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LIPID METABOLITES AND CORONARY PLAQUE VULNERABILITY

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Metabolomics has been increasingly recognized as an enabling technique with the potential to identify key metabolomic features in an attempt to understand the pathophysiology and differentiate different stages of Coronary Heart Disease (CHD). We performed comprehensive metabolomic analysis in human plasma from 28 human subjects with Stable Angina (SA), Myocardial Infarction (MI), and Healthy Control (HC). Subsequent analysis demonstrated a uniquely altered metabolic profile in these CHD: a total of 18, 37 and 36 differential metabolites were identified to distinguish SA from HC, MI from SA, and MI from HC groups respectively. Among these metabolites, glycerophospholipid (GPL) metabolism emerged as the most significantly disturbed pathway. We used a targeted metabolomic approach to systematically analyse GPL, oxidized phospholipid (oxPL), and downstream metabolites derived from polyunsaturated fatty acids (PUFAs), such as arachidonic acid and linoleic acid. Surprisingly, lipids associated with lipid peroxidation (LPO) pathways including oxidized PL and isoprostanes, isomers of prostaglandins, were significantly elevated in plasma of MI patients comparing to HC and SA, consistent with the notion that oxidative stress-induced LPO is a prominent feature in CHD. Optical coherence tomography (OCT) has been considered as the ideal tool for the evaluation of atherosclerotic plaques. Circulating trimethylamine-N-oxide (TMAO), which is a metabolite of the dietary lipid phosphatidylcholine by gut microbiota, has recently been linked to elevated CHD risk. A total of 26 patients with CAD were recruited to assess coronary plaque using OCT and measure plasma TMAO level. According to plaque rupture status, patients were divided into plaque rupture group (n=12) and non-plaque rupture group (n=14). Plasma TMAO level was significantly higher in patients with plaque rupture than in those with non-plaque rupture (8.6±4.8 μmol/L vs. 4.2±2.4 μmol/L, p=0.011). In conclusion, circulating TMAO level may reflect coronary plaque vulnerability and progression.

Biography

Lemin Zheng has completed his PhD in 2005 from Cleveland Clinic/Cleveland State University in Clinical/Bioanalytical Chemistry. Currently, he is the Lab Director and Professor in Institute of Cardiovascular Sciences, Peking University (China), Key Laboratory of Molecular Cardiovascular Science of Ministry of Education. His main field of research is lipoprotein, lipids, vascular function and bio-material. He has published more than 60 papers in reputed journals, such as the *Journal of Clinical Investigation, Clin. Cancer Res. Advanced Functional Materials, Nanoscale, ACS APPL MATER INTERFACES, Free Radical Biology and Medicine, JAHA, International Journal of Cardiology, BBA-Molecular and Cell Biology of Lipids, JBC, Nature Structural & Molecular Biology, ATVB, Int. J Cancer, Cardiovasc Diabetol, Journal of Translational Medicine, AJP,* etc. He has more than 1600 SCI citations. He has served as an Editorial Board Member of *Cardiovascular & Hematological Disorders - Drug Targets, and Lipid & Cardiovascular Research*. He has a US patent.

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