8 weeks between the obese patients (n=153) and the non-obese patients (n=84).

Results: Baseline hsCRP ≥ 2 mg/dL and RSV 20 mg/AML 5 mg were independent predictors of hsCRP responder. Their median hsCRP % change rates were -53.11 % and -40.0 %, respectively. Normal weight, pre-obesity, and obesity were not independent predictors of the hsCRP responder. Median hsCRP % reduction rates among normal weight, pre-obese, and obese patients were less than 40% in all groups, and the differences among each group were not significant (-20.0% vs. -33.33 vs. -23.08%, p=0.289).

Conclusions: In patients with ATV, RSV/AML polypill, baseline hsCRP ≥ 2 mg/dL and RSV 20 mg/AML 5 mg were independent predictors of significant hsCRP reduction. BMI was not associated with hsCRP reduction. (Clinical trial: NCT03951207).

Keywords: Amlodipine, C-reactive protein, Statin, Obesity

MPP-39

Vasoprotective effect of moringa oleifera root ethanolic extract on thrombospondin-1 expression in aortic tissue of metabolic syndrome-induced Wistar rats

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Objectives: The high incidence of metabolic syndrome can lead to various diseases, particularly affecting the cardiovascular system. In addition to lifestyle modifications and pharmacological treatments, secondary metabolites from herbal plants, such as *Moringa oleifera*, have shown potential in improving metabolic syndrome conditions. This study aims to evaluate the impact of ethanolic extract from *Moringa* root on thrombospondin-1 (TSP-1) protein expression in the aortic tissue of Wistar rats, focusing on the effectiveness of different dosages.

Methods: In this laboratory study, white rats (*Rattus norvegicus*) were divided into five groups to investigate the effects of *Moringa* root extract on metabolic syndrome. The control group (G1) received standard feed, while group G2, G3, G4, and G5 was subjected to a high-fat diet and induced metabolic syndrome using Streptozotocin-Nicotinamide. Groups G3, G4, and G5 were treated with *Moringa* root extract at doses of 150 mg/KgBB, 250 mg/KgBB, and 350 mg/KgBB, respectively. This post-test only control group design allowed for the examination of TSP-1 expression in aortic tissue after the interventions. Data analysis utilized one-way ANOVA and post hoc LSD tests to assess differences in TSP-1 expression, with linear regression tests applied to explore relationships between variables. Understanding these dynamics is crucial for elucidating the potential therapeutic effects of *Moringa* root extract on aortic tissue in metabolic syndrome condition.

Results: There is a significant difference between the TSP-1 expression on G2 and G1, G3, G4, G5. On the other hand, there is no significant difference between G1 and G5, it shows 350 mg/KgBB extract of *Moringa* root is more effective. The extract of *Moringa* root showed strong negative regression relationship to the aortic tissue TSP-1 expression.

Conclusions: *Moringa* roots extract at a dose of 150 mg/Kg, 250 mg/Kg, and 350 mg/Kg repair the injury of aorta in metabolic syndrome. With a dose of 350 mg/Kg is the most effective.

Keywords: Metabolic syndrome, Atherosclerosis, *Moringa oleifera*, Ethanolic extract, Thrombospondin-1

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Moderated Poster Presentation 6

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CHAIRPERSON : Jong Shin Woo (Kyung Hee University, Republic of Korea)



MPP-40

Effects of cilostazol on prognosis of peripheral arterial disease in patients with diabetes mellitus in Korea: a nationwide population-based study

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Objectives: Cilostazol increases pain-free walking distance in patients with peripheral arterial disease (PAD). However, the effect of cilostazol in improving the prognosis of PAD in patients with diabetes remains unclear. We analyzed its effect on long-term prognosis of Korean patients with diabetes and PAD.

Methods: Data from patients with diabetes and PAD between 2009 and 2018 were collected from the Korean National Health Information database. The primary outcome was all-cause mortality from baseline to death or until December 31, 2022. The secondary outcomes were major adverse cardiac and cerebrovascular events (MACCE) and recurrent major adverse limb events (MALEs). Multiple Cox proportional hazard regression analyses were performed.

Results: We enrolled 14,768 patients with diabetes before PAD diagnosis with complete health screening data within 2 years of PAD diagnosis (5,382 patients receiving cilostazol and 9,386 controls). All-cause mortality was significantly lower in patients treated with cilostazol than in controls. Antiplatelet treatment significantly lowered the risk of all-cause mortality compared with untreated controls, although the risk reduction did not differ significantly between antiplatelet agents. However, cilostazol did not reduce the risk of MACCEs. Treatment with cilostazol alone or in combination with antiplatelet agents was associated with a significant reduction in recurrent MALEs, without increasing the risk of major bleeding.

Conclusions: In patients with diabetes and PAD, cilostazol may improve the prognosis without increasing the risk of bleeding. These findings provide epidemiological evidence of better PAD outcomes in patients with diabetes, although further research is required to elucidate the underlying mechanisms.

Keywords: Cilostazol, Peripheral artery disease

MPP-41

Associations between multiple levels of advanced glycation end products and cardiovascular disease risk factors in Singapore older adults

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Objectives: Advanced glycation end products (AGEs) can be obtained either endogenously, which is produced within the body, or exogenously, which is acquired from external sources like foods. Moreover, they can accumulate in skin due to the long turnover rate of collagen. It has been suggested that AGEs are one of the risk factors for cardiovascular disease (CVD) development since AGEs can induce CVD-related conditions like hypertension and vascular stiffening as they cause oxidative stress and inflammation. However, no studies investigated the relationship between multiple AGE levels and CVD risk factors particularly in the Asian older population. Thus, this study aimed to investigate the associations between blood, skin and dietary levels of AGEs and CVD risk factors in Singapore older adults.

Methods: This is a secondary analysis from a single-blinded, parallel design, 16-week randomized controlled trial with Singapore older adults. Data of blood AGE levels (N(6)-(1-carboxymethyl)-L-lysine (CML), N(6)-(1-carboxyethyl)-L-lysine (CEL), pentosidine and pyrraline), dietary AGEs content, skin AGEs status and CVD risk factors (anthropometric measurement, blood pressure, blood lipid-lipoprotein, and flow mediated dilation (FMD)) were collected at pre- and post-intervention. Spearman correlation was applied to assess association between blood, skin, and dietary AGEs levels and CVD risk factors.

Results: Skin AGEs level was negatively associated with blood glucose level (p-value=0.04) and positively associated with systolic FMD (p-value=0.02), diastolic FMD (p-value=0.03) and waist circumference (WC) (p-value=0.01). Among the blood AGEs, only blood CEL level was negatively associated with total cholesterol level (p-value=0.03) and positively associated with systolic FMD (p-value=0.04), WC (p-value=0.02) and high-sensitivity C-reactive protein level (p-value=0.02). However, dietary AGEs level was not associated with any CVD risk factors.

Conclusions: Long-term AGEs status and circulating AGEs levels were associated with CVD risk factors but no associations were observed with dietary AGEs, suggesting management of endogenous AGEs metabolism may be critical in CVD risk.

Keywords: Cardiovascular disease, Advanced glycation end products, Older adults, Nutrition

MPP-42

Effects of portulaca oleracea L. extract on inflammation and mitochondrial biogenesis in rats fed a high-fat diet

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Objectives: Portulaca oleracea L., a medicinal herb belonging to the Portulacaceae family, is rich in bioactive compounds such as polyphenols, omega-3 fatty acids, minerals, and vitamins. This study investigated whether Portulaca oleracea L. extract (PO) could attenuate high-fat diet (HFD)-induced inflammation and enhance muscle mitochondrial biogenesis through regulation of inflammatory microRNAs (miRs) and activation of the AMP-activated protein kinase (AMPK) and sirtuin (SIRT) pathways.

Methods: Four-week-old male Sprague-Dawley rats were randomly divided into four dietary groups for 12 weeks: normal chow diet, 45% HFD, HFD with 0.2% PO, and HFD with 0.4% PO. During the experimental period, body weight and food intake were recorded weekly. After sacrifice, serum and liver were collected for lipid profile analysis, and WAT and skeletal muscle were obtained to analyze the expression of mRNA and miR-221/222. Moreover, AMPK and SIRT activities in muscle were evaluated using commercial assay kits.

Results: PO supplementation showed reduced body weight gain, WAT mass, and adipocyte size compared to the HFD-fed rats, along with improvements in serum and hepatic lipid profiles. In WAT, PO suppressed the mRNA levels of proinflammatory cytokines (Tnf- α , Il-6, and Mcp-1), NF- κ B activity, and the miR-221/222 expression. Furthermore, PO down-regulated the expression of proinflammatory M1 macrophage markers (Cd68, Nos2, and Cd11c) while upregulating the anti-inflammatory M2 marker Arg1, thereby promoting macrophage polarization from the M1 to the M2 phenotype. In skeletal muscle, PO increased mitochondrial DNA content and upregulated the mitochondrial biogenesis-related genes (Ppar δ , Nrf1, Tfam, Pgc-1 α , Sirt1, and Cpt-1B). PO also activated AMPK and SIRT.

Conclusions: These results suggest that PO may attenuate obesity-induced inflammation and promote muscle mitochondrial biogenesis via partial modulation of the miR-221/222 and AMPK/SIRT pathways.

Keywords: Obesity, Portulaca oleracea L. extract, Inflammation, Mitochondrial biogenesis, AMPK/SIRT, MicroRNA

MPP-43

Impact of gut microbiota-based therapies on cardio-metabolic risk factors induced by atypical antipsychotics: a network meta-analysis

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Objectives: The use of atypical antipsychotics has been linked to an elevated likelihood of cardio-metabolic risk factors, such as diabetes mellitus, weight gain, and hyperlipidemia. Alteration of gut microbiota may be the mechanism behind these metabolic abnormalities. This study aims to examine the impact of dietary gut-microbiota based interventions, including fiber and probiotics, on metabolic abnormalities that result from atypical antipsychotic.

Methods: A comprehensive search was conducted across many databases, including PubMed, Scopus, Web of Science, and Cochrane Controlled Register of Trials (CENTRAL), to identify potential papers. 324 abstracts were screened, identifying eight full-text articles which were assessed for eligibility. Five articles were finally included in the meta-analysed. The primary outcomes were changes in body weight and body mass index (BMI), whereas secondary outcomes included changes in lipid parameters and other cardiovascular risk factors.

Results: The effect size utilized was the mean difference (MD), accompanied by a 95% confidence interval. A total of 5 randomized controlled trials with 389 patients were included. Probiotics + fibers (MD -3.95, 95% CI [-5.15, -2.76]) and probiotics only (MD -1.35, 95% CI [-2.04, -0.66]) significantly decreased body weight, compared to placebo. Probiotics + fibers was also associated with significant changes in secondary outcomes like cholesterol (MD: -0.37, 95% CI [-0.67, -0.07]), insulin levels (MD: -5.78, 95% CI [-8.49, -3.06]), and insulin resistance index (MD: -1.34, 95% CI [-1.93, -0.76]). None of the treatments significantly improved LDL, HDL, or triglycerides.

Conclusions: This study found that dietary gut-microbiota based therapies like probiotics and fibers may be effective in counteracting cardiometabolic risk factors induced by atypical antipsychotics.

Keywords: Probiotics

MPP-44

Ellagic acid alleviates MASLD by reducing hepatic lipogenesis and fibrosis in cholesterol-fed apoE-deficient mice

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Objectives: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a chronic liver condition with a rising global incidence, closely linked to metabolic risk factors such as dyslipidemia. Apolipoprotein E knockout (apoE^{-/-}) mice fed a Paigen diet serve as a relevant model for MASLD and atherosclerosis research. This study investigated the effects of ellagic acid (EA) on MASLD progression in apoE^{-/-} mice fed a Paigen diet.

Methods: Wild-type (WT) and apoE^{-/-} mice were fed a Paigen diet for 10 weeks to induce metabolic dysregulation. ApoE^{-/-} mice were concurrently administered either ellagic acid (10 mg/kg/day orally) or no treatment.

Results: After 10 weeks, apoE deficiency markedly elevated circulating total cholesterol (TC) and LDL cholesterol levels. ApoE^{-/-} mice also exhibited significant hepatic lipid accumulation, demonstrated by increased Oil Red O staining and elevated perilipin 2 (PLIN2) expression. EA treatment significantly reduced circulating TC and LDL levels, hepatic lipid accumulation, and lipid peroxidation. Furthermore, EA administration decreased hepatic expression of lipogenic proteins, including SREBP1, FAS, and C/EBP α . Additionally, hepatic fibrogenic markers, including TGF- β , α -SMA, and collagen, which were elevated in apoE^{-/-} mice compared to WT, were significantly reduced by EA. Levels of phosphorylated SMAD2, as well as total SMAD2, SMAD3, and SMAD4 proteins, were also significantly attenuated by EA treatment.

Conclusions: EA effectively ameliorates MASLD and hypercholesterolemia in apoE^{-/-} mice fed a Paigen diet. The beneficial effects of EA may be mediated through downregulating lipogenic pathways and suppressing TGF- β /SMAD signaling.

Keywords: Ellagic acid, ApoE^{-/-} mice, Hypercholesterolemia, Metabolic dysfunction-associated steatotic liver disease

MPP-45

Adiponectin, dyslipidemia, nutritional indices and atherogenic indices in relation to muscle quality in sarcopenic and non-sarcopenia elderly diabetic patients

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Objectives: To assess the relationship between adiponectin levels, dyslipidemia, atherogenic indices, objective nutritional indices, and muscle quality in sarcopenic versus non-sarcopenic diabetic patients, and to determine the association between these parameters.

Methods: A cross-sectional study was conducted involving 160 diabetic patients, divided into sarcopenic (n=80) and non-sarcopenic (n=80) groups based on AWGS (skeletal muscle index and muscle strength or walking speed). Anthropometric measurements, biochemical analyses including adiponectin levels, lipid profiles, and calculation of atherogenic indices were performed. Nutritional status was assessed using Prognostic Nutritional Index (PNI), Geriatric Nutritional Risk Index (GNRI), and Controlling Nutritional Status (CONUT) scores. Ultrasound measurements included assessments of muscle thickness and echo intensity. Correlation analyses were performed to determine relationships between variables.

Results: Sarcopenic diabetic patients demonstrated significantly higher adiponectin levels compared to non-sarcopenic patients (12.8 ± 3.4 vs 8.4 ± 2.1 μ g/mL, $p < 0.001$). Nutritional assessment revealed that sarcopenic patients had significantly lower PNI and GNRI scores, indicating poorer nutritional status, whereas CONUT scores were significantly higher (5.2 ± 2.1 vs 2.8 ± 1.4 , $p < 0.001$), reflecting an increased nutritional risk. Sarcopenic patients demonstrated significantly higher atherogenic coefficient (3.4 ± 0.8 vs 2.6 ± 0.6 , $p < 0.001$) and increased echo intensity (33.5 ± 4.2 vs 24.7 ± 4.1 , $p < 0.001$) in sarcopenic patients. Correlation analysis revealed that adiponectin levels showed significant negative correlations with HOMA-IR ($r = -0.524$, $p < 0.001$) and skeletal muscle index ($r = -0.672$, $p < 0.001$), as well as positive correlations with atherogenic coefficient ($r = +0.445$, $p < 0.001$) and echo intensity.

Conclusions: Sarcopenic diabetic patients exhibit elevated adiponectin levels, compromised nutritional status, increased atherogenic risk, and severely deteriorated muscle quality as evidenced by increased echo intensity, suggesting complex pathophysiological mechanisms linking muscle deterioration with cardiovascular and metabolic dysfunction.

Keywords: Sarcopenia, Adiponectin, Nutritional indices, Muscle quality, Diabetic

MPP-46

Effects of RAS and SGLT2 inhibitors alone or in combination on end-stage kidney disease and/or all-cause death in patients with both diabetes and hypertension: a nationwide cohort study

Renin-angiotensin-aldosterone system (RAS) inhibitors and sodium-glucose cotransporter 2 (SGLT2) inhibitors are key

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Objectives: Renin-angiotensin-aldosterone system (RAS) inhibitors and sodium-glucose cotransporter 2 (SGLT2) inhibitors are key treatments for diabetic kidney disease. However, their independent and combined effects on end-stage kidney disease (ESKD) and mortality remain unclear. This study evaluates their impact, alone or in combination, on ESKD and all-cause mortality in patients with diabetes and hypertension.

Methods: A nationwide cohort study using the Korean National Health Database included 261,783 individuals with type 2 diabetes and hypertension (2015–2017). Participants were grouped into (1) no RAS or SGLT2 inhibitors (reference), (2) SGLT2 inhibitors alone, (3) RAS inhibitors alone, and (4) combination therapy. Cox regression models were used to estimate hazard ratios (HRs) for ESKD, mortality, and their composite.

Results: Over 5.38 years, 2,674 (1.02%) developed ESKD and 20,866 (7.97%) died. Combination therapy showed the greatest risk reduction for composite outcomes [HR: 0.68, 95% confidence interval (CI): 0.56–0.82] and mortality (HR: 0.68, 95% CI: 0.56–0.83). SGLT2 inhibitors alone reduced composite risk (HR: 0.71, 95% CI: 0.61–0.84) and mortality (HR: 0.68, 95% CI: 0.57–0.81). RAS inhibitors alone had modest effects (HR: 0.96, 95% CI: 0.93–0.98) on composite outcomes and mortality (HR: 0.94, 95% CI: 0.91–0.97). Notably, only combination therapy was associated with lower ESKD risk (HR: 0.63, 95% CI: 0.37–1.07), but this was not statistically significant. SGLT2 inhibitors consistently reduced ESKD and mortality, while RAS inhibitors were beneficial mainly in non-SGLT2 inhibitor users.

Conclusions: Combination therapy may provide the greatest renal and survival benefit for diabetic patients with hypertension. SGLT2 inhibitors alone significantly reduced mortality, while RAS inhibitors alone had a modest impact.

Keywords: End-stage kidney disease, Sodium-glucose cotransporter-2 inhibitors, Renin-angiotensin system inhibitors, Type 2 diabetes, Hypertension, All-cause mortality

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General Poster Display



PE-01

Integrative fine-mapping and functional annotation identify novel genetic determinants of LDL cholesterol regulation in Koreans

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Objectives: Low-density lipoprotein cholesterol (LDL-C) is a major modifiable risk factor for cardiovascular disease, yet much of its genetic architecture remains underexplored in East Asian populations.

Methods: We conducted a meta-analysis of LDL-C GWAS summary statistics from two large Korean cohorts (KCPS-II and KoGES) and compared the findings with results from the UK Biobank. Fine-mapping analyses, colocalization with tissue-specific eQTL data, SMR-HEIDI testing, MAGMA pathway enrichment, and phenome-wide association studies (PheWAS) were applied to prioritize and functionally characterize putative causal variants and genes.

Results: We identified multiple genome-wide significant loci, including known LDL-C regulators such as HMGCR, PCSK9, APOE, and ABCA1. Novel loci uniquely detected in the Korean data, including FABP1, CFHR1, and MACO1, were further supported by colocalization and expression-based analyses. Phenome-wide association studies (PheWAS) for FABP1 and CFHR1 revealed additional associations with metabolic and immune-related traits, providing biological plausibility for their roles in systemic disease pathways. Integrative analysis highlighted functional pathways related to lipid metabolism, immune regulation, and neuronal systems. Notably, fine-mapping and functional annotation converged on several key genes (e.g., FABP1, CFHR1, MACO1, APOE, APOC1, and PCSK9) through independent analytical pipelines.

Conclusions: Our integrative genomic analyses identified both novel and established genetic determinants of LDL-C regulation specific to Korean populations. These findings expand the understanding of LDL-C genetics beyond European ancestry and provide a foundation for future precision medicine strategies targeting lipid disorders in East Asian populations.

Keywords: LDL cholesterol, Genome-wide association study, Fine-mapping, Colocalization, SMR-HEIDI, Korean population, Lipid metabolism

PE-02

Torin-1 administration enhances cognitive function by regulating autophagy and cholesterol metabolism in hepatic encephalopathy

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Objectives: Patients with acute or chronic liver diseases commonly suffer from hepatic encephalopathy (HE), which is featured by neurological disorders. In the development of HE, it has been known that hyperammonemia, abnormal energy metabolism, and inflammation are considered as contributing factors. Recently, aberrant autophagic system has been reported contribute to the development of HE. Thus, we hypothesized whether the autophagy inducer torin-1 show beneficial effects against HE.

Methods: In present study, C57BL/6 mice were subjected to bile duct ligation (BDL) for 14 days to induce HE. Torin-1 was intraperitoneally injected daily for 14 days. Liver function was estimated by aspartate aminotransferase, alanine aminotransferase, total bilirubin levels. Liver histology was assessed by hematoxylin and eosin staining. Autophagy, cholesterol metabolism, and the cytokine/chemokine profile were measured in the cerebral cortex. Cognitive function was evaluated by using elevated plus maze, novel object recognition test, marble-burying test, clasping test, and passive avoidance test.

Results: Torin-1 administration decreased the BDL-induced elevation in serum cholesterol and ammonia levels, although it showed no effects on liver enzyme levels. Aberrantly increased expression of autophagy- and cholesterol-associated molecules in the cerebral cortex was decreased by torin-1 treatment. Mice undergoing BDL with torin-1 treatment displayed improved cognitive function.

Conclusions: These results suggest that the autophagy induction inhibit detrimental effects found in HE by reducing cholesterol metabolism in the cerebral cortex. Therefore, torin-1 might be a potential approach to alleviate metabolic changes and neurological symptoms in HE.

Keywords: Autophagy, Cholesterol metabolism, Hepatic encephalopathy, Liver disease

PE-04

Precision hepatometabolic therapy: integrating PPAR α agonists and omega-3 fatty acids in the treatment of diabetic lipotoxic liver disease

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Objectives: Non-alcoholic fatty liver disease (NAFLD) and its progressive form, non-alcoholic steatohepatitis (NASH), are frequently observed in patients with type 2 diabetes mellitus (T2DM) and are closely linked to atherogenic dyslipidemia. This dyslipidemia, characterized by elevated triglycerides, increased small dense LDL (sdLDL), and low HDL levels, contributes to hepatic lipotoxicity, systemic inflammation, and cardiometabolic risk. This study aims to investigate the relationship between diabetic dyslipidemia and hepatic lipotoxicity and evaluate novel therapeutic strategies targeting these mechanisms.

Methods: A translational cohort of 90 T2DM patients with biopsy-proven NAFLD/NASH and 30 healthy controls was analyzed. Lipid parameters, liver enzymes, and inflammatory markers (IL-6, TNF- α , hsCRP) were measured. Liver steatosis and fibrosis were assessed using transient elastography and MRI-PDFF. A treatment subgroup (n=30) received either pemafibrate or high-dose omega-3 fatty acids for six months.

Results: Higher sdLDL and remnant cholesterol levels were significantly associated with liver stiffness (r=0.54, p<0.01) and hepatic fat content (r=0.48, p<0.01). Pemafibrate reduced triglycerides by 45 %, improved ALT/AST levels, and decreased liver stiffness (mean Δ =-2.3 kPa, p<0.05). Omega-3 therapy decreased IL-6 and TNF- α concentrations. Both interventions showed modest improvement in MRI-PDFF scores. Multivariate regression confirmed that improvement in lipotoxicity markers predicted hepatic function improvement independently of glycemic control.

Conclusions: The findings reinforce the emerging concept of precision hepatometabolic therapy, emphasizing the need to address both dyslipidemia and inflammation in diabetic liver disease. Integrative treatment using PPAR α agonists alongside omega-3 fatty acids may synergistically attenuate hepatic damage, representing a promising strategy to lower the burden of hepatic and cardiovascular complications in patients with type 2 diabetes.

Keywords: Diabetic dyslipidemia, Lipotoxicity, NAFLD, PPAR α agonist

PE-05

Chronic intermittent hypoxia induces aortic inflammation via NLRP3 inflammasome activation in normal C57BL/6 mice

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Objectives: Obstructive sleep apnea (OSA) with chronic intermittent hypoxia (CIH) is increasingly linked to vascular inflammation and atherogenesis. However, the mechanisms in normolipidemic animals remain unclear. This study investigates the impact of CIH on aortic inflammation and NLRP3 inflammasome activation in wild-type C57BL/6 mice. To determine the impact of chronic intermittent hypoxia on vascular inflammation and inflammasome activation in normolipidemic C57BL/6 mice.

Methods: Male C57BL/6 mice (n=24) were exposed to either normoxia or CIH (21-5% FiO₂, 6-min cycles, 8 hours/day) for 28 days. Systolic blood pressure (tail-cuff), body weight, and fasting lipids were recorded. Aortas were harvested to assess histology, oxidative stress markers (MDA, SOD), IL-1 β , IL-18, NLRP3, caspase-1, and VCAM-1 via qPCR and Western blot. Endothelial function was assessed using acetylcholine-induced vasorelaxation in isolated aortic rings.

Results: CIH mice developed moderate hypertension (+20 mmHg, p<0.01).

Conclusions: CIH triggers aortic inflammation in normal C57BL/6 mice through NLRP3 inflammasome activation, independent of dyslipidemia. This suggests that OSA may promote vascular inflammation and endothelial dysfunction even in metabolically healthy individuals, highlighting the need for early intervention strategies.

Keywords: Chronic intermittent hypoxia, Vascular inflammation, NLRP3 inflammasome, Endothelial dysfunction

PE-06

Perivascular adipose tissue browning in early atherosclerosis

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Objectives: Perivascular adipose tissue (PVAT), which anatomically surrounds blood vessels, has emerged as a regulator of vascular function and a potential marker of vascular health. While phenotypic alterations in other adipose tissues have been well studied, the pathological role of PVAT remains poorly defined. Notably, PVAT whitening has been reported in chronic cardiovascular disease, studies on the phenotype of PVAT in the early atherosclerosis are still underexplored. This study investigates PVAT browning in early atherosclerosis and to distinguish its phenotype under various metabolic conditions.

Methods: To examine the phenotype of PVAT during early atherosclerosis, ApoE KO and C57BL/6 mice were fed a high-fat diet for 8 weeks. A separate group of C57BL/6 mice received daily s.c. injections of CL316,243, β_3 adrenoceptor agonist, or PBS for 7 days to induce systemic browning. Atherosclerotic lesion was assessed using en face and aortic root Oil Red O staining. PVAT phenotype was evaluated through qPCR for gene expression analysis, holotomographic imaging and H&E staining for lipid droplet.

Results: In ApoE KO mice, expression of UCP1, a browning marker, was elevated in PVAT as atherosclerosis progressed. This phenotype was not observed in the inguinal white adipose tissue (iWAT) of ApoE KO mice. Holotomographic imaging revealed differences in lipid droplet morphology between strains in ApoE KO PVAT compared to C57BL/6 controls. Furthermore, the pattern of brown adipose-related gene expression in PVAT of ApoE KO mice differed from that induced by β_3 adrenergic stimulation, suggesting a distinct browning mechanism in early atherosclerosis.

Conclusions: PVAT undergoes browning in early atherosclerosis. This browning response appears to be vascular-localized, distinct from systemic browning induced by sympathetic activation, as evidenced by the absence of browning in iWAT. These findings highlight the potential role of PVAT browning as an adaptive or pathological response during atherosclerosis and underscore the need for further investigation into its mechanisms.

Keywords: Perivascular adipose tissue, Browning, Atherosclerosis, Adipose tissue

PE-07

Dietary flaxseed oil restores lipid homeostasis and reduces atherogenic index in fructose-induced metabolic syndrome in Wistar rats

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Objectives: To investigate whether flaxseed oil supplementation can reverse lipid abnormalities and aortic vascular dysfunction in a fructose-induced metabolic syndrome model using normal Wistar rats.

Methods: Male Wistar rats (n=30) were divided into 3 groups: control (normal diet), MetS (10% fructose in drinking water), and MetS+flaxseed oil (2 mL/kg/day orally for 8 weeks). Body weight, fasting glucose, insulin, lipid profile, and HOMA-IR were measured. Atherogenic index of plasma (AIP), serum adiponectin, and CRP were evaluated. Targeted plasma lipidomics using LC-MS/MS was performed. Thoracic aorta samples were assessed histologically and for eNOS and VCAM-1 expression by Western blot.

Results: Fructose-fed rats exhibited significant weight gain, insulin resistance (HOMA-IR $\uparrow$ 2.5-fold), elevated triglycerides (+65%) and LDL (+58%) with reduced HDL (-35%) vs controls ($p < 0.01$). Flaxseed oil reversed these trends: TG and LDL decreased by 40% and 35% respectively, while HDL increased by 25% ($p < 0.05$). AIP reduced from 0.42 to 0.21 ($p < 0.01$). Plasma lipidomics revealed decreased ceramide (C16:0) and increased sphingomyelin and phosphatidylcholine species. Adiponectin levels rose 1.8-fold, CRP dropped by 45%, and aortic histology showed preserved elastin and reduced intimal thickening. eNOS expression improved (1.6-fold $\uparrow$) and VCAM-1 decreased by 50%.

Conclusions: Oral flaxseed oil significantly improves insulin sensitivity, lipid profiles, and vascular integrity in fructose-induced MetS in Wistar rats. Its lipidomic remodeling potential, including lowering pro-atherogenic ceramides, supports its preventive role against atherosclerosis in diet-induced metabolic syndrome.

Keywords: Metabolic syndrome, Lipidomics, Atherosclerosis

PE-08

Mucosal invariant T-Cells link CCBE1 expression to clinically actionable CAD

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Objectives: Coronary artery disease (CAD) remains the leading global cause of death, despite therapies targeting standard modifiable risk factors (SMuRFs). However, the presence of CAD in SMuRF-less individuals and results from the CANTOS trial targeting IL-1 β highlight the need to explore alternative pathological drivers. Single-cell RNA sequencing (scRNA-seq) enables high-resolution characterization of immune and vascular cell states, including endothelial colony-forming cells (ECFCs), which may uncover novel contributors to CAD pathogenesis.

Methods: The BioHEART-CT study is a prospective, cross-sectional study of stable patients referred for CT angiography - with advanced CT imaging of coronary atherosclerosis, and matched blood samples for peripheral blood mononuclear cells (PBMCs) preparation (n=873 patients). From this cohort, PBMCs were isolated and analysed using scRNA-seq as part of the TenK10K project. We focused on CCBE1 (Collagen and Calcium-Binding EGF Domain-Containing Protein 1), a gene previously implicated in CAD via mitochondrial dysfunction in endothelial cells. To understand its immune relevance, we assessed CCBE1 expression across 28 immune cell subpopulations identified by canonical markers. Expression patterns were compared between patients with high coronary artery calcium scores (CACS >100) and those with CACS = 0. Differential expression analysis was conducted using MAST, with Benjamini-Hochberg correction applied to adjust for multiple comparisons.

Results: We found that mucosal-associated invariant T cells (MAITs) had significantly higher expression of CCBE1 in patients with CAD compared to non-CAD (mean difference: 2.2×10^{-4} log-transformed counts; adjusted P=4.4x10⁻²; Figure 1C). No other cell subpopulation had CAD-dependent differences in CCBE1 expression. MAITs are a unique subset of T cells with both adaptive and innate properties, implicated in various inflammatory and metabolic diseases⁴. While the role of CCBE1 in MAITs and its broader significance in CAD remain unclear, these findings highlight the need for functional experiments.

Conclusions: Our results have significant implications for the value of scRNA-seq in identifying novel molecular contributors to cardiovascular disease.

Keywords: Single-cell RNA sequencing, Single-cell transcriptomics, Atherosclerosis, Coronary artery disease, CAD

PE-09

Elucidating the renoprotective effects of Boerhaavia diffusa in a high-fat diet-induced chronic kidney disease mouse model

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Objectives: Chronic kidney disease (CKD) remains a critical global health challenge characterized by progressive renal dysfunction. Our previous study demonstrated that Boerhaavia diffusa (BD) exhibits promising renoprotective effects by mitigating oxidative stress and lipid accumulation in vitro. Building on these findings, we extended our investigation to an in vivo model to elucidate the potential of BD in attenuating CKD progression induced by a high-fat diet. This study aims to evaluate the renoprotective efficacy of BD in C57BL6/J male mice subjected to a high-fat diet (HFD) and to characterize renal histopathological changes and lipid profile associated with BD treatment.

Methods: Male C57BL6/J mice were fed either a high-fat diet (60% kcal from fat) for 12 weeks to induce CKD-like pathology or a standard chow diet for control comparisons. BD was administered daily (500mg/kg) via oral gavage throughout the feeding period. For lipidomic profiling, total lipids were extracted from kidney tissues using a modified Bligh and Dyer method. Lipid extracts were analyzed using liquid chromatography-mass spectrometry (LC-MS), employing a reverse-phase C18 column coupled with a high-resolution Q-TOF mass spectrometer.

Results: Preliminary histological analysis revealed that BD treatment significantly ameliorated the pathological changes in kidneys compared to untreated HFD mice. These findings corroborate previous in vitro data indicating BD's antioxidative and hypolipidemic properties. Lipidomics analysis using LC-MS revealed extensive alterations in renal lipid composition including restored lipid metabolism, reduced lipotoxic lipids, reduced inflammation.

Conclusions: Our in vivo study confirms that Boerhaavia diffusa exerts significant renoprotective effects in a diet-induced CKD mouse model. The mitigation of lipid accumulation, oxidative damage, and fibrosis highlights BD's therapeutic potential for CKD management. It supports that BD treatment in HFD-fed mice upregulates beneficial lipid species and restores metabolic homeostasis. Future work will focus on molecular pathways involved in BD-mediated renoprotection and its translation to clinical applications.

Keywords: Boerhaavia diffusa, Nephroprotection, Oxidative stress, Anti-inflammatory, Anti-oxidant, Lipidomics, Omics

PE-10

Boerhaavia diffusa ameliorates renal lipid deposition and attenuates kidney injury in high-fat diet-induced chronic kidney disease mouse models

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Objectives: Chronic kidney disease (CKD) is a major global health issue characterized by progressive renal decline. Preliminary studies suggested that Boerhaavia diffusa (BD), a traditional medicinal plant, exhibits therapeutic potential for CKD due to its lipid-lowering and antioxidant effects in vitro. Building on this evidence, an in vivo model of high-fat diet (HFD)-induced dyslipidaemia was employed to investigate whether BD attenuates CKD progression. This study aims to evaluate BD's renoprotective effects, particularly its ability to reduce renal lipid deposition, while examining associated improvements in renal histopathology, as well as examining any potential hepatotoxicity linked to BD treatment.

Methods: BD (500 mg/kg) was administered via oral gavage to male C57BL/6 mice with HFD-induced CKD for 12 weeks. HFD-induced CKD C57BL/6 mice without BD administration were used for control comparisons. Mice were euthanized at 32 weeks, and kidneys and livers were collected for histology analysis.

Results: In kidneys, Haematoxylin and Eosin (H&E) stain demonstrated reduced lipid deposition in the BD group versus HFD controls. Periodic Acid-Schiff (PAS) staining showed normalized mesangial matrix in BD-treated groups, contrasting with HFD-induced mesangial expansion. Periodic Schiff-Methenamine Silver (PASM) stain in the BD group revealed attenuated glomerular basement membrane (GBM) thickening, while Sirius Red (SR) staining indicated reduced renal fibrosis with BD treatment (Figure 1). BD treatment exhibited no hepatotoxicity, as evidenced by significantly reduced cytoplasmic vacuolation on H&E staining and absence of exacerbating fibrosis on SR staining (Figure 2).

Conclusions: Results affirm BD treatment provides kidney protection in HFD-induced CKD, significantly reducing lipid deposition while improving renal pathologies including GBM thickening, mesangial expansion and fibrosis, without evidence of hepatotoxicity. These therapeutic effects correlate with BD's antioxidative and hypolipidemic properties, demonstrating its capacity to concurrently address metabolic dysregulation via lipid modulation and structural damages in HFD-induced CKD progression. Thus, BD emerges as a promising therapeutic agent for dyslipidaemia-related CKD management.

Keywords: Antioxidant, Chronic kidney disease, Boerhaavia diffusa, Anti-inflammatory

PE-11

Lactoferrin alleviates renal oxidative stress and lipid accumulation in high-fat diet-induced diabetic nephropathy

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Objectives: Diabetic nephropathy (DN) is a serious complication of diabetes mellitus and a leading cause of end-stage renal disease (ESRD). As there is currently no definitive cure, preserving renal function remains the primary strategy to delay disease progression. A central pathological feature of DN is the excessive production of reactive oxygen species (ROS) induced by chronic hyperglycemia, which damages proximal tubule cells (PTCs), promoting inflammation and fibrosis. Additionally, dyslipidaemia intensifies oxidative stress and contributes to lipid accumulation in renal tissues, aggravating kidney injury. Lactoferrin (Lf), a milk-derived glycoprotein with antioxidant, anti-inflammatory, and iron-binding properties, may influence both oxidative stress and lipid metabolism. This study aimed to assess whether Lf supplementation can reduce renal ROS levels and improve lipid handling, thereby protecting against renal damage in DN.

Methods: The AnOxPePred deep-learning algorithm was used to predict the antioxidant potential of human and bovine Lf, specifically their free radical scavenging (FRS) and metal-chelating activities (MCA), based on full peptide sequences (NP_002334.2 and NP_851341.1). Key antioxidant domains were identified within the transferrin-like 1 and 2 regions. In vivo, male C57BL/6 mice with high-fat diet (HFD)-induced chronic kidney disease (CKD) received Lf via oral gavage at low (300 mg/kg) and high (900 mg/kg) doses for 12 weeks. Urine samples were collected before sacrifice at 32 weeks. Kidneys and livers were harvested for histological and lipidomic analyses.

Results: The predicted antioxidant properties were consistent with peptide-level scores, with the top bovine Lf fragment exhibiting FRS and MCA values of 0.60836 and 0.32879, respectively. Histological analysis revealed improved renal architecture and reduced KIM-1 expression in Lf-treated groups. Lipid staining indicated decreased ectopic lipid deposition in PTCs, particularly in high-dose Lf-treated mice.

Conclusions: Lf demonstrates both antioxidant and lipid-lowering properties that contribute to renal protection in DN. These findings highlight its potential as a supportive therapeutic candidate for delaying disease progression.

Keywords: Renal, Lactoferrin, Lipid metabolism, Lipidomics

PE-12

Smad7 deletion in smooth muscle cells promotes the contractile phenotype and results in more stable atherosclerotic plaques

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Objectives: During atherogenesis, smooth muscle cells (SMCs) lose their contractile phenotype, proliferate, and adopt alternative states that contribute to plaque growth. SMAD7, an inhibitor of TGF- β signaling, is known to suppress the formation of vascular contractile SMCs. Based on this, we investigated whether SMC-specific Smad7 deletion could restore contractile identity and limit plaque development.

Methods: We knocked down Smad7 in cultured aortic SMCs and evaluated contractility, proliferation, migration, and gene expression. Additionally, we used transgenic mice with SMC-specific Smad7 deletion (Smad7SMC-KO) along with tdTomato-based SMC-lineage tracing. Atherosclerosis was induced via AAV-PCSK9 and a 20-week high-fat diet.

Results: Smad7-deficient SMCs upregulated contractile genes, showed stronger contraction in collagen gel assays, and reduced proliferation, migration, and expression of markers associated with modulated SMC states. In vivo, Smad7 deletion in SMCs led to elevated plasma cholesterol and increased hepatic lipid accumulation compared to wild-type littermates. However, after adjusting for total cholesterol using multiple linear regression, Smad7SMC-KO mice developed significantly smaller plaques. These plaques were also enriched in collagen content, suggesting greater stability despite the lipid-rich milieu. Single-cell RNA sequencing of atherosclerotic plaques revealed diverse SMC-derived populations—including contractile SMCs, fibromyocytes, chondromyocytes, and pericyte-like cells. Among these, WT chondromyocytes displayed the highest Smad7 expression. Moreover chondromyocytes exhibited the most pronounced gene expression differences between genotypes. In Smad7SMC-KO mice, this population showed enrichment in Smooth Muscle Contraction and Focal Adhesion pathways, consistent with the enhanced contractile features and increased plaque stability observed in vitro and in vivo.

Conclusions: SMAD7 restrains the contractile program in SMCs. Its loss promotes a more stable, contractile phenotype. In vivo, this phenotypic shift results in smaller, collagen-rich plaques, even under high-cholesterol conditions. Targeting SMAD7 may complement cholesterol-lowering therapies to stabilize plaques and reduce rupture risk.

Keywords: Smooth muscle cells, SMAD7, Phenotypic switch

PE-13

Adenylyl cyclase-associated protein 1 and caveolin complex regulates adhesion molecules on endothelial cells

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Objectives: Adenylyl cyclase-associated protein 1 (CAP1) serves as a receptor for human resistin in monocytes, yet its role in endothelial-monocyte interactions and regulation of adenylyl cyclase (AC) in endothelial cells remains undefined. We sought to (1) determine which AC isoforms predominate in endothelial cells, (2) elucidate how CAP1 interacts with and modulates AC in the presence or absence of resistin, and (3) assess the impact of endothelial CAP1 on trans-endothelial monocyte migration both in vitro and in vivo.

Methods: Human umbilical vein endothelial cells (HUVECs) were subjected to membrane fractionation and Opti-PrepTM gradient centrifugation to localize AC isoforms and lipid-raft components. Co-immunoprecipitation and proximity ligation assays (PLA) examined CAP1-AC-caveolin-1 (CAV1) complex formation. Intracellular cAMP levels were measured by ELISA, and downstream PKA, CREB, and NF- κ B activation were evaluated by immunoblotting. Transwell assays quantified monocyte (THP-1) migration across HUVEC monolayers after CAP1 knockdown or resistin treatment. In vivo, CAP1^{+/+} mice—with or without wild-type bone marrow transplantation—were analyzed in a λ -carrageenan-induced paw edema model for monocyte extravasation (DiI labeling, GFP chimerism) and inflammatory responses.

Results: AC3 and AC6 were enriched in HUVEC lipid rafts alongside CAP1 and CAV1. Under basal conditions, CAP1 formed a suppressive complex with AC3/CAV1 that maintained low cAMP. Resistin binding disrupted this assembly, elevating cAMP and activating PKA-CREB and NF- κ B, which in turn upregulated ICAM-1 and VCAM-1. CAP1 knockdown mimicked resistin effects, enhancing monocyte transmigration in vitro. In CAP1^{+/+} mice reconstituted with wild-type monocytes, endothelial CAP1 deficiency markedly increased leukocyte extravasation and edema formation.

Conclusions: CAP1 functions as a gatekeeper in endothelial cells by restraining AC activity within caveolae. Resistin-mediated disassembly of the CAP1-AC-CAV1 complex unleashes cAMP-PKA-NF- κ B signaling, driving adhesion molecule expression and monocyte infiltration. These findings reveal a novel mechanistic axis of CAP1 in vascular inflammation and highlight CAP1 as a potential therapeutic target in cardio-metabolic disease.

Keywords: Caveolin, Inflammation, Endothelial cell, Resistin, Adenylyl cyclase

PE-14

ANGPTL4 suppresses endothelial inflammation and EndMT to preserve vascular integrity in atherosclerosis

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Objectives: Endothelial dysfunction is a critical driver of atherosclerosis, promoting vascular inflammation, endothelial-to-mesenchymal transition (EndMT), and plaque instability. While ANGPTL4 is known to modulate smooth muscle phenotype, its role in endothelial integrity and EndMT remains unclear.

Methods: We assessed the protective effects of ANGPTL4 on endothelial dysfunction using Apoe^{-/-} mice fed a Western diet and cultured human umbilical vein endothelial cells (HUVECs). EndMT marker expression was also evaluated in human atherosclerotic plaques.

Results: ANGPTL4 significantly suppressed TNF- α - and IL-1 β -induced endothelial inflammation in vitro by downregulating ICAM1 and VCAM1. In Apoe^{-/-} mice, ANGPTL4 reduced vascular inflammation and preserved endothelial barrier function. ANGPTL4 inhibited EndMT induced by pro-inflammatory cytokines and TGF- β , as shown by preserved expression of endothelial markers (CD31, VE-Cadherin) and reduced mesenchymal markers (SM22 α , fibronectin, α SMA). Mechanistically, ANGPTL4 attenuated TGF- β -Smad2 signaling and restored expression of the transcription factor Krüppel-like factor 2 (KLF2). Knockdown of KLF2 abrogated the anti-inflammatory and anti-EndMT effects of ANGPTL4, highlighting its essential role in endothelial protection. In human plaques, increased EndMT marker expression was associated with morphologically complex lesions, suggesting a link between EndMT and advanced atherosclerosis.

Conclusions: ANGPTL4 protects against endothelial inflammation and EndMT by suppressing TGF- β -Smad2 signaling and restoring KLF2 expression. These findings suggest that ANGPTL4 plays a critical role in preserving vascular integrity and preventing atherosclerosis progression.

Keywords: ANGPTL4, Atherosclerosis, Endothelial dysfunction, EndMT

PE-15

Eicosapentaenoic acid suppresses vascular smooth muscle cell calcification through regulation of miR-30c1-3p

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Objectives: The role of high-dose icosapent ethyl in reducing cardiovascular risk has been reported. However, the cardiovascular effect of its active component, eicosapentaenoic acid (EPA), has not yet fully understood. The aim of this study was to investigate the effect of EPA on calcification of vascular smooth muscle cells (VSMCs) and specific genes/pathways associated with beneficial effect of EPA.

Methods: Rat VSMCs were treated with β -glycerophosphate (β -GP) and treated or untreated with EPA thereafter to analyze calcification and its potential suppression, respectively. The degree of calcification was assessed by Alizarin Red staining and validated by qPCR of key osteogenic genes such as Runx2 or ALP. Whole genome sequencing covering using Illumina NovaSeq 6000 system to identify regulated genes in each group of cells. Top-ranked candidate genes were selected and their effects on calcification were verified by specific inhibitors.

Results: VSMC calcification by β -GP was reduced after EPA treatment by the mean of 50% (p=0.029), whereas ALP expression was lower by the mean of 44% on EPA treatment. A total of 254 genes showed significant expression changes after treating EPA. Pathways of ossification revealed significant changes in the cells with EPA treatment. Among top-ranked upregulated genes with EPA, miR-30c-1-3p was tested for potential role in attenuating calcification. When an inhibitor of miR-30c-1-3p was added, effect of EPA on VSMC calcification was lowered by the mean of 56% (p=0.029). Further analysis exhibited three genes co-regulated by EPA and miR-30c-1-3p.

Conclusions: These results indicate the role of miR-30c-1-3p in anti-calcifying effect of EPA. Further studies may provide more solid evidence on beneficial effects of EPA and associated miRNAs in vascular calcification.

Keywords: Omega 3 PUFA, Non coding RNA

PE-16

Effects of semaglutide on glucose metabolism in the streptozotocin-high fat diet-induced diabetic mouse model

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Objectives: Semaglutide, a glucagon-like peptide-1 receptor agonist, is a medication used for glucose lowering and weight loss. We aimed to investigate the effects of semaglutide on glucose metabolism in a streptozotocin (STZ)-high fat diet (HFD)-induced diabetic mouse model.

Methods: Twelve 8-week-old male C57BL/6J mice were randomly divided into three groups: HFD, STZ-HFD, and STZ-HFD + semaglutide (Sema group, receiving semaglutide at 0.1 mg/kg every other day for 9 weeks). STZ (100 mg/kg) was administered intraperitoneally for 3 consecutive days to the 8-week-old male C57BL/6J mice. Serum samples were collected from the mice to measure blood glucose and insulin levels, while urine samples were collected to assess albuminuria.

Results: The Sema group exhibited significantly reduced water intake, food intake, and body weight compared to the STZ-HFD group. The Sema group also showed significantly lower levels of serum glucose and insulin, along with improved glucose tolerance and insulin sensitivity, compared to the STZ-HFD group. Additionally, the Sema group had a significantly larger beta cell area compared to the STZ-HFD group. Serum IL-1 β levels were significantly reduced in the Sema group compared to the STZ-HFD group. However, no significant difference in albuminuria levels was observed between the Sema and STZ-HFD groups.

Conclusions: Semaglutide treatment improved glucose metabolism in the STZ-HFD-induced diabetic mouse model. The lack of improvement in albuminuria with semaglutide needs further investigation.

Keywords: Glucose metabolism, Semaglutide

PE-17

Multitarget modulation of LOX-1, 15-LOX-1, and NLRP3 by thiazole-coumarin-azomethine derivatives: a pharmacoinformatic strategy to disrupt lipid peroxidation-driven inflammation in atherosclerosis

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Objectives: Atherosclerosis is a chronic immunometabolic disease driven by vascular inflammation initiated by oxidized low-density lipoprotein (oxLDL). The endothelial receptor LOX-1 facilitates oxLDL uptake and triggers oxidative stress. Concurrently, 15-lipoxygenase-1 (15-LOX-1) catalyzes lipid peroxidation, generating pro-inflammatory lipid mediators that activate the NLRP3 inflammasome, promoting IL-1 β and IL-18 release. Targeting this interconnected axis of lipid uptake, oxidation, and innate immune activation offers a promising multitarget strategy to disrupt atheroinflammation and stabilize plaques.

Methods: Five thiazole-coumarin-azomethine derivatives were docked against LOX-1 (PDB: 1YPQ), 15-LOX-1 (PDB: 1LOX), and a homology model of NLRP3's NACHT domain (based on PDB: 7ALV) using AutoDock Vina. ADMET properties were predicted using pkCSM. The lead compound was further evaluated via β -cyclodextrin inclusion complex modelling to assess bioavailability enhancement.

Results: Compounds 6i and 6k demonstrated favorable binding affinities across all targets: LOX-1 (-10.4 and -8.4 kcal/mol), 15-LOX-1 (-9.6 and -8.3 kcal/mol), and NLRP3 (-10.7 and -11.1 kcal/mol), respectively. The lead molecules interacted with key active sites, including π - π stacking with Tyr166 in LOX-1, coordination with the catalytic iron in 15-LOX-1, and stable binding at the ATP cleft of NLRP3. ADMET profiling revealed high intestinal absorption ($\geq 90\%$), no CYP1A2 inhibition, low BBB permeability (log BB ~ -1.1), and acceptable oral toxicity (LD₅₀: 2.56-3.21 mol/kg). Lipophilicity values ranged from 6.0-6.8 (LogP), while water solubility was moderate (log S: -3.3 to -4.7). All compounds showed one or no Lipinski rule violations and bioavailability scores of 0.55. Although lead-likeness violations (2-3) indicated moderate complexity, β -cyclodextrin complexation provided favorable binding energy (-7.5 kcal/mol), supporting improved bioavailability.

Conclusions: Thiazole-coumarin-azomethine derivatives exhibited multitarget activity against LOX-1, 15-LOX-1, and NLRP3, addressing lipid uptake, lipid peroxidation, and inflammasome activation. This integrated pharmacoinformatic strategy highlights a novel scaffold for next-generation anti-atherosclerotic immunotherapies that simultaneously target oxidative stress and innate immunity.

Keywords: Atherosclerosis, Lipid peroxidation, Inflammasome modulation

PE-18

Mathematical modeling of palmitic acid-induced super-enhancer and noncoding RNA crosstalk regulating atherogenic inflammation in human monocytes

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Objectives: Palmitic acid (PA), a saturated fatty acid elevated in Western diets, contributes to vascular inflammation through complex epigenetic mechanisms. The interactions between PA-induced super-enhancers (SEs), long noncoding RNA PARAIL, and miR-204-5p signaling in monocyte dysfunction remain unexplored through integrated computational modeling. This study aims to construct a systems model to simulate how PA alters SE activity and noncoding RNA crosstalk in regulating inflammatory state transitions in human monocytes.

Methods: Enhancer and transcriptomic datasets were obtained from the Gene Expression Omnibus (GEO), including ChIP-seq and RNA-seq of PA-treated CD14⁺ human monocytes (GSE281939, GSE214160) and exosomal miRNA data related to endothelial-smooth muscle signaling (GSE260844). A system of 14 nonlinear ordinary differential equations was constructed to simulate feedback interactions among SE activation, PARAIL-HuR binding, and miR-204-5p-mediated communication. Parameter estimation was performed using a hybrid of particle swarm optimization and simulated annealing (1,000 iterations), with model performance validated by 5-fold cross-validation and an 80:20 train-test split using normalized enhancer occupancy and gene expression values. Bifurcation and global sensitivity analyses were conducted to define PA concentration thresholds and identify dominant regulatory interactions.

Results: The model identified a bistable inflammatory switch at $\geq 200 \mu\text{M}$ PA, characterized by $>78\%$ SE occupancy across 134 loci (95% CI: 74.3–81.6), 5.3-fold upregulation of PARAIL ($p < 0.0001$), and 41.8% suppression of miR-204-5p, leading to a 42.1% decrease in anti-calcification exosomal signaling ($p = 0.002$). This transition was associated with sustained NF- κ B activation, IRF1 expression and chromatin accessibility, and IL1B transcriptional amplification, consistent with enhancer looping to inflammatory gene promoters observed in time-course ChIP-seq simulations. Sensitivity analysis indicated that BRD4-SE dynamics and PARAIL-HuR interactions contributed 65.2% of the variance in inflammatory output, suggesting key regulatory control points.

Conclusions: This modeling framework describes a PA-driven, dose-dependent epigenetic mechanism regulating monocyte inflammation and proposes specific noncoding RNA-enhancer axes as potential targets in lipid-associated atherogenic pathways.

Keywords: Super-enhancer remodeling, Noncoding RNA regulation, Mathematical modeling, Palmitic acid-induced inflammation

PE-19

MicroRNA-17, -21, and -92a as novel predictive biomarkers for cardiovascular disease risk: A STAT3-mediated pathway analysis

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Objectives: Cardiovascular disease (CVD) remains the leading cause of death globally, accounting for approximately 19.41 million deaths as of 2021. Atherosclerosis, a major contributor to CVD, is characterized by the accumulation of lipids in large arteries. STAT3 activation promotes cytokine production and endothelial dysfunction. MicroRNAs such as miR-17, miR-21, and miR-92a may modulate STAT3 activity, thereby influencing the progression of atherosclerosis. This study aims to explore their regulatory roles to identify potential biomarkers for improving early CVD risk prediction and enabling personalized interventions.

Methods: A cross-sectional analysis was conducted involving 46 participants stratified according to Framingham Risk Score (FRS) categories. Plasma expression levels of miR-17, miR-21, and miR-92a were quantified using qRT-PCR. Correlations between miRNAs were assessed using Spearman's correlation analysis. In silico analyses were performed to investigate pathways influenced by these microRNAs and their shared target gene, STAT3, using the Human microRNA Disease Database (HMDD). Pathway interactions were further explored using KEGG and Gene Ontology (GO) enrichment tools.

Results: miRNA expression levels decreased across increasing FRS categories: miR-17-5p (1.17 to 0.25), miR-21-5p (2.22 to 1.39), and miR-92a-3p (1.03 to 0.00), respectively. A strong positive correlation was observed between miR-17-5p and miR-21-5p ($r = 0.961$, $p < 0.001$), with a moderate correlation between miR-17-5p and miR-92a-3p ($r = 0.326$, $p = 0.027$). In silico analysis using the Human microRNA Disease Database (HMDD) identified shared target genes, including CCND1, HMGB1, and STAT3. KEGG pathway analysis highlighted STAT3's involvement in atherosclerosis via the cytokine-cytokine receptor interaction and JAK-STAT signaling pathways.

Conclusions: The downregulation of miR-17, miR-21, and miR-92a levels may affect plaque progression by modulating STAT3-mediated signalling pathways. These findings highlight the importance of these molecules in various cellular processes and their potential roles in CVD, and should be further explored in larger cohorts and through longitudinal studies.

Keywords: microRNA, Cardiovascular diseases, Atherosclerosis, Biomarkers, STAT3

PE-20

Non-coding RNAs differently expressed in cell lines with normal and defective mitophagy

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Objectives: Mitophagy is the selective degradation of damaged mitochondria. Defective mitophagy leads to the accumulation of abnormal mitochondria and may cause various inflammatory diseases, including atherosclerosis. The role of lncRNAs in mitophagy regulation is understudied. The purpose of our study is to determine lncRNA differentially expressed in cell lines with defective and normal mitophagy.

Methods: We obtained 13 cybrid cell lines by fusion of p0 THP-1 with platelets of patients with atherosclerosis and determined mitophagy level via FCCP stimulation. We performed a transcriptomic analysis of cell lines with normal and defective mitophagy in three biological replications. We mapped the reads to the human genome assembly GRCh38 with Ensembl annotation using STAR (v2.7.0a). To analyze the differential gene expression we used the DESeq2 R package (1.12.3). We obtained RNA indexes from RNAcentral database and selected differentially expressed lncRNAs ($p\text{-adj} < 0.05$, $\log_2\text{FoldChange} > 1$).

Results: We identified 5 differently expressed lncRNAs genes. They are listed in the table. ST3GAL3-AS1 is antisense RNA to ST3GAL3, a glycosyltransferase that transfers sialic acid residues to various substrates, including phospholipids. There is evidence that sialylated glycosphingolipids are involved in the regulation of autophagy. CASC9 is lncRNA which deficiency increases autophagy via the AKT/mTOR pathway. LINC01857 is an endogenous competing RNA. It binds to miR-1281, preventing its interaction with the target deubiquitinase USP39, which is involved in the assembly of the spliceosome. ZEB1-AS1 is an antisense RNA to the ZEB1 transcription factor, which regulates the expression of a large number of genes, including those related to autophagy. PCAT7 is an RNA sponge for miR-134-5p and miR-324-5p.

Conclusions: We identified 5 differently expressed lncRNAs genes, several of which are known to be involved in autophagy regulation. Future studies may shed light on their role in mitophagy regulation and atherosclerosis pathogenesis. This work was supported by RSF Grant #25-25-00364.

Keywords: Mitophagy, lncRNA, Differential gene expression analysis

PE-21

HDAC inhibitor YAK577 mitigates vascular calcification through regulation of MMP14

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Objectives: Vascular calcification is a pathophysiological phenomenon in which calcium phosphate accumulates in blood vessels, and the process resembles a similar mechanism to bone formation. Vascular calcification is prevalent in various diseases, including atherosclerosis, diabetes, aortic valve stenosis, and chronic kidney failure. Histone deacetylases (HDACs) act as erasers of histone acetylation, affect gene expression, and have been reported to be involved in many diseases, including cancer and cardiovascular diseases, and therefore HDAC inhibitors have been shown to be effective in inhibiting the progression of these diseases. We recently reported that HDAC inhibitor YAK577 inhibits heart failure by reducing the expression of matrix metalloproteinase (MMP)12, but whether this HDAC inhibitor affects vascular calcification has not been studied.

Methods: Vascular calcification was induced by inorganic phosphate plus ascorbic acid treatment in vascular smooth muscle cells (VSMCs) and subcutaneously injection of vitamin D3 for 5 days in C57BL/6 male mice. Calcification was confirmed by Alizarin Red staining, calcium assay, and pro-calcification marker gene expression. Knockdown of MMP14 was performed in VSMCs. In vivo experiments, HDAC inhibitor YAK577 (10 mg/kg/day) was intraperitoneally injected to vitamin D3-treated mice for 7 days.

Results: YAK577 treatment reduced inorganic phosphate plus ascorbic acid-induced calcification, demonstrated by Alizarin Red staining and calcium assay. YAK577 treatment attenuated calcification medium (CM)-induced upregulation of pro-calcification marker genes (Bmp2, Runx2, and Msx2). The mRNA levels of MMP 1, 2, 3, 7, 9, 10, 12, and 14 showed a significant increase in CM conditions, however only MMP14 was reduced upon YAK577 treatment. Small interfering RNA for MMP14 significantly reduced the CM-induced calcium deposition and the expression of BMP2 and RUNX2 in VSMCs. YAK577 administration suppressed the vitamin D3-induced calcium accumulation, pro-calcification-related gene expression, and MMP14 expression in aorta tissues.

Conclusions: We suggest that HDAC inhibitor YAK577 could be new therapeutic target for the treatment of vascular calcification through.

Keywords: YAK577, Vascular calcification

PE-22

Prdx1-dependent stroke-associated microglia attenuate ischemic damage in a photothrombotic stroke model

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Objectives: Ischemic stroke accounts for 85% of all stroke cases and remains the second leading cause of mortality worldwide. Microglia, the resident immune cells of the central nervous system, play a pivotal role in post-stroke recovery. Stroke-associated microglia (SAM) have been identified as a unique subpopulation with antioxidant properties, largely mediated by peroxiredoxin 1 (Prdx1), and share phenotypic markers with disease-associated microglia (DAM). Notably, SAM has been characterized thus far only in the tMCAO model. This study aims to determine whether SAM also emerges in other ischemic stroke models, specifically the photothrombotic (PT) stroke model.

Methods: To simulate ischemic stroke, we induced PT stroke in WT mice expressing Prdx1. SAM marker expression was assessed in three brain regions—contralateral, infarct, and peri-infarct (non-infarct)—via Western blotting. Immunohistochemical analysis was performed to visualize the spatial distribution of SAM. To evaluate the functional role of Prdx1, infarct volumes were quantified using TTC staining in both WT and Prdx1 knockout mice.

Results: Due to the relatively small infarct size characteristic of the PT stroke model, no significant difference in SAM marker protein levels was observed when comparing the contralateral and ipsilateral hemispheres. Therefore, the brain was subdivided into three distinct regions for further analysis. Three days post-PT stroke, SAM markers were elevated at the protein level in the peri-infarct (non-infarct) region. Co-expression of CD63, a known SAM marker, and Iba1 was observed in microglial cells located in the peri-infarct part. Notably, Prdx1-deficient mice exhibited significantly larger infarct volumes compared to WT, indicating a protective role of Prdx1 in ischemic injury.

Conclusions: Our findings demonstrate that SAM, characterized by Prdx1-dependent antioxidant activity, is present and functionally relevant in the PT stroke model. These results suggest that SAM may represent a conserved neuroprotective mechanism across multiple ischemic stroke paradigms.

Keywords: Ischemic stroke, Prdx1

PE-23

Comparing cost-effectiveness of obesity interventions: insights into surgery, medications, and diets

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Objectives: Obesity poses significant health and economic burdens globally. Bariatric surgery is the gold standard for severe obesity, but emerging therapies like GLP-1 receptor agonists and dietary interventions offer alternatives. This study evaluates the cost-effectiveness of these interventions across diverse populations, stratified by age groups.

Methods: Data were sourced from systematic reviews, WHO, and UNDP reports. Cost-effectiveness was measured as cost per quality-adjusted life year (QALY) gained over 12 months. Interventions included bariatric surgery, GLP-1 therapies, and dietary interventions. Control variables were baseline BMI, comorbidities, and socioeconomic status. Subgroup analysis examined age-specific differences (18-34, 35-49, 50-64, ≥65 years). Regression models assessed cost-effectiveness, and descriptive statistics summarized costs and health outcomes.

Results: Bariatric surgery showed the highest QALYs gained (9.5) but at higher costs (\$15,000), achieving \$1,579 per QALY. Emerging therapies yielded 5.8 QALYs at \$8,500, with \$1,655 per QALY. Dietary interventions, the least costly (\$2,000), produced 3.2 QALYs at \$625 per QALY. Subgroup analysis revealed dietary interventions were most cost-effective for younger populations (\$500 per QALY for ages 18-34), while bariatric surgery excelled in older groups (\$2,100 per QALY for ages ≥65). Regression confirmed bariatric surgery's superiority in severe obesity ($p < 0.001$).

Conclusions: Bariatric surgery offers the greatest health benefits but is costlier, making it most suitable for older populations with severe obesity. Emerging therapies are viable for moderate obesity or patients ineligible for surgery, while dietary interventions are best for younger individuals. Policymakers should prioritize accessibility to these interventions based on demographic needs. Future research should explore combination therapies and long-term societal impacts.

Keywords: Bariatric surgery, Cost-effectiveness, Emerging therapies

PE-24

Healthy fat expansion is induced by extracorporeal shockwave treatment (ESW-treatment) under high-fat diet (HFD) in mouse

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Objectives: Obesity is a serious medical condition in which body fat mass is increased and accelerates the risk of metabolic diseases such as type 2 diabetes and cardiovascular diseases. Some people who are considered 'metabolically healthy obese' are protected from many adverse metabolic effects. However, there is no accepted definition of metabolically healthy obesity (MHO) and the precise mechanisms are not known. Extracorporeal shockwave (ESW) is a sequence of mechanical pulses characterized by high peak pressure (100 MPa), fast rise (<10 ns), and short lifecycle (10 μ s). Low-dose energy shockwave therapy has been proven to be beneficial for several medical conditions including orthopedic diseases. The objectives of this study is to investigate the effects of ESW in obesity and type 2 diabetes.

Methods: C57BL/6J mice on a high fat-diet (HFD) were treated with or without ESW targeting epididymal white adipose tissue (eWAT) twice per week for 16 weeks. Glucose tolerance test (GTT) and insulin tolerance test (ITT) were performed. After sacrifice mice, all organs were weighed and frozen for analysis. RNA-transcriptome sequencing analysis of adipose tissues from mice with or without ESW treatment and qRT-PCR were performed.

Results: ESW-treatment improves glucose tolerance and promotes insulin sensitivity under diet-induced obese wild-type (WT) mice by inducing white adipose tissue (WAT) expansion. ESW-treatment affects the pathways for mitochondria, fatty acid metabolism, adipogenesis, inflammation and extracellular matrix.

Conclusions: Taken together, we concluded that ESW treatment regulates metabolism and leads to metabolically healthy obesity (MHO).

Keywords: Extracorporeal shockwave, Obesity

PE-26

Trends and factors associated with LDL-C treatment target attainment rates among patients with secondary prevention setting of atherosclerotic cardiovascular disease: assessments from Japanese health checkup data

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Objectives: According to the latest Japanese Guidelines for the Prevention of Atherosclerotic Disease, the target for secondary prevention of low-density lipoprotein cholesterol (LDL-C) in patients with coronary artery disease is LDL-C <100 mg/dL, and in patients with acute coronary syndrome, familial hypercholesterolemia, diabetes, or atherothrombotic stroke is LDL-C <70 mg/dL. However, it is still unclear if the LDL-C treatment target attainment rates are in upward trend, and what are the factors associated with target attainment.

Methods: We aimed to investigate trends and factors associated with LDL-C treatment target attainment. Using the Health Checkup data in Kanazawa City in 2008, 2012, 2017, and 2022 (n=38,146-51,969), we analyzed the trends of secondary prevention of LDL-C treatment target attainment rates by Cochran-Armitage trend test, and the factors associated with target attainment by logistic regression analysis from the 2022 health examination.

Results: The LDL-C treatment target attainment rates were 24.6% in 2008, 30.2% in 2012, 33.0% in 2017, and 34.7% in 2022. We found that there was a significantly upward trend over time ($P<0.001$). Next, we also found that, male sex (odds ratio: 2.64, 95% confidence interval: 1.72-4.06, $P<0.001$), and several lifestyles were significantly associated with LDL-C treatment target attainment.

Conclusions: The LDL-C treatment target attainment rates tended to increase over time, and there are several factors associated with LDL-C treatment target attainment.

Keywords: Secondary prevention, Health checkup data, LDL-C

PE-27

Effect of statin and ezetimibe combination therapy on small dense LDL in patients undergoing coronary angiography

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Objectives: Small dense low-density lipoprotein (sdLDL) is recognized as a more atherogenic subfraction of LDL, particularly in patients with metabolic abnormalities. Although combination therapy with statins and ezetimibe has demonstrated superior efficacy in lowering LDL-C levels and reducing atherosclerotic cardiovascular disease (ASCVD) events, limited data exist regarding its specific effects on sdLDL reduction.

Methods: We conducted a retrospective analysis of 245 patients who underwent coronary angiography (CAG) for suspected coronary artery disease. Patients were categorized based on their lipid-lowering therapy: statin monotherapy versus statin plus ezetimibe combination therapy. Baseline statin intensity was classified as high (20.0%), moderate (62.4%), low (1.2%), or statin-naïve (15.5%). Ezetimibe was used in approximately 20% of the cohort. Lipid profiles, including sdLDL concentrations, were compared between groups according to statin intensity.

Results: Among patients receiving high-intensity statins, those on combination therapy showed significantly lower LDL-C levels (47.8 ± 24.3 mg/dL vs 84.9 ± 36.4 mg/dL, $p=0.02$) and sdLDL levels (13.3 ± 5.7 mg/dL vs 24.7 ± 11.7 mg/dL, $p=0.02$) compared to those on statin monotherapy. Similarly, in the moderate-intensity statin group, combination therapy resulted in greater reductions in LDL-C (61.7 ± 22.7 mg/dL vs 83.9 ± 37.9 mg/dL, $p=0.001$) and sdLDL (18.0 ± 7.2 mg/dL vs 25.5 ± 11.3 mg/dL, $p<0.001$) compared to those on statin monotherapy.

Conclusions: Statin and ezetimibe combination therapy is more effective than statin monotherapy in lowering both LDL-C and sdLDL levels in patients performing coronary angiography. These findings support the potential benefit of combination therapy in reducing atherogenic lipid subfractions, particularly in high-risk populations.

Keywords: LDL, Small dense LDL, Statin

PE-28

Bibliometric mapping of single nucleotide polymorphisms and lipoprotein(a) towards cardiovascular disease: evolution, contribution, and knowledge gaps

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Objectives: The role of lipoprotein(a) [Lp(a)] as a genetic risk factor for cardiovascular disease (CVD), largely determined by single nucleotide polymorphisms (SNPs) in the LPA gene, is a research area of growing clinical importance. However, its intellectual structure, evolution, and knowledge gaps have not been comprehensively mapped. This study performs a bibliometric analysis to systematically chart the global scientific landscape of research connecting SNPs, Lp(a), and CVD, identifying its historical evolution, key contributors, and thematic frontiers.

Methods: A total of 465 documents published from 1992 to 2025 were retrieved from the Scopus and Web of Science databases. This study used ScientoPy for quantitative analysis and VOSviewer for network visualization to assess publication trends, influential countries and journals, and core research themes derived from author keywords.

Results: The analysis reveals a field that has matured from foundational work into a phase of exponential growth, with a significant increase in publications since 2009. United States, China, and United Kingdom lead research productivity, with journals such as *Atherosclerosis* and the *Journal of the American College of Cardiology* serving as the primary outlets for impactful studies. Network visualization of research themes uncovered a sophisticated, multi-layered knowledge structure. The field has progressed from broad, foundational studies (Cluster 1: "genetics," "risk factors") to highly specific mechanistic (Cluster 4) and clinical (Cluster 3) investigations, all aimed at validating causal factors (Cluster 2) to ultimately refine cardiovascular risk management (Cluster 5).

Conclusions: This study provides the first comprehensive bibliometric roadmap of the SNP-Lp(a)-CVD research landscape. By mapping its key contributions and thematic evolution, this research identifies critical knowledge gaps and provides a strategic guide for future research, particularly in areas such as ethnic-specific risk and the pharmacogenomics of emerging Lp(a)-lowering therapies.

Keywords: Lipoprotein(a), Single nucleotide polymorphism, Cardiovascular disease, Bibliometric analysis, Scopus, Web of Science

PE-29

HLA DR genome editing with TALENs in human iPSCs produced immune-tolerant dendritic cells

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Objectives: To investigate the role of embryonic Ras (E-Ras) in enhancing the efficiency of somatic cell reprogramming and to elucidate the underlying molecular mechanism, particularly its effect on cell cycle regulation.

Methods: Mouse embryonic fibroblasts (MEFs) were transduced with E-Ras and Yamanaka's four factors (Oct4, Sox2, Klf4, c-Myc). Cell proliferation and cell cycle progression were assessed via WST-1 assay, cell counting, and flow cytometry. Cyclin expression and transcriptional regulation were analyzed using qPCR, Western blotting, and luciferase reporter assays. Inhibitors were used to dissect the involvement of JNK signaling. iPSC induction efficiency was evaluated by alkaline phosphatase staining and pluripotency marker expression.

Results: E-Ras expression significantly increased cell proliferation and accelerated G1-to-S phase transition by upregulating cyclins D and E, without affecting cyclins A and B. This effect was mediated by activation and phosphorylation of the transcription factor Sp1, which enhanced promoter activity of cyclins D and E. Mechanistically, E-Ras selectively activated JNK (but not ERK or p38), and inhibition of JNK abolished Sp1 activation, cyclin expression, and iPSC generation. Co-expression of E-Ras with four reprogramming factors substantially increased iPSC colony formation, which was reversed by JNK inhibition.

Conclusions: E-Ras enhances somatic cell reprogramming efficiency by stimulating cell cycle progression via the JNK-Sp1-cyclin D/E axis. This study identifies a novel E-Ras-dependent mechanism linking cell proliferation to reprogramming and suggests E-Ras as a potential modulator to improve iPSC generation protocols.

Keywords: iPSC

PE-30

FFAR-Based polygenic risk score and its interaction with macronutrient intake in relation to MAFLD risk in Korean adults

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Objectives: Metabolic dysfunction-associated fatty liver disease (MAFLD) is defined as hepatic steatosis accompanied by metabolic dysfunction. This study aim to investigate the association between a polygenic risk score (PRS) based on FFAR gene variants and the risk of MAFLD, as well as to examine whether macronutrient intake modifies this association in Korean adults.

Methods: A total of 2,667 adults aged 40-69 years from the Korean Genome and Epidemiology Study (KoGES) were included. Additionally, 7 single nucleotide polymorphisms (SNPs) within FFAR loci were selected to construct the PRS. Participants were categorized into tertiles based on their PRS. Cox proportional hazard models were used to estimate hazard ratios (HRs) for MAFLD, adjusting for demographic and dietary factors. Interaction between PRS and macronutrient intake quartiles was also evaluated.

Results: Higher PRS tertiles were significantly associated with increased MAFLD risk. In fully adjusted models, HRs for the second and third tertiles were 1.437 (1.115-1.851) and 1.349 (1.045-1.741), respectively (p-trend=0.0124). A significant interaction was found with fat intake ratio, where the genetic risk was more pronounced in individuals with higher fat intake (p-interaction=0.0033). Similarly, a significant interaction was observed between PRS and carbohydrate intake ratio, with a genetic effect being stronger in the lowest intake group (p-interaction=0.0101). Expression quantitative traits loci (eQTL) analysis showed that selected SNPs were associated with FFAR gene expression.

Conclusions: FFAR gene-based PRS value was significantly associated with MAFLD risk in Korean adults, with genetic risk being more prominent among individuals with high fat and low carbohydrate intake. These findings support a gene-diet interaction in MAFLD development and highlight the potential utility of integrating genetic and dietary information into personalized prevention strategies.

Keywords: Free fatty acid receptors (FFAR), Metabolic dysfunction-associated liver disease (MAFLD), Polygenic risk score (PRS), Dietary fats, Dietary carbohydrates, Dietary proteins

PE-32

Mapping the scientific landscape of sitosterolemia: a bibliometric and visualization study

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Objectives: Sitosterolemia is a rare autosomal recessive lipid metabolism disorder characterized by elevated absorption and decreased excretion of plant sterols leading to their excessive accumulation in the plasma and tissues. Despite its rarity, the clinical relevance of phytosterolemia has gained increasing attention in recent years, particularly due to its phenotypic overlap with familial hypercholesterolemia and the risk of misdiagnosis. This study aims to conduct bibliometric and visual analysis of global research trends in phytosterolemia. The findings will inform future investigations and enhance awareness among researchers, clinicians, and policy makers regarding sitosterolemia research.

Methods: This study employs a bibliometric analysis approach using publication data retrieved from Scopus database. The data will be processed using tools such as VOSviewer for visualizing and Bibliometrix (R package) for advanced statistical analysis and thematic mapping.

Results: A total of 677 journal publications from 1974 to 2025 were found on sitosterolemia, with an average of 47 citations per article. The number of publications and the related citations show an obvious fluctuating trend with annual growth rate of 6.51%. The author with the most documents published is Salen, G with 43 documents from 1981 to 2018 followed by Patel, S. B. with 35 documents and Tada, H. with 34 documents. Journals that had published the most are Journal Of Lipid Research, Journal Of Clinical Lipidology, and Journal Of Atherosclerosis And Thrombosis. USA, Japan, and China are the biggest contributors for publications. Publications on "Lipid metabolism, inborn errors" and "familial hypercholesterolemia" have increased rapidly this year.

Conclusions: This bibliometric analysis highlights a notable rise in sitosterolemia research, reaching its highest point in 2022, with the United States emerging as the top contributor and showing a strong emphasis on collaboration. The study outlines the present state of research in this field and provides a foundation for future studies aimed at improving.

Keywords: Sitosterolemia, Hypercholesterolemia, Bibliometric analysis

PE-33

Eight-year trends in random urine sodium-to-potassium ratio among Korean adults: findings from KNHANES 2016-2023

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Objectives: Recent Japanese guidelines recommend using the average sodium-to-potassium (Na/K) ratio from casual urine samples to assess hypertension and cardiovascular risk, suggesting cutoffs of 2 (optimal) and 4 (feasible). We aimed to evaluate the proportion of Korean individuals who would be classified as having elevated Na/K ratios using these cutoffs, based on random urine Na/K measurements obtained from the nationally representative Korea National Health and Nutrition Examination Survey (KNHANES) dataset.

Methods: We analyzed data from 50,440 participants in KNHANES from 2016 to 2023, all of whom had available random urine Na and K measurements. For each survey year, we calculated the urinary Na/K ratio and assessed the proportion of individuals classified as high risk using the established cutoffs of 2 and 4, stratified by age and sex. All analyses were performed without applying complex sampling weights.

Results: Median Na/K ratios were consistently lower than the mean values, indicating skewed distributions. Over the 8-year period, both median and mean Na/K ratios gradually increased from 2.28 and 2.66 in 2016 to 2.84 and 3.25 in 2023, respectively. The prevalence of Na/K ratios ≥ 2 and ≥ 4 also increased from 59.2% and 16.3% in 2016 to 71.3% and 28.1% in 2023. When stratified by age, the prevalence of Na/K ratios ≥ 4 showed a U-shaped pattern, with higher rates observed among individuals aged <20 and ≥ 70 years. When stratified by sex, males consistently showed a higher prevalence of Na/K ratios ≥ 4 than females across all age groups, except for those aged 20-29 years.

Conclusions: A substantial proportion of the Korean population exceeded the proposed urinary Na/K ratio thresholds of 2 and 4, with an increasing trend over time. The observed distribution patterns varied by age and sex, underscoring the need for demographic-specific interpretation when applying these cutoffs for hypertension and cardiovascular risk assessment in public health surveillance.

Keywords: Hypertension risk, Korea National Health and Nutrition Examination Survey, Sodium-to-potassium ratio

PE-34

Cardioprotective effects of β -Lapachone on Isoproterenol-Induced heart failure mice

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Objectives: This study aimed to elucidate the therapeutic effects and underlying molecular mechanisms of β -Lapachone in the context of isoproterenol-induced cardiac hypertrophy and heart failure in mice.

Methods: Cardiac hypertrophy and fibrosis were induced in 8-week-old male C57BL/6 mice by subcutaneous administration of isoproterenol (ISO) at a dose of 100 mg/kg/day for two consecutive weeks. Prior to ISO induction, mice were pre-treated with β -Lapachone for one week at two different doses (20 mg/kg/day and 80 mg/kg/day). β -Lapachone treatment was continued concomitantly with ISO injection and maintained until the mice reached 15 weeks of age. Body weight was monitored weekly, and cardiac function was assessed via echocardiography. Following the treatment period, blood samples were collected, and the heart tissues were harvested for histological and molecular analyses.

Results: β -Lapachone treatment significantly attenuated isoproterenol (ISO)-induced cardiac dysfunction, hypertrophy, and fibrosis in mice. Serum levels of creatine, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) remained unaffected by either ISO or β -Lapachone treatment, although a slight reduction in body weight was observed. Mechanistically, β -Lapachone enhanced mitochondrial function, likely through activation of NAD(P)H:quinone oxidoreductase 1 (NQO1) and the AMPK/NRF2/HO-1 signaling pathway. Additionally, β -Lapachone treatment ameliorated ISO-induced cardiomyocyte apoptosis. Furthermore, β -Lapachone exerted cardioprotective effects by promoting the activation and phosphorylation of CaMKK2/CaMK4/CREB signaling, thereby contributing to the mitigation of ISO-induced heart failure.

Conclusions: This study provides evidence that β -Lapachone exerts cardioprotective effects in isoproterenol-induced heart failure by reducing fibrotic remodeling and restoring mitochondrial function, primarily through the NQO1- and CaMKK2/CaMK4/CREB-dependent pathways.

Keywords: β -Lapachone, Heart failure

PE-35

ASR-ZnO nanoparticles mitigate oxidative and inflammatory stress in human umbilical vein endothelial cells

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Objectives: Green synthesis of nanoparticles is favoured for its eco-friendly and cost-effective nature. Zinc oxide nanoparticles (ZnO NPs) are considered biocompatible, as zinc is an essential trace element. In this study, *Alternanthera sessilis* Red (ASR), a medicinal plant with antioxidant, anticancer, antidiabetic, and lipid-lowering properties, was used to synthesize ZnO NPs for potential drug delivery applications. Given the role of vascular inflammation and oxidative stress in endothelial dysfunction, which may lead to atherosclerosis, this study aimed to evaluate the potential therapeutic effects of ASR-derived ZnO NPs on hydrogen peroxide (H₂O₂)- and lipopolysaccharide (LPS)-induced damage in human umbilical vein endothelial cells (HUVECs), a novel approach not previously explored.

Methods: Three groups were tested: ASR-ZnO NPs, ASR crude extract, and chemically synthesized ZnO, along with untreated and stimulated control groups. Cytotoxicity in HUVECs was assessed using the MTT assay over 24 hours, and non-toxic concentrations (1-4 μ g/mL) were selected for further experiments. For the reactive oxygen species (ROS) assay, cells were pre-treated with the test groups for 4 hours, followed by exposure to H₂O₂ (600 μ M) for 2 hours. Anti-inflammatory effects were evaluated by measuring VCAM-1 and ICAM-1 levels via ELISA after LPS (1 μ g/mL) stimulation for 6 hours in pre-treated cells.

Results: ASR-ZnO NPs (1-4 μ g/mL) significantly reduced H₂O₂-induced ROS levels in a concentration-independent manner, outperforming ASR crude extract (4 μ g/mL). In contrast, chemically synthesized ZnO (4 μ g/mL) had no significant effect on ROS reduction. LPS (1 μ g/mL, 6 hours) stimulation elevated both VCAM-1 and ICAM-1 levels. ASR-ZnO NPs (1-4 μ g/mL) effectively prevented VCAM-1 elevation, comparable to ASR crude extract (4 μ g/mL), whereas ZnO (4 μ g/mL) showed no effect. However, none of the test groups inhibited ICAM-1 elevation.

Conclusions: Our study demonstrated that ASR-ZnO NPs possess promising antioxidant and VCAM-1-selective anti-inflammatory effects in HUVECs, warranting further investigation.

Keywords: Green synthesis, *Alternanthera sessilis* red, Endothelial dysfunction, Zinc oxide nanoparticle, Drug delivery

PE-36

A novel mannose receptor agonist enhances self-renewal and therapeutic potential of mesenchymal stem cells

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Objectives: Mesenchymal stem cell (MSC) expansion in vitro is limited by senescence and loss of potency. We found that quinic acid, a mannose receptor bioisostere, enhances MSC proliferation, self-renewal, and anti-aging effects without impairing differentiation. Quinic acid activates the MKK-ERK/P38-CREB pathway, inducing Sox2, Nanog, Oct4, and Tert. It also promotes Cyclin E-mediated proliferation and improves heart regeneration in a mouse myocardial infarction model. These findings suggest quinic acid as a promising agent for MSC expansion and regenerative applications.

Methods: Cells or heart tissues were lysed in RIPA buffer supplemented with protease and phosphatase inhibitors. Protein concentration was quantified using the BCA assay. Equal amounts of protein were resolved by SDS-PAGE and transferred to PVDF membranes. Membranes were blocked with 5% BSA and probed with primary antibodies overnight at 4°C, followed by HRP-conjugated secondary antibodies and ECL detection. Cell viability was assessed using the WST-1 assay (Roche) according to the manufacturer's protocol. Absorbance at 450 nm was measured after 2 h incubation. For myocardial infarction (MI), 10-week-old male C57BL/6J mice underwent permanent ligation of the left anterior descending (LAD) coronary artery under isoflurane anesthesia. Cardiac function was evaluated by echocardiography at indicated time points. All animal procedures were approved by the Institutional Animal Care and Use Committee.

Results: 1. QA enhances MSC proliferation, stemness, and self-renewal. 2. QA enhances MSC-based regenerative therapies through MR activation. 3. QA enhances MSC-based regenerative therapies through downstream MAPK/CREB signaling.

Conclusions: This study demonstrates that quinic acid (QA) effectively enhances the stemness, proliferation, and self-renewal capacity of both adipose-derived stem cells (ADSCs) and human embryonic stem cell-derived MSCs through mannose receptor-mediated activation of the MKK-ERK/P38-CREB signaling pathway. QA treatment upregulated key pluripotency genes, extended telomere length, and suppressed cellular aging without compromising differentiation potential.

Keywords: Mesenchymal stem cell, Mannose receptor

PE-37

Elevated lipoprotein(a) levels are associated with increased arterial stiffness in hypertension

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Objectives: Lipoprotein(a) [LP(a)] is an emerging cardiovascular risk factor that has been associated with arterial stiffness, a predictor of cardiovascular events. However, the relationship between LP(a) and arterial stiffness in hypertensive patients remains unclear.

Methods: A total of 369 hypertensive patients without cardiovascular disease (mean age: 60.6 ± 12.4 years; female: 37.7%) were analyzed. Arterial stiffness was assessed using brachial-ankle pulse wave velocity (baPWV). Blood levels of LP(a) were measured after overnight fasting, and baPWV measurements were performed on the same day following blood sampling.

Results: Patients with higher baPWV ($\geq 1,800$ cm/s) had significantly elevated LP(a) levels compared to those with lower baPWV values (82.9 ± 83.8 mg/dL vs. 43.7 ± 66.1 mg/dL, $P < 0.001$). There was a significant positive correlation between log-transformed LP(a) and baPWV ($r = 0.401$, $P < 0.001$). Multiple logistic regression analysis confirmed that elevated LP(a) levels (≥ 50 mg/dL) were independently associated with higher baPWV (odds ratio: 5.7; 95% confidence interval: 2.61-10.64; $P < 0.001$), even after adjusting for traditional cardiovascular risk factors.

Conclusions: Elevated LP(a) levels are independently associated with increased arterial stiffness in hypertensive patients. LP(a) may serve as a valuable tool for risk stratification in hypertension. Further studies with larger sample sizes and longitudinal designs are needed to confirm our findings.

Keywords: Arterial stiffness, Hypertension, Lipoprotein (a)

PE-38

The impact of high protein diet with different source on cardiovascular disease risk factors in good and poor sleepers: a randomized controlled trial

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Objectives: Consuming high protein diet (HPD) is suggested to improve cardiovascular health particularly in older adults and recent review indicated that protein sources may play a role in cardiovascular disease (CVD) risk factors. Sleep status is essential for cardiovascular health. However, limited studies determined the effects of HPD with different sources on CVD risk factors in individuals with different sleep quality status. Therefore, this study aimed to assess the impact of HPD with different protein sources on CVD risk factors in Singapore older adults with different sleep quality status.

Methods: This was a 16-week single-blind, parallel randomized controlled trial and older adults were randomly assigned into control (n=19), HPD with 20g/d casein protein (n=18), and HPD with 20g/d soy protein (n=18). Global sleep score (GSS) from Pittsburgh Sleep Quality Index questionnaire was used to define good sleepers (GSS≤5) and poor sleepers (GSS>5). CVD risk factors including anthropometric measurement, blood pressure, lipid-lipoproteins and flow-mediated dilation were measured before and after intervention.

Results: In good sleepers, time (P<0.01) and interaction (P=0.03) effects on total cholesterol (TC) level were found and both casein and soy groups showed a significant reduction. Also, there was a time effect (P=0.01) in low-density lipoprotein cholesterol level, with a significant reduction in soy group. However, the time effect (P<0.01) in high-density lipoprotein (HDL) level indicated a significant reduction in control and soy groups. For poor sleepers, time effects were found in triglycerides level (P<0.01) and TC/HDL ratio (P=0.01) with a significant increase in control group while maintenance in HDP groups.

Conclusions: Consuming HPD is beneficial to cardiovascular health in Singapore older adults and particularly HPD with plant-based source exerts lipid-lipoprotein management effect in older adults with poor sleep quality.

Keywords: High protein diet, Protein source, Lipid profile, Cardiovascular disease risk factors, Sleep

PE-39

Prognostic significance of endothelial dysfunction in coronary heart disease patients

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Objectives: To evaluate the prognostic significance of biochemical markers of endothelial dysfunction in coronary heart disease (CHD) patients during two years of observation.

Methods: Primary the study included 173 CHD patients (the average age was 57.24±5.12 years): 92 patients with stable CHD 2nd-3rd functional classes and 81 patients - with acute coronary syndromes. During 2 years of observation information about 11 patients was lost. Secondary the data of 162 CHD patients observation were evaluated: 135 patients with uncomplicated disease and 27 - with an unfavorable outcome. All biochemical markers were estimated at the first contact with the patient. Endothelial function was assessed by the concentration of endothelin-1 (ET-1), soluble vascular cell adhesion molecules (sVCAM) and pregnancy-associated plasma protein A (PAPP-A) using ELISA method.

Results: The patients with unfavorable outcome had initially higher levels of endothelial dysfunction biomarkers than patients with uncomplicated course (p<0.001). There was no significant impact of the patient's age, gender, smoking status, BMI, and the presence of concomitant hypertension on the CHD prognosis (p>0.05). At the same time, the prognosis was significantly influenced by a decrease in left ventricular ejection fraction (p<0.01) and the course of CHD at the beginning of the study, with the highest reliability in the group of patients with acute myocardial infarction (p<0.05). There was only a tendency to reliability of previous myocardial infarction that had occurred more than 6 months before the study start (0.05<p<0.1). Assessment of odds ratio had confirmed the significant effect of endothelial dysfunction markers on the CHD patients prognosis (for sVCAM>1300.0 ng/mL OR=3.35, p=0.011; for ET-1>9.0 ng/mL OR=2.87, p=0.024; for PAPP-A>6.0 mIU/L OR=6.40, p<0.001).

Conclusions: An unfavorable outcome of CHD patients was associated with an increasing of endothelial dysfunction severity. Thus, markers of endothelial dysfunction (sVCAM, ET-1 and PAPP-A) are independent predictors of adverse cardiovascular events with long-term prediction in CHD patients.

Keywords: Coronary heart disease, Endothelin-1 (ET-1), Pregnancy-associated plasma protein A (PAPP-A), Soluble vascular cell adhesion molecules (sVCAM), Endothelial dysfunction, Atherosclerosis, Patients' prognosis, Patient' prognosis

PE-41

Patient-specific hemodynamic evaluation of coronary stenting for atherosclerosis

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Objectives: This study aims to evaluate the hemodynamic effects of customized stent implantation in patient-specific coronary arteries using computational fluid dynamics (CFD). It also seeks to establish a simulation-based framework that can predict potential post-procedural complications following percutaneous coronary intervention (PCI). By assessing changes in flow characteristics induced by stent geometry and placement, this approach intends to support safer and more effective interventional strategies for patients with atherosclerosis.

Methods: Coronary artery geometries were reconstructed from patient-specific computed tomography (CT) data. Customized stent models were virtually deployed in the affected segments using CAD-based modeling. Pulsatile flow conditions were applied to replicate physiological states, and transient CFD simulations were performed. Hemodynamic parameters including time-averaged wall shear stress (WSS), oscillatory shear index (OSI), relative residence time (RRT), and WSS-based divergence (WSSBD) were calculated and visualized. These indices were compared across pre- and post-stenting conditions to assess local flow disturbance, regions of risk, and the overall effect of stent placement on vascular dynamics.

Results: The simulations revealed meaningful differences in hemodynamic behavior between the unstented and stented models. Variations observed in the calculated indices indicate that the deployed stent affected local flow environments in a clinically relevant manner. These changes were consistent with known risk factors for restenosis, thrombosis, or disturbed vascular remodeling. The results confirm the utility of multi-index CFD analysis in detecting subtle yet significant flow alterations related to intervention design.

Conclusions: CFD-based evaluation incorporating multiple shear-related metrics provides a non-invasive and patient-specific approach to assessing the impact of coronary stenting. This method can guide optimization of stent design and positioning, offering valuable insights into improving procedural planning and long-term vascular outcomes in patients with atherosclerotic disease.

Keywords: Computational fluid dynamics (CFD), Coronary artery disease

PE-42

Predicting future cardiovascular events by lipid core burden index (LCBI)

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Objectives: Vulnerable coronary plaques, particularly those rich in lipid content, are key contributors to the onset of acute coronary syndromes. The Lipid Core Burden Index (LCBI) has gained attention as a promising imaging biomarker for detecting patients who may be at increased risk of experiencing future major adverse cardiovascular events (MACE). Near-infrared spectroscopy (NIRS) is a novel imaging technique that detects plaques with high lipid content, which is a critical indicator of plaque vulnerability. This study evaluates the prognostic utility of NIRS-derived LCBI in predicting MACE in patients undergoing coronary angiography.

Methods: We conducted a prospective study at National Institute of Cardiovascular diseases involving 100 patients undergoing NIRS-IVUS imaging during clinically indicated coronary catheterization. The primary endpoint was a composite of MACE (defined as cardiac death, myocardial infarction, or unplanned revascularization) at 12 months. Non-culprit lesions were imaged using Makoto NIRS-IVUS, and the highest LCBI was recorded for each patient. Patients were stratified into two groups based on LCBI values: high-risk (≥ 400) and low-risk (< 400). Event rates were compared using Kaplan-Meier analysis, and Cox proportional hazards modeling was used to assess the independent predictive value of LCBI.

Results: Among 100 patients (mean age 64 ± 9 years; 72% male), 23 % had a non-culprit lesion with LCBI ≥ 400 . At 12 month follow-up, the incidence of MACE was significantly higher in the high-LCBI group compared to the low-LCBI group (11.2% vs. 4.2%, $p < 0.001$). LCBI ≥ 400 remained an independent predictor of MACE (adjusted HR 3.6; 95% CI 2.1–6.2; $p < 0.001$). Notably, most events occurred at sites with $< 70\%$ angiographic stenosis.

Conclusions: NIRS-derived LCBI is an independent predictor of future MACE in patients undergoing coronary angiography. An LCBI threshold of ≥ 400 identifies high-risk plaques in non-culprit segments often missed by angiography alone. Incorporating NIRS imaging into routine clinical may enhance risk stratification and guide targeted preventive therapies in atherosclerotic patients.

Keywords: Lipid core burden index

PE-43

Ginsenoside compound K, a panax ginseng metabolite, attenuates macrophage phagocytosis by inhibiting histone deacetylase 6

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Objectives: This study aimed to determine whether ginsenoside compound K (GCK), a major metabolite of *Panax ginseng*, can prevent lipopolysaccharide (LPS)-stimulated macrophage phagocytosis by inhibiting histone deacetylase 6 (HDAC6), a class IIb deacetylase. As macrophage phagocytosis drives intracellular oxidized LDL accumulation, foam cell formation, and plaque development in atherosclerosis, GCK's potential to mitigate these processes via histone modification was assessed.

Methods: Macrophages were pretreated with GCK or the HDAC6-selective inhibitor Tubastatin A, then stimulated with LPS. Phagocytic activity was quantified by uptake of Giemsa-stained latex beads to calculate the phagocytic index. Expression of scavenger receptors and phagocytic activation markers were assessed. *In silico* docking simulations were performed to explore GCK binding to HDAC6, focusing on both catalytic and allosteric domain interactions.

Results: GCK treatment attenuated LPS-induced increases in phagocytic index, scavenger receptor and activation marker expression, and HDAC6 levels in macrophages, resulting in reduced bead uptake. Both GCK and Tubastatin A disrupted actin remodeling, thereby inhibiting phagocytosis, with combined treatment yielding even greater suppression. *In silico* docking revealed dual-site engagement of HDAC6 by GCK, with a catalytic-site hydrogen bond to His614 in catalytic domain 2 (CD2) and an allosteric interaction at Arg485 in the CD1-CD2 interface, supporting two complementary inhibition strategies.

Conclusions: GCK inhibits HDAC6-mediated deacetylation to attenuate LPS-induced macrophage phagocytosis, thereby reducing foam cell formation and underscoring its potential as a nutraceutical candidate for atherosclerosis.

Keywords: Ginsenoside compound K, Histone deacetylase 6, Phagocytosis, Macrophage, Atherosclerosis

PE-44

Association between dietary niacin intake and dyslipidemia in Korean adults: a cross-sectional analysis based on KNHANES 2016-2018

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Objectives: Although niacin has been linked to lipid metabolism and cardiovascular risk in Western populations, evidence regarding its effects on dyslipidemia in Korean adults is limited. This study aimed to examine the association between dietary niacin intake and the risk of dyslipidemia.

Methods: A cross-sectional analysis was conducted using data from the 7th Korea National Health and Nutrition Examination Survey (2016-2018). Dietary niacin intake was assessed by 24-hour recall and categorized as below or at or above the Estimated Average Requirement (EAR) threshold. Each component of dyslipidemia was defined as follows: 1) hypercholesterolemia, total cholesterol ≥ 240 mg/dL; 2) hypertriglyceridemia, triglycerides ≥ 200 mg/dL; 3) hypo-high-density lipoprotein (HDL)-cholesterolemia, HDL-cholesterol < 40 mg/dL for men, < 50 mg/dL for women; and 4) hyper-low-density lipoprotein (LDL)-cholesterolemia, LDL-cholesterol ≥ 160 mg/dL. Multivariable logistic regression was conducted to examine the association.

Results: After adjusting for age, body mass index, energy intake, household income, smoking, alcohol consumption, and physical activity, dietary niacin intake at or above the EAR was associated with a lower risk of hypertriglyceridemia among middle-aged adults aged 30-49 years (odds ratio [OR] 0.73, 95% confidence interval [CI] 0.57-0.93 in men; OR 0.70, 95% CI 0.50-0.98 in women) and older women aged 65-74 years (OR 0.54, 95% CI 0.36-0.82).

Conclusions: These findings suggest that sufficient dietary niacin intake may help reduce the risk of hypertriglyceridemia especially in middle-aged adults and older women.

Keywords: Niacin, Dyslipidemia, Triglycerides, Nutrition survey

PE-45

Association between laver consumption and dyslipidemia in Korean adults: a community-based prospective cohort study

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Objectives: Dyslipidemia is a major risk factor for cardiovascular disease, and dietary factors play a critical role in contributing to the risk of dyslipidemia. Laver (*Porphyra* spp.), a type of seaweed commonly consumed in East Asia, contains various bioactive compounds such as omega-3 fatty acids particularly eicosapentaenoic acid (EPA) and porphyran, which may exert beneficial effects on dyslipidemia. This study investigated the longitudinal association between laver consumption and the risk of dyslipidemia.

Methods: We conducted a 14-year prospective cohort study using data from the Korean Genome and Epidemiology Study (KoGES), including 2,361 adults aged 40-69 years. Dietary intake was assessed at baseline using a validated semi-quantitative food frequency questionnaire. Laver intake was categorized into tertiles based on daily consumption. Dyslipidemia was defined according to clinical lipid profile criteria during follow-up. Multivariable-adjusted Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs).

Results: During the 14-year follow-up, 1,810 incident cases of dyslipidemia were identified. Higher laver consumption was positively associated with intakes of EPA and porphyran. The cumulative incidence of dyslipidemia was significantly lower in the group with higher laver consumption ($P=0.0354$). Moreover, the highest tertile of laver consumption was associated with a reduced risk of dyslipidemia compared to the lowest tertile (HR: 0.85; 95% CI: 0.75-0.95; $P=0.0059$). In subgroup analyses, the inverse association remained significant in men ($P=0.0237$), women ($P=0.0339$), and particularly in postmenopausal women ($P=0.0447$).

Conclusions: Our findings show that higher laver consumption is associated with a lower risk of dyslipidemia, suggesting that laver consumption may serve as a beneficial dietary strategy for improving dyslipidemia.

Keywords: Dyslipidemia, Porphyran, Omega-3 fatty acids, Laver

PE-46

Biglycan enhances muscle regeneration and function in dystrophic muscle: evidence from mdx mice and human population data

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Objectives: Biglycan (BGN) has recently emerged as a novel myokine, accompanied by evidence supporting its role in muscle regeneration. However, its functional role in muscular dystrophy has not been investigated. Herein, the mdx mouse, a well-characterized model for DMD (Duchenne Muscular Dystrophy) was used to evaluate the effect of BGN on both muscle regeneration and functional improvement. Moreover, in vitro analyses were performed to explore the underlying mechanisms. To extend these findings to humans, we further assessed its association with muscle health indicators using large-scale human data from the UK biobank (UKB).

Methods: C57BL/10ScSn-Dmdmdx/J mice were stratified based on grip strength and randomly allocated to receive vehicle subcutaneously (s.c.) or BGN (2 mg/kg) via intraperitoneal (i.p.) injection for 10 weeks. To comprehensively assess motor function, rotarod tests and grip tests were conducted. To further elucidate the underlying mechanisms, immunoblotting was performed on undifferentiated C2C12 using total and phospho-S6/Akt antibodies. Linear regression analysis using UKB data assessed the association between circulating BGN levels and appendicular lean mass (ALM) and ALM/BMI.

Results: In the accelerating rotarod test, BGN-administered groups exhibited a statistically significant improvement ($p<0.001$).

Conclusions: This study demonstrates that BGN improves muscle function in dystrophic model and its circulating levels are associated with muscle mass, suggesting its potential as a marker of muscle regeneration across experimental and human contexts.

Keywords: Biglycan, DMD mouse model, Muscular endurance improvement

PE-47

Development of polyvinyl alcohol nanofiber-covered stents for coronary perforation

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Objectives: Coronary artery perforation can lead to serious outcomes. Although covered stents are available for emergency vessel sealing, limitations such as poor biocompatibility, restenosis, and thrombosis persist. This study aims to fabricate polyvinyl alcohol (PVA)-coated stents designed to provide both effective vessel sealing and sustained local drug delivery.

Methods: PVA nanofibers were coated onto bare-metal stents (Chonnam National University Hospital Stent, CNUH Stent) by electrospinning, and dual-coating was performed to improve balloon adhesion. Scanning electron microscopy (SEM) was used to assess surface morphology. Drug release kinetics from sirolimus-loaded PVA fibers were measured in vitro. For functional testing, the stents were placed in the left anterior descending diagonal branch of the coronary artery in a porcine model, and vessel closure was evaluated by angiography. Follow-up at 4 weeks was performed using OCT analysis to evaluate percent neointimal area and to compare with the GraftMaster stent group.

Results: SEM analysis demonstrated that both PVA and sirolimus-loaded PVA nanofibers formed uniform, bead-free coatings. In vitro drug release test of sirolimus-loaded PVA fibers confirmed continuous drug release for one month. Angiography confirmed effective vessel closure with PVA-coated stents. Four-week OCT evaluation suggested that percent neointimal stenosis was lower in the PVA-coated stent group compared to the GraftMaster stent group.

Conclusions: PVA-coated stents achieved effective occlusion of blood flow in the target branch, while sirolimus-loaded PVA fibers enabled sustained drug delivery. The combination of these features suggests a dual strategy for both vessel closure and future restenosis prevention. As sirolimus-loaded fibers were evaluated only in vitro in this study, further in vivo and long-term studies are necessary to confirm the safety and efficacy.

Keywords: Polyvinyl alcohol, Sirolimus

PE-48

Safety and efficacy evaluation of bioresorbable vascular scaffolds according to changes in a porcine coronary artery vessel size

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Objectives: The drug-eluting stents developed so far are permanently implanted, leaving the risk of stent fracture, local inflammation, and stent thrombosis. Therefore, it is very necessary to develop a new bioresorbable vascular scaffold (BVS) that can replace it. In this study aimed to investigate the safety and efficacy of BVS insertion in small vessels (reference vessel diameter ≤ 2.75 mm) and normal vessels (reference vessel diameter ≤ 2.75 -3.0 mm) in the coronary arteries of pigs.

Methods: BVS was fabricated from a PLLA tube using a femtosecond laser cutting machine (Coherent, Starcut Tube SL, Hamburg, Germany). sirolimus-eluting BVS [poly(L-lactide) (PLLA) scaffold backbone with drug sirolimus and polymer poly(D,L-lactide) (PDLLA) (SE-BVS) 2.75 $\times$ 18 mm, n=3 and 3.0 $\times$ 18 mm, n=3] was implanted in pig coronary arteries. The stents were implanted in the coronary artery at a 1.1:1 stent to artery ratio. Three stented coronary arteries in each group were finally analyzed using X-ray angiography, optical coherence tomography (OCT), and histopathologic evaluation 4 weeks after stenting.

Results: Histomorphologic findings showed no significant differences in neointima area, inflammation and injury scores between the two groups. Internal Elastic Lamina (3.69 ± 0.750 vs. 4.68 ± 0.754 , $p < 0.001$) and lumen area (2.31 ± 0.779 vs. 3.49 ± 0.787 , $p < 0.001$) were significantly higher in the normal vessels 3.0 mm group. Percent area restenosis (38.84 ± 12.941 vs. 26.025 ± 7.558 , $p < 0.001$) and Fibrin score (2.5 ± 0.51 vs. 1.8 ± 0.54 , $p < 0.001$) were significantly higher in the small vessels 2.75 mm group. Also, no significant change in strut thickness was observed in histological and OCT analysis.

Conclusions: BVS significantly reduced the rate of restenosis in the treatment of coronary arteries with a reference vessel diameter of 2.75 mm or more, and was relatively safe and effective.

Keywords: Bioresorbable vascular scaffolds, Porcine, Coronary artery, Vessel

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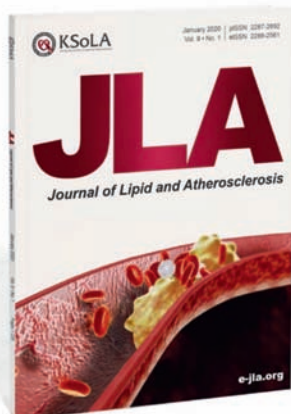
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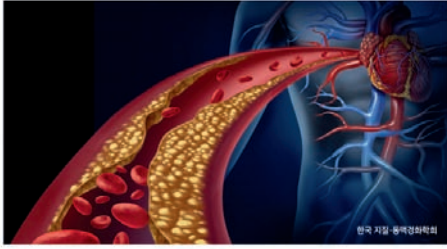
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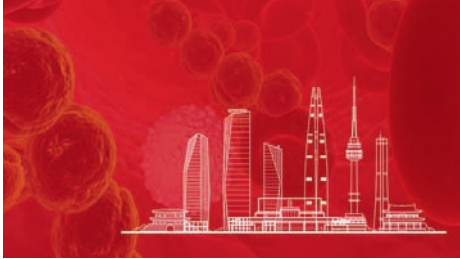
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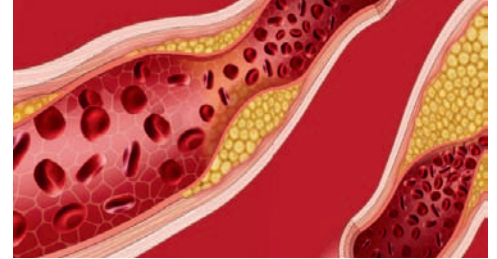


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References 1. Bowman AJ, et al. Nitric oxide mediated venodilator effects of nebivolol. *Br J Clin Pharmacol*. 1994 Sep;38(3):199-204. 2. Broeders MA, et al. Nebivolol: a third-generation beta-blocker that augments vascular nitric oxide release, endothelial beta(2)-adrenergic receptor-mediated nitric oxide production. *Circulation*. 2000 Aug 8;102(6):677-84. 3. IQVIA data 2023 based on global data, supported by IQVIA as retrieved on 8 Nov 2023.

* 75 countries with applicable scope, reflecting estimates of real-world activity in retail/hospital pharmacy channels.

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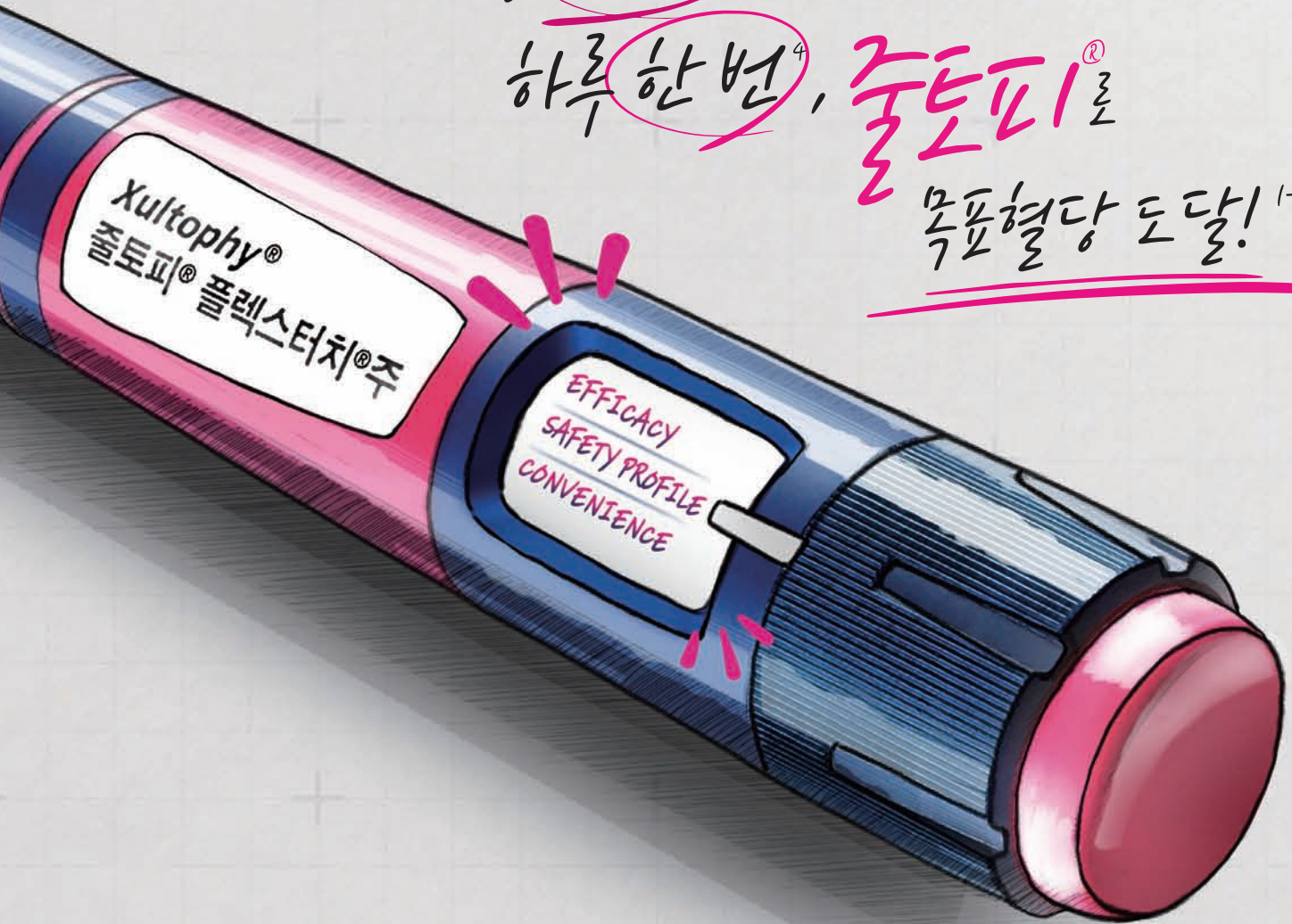
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***DUAL V:** 줄토피®(IDegLira) 투여 26주 차에서 HbA_{1c} 추정 치료 효과 차이는 -0.59%로 나타났습니다[ETD -0.59% (95% CI, -0.74 to -0.45), meeting criteria for statistical superiority ($P < 0.001$)]. 2차 평가 변수 중 26주 차 체중 변화 결과, IGLar U100 투여군은 baseline 대비 +1.8 kg의 변화를 나타냈으며, 줄토피®(IDegLira) 투여군은 baseline 대비 -1.4 kg의 변화를 나타내어 체중 증가 없이 유의한 혈당 강하 효과를 보였습니다[ETD -3.20 kg (95% CI, -3.77 to -2.64), $P < 0.001$].²

***DUAL VII:** 줄토피®(IDegLira)는 basal-bolus 대비 저혈당 및 체중 증가 없이 HbA_{1c} <7%를 달성한 환자 비율이 유의하게 높았습니다 (IDegLira 38.2% vs. IGLar U100 + IAsp 6.4%, $P < 0.0001$).³

***Confirmatory secondary end points:** proportion of patients achieving HbA_{1c} <7.0% after 26 weeks of treatment without severe or BG-confirmed symptomatic hypoglycemia in the last 12 weeks without weight gain.

BG, blood glucose; CI, confidence interval; ETD, estimated treatment difference; HbA_{1c}, hemoglobin A1c; IAsp, insulin aspart; IDegLira, insulin degludec/liraglutide; IGLar, insulin glargine; TZD, type 2 diabetes.

Study design

DUAL I: In a 26-week, double-blind trial, patients with T2D (A1C 7.5-10.0% [58-86 mmol/mol]) on basal insulin (20-40 units) and metformin with or without sulfonylurea/glinides were randomized (1:1) to once-daily (IDegLira + metformin or IDeg + metformin with titration aiming for fasting plasma glucose between 4 and 5 mmol/L. Maximum allowed doses were 50 dose steps (equal to 50 units IDeg plus 1.8 mg liraglutide) and 50 units for IDeg. The primary end point was change from baseline in A1C after 26 weeks of treatment. Secondary efficacy end points included doses of IDegLira and IDeg after 26 weeks, achievement of A1C levels of <7.0% (53 mmol/mol) and ≤6.5% (48 mmol/mol), and achievement of these A1C levels with or without confirmed hypoglycemia or weight gain, changes in laboratory-measured FPG, 9 point plasma glucose (PG) profiles, and body weight.¹

DUAL VI: Phase 3, multinational, multicenter, 26-week, randomized, open-label, 2-group, treat-to-target trial. 1:1 randomization to degludec/liraglutide ($n=278$; maximum dose, 50 U of degludec/1.8 mg of liraglutide) or glargine ($n=279$; no maximum dose), with twice-weekly titration to a glucose target of 72 to 90 mg/dL. Primary outcome measure was change in HbA_{1c} level after 26 weeks, with a noninferiority margin of 0.3% (upper bound of 95% CI, <0.3%). If noninferiority of degludec/liraglutide was achieved, secondary end points were tested for statistical superiority and included change in HbA_{1c} level, change in body weight, and rate of confirmed hypoglycemic episodes.²

DUAL VII: DUAL VII was a phase 3b, multinational, open-label, two-arm parallel, randomized trial in patients with type 2 diabetes conducted at 89 sites in 12 countries from July 2015 to October 2016. Patients were randomized to either IDegLira (insulin degludec 100 units/mL and liraglutide 3.6 mg/mL) or IGLar U100 (insulin glargine 100 units/mL) and insulin aspart (IAsp) (100 units/mL). Metformin was continued at pretrial doses. The primary end point was change in HbA_{1c} from baseline to week 26 of treatment. Confirmatory secondary end points included number of treatment-emergent severe or BG-confirmed symptomatic hypoglycemic episodes during 26 weeks of treatment and change in body weight from baseline after 26 weeks of treatment.³

References 1. Buse JB, et al. *Diabetes Care*. 2014;37(11):2926-2933. 2. Lingvay I, et al. *JAMA*. 2016;315(9):898-907. 3. Billings LK, et al. *Diabetes Care*. 2018;41(5):1009-1016. 4. 줄토피® 플렉스터치® 주 (insulin degludec/liraglutide). 국내제품설명서. <https://nedrug.mfds.go.kr/pbp/CCBB01/getItemDetailCache?cacheSeq=201905977&updateTs2023-07-03%2017:55:00.0b> (accessed 04 Feb 2024).



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[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.

[제품요약 정보] 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 병용투여하고 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)(황문근용해증이 나타날 수 있다.) 6) 담낭질환 환자(선생성 담낭 질환 환자 포함) 7) 피브레이트 또는 케토프로펜으로 치료하는 동안 광알레르기 또는 광독성을 경험한 환자 8) 소아 9) 담관간경화증 환자 10) 체장염 환자(중증 고중성지질혈증으로 인한 급성 체장염 제외) 11) 이 약은 유당을 함유하고 있으므로, 갈락토스 불내성(galactose intolerance), lapp(유당 분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토스 흡수장애(glucose-galactose-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)(황문근용해증이 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(신중후군) 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 포함 한다. **포장단위** 30정/PTP (10정/PTP X3) **저장방법** 기밀용기, 실온(1~30°C) 보관 제품에 대한 자세한 사항은 사용설명서를 참고하시기 바랍니다. 가장 최근 개정된 제품설명서의 내용은 현대약품 홈페이지를 통해 확인하실 수 있습니다. 설명서 작성년월: 2021년 10월

[제약사명]



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References 1. Grundy SM, et al. 2018 AHA/ACC/AACVPR/CAPA/PBS/ADA/AGS/AHA/APA/ASPC/LAN/APCA guideline on the management of blood cholesterol: Executive summary: A report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. *Circulation*. 2019;73(24):3169-209. 2. Data on file. 임상심리학회지 한국심리학회지 심리학과 상담심리 연구소. 원본상표 국제등록번호출원 번호 제 2대상은 AGT, AGZ의 병용요법의 유효성과 안전성을 비교평가하기 위한 다기관, 무작위화평행, 이중눈속임, 위약대조, 요인배제 제 3상 임상시험. 안국약품. 2023. 3. Choi JY, et al. Effect of pitavastatin compared with atorvastatin and rosuvastatin on new-onset diabetes mellitus in patients with acute myocardial infarction. *Am J Cardiol*. 2018;122:922-8. 4. 의약품안전나라. 의약품 제 제품정보 검색(제품명 : 페비로정). Available at: <https://nedrug.mfds.go.kr/searchDrug>. Accessed on 25 Oct. 2024.

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효능효과	관상동맥심질환(CHD) 고위험이 있는 성인환자에서 피타바스타틴 2mg 단일 치료요법시 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
약가	872원	저장방법	기밀용기, 실온(1~30℃)보관
보험코드	645306120	포장단위	30캡슐/상자(10캡슐/PTPX3), 300캡슐/병
		제조/판매원	한림제약

Ref. 1. Clinical Therapeutics/Volume 42, Number 10,2020(한림제약 스타펜 임상3상)

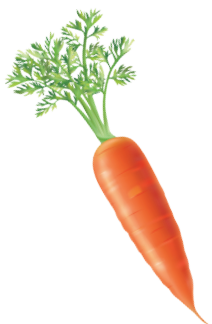
* **Original STAFEN** STAFEN은 Pitavastatin과 Fenofibrate의 복합제로서 최초로 승인된 한림제약의 개량신약입니다.

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(2024.10.01 기준)

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에제티미브 복합제
듀오논정 5/10 mg이**
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이상지질혈증 치료,

타바로젯 한 알로

(Pitavastatin/Ezetimibe)

바로 보다¹



References. 1, 식품의약품안전처 의약품통합정보시스템, 타바로젯정2/10 mg, 4/10 mg 의약품 상세정보, <https://nedrug.mfds.go.kr/> (accessed on 2025, 03,19), 2, Data on File, (원발성 고콜레스테롤혈증 환자를 대상으로 AGT, AGZ의 병용요법과 AGT 단일요법의 유효성 및 안전성을 비교평가하기 위한 다기관, 무작위배정, 이중눈가림, 활성대조, 요인설계 제 3상 임상시험) 3, Choi JY, et al, Am Cardiol, 2018;122:922-8.

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[효능·효과] 1. 만성동맥색증(허거씨병, 폐색성 동맥경화증, 당뇨병성 말초혈관병증 등)에 따른 껴임, 통증 및 냉감 등 허혈성 증상상의 개선 2. 뇌경색심인성뇌색전증 제외) 발증 후 재발억제

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3P-MACE, 3-point major adverse cardiovascular events; eGFR, estimated glomerular filtration rate

References. 1. Zinman B, et al. *N Engl J Med*. 2015;373(22):2117-2128. 2. Hadjadj S, et al. *Diabetes Care*. 2016;39(10):1718-1728. 3. The EMPA-KIDNEY Collaborative Group, et al. *N Engl J Med*. 2023;388(2):117-127.

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*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, myocardial 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:http://10.1161/CIRCULATIONAHA.118.038840. 4. 프랄루엔트®펜주 식약처 허가사항(75 mg 개정년월일: 2024.09.30/150 mg 개정년월일: 2022.12.20/300 mg 개정년월일: 2023.02.13)

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91.2%의 예방효과²

(VE 97.2%, 95% CI 93.7-99.0, p<0.001³; VE 91.2%, 95% CI 81.1-96.6²)

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¹2023 Global IQVIA data (J07E2 VARICELLA VACCINES, MOLECULE: VACCINE, VARICELLA ZOSTER) Herpes zoster vaccine 부문, Value 기준; ²2023 국내 IQVIA data (J07E2 VARICELLA VACCINES, MOLECULE: VACCINE, VARICELLA ZOSTER) Herpes zoster vaccine 부문, Value 기준



싱그릭스주
대상포진바이러스백신 (유전자재조합)

CI, confidence interval; VE, vaccine efficacy.

References 1. Huang CT, et al. Association Between Diabetes Mellitus and the Risk of Herpes Zoster: A Systematic Review and Meta-analysis. J Clin Endocrinol Metab. 2022;107(2):586-597. 2. Oostvogels L, et al. Medical conditions at enrollment do not impact efficacy and safety of the adjuvanted recombinant zoster vaccine: a pooled post-hoc analysis of two parallel randomized trials. Hum Vaccin Immunother. 2019;15(12):2865-2872. 3. Lal H, et al. Efficacy of an adjuvanted herpes zoster subunit vaccine in older adults. N Engl J Med. 2015;372(22):2087-2096. 4. 2023 Global IQVIA data (J07E2 VARICELLA VACCINES, MOLECULE: VACCINE, VARICELLA ZOSTER) Herpes zoster vaccine 부문, Value 기준. 5. 2023 국내 IQVIA data (J07E2 VARICELLA VACCINES, MOLECULE: VACCINE, VARICELLA ZOSTER) Herpes zoster vaccine 부문, Value 기준. 6. 싱그릭스 국내제품설명서.

[Integrated safety information] 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 열성 질환 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 투여하여야 한다.) 3. 약물이상반응 ○ 만 50세 이상의 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 주사부의 통증 및 종창, 피로, 근육통, 두통, 팔꿈치 및 위장관증상의 발생률이 더 높았다. 이들 약물의 이상반응 대부분은 오래 지속되지 않았다(지속시간 중앙값 2-3일). 중증 약물이상반응으로 보고된 반응은 1-2건만 지속되었다. ○ 만 18세 이상의 질병 혹은 치료로 인한 면역저하 또는 면역억제제로 대상포진의 위험이 높거나 높은 것으로 예상되는 자 (이하 "면역저하자 (immunocompromised)"로 지칭함) 예상된 약물이상반응을 자기조혈모세포이식 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%), 열림 (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 적용중으로 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 길랭-바레증후군의 발생 위험 증가가 관찰되었다(백신 투여당 1000,000 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 어머니 모체에 미치는 영향은 연구된 바 없다. 이 백신이 사람의 모유로 분비되는지 여부는 알려져 있지 않다. 3) 수태능: 동물시험에서 수컷 또는 암컷의 수태능 면에서 직접적 또는 간접적인 유해한 영향이 나타나지 않았다.

수입판매원: (주)글락소스미스클라인 | 서울특별시 용산구 한강대로 92 Tel. 080-901-4100 / **공동판매원:** GC녹십자 | 경기도 용인시 기흥구 이현로30번길 107 (보정동), 광동제약(주) | 경기도 과천시 과천대로74길 52 (갈현동) 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 companies or its licensor.

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LIPIDIL[®] NT
145mg Fenofibrate

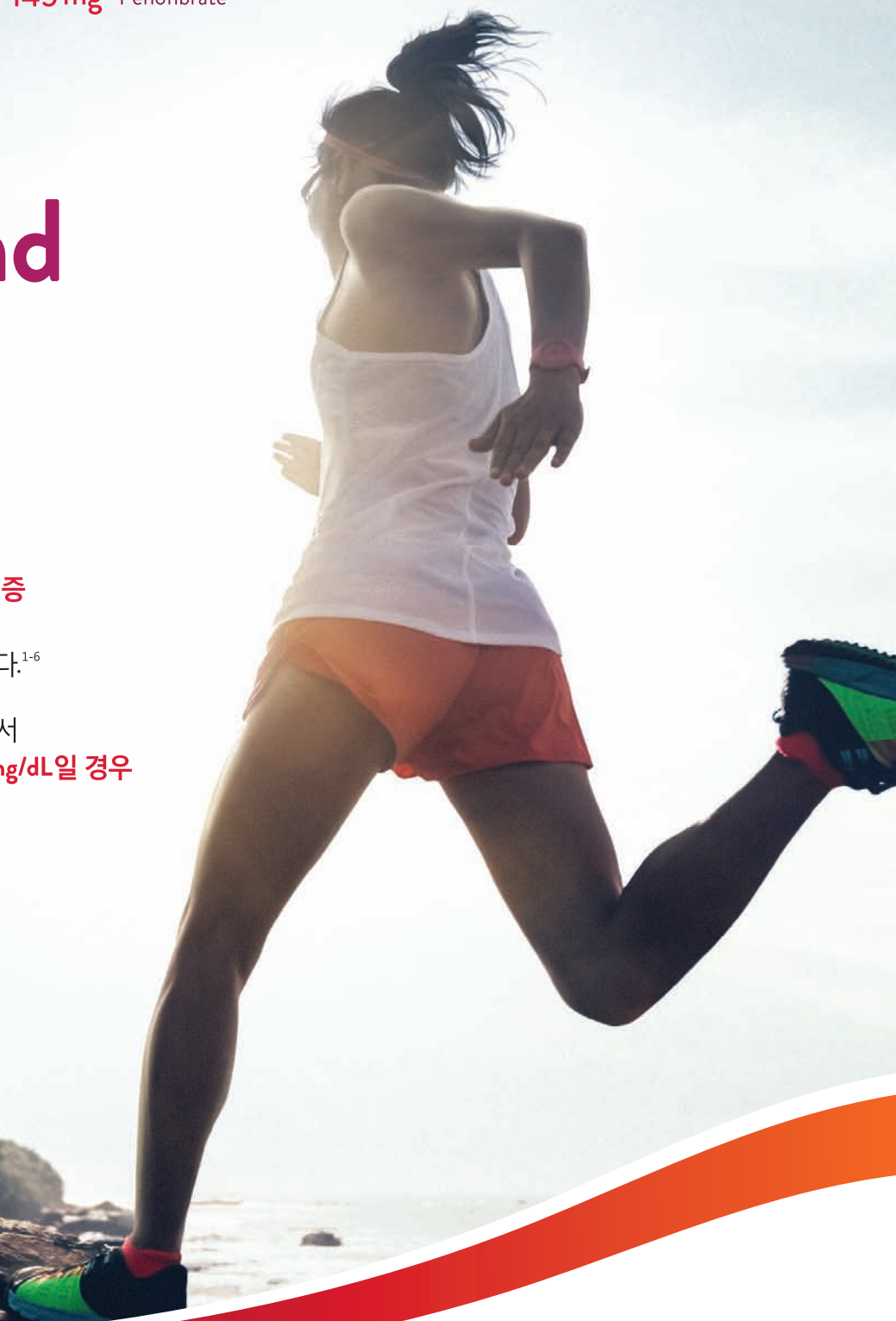
Go Beyond LDL-C



국내외 연구를 통해
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CV Risk의 독립인자가
될 수 있음이 확인되었습니다.¹⁻⁶



AACE 2023 가이드라인에서
Statin 복용에도 TG $\geq 200\text{mg/dL}$ 일 경우
Fenofibrate를 추가하도록
권고하고 있습니다.⁶



TG, triglyceride; CV, cardiovascular

* fibrat와 HMG-CoA reductase inhibitor의 병용요법은 혈문근용해증, 크레아틴키나아제(CK) 레벨 상승, 미오글로빈뇨 등의 이상반응이 나타날 수 있으므로, 신중히 투여되어야 합니다.
보다 자세한 내용은 제품설명서를 참고해 주세요. 한국에보트는 제품설명서 이외의 사용을 권장하지 않습니다.

References) 1. Kim NH, Han KH, Choi J, Lee J, Kim SG. Use of fenofibrate on cardiovascular outcomes in statin users with metabolic syndrome: propensity matched cohort study. *BMJ*. 2019;366:l5125. 2. SH Jo, et al. *Diabetes Care*. 2021 Aug;44(8): 1868-1876. doi: 10.2337/dc20-1533. 3. KS Kim, et al. *Metabolism*. 2022 Dec;137:155327. 4. Ting RD, et al. *Diabetes Care*. 2012 Feb;35(2):218-25. 5. ACCORD Study Group. Effects of combination lipid therapy in type 2 diabetes mellitus. *N Engl J Med*. 2010;362(17):1563-1574. doi:10.1056/NEJMoa1001282. 6. S.L. Samson, P. Vellanki, L. Blonde et al. *Endocrine Practice*. 2023;29(5):305-340. doi: 10.1016/j.eprac.2023.02.001.

* 자세한 제품 정보는 하단의 QR 코드를 통해 참고해 주시기 바랍니다.



리피딜[®]슈프라 정
(Fenofibrate 160mg)



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(Fenofibrate 145mg)

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다시 주목해야 할 분명한 이유입니다.

Reappraise,
메바로친®

[References] 1. Shepherd, J et al. "Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia. West of Scotland Coronary Prevention Study Group."

The New England Journal of medicine vol. 333,20 (1995): 1301-7.

2. Ford, Ian et al. "Long-Term Safety and Efficacy of Lowering Low-Density Lipoprotein Cholesterol With Statin Therapy: 20-Year Follow-Up of West of Scotland Coronary Prevention Study." Circulation vol. 133,11 (2016): 1073-80.

메바로친® 정 5, 10, 20, 40 mg

[주성분] 프라바스타틴 나트륨 [효능·효과] 1. 원발성고지혈증: 고콜레스테롤혈증(Ia형), 고콜레스테롤혈증과 고트라이글리세라이드혈증의 복합형(IIb형) 2. 고콜레스테롤혈증 또는 복합성고콜레스테롤혈증을 갖고 있는 환자 중 다음의 고위험군 환자에서 심근경색의 초발, 관상동맥심장질환 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 고콜레스테롤혈증에는 투여하지 않는다. 9) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당분해효소결핍증(Laplactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption)등의 유전적인 문제가 있는 환자에게는 투여하면 안 된다. 2. 이상반응 1) 과민증: 발진, 아나필락시스, 혈소판감소, 백혈구 감소, 용혈성 빈혈, 항핵항체(ANA) 양성, 혈액침착속도 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: 횡문근융해증, 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] 2023년 3월 1일 ※ 본 정보는 요약된 일부의 정보입니다. 따라서 최신 변경된 허가사항이나, 보다 자세한 내용은 한국다이이찌산쿄 홈페이지(www.daiichisankyo.co.kr)의 제품 설명서나 의약품안전나라(nedrug.mfds.go.kr)를 참고하시기 바랍니다.

[판매원]

[공동판매원]



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원인을 치료해야 한다. 지질 검사시에는 총콜레스테롤, LDL-콜레스테롤, HDL-콜레스테롤 및 트리글리세라이드를 포함해야 한다. 트리글리세라이드 수치가 400mg/dL 이상(4.5mmol/L 이상)인 경우에는 초원심분리로 LDL-콜레스테롤 농도를 측정해야 한다. 급성 관상동맥 사고로 입원할 경우에는 입원 시 혹은 입원 후 24시간 이내에 지질을 측정해야 한다. 환자의 퇴원 전 혹은 퇴원 시에 LDL-지하치료를 시작하는데 있어 이 측정치가 참고가 될 수 있다. **[용법·용량]** 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. **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 발생할 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 주의사항) *기타 자세한 사항은 제품 설명서를 참조하십시오.

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†† 기저대비 HbA1c 감소

Met, Metformin; Gemi, Gemigliptin; Dapa, Dapagliflozin; HbA1c, Glycated hemoglobin.

[References] 1. 식품의약품안전처 의약품통합정보시스템 (<https://nedrug.mfds.go.kr>). 2. Data on file, Clinical Phase III trial in Korea (Pivotal Study), LG-DPCL019 (2022), LG Chem. 3. Lee BW et al., (2022). Efficacy of Gemigliptin Add-on to Dapagliflozin and Metformin in Type 2 Diabetes Patients: A Randomized, Double-blind, Placebo-controlled Study (SOLUTION I) [Unpublished manuscript]. 4. Data on file, Clinical Phase III trial in Korea (Pivotal Study), LG-GLCL001 (2023), LG Chem.

제미다파[®] (제미글립틴/다파글리플로진) 50/10 mg (제품 허가일: 2022.06.21, 제품 출시일: 2023.04.08 / 제품의 Product Information 전문은 QR 코드를 통해 참고 하시기 바랍니다.)

[효능·효과] 이 약은 제미글립틴과 다파글리플로진의 병용투여가 적합한 제2형 당뇨병 환자의 혈당조절을 향상시키기 위해 식사요법 및 운동요법 보조제로 투여, [용법·용량] 이 약은 제미글립틴 50 mg과 다파글리플로진 10 mg을 투여하는 환자에게 식사와 관계없이 1일 1회 1정을 투여, 추정 사구체 여과율(eGFR)이 45 mL/min/1.73m² 미만인 경우에는 이 약의 투여는 권장되지 않음, 종종 간장에 황지 대상으로 이 약의 안전성과 유효성은 연구된 바 없음, [사용상 주의사항] 이 약의 주성분인 제미글립틴이나 다파글리플로진, 또는 첨가제에 대한 과민반응 병력이 있는 환자, 제1형 당뇨병 및 당뇨병성 케톤산증 환자, 갈락토오스 불내성, Lapp 유당 분해효소 결핍증 또는 포도당-갈락토오스 흡수장애 환자, 투약중인 환자에게 금기, [이상반응] 에트포르민과 다파글리플로진에 제미글립틴을 24주간 추가 병용 투여한 임상시험에서 1% 이상의 환자에게서 보고된 이상반응은 리파아제 증가, 만성위염, 어지러움, 소화불량, 기침, 당뇨병 신장병증, 위염, 치은염, 대장염증, 두드러기, 소파불안임, 24주 이후 52주까지 연장하여 진행된 기간 (후반 28주)동안 새롭게 확인된 이상반응 (연관관계 평가와 관계없이 보고된 이상반응)은 고열병증과 두통임 [제품 허가일 2022.06.21].



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리피로우정 (아토르바스타틴칼슘수화물)

【조성·성상】 1. 원료약품의 분량 1정 중 유효성분: 리피로우정 10 mg - 아토르바스타틴칼슘수화물(별규) 10.85 mg (아토르바스타틴으로서 10 mg), 리피로우정 20 mg - 아토르바스타틴칼슘수화물(별규) 21.70 mg (아토르바스타틴으로서 20 mg), 리피로우정 40 mg - 아토르바스타틴칼슘수화물(별규) 43.40 mg (아토르바스타틴으로서 40 mg), 리피로우정 80 mg - 아토르바스타틴칼슘수화물(별규) 86.80 mg (아토르바스타틴으로서 80 mg). 2. 성상: 흰색의 달걀형 필름코팅정제. **【효능·효과】** 1. 다음의 심장혈관 질환에 대한 위험성 감소. 2. 고지혈증. 3. 식이요법에도 불구하고 여전히 아래의 기준에 해당되는 이형지방 가축형 고콜레스테롤혈증을 가진 10~17세의 소아환자(여성의 경우 초경 이후의 환자의 총콜레스테롤, LDL-콜레스테롤, 아포-B 단백질 수치를 감소시키는 식이요법의 보조제(10, 20 mg에 한함)). **가.** LDL-콜레스테롤이 여전히 190 mg/dL 이상 (≥190 mg/dL)이거나 **나.** LDL-콜레스테롤이 여전히 160 mg/dL 이상 (≥160 mg/dL)이고 조기 심장혈관 질환의 가족력이 있는 경우 또는 해당 소아환자에서 두 가지 이상의 다른 심장혈관 질환의 위험인자가 있는 경우. **【용법·용량】** 1. 고지혈증 환자: 1일 1회 10~80 mg 범위로 투여, - 동형지방 가축형 고콜레스테롤혈증 환자: 1일 1회 10~80 mg. 2. 이형지방 가축형 고콜레스테롤혈증 소아환자(10~17세): 권장 초회용량 1일 10 mg, 권장 최대용량 1일 20 mg(10, 20 mg에 한함).

※ 자세한 내용은 제품설명서를 참고하시기 바랍니다.

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10/10 mg (가격: 637원/정)



구분	장축	단축	두께	총중량
기존설계	13	5	4	309.4
발매제품	11.3	4.5	4.5	257.50

17%

Ezetimibe/Atorvastatin
10/20 mg (가격: 808원/정)



구분	장축	단축	두께	총중량
기존설계	15	6	5	414.7
발매제품	13.2	5.3	4.7	360.50

13%

Ezetimibe/Atorvastatin
10/40 mg (가격: 1,415원/정)



구분	장축	단축	두께	총중량
기존설계	17	7	7	625.7
발매제품	15.5	6.0	5.6	566.50

Abbreviations API: Active Pharmaceutical Ingredient (유통이약품)

References 1 리피투®플러스정 허가증 2 Data on file 3 리피투®플러스정 국내 허가사항 식품의약품안전처 의약품 안전나라 Available at <https://medrug.mfds.go.kr/searchDrug> Accessed Oct 04 2022

리피토® 플러스정 10/10 mg, 10/20 mg, 10/40 mg (에제티미브/아토르바스틴 칼슘산수화물) 제품설명서 요약정보

[illegible]

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ASCVD, atherosclerotic cardiovascular disease.

Reference. 레파타[®] 주 프리필드펜(에볼로쿠맙) 제품설명서, 식품의약품안전처 의약품통합정보시스템.

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References 1. 리피토정 품목허가증(1999.12.01) 2. IQVIA Sales audit data, 2003년~2024년 Full year, 2023 3Q Total 기준 처방량 1위, (스타틴 단일제+복합제 전체 기준, ATC: C10A1+C10C0) 3. 한국지질·동맥경화학회 진료지침위원회. 이상지질혈증 진료지침 제5판. 2022.



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* Diabetes Ther. 2020 Apr;11(4):859-871(rosuvastatin 10mg monotherapy 대비 로수바미브 10/5mg의 유효성과 안전성을 확인). ⁶ 2022년 유비스트 '로수바미브정' 처방건 수 기준

전문의약품

로수바미브정(에제티미브/로수바스타틴합제) 10/5mg, 10/10mg, 10/20mg [참조약물 및 분량] • 로수바미브 10/5mg : 에제티미브(USP) 10.0mg, 로수바스타틴합제(분량) 5.2mg(로수바스타틴으로서 5mg) • 로수바미브 10/10mg : 에제티미브(USP) 10.0mg, 로수바스타틴합제(분량) 10.4mg(로수바스타틴으로서 10mg) • 로수바미브 10/20mg : 에제티미브(USP) 10.0mg, 로수바스타틴합제(분량) 20.8mg(로수바스타틴으로서 20mg) [작용] • 로수바미브 10/5mg : 분홍색의 장방형 필름코팅정 • 로수바미브 10/10mg : 도관색의 장방형 필름코팅정 • 로수바미브 10/20mg : 분홍색의 장방형 필름코팅정 [효능·효과] 항지방 고콜레스테롤혈증(이형지방 과다증 및 비가속형) 또는 혼합형 이상지질혈증 환자의 상승된 총 콜레스테롤(Total-C), LDL-콜레스테롤(LDL-C), apoB 단백질(ApoB), 트리글리세라이드(TG) 및 non-HDL-콜레스테롤을 감소시키고, HDL-콜레스테롤(HDL-C)을 증가시키기 위한 식이요법만 보조제로서 이 약을 투여한다. 고콜레스테롤혈증에 기인한 동맥경화성 혈관 질환의 위험성이 증가한 환자에게 지질조절약물을 투여할 때에는 모든 위험 인자를 고려해야 한다. 지질조절약물은 적절한 식이요법(포화지방 및 콜레스테롤 제한을 포함)과 함께 사용되고, 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, 만성 신부전, LDL-콜레스테롤을 증가시키는 약물 및 HDL-콜레스테롤을 감소시키는 약물(progestin, anabolic steroid, 및 corticosteroid))을 확인하여야 하며, 필요한 경우 이차적 원인을 치료해야 한다. 지질 검사에는 총콜레스테롤, LDL-콜레스테롤, HDL-콜레스테롤 및 트리글리세라이드를 포함해야 한다. 트리글리세라이드 수치가 400mg/dL 이상(4.5mmol/L) 이상인 경우에는 초완전분리 LDL-콜레스테롤 농도를 측정해야 한다. 급성 관상동맥 사고로 입원할 경우에는 입원 후 24시간 이내에 지질을 측정해야 한다. 환자의 퇴원 시 혹은 퇴원 시에 LDL 저하치료를 시작하는데 있어 이 측정치가 참고가 될 수 있다. [용법·용량] 이 약은 식사와 관계없이 1일 1회 투여한다. 이 약을 투여하기 전 또는 투여 중인 환자는 반드시 표준 콜레스테롤 저하제를 지속적으로 해야 한다. 이 약의 투여량은 환자의 LDL-콜레스테롤을 기저치, 권장되는 치료목표치 및 환자의 반응에 따라 조절되어야 한다. 완발성 고콜레스테롤혈증: 이 약의 용량범위는 1일 10/5mg~10/20mg이다. 초회용량으로 1일 10/5mg이 권장된다. LDL-콜레스테롤을 감소가 더 많이 요구되는 환자의 경우 용량을 조정하여 투여할 수 있다. 이 약의 투여를 시작한 후 또는 용량을 조정한 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 구성분 함량이 동일한 복합제)으로 전환할 수 있다. [사용상의 주의사항] 1. 다음 환자에는 투여하지 마십시오. 1) 이 약의 구성분 또는 구성성분에 과민반응이 있는 환자 2) 활동성 간질환 환자 또는 혈장 아미노산(알부민) 수치가 전인량으로 높은 용량을 수반한 환자 5. 알코올, 우유, 지방 3) 근질환 환자 4) 사이클로포스파이드 병용투여 환자 5) 중증의 신부전의 신장에 환자(creatinine clearance (CLcr)<30mL/min) 6) 일부 또는 일신하고 있을 가능성¹이 있는 여성 및 수유부 7. 일부 및 수유부에 대한 투여 참조 7) 근발증/혈당-갈락토스에 걸리기 쉬운 환자(혈당에 로수바스타틴 40mg과 함께 투여하는 글리제민). 이러한 인자들은 약제와 같습니다. (1) 중증의 신장장애 (크레아티닌 청소율 < 60mL/min) (2) 갑상선기능저하증 (3) 유전적인 근질환 병력 또는 가족력이 있는 경우 (4) 다른 스타틴계 약물(MG-CoA 전환효소, 치매) 또는 피브라이트 계열 약물에 대한 근육 독성의 병력이 있는 경우 (5) 알코올 중독 (6) 혈장 농도가 증가할 수 있는 상황 (7) 아시아계 환자 (8) 피브라이트 계열 약물을 병용하여 8) 이 약은 유당을 함유하고 있으므로, 갈락토스 불내성(galactose intolerance), Lapp 유당분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토스 흡수장애(glucose-galactose malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안 됩니다. (자세한 내용은 제품설명서 참조) [저장방법] 기밀용기, 실온(1~30도) 보관 [포장방법] 30정(PTP), 100정(병) [대용량] 22,1021 ※ 제품에 대한 자세한 내용은 최신의 제품설명서 또는 식약처 의약품통합정보시스템 홈페이지(<https://nedrug.mfds.go.kr/>)를 참조해 주시기 바랍니다.



제조판매사 **유한양행** • 본사: 서울 동작구 노랑진로 74 • 공장: 충청북도 청주시 청원구 오창을 연구단지 219
• 홈페이지: www.yuhan.co.kr • 소비자상담실: 080-024-1188 (수신자 요금부담)

로수바스타틴과에제티미브의 복합제
로수바미브정

KR-RSM-2300002



1년에 2번*, 일상을 돌려주는 6개월의 약속

LDL-C reduction

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

한국인 포함 대규모 3상 임상에서 LDL-C **57.2%** 감소⁴
실제 진료 환경에서 LDL-C 치료목표 **94.7%** 달성^{5,*}

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

Long-lasting (6 months)

* 1회 1회 1프리필드시린지 (인클리시란으로서 284 mg)를 피하 투여하며, 최초, 3개월 후, 이후 6개월마다 투여합니다.¹

[†] 초기 두 번의 인클리시란 투여 후, 각 6개월 간격의 투여 기간 동안 LDL-C 감소가 유지되었습니다.

References 1. (레비오프리필드시린지 (인클리시란트름)) 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. Huo 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.



위험도 분류에
치료 환자군

초고위험군은 심혈관질환이 있는 환자, 즉 이미 관상동맥질환

말초혈관질환으로 관상동맥질환으로

고위험군은

당뇨병 환자는 고위험군의

이험군은 심혈관질환

...동맥질

중등도위험군은 심혈관질환의 위험요소(흡연, 고혈압, 고지혈증, 당뇨병, 환자는 고위험군으로 분류한다.)

중등도위험군은 심혈관질환의 조기발병 가족력(부모 중 1명 이상)을 가진 경우

여자 55세 이상, 관상동맥질환이 발병한 경우) 중에서 LDL 콜레스테롤 수치가 160~190mg/dl인 경우

관상동맥질환이 발병한 경우) 중에서 LDL 콜레스테롤 수치가 160~190mg/dl인 경우

중등도위험군에 속합니다.

중등도위험군은 LDL 콜레스테롤을 제외한 주요 위험인자가 1~2개인 경우

저위험군은 LDE

스타틴에 의한

당뇨병 신규 발생을 줄이는 데 있어

대부분의 당료
고용량의 스타틴을 식

리바로젯®

- 복용 후 50% 이상 LDL-C 감소효과 입증¹⁾
- 저·중등위험군은 물론, 고위험군 이상으로 넓어진 치료범위²⁾

- 당뇨병 안전성을 공인 받은 유일한 스타틴
- 32개국 당뇨병 안전성 공인³⁾



1), 2) 리바로렛 3상 허가임상 결과 3) 32개 국가 현황 • 유럽(13개국): 영국, 독일, 프랑스, 스페인, 이탈리아, 핀란드, 네덜란드, 스웨덴, 오스트리아, 아일랜드, 포르투갈, 그리스, 노르웨이 • 동유럽(5개국): 러시아, 폴란드, 우크라이나, 조지아, 아르메니아 • 동아시아(5개국): 싱가포르, 대만, 인도네시아, 말레이시아, 카자흐스탄 • 중동(8개국): 사우디아라비아, UAE, 쿠웨이트, 카타르, 요르단, 오만, 레바논, 바레인 • 아프리카(1개국): 모로코