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[논문]

1. Inhibitory Effects of Ginsenoside Compound K on Lipopolysaccharide-Stimulated Inflammatory Responses in Macrophages by Regulating Sirtuin 1 and Histone Deacetylase 4. H. Kang, S. Kim, J-Y. Lee, B. Kim. *Nutrients*. 2023 15(7): 1625.
2. Bioactive Compounds as Inhibitors of Inflammation, Oxidative Stress and Metabolic Dysfunctions via Regulation of Cellular Redox Balance and Histone Acetylation State. H. Kang and B. Kim. *Foods* 2023 12(5): 925.
3. DGKB mediates radioresistance by regulating DGAT1-dependent lipotoxicity in glioblastoma. H. Kang, H. Lee, K. Kim, E. Shin, B. Kim, JH Kang, B. Kim, J. S. Lee, J-M Lee, HS. Youn, BH Youn. *Cell Rep Med* 17: 4(1):100880.
4. The protective effects of *Aster yomena* (Kitam.) Honda on high-fat diet-induced obese C57BL/6J mice. M. J. Kim, J. H. Kim, S. Lee, B. Kim, H. Y. Kim. *Nutr Res Pract*. 2022 16(1): 46.
5. The Effects of Anthocyanin-Rich Bilberry Extract on Transintestinal Cholesterol Excretion. J. Hong, M. Kim, B. Kim. *Foods* 2021 10(11), 2852.

Trust your gut for cholesterol metabolism

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Hypercholesterolemia is one of the primary and modifiable risk factors for cardiovascular disease (CVD). Disrupted cholesterol homeostasis is highly associated with hypercholesterolemia. Cholesterol metabolism is regulated by de novo synthesis, uptake, efflux and excretion. The interplay between the liver and the intestine maintains cholesterol homeostasis. The liver, a primary site for cholesterol metabolism, contributes to de novo cholesterol synthesis, uptake of lipoprotein-derived cholesterol, and conversion of cholesterol to bile acids for biliary cholesterol secretion. Therefore, most of the studies about cholesterol homeostasis have been focused on the liver [4,5]. The absorption of dietary and biliary cholesterol is the primary role of the intestine in cholesterol balance. Cholesterol excretion is critical in human cholesterol metabolism, as there is a lack of the enzymes responsible for the degradation of the cholesterol ring. Classically, hepatobiliary cholesterol excretion mediated by high-density lipoprotein (HDL)-driven reverse cholesterol transport has been accepted as the only way to remove cholesterol from the body. In this

pathway, biliary cholesterol is subsequently excreted as fecal-neutral sterols. Transintestinal cholesterol excretion (TICE) is the nonbiliary cholesterol excretion, the second major pathway for cholesterol elimination from the body. In the alternative pathway to cholesterol removal, enterocytes directly uptake circulating lipoprotein-derived cholesterol from plasma for subsequent removal into the intestinal lumen for cholesterol excretion. The underlying mechanisms of TICE remain largely unknown. However, several studies reported that 30-40% of fecal sterol excretion is from TICE in both mice and humans under normal conditions. Recent studies support that the role of the intestine in cholesterol net balance has been underestimated. Therefore, dynamics of the alteration of intestinal cholesterol metabolism provide attractive intestine-specific nutritional strategies to lower hypercholesterolemia for protection against CVD. The recent insights into intestinal cholesterol metabolism and stimulation of TICE through nutrition will be reviewed and discussed.