Editorial

Lipids as Effectors

Lipid, one of the major structural components of living cells, is also the most concentrated source of energy of all the nutrients. Lipids have also been recognized as signaling molecules. Lipid molecules such as fatty acids, eicosanoids, phosphoinositides and sphingolipids are known to control important cellular processes including cell proliferation, apoptosis and metabolism. They play key roles in inflammation, cancer and metabolic disease [1]. In this issue, effects and functions of various lipids on health are addressed and wide range of research results is presented.

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Alzheimer's disease (AD), which causes one of the greatest threats to the future healthcare system, is a degenerative and terminal disease with no current cure. Here, Corsinovi et al. gives comprehensive review on the modulation of AD development by dietary lipids focusing on the pathogenetic role of lipid oxidation products. Oxidative breakdown products of ω-6 polyunsaturated fatty acids (ω-6 PUFAs), and cholesterol oxidation products, might play a role in favoring Amyloid beta (Aβ) deposition, a hallmark of AD's onset and progression. Conversely, ω-3 PUFAs appear to contribute to preventing and treating AD. Generally, Aβ has been revealed as triggers of oxidative stress and cell apoptosis. Oxidative stress causes the damage to nucleic acids, lipids, and proteins, and consequently leading to the cell death. Oxidative stress in AD is generally attributed to the generation of reactive oxygen species (ROS) as well as reactive nitrogen species (RNS). Besides the cell damage or death caused by these free radicals, cis-trans isomerization of unsaturated fatty acids in cell membranes have also been found under free radical stress. As far as known, configuration may enhance membrane rigidity, meanwhile, it has demonstrated that Aβ impair synaptic plasticity [2]. Whether *trans*-fatty acids occur in synapse because of oxidative stress, what is the correlation of endogenous *trans*-fatty acids and AD are questions needed to be answered.

High-fat diet (HFD) has widely been used to induce diseases such as metabolic syndrome, diabetes, obesity and cancers in animal models. Using mice model, Do et al. investigated the global transcriptional and metabolic changes occurring in response to a high-fat diet. In 24 wk high-fat fed mice developed early clinical indicators of obesity-related complications including fatty liver, insulin resistance, hyperglycemia and hypercholesterolemia. Glucose regulating enzyme activity and gene expression were altered early in the HFD-fed mice. Fatty acid and triglyceride accumulation in combination with inflammatory changes appear to be likely candidates contributing to hepatic insulin resistance. In another study, using rat model Yamane et al. showed that HFD reduced ceramide and lipid synthesis in the skin and appeared to attenuate molecular mechanisms that underlie skin functionality.

The impairment of HFD can be improved through exercise and dietary supplementation with phytochemicals. In this issue, Ringseis et al. showed that regular endurance exercise improved the HFD-induced impairment of carnitine status through stimulating the expression of hepatic genes involved in carnitine synthesis and uptake. Beattie et al. showed that phytochemicals from the rhizomes of ginger family plants significantly diminished obesogenesis in mice on an HFD.

In another study, Bhuiyan et al. investigated the effects of ERS on glucose and lipid metabolism with mice fed an HFD containing an enzymatically modified rice starch (ERS). Four weeks of ERS feeding showed hypoglycemic effects with a significant reduction in fasting glucose, insulin, and leptin levels; improved glucose tolerance; and increased adiponectin concentrations compared with the HFD group.

Most of the various studies presented in this issue show the wide range biological effects of lipid

molecules such as *trans*-fatty acids, conjugated linoleic acid, alkylresorcinols and phytosterols in the body.

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References

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