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Dyslipidemia and Cognitive Impairment

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In the brain, cholesterol is made in the astrocyte and forms a complex with ApoE, which is then internalized through the LDLRc of neuronal cells. The cholesterol, which enters neuron, plays a pivotal role in maintaining cell membranes such as forming lipid rafts. Thus, alteration of cholesterol metabolism in the brain may lead to both structural and functional abnormalities in neuronal cell bodies and myelin.

Studies on the relationship between changes in serum cholesterol levels and cerebral amyloidosis have been made based on this theoretical basis. Recently, the correlation between visit-to-visit variability of LDL cholesterol and cognitive performance has been also studied along with white matter hyperintensities.

Statin increased alpha-secretase activity and decreased amyloid beta production in addition to its lipid-lowering effects. Statin also decreased serum amyloid beta levels in a dose dependent manner. Autopsy data showed that typical AD pathology was reduced in statin users. However, the protective effects of statins on cognitive function in the recent meta-analysis was not clear. Rather, it has been reported to have cognitive side effects, so it is necessary to have a good understanding and knowledge of its efficacy and side effects.

There is a need to better understand the mechanism between dyslipidemia and cognitive impairment and continue to apply it to the development of therapies.