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Editorial

Editorial: Dietary Fat

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Cardiovascular disease is still the world's leading cause of death [1]. Dyslipidemia is one of the most important risk factors for cardiovascular disease. Treatment of dyslipidemia with pharmaceutical or lifestyle therapies has been found to reduce cardiovascular disease-related mortality [2,3], emphasizing the relevance of addressing dyslipidemia in the prevention and treatment of cardiovascular disease. Dyslipidemia is caused by a disruption in lipoprotein metabolism, of which dietary fat absorption is a prominent component. Therefore, it is essential to understand how the small intestine absorbs dietary lipids and transport them as lipoproteins, namely chylomicrons and very low-density lipoproteins.

Cifarelli., *et al.* recently conducted a Research Topic on the lymphatic system's involvement in lipid metabolism [4]. The small intestine governs dietary fat absorption and the generation of chylomicrons and very low-density lipoproteins.

[5] examine congenital dietary fat absorption deficiencies and how these defects impact our understanding of chylomicron and intestinal very low-density lipoproteins production in the Review article. MTTP gene mutations that cause abetalipoproteinemia, APOB gene modifications that cause familial hypobetalipoproteinemia, SAR1B gene flaws that cause chylomicron retention disorder, and CD36 deficiency that causes elevated plasma triglycerides are only a few of the genetic defects described. Our knowledge of chylomicron and intestinal very low-density lipoproteins synthesis has aided in the creation of a number of essential lipid-lowering medicines, including MTP inhibitors (lomitapide) and cholesterol absorption inhibitors (ezetimibe). NPC1L1, the ezetimibe target, is important for cholesterol uptake as well as cholesterol esterification and intracellular trafficking [6].

Dietary fat absorption is dependent on cholesterol-derived bile acid, which is arguably one of the most essential physiological fat emulsifiers. Bile acid sequestering drugs can be used to decrease cholesterol levels and are safe for pregnant and nursing women [7]. Bile acid malabsorption occurs in inflammatory bowel disease, particularly Crohn's disease. [8], examine this issue from both a preclinical and clinical perspective in their Mini Review article. Importantly, people with Crohn's disease who have severe bile acid malabsorption have lower intraduodenal bile acid levels and poor micelle production. Despite the fact that bile acid malabsorption is widespread in these patients, it is not regularly evaluated in clinical practice [9].

The atherogenic lipoproteins produced by the small intestine are chylomicrons and very low-density lipoproteins, as previously stated. The intestine atherogenic lipoproteins contain ApoB48 rather than ApoB100, which is found in hepatic lipoproteins [10]. earlier provided one of the reasons why the small intestine produces ApoB48 instead of ApoB100. When challenged with a moderate dose (6 moles/h) of triglycerides, ApoB48 may facilitate more dietary lipid transport from the gut to the lymph than ApoB100 [11]. Lo and Coschigano report in the Perspective article that the trans-

fer of dietary lipids to the lymphatic circulation was comparable between the ApoB48-producing gut and the ApoB100-producing intestine when challenged with a high dosage (8 moles/h) of triglycerides. As a result, their findings show that the enhanced efficiency of ApoB48 in mediating dietary lipid transport from the colon seemed to vanish when a higher dose of triglycerides was given.

[12] present unique findings indicating that intestinal lymphatic flow, lipid transfer, and medication transport in larger animals, such as dogs, are more human-like. This is especially important because studies in small animals, such as mice, often underestimate the lymphatic drug transport in humans. The authors also propose that allometric scaling can be utilized to estimate lymphatic drug transport in humans based on data acquired from larger animal models.

[13], report in the Brief Research Report that patients with spinal cord injury have a lower peak in the increase of dietary triglycerides in their blood. The level of spinal cord injury was substantially connected with time-to-peak dietary triglyceride in the blood, despite the fact that the difference in amplitude was not statistically significant. To put it another way, the higher the location of the damaged spinal cord, the slower the dietary triglycerides in the blood reached their peak. According to their findings, spinal cord injury may cause dietary fat absorption to be disrupted.

Despite the fact that the basic physiological processes of dietary fat absorption are well recognized, there are still a few issues to be resolved. More research is needed to have a better understanding of the mechanisms of chylomicron packaging and release by enterocytes from the standpoint of cell and molecular biology. It's crucial to understand how dietary fat absorption contributes to the course of dyslipidemia from an integrative physiology standpoint. As previously stated, spinal cord damage has the potential to impact both dietary fat absorption and lipoprotein metabolism, but the relationship between the two has to be clarified. Dietary fat absorption, body fat deposition, and lipoprotein metabolism varies significantly between men and women [14]. All of these complicated relationships should be investigated further in order to gain a better knowledge of these physiological processes.

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