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Lipoprotein lipase is a novel biomarker of insulin sensitivity and is produced by lipid- and glucose-targeting drugs through different mechanism in adipocytes and skeletal muscle cells

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Lipoprotein lipase (LPL) plays an important role in the absorption of TG from the bloodstream into the tissues. LPL abnormalities cause hypertriglyceridemia and low high-density lipoprotein-cholesterol (HDL-C) levels. Serum LPL mass is usually measured after a heparin injection. However, LPL mass was found to exist in the serum before heparin injection (preheparin serum).

The administration of troglitazone, an insulin-sensitizer, increased the preheparin LPL mass. Metabolic syndrome severity is negatively related to the preheparin LPL mass. The homeostasis model assessment of insulin resistance (HOMA-IR) is also correlated with the preheparin LPL mass. These data suggest that the preheparin LPL mass might be a good marker of insulin sensitivity.

Metformin increases preheparin LPL mass in type 2 diabetes mellitus patients. In an in vitro study, metformin enhanced the LPL production in skeletal muscle cells through the activation of adenosine monophosphate-activated protein kinase (AMPK); however, this effect was not observed in adipocytes.

3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins) reduce serum low-density lipoprotein-cholesterol (LDL-C) levels. Some statins reduce serum TG levels and increase serum HDL-C levels. Pravastatin and atorvastatin increase preheparin LPL mass in patients with type 2 diabetes. Simvastatin and pitavastatin increase LPL activity in adipocytes. In skeletal muscle cells, atorvastatin and pitavastatin enhance LPL production through the activation of AMPK.

This presentation will define the preheparin LPL mass and mention the effects of several lipid- and glucose-targeting drugs on serum LPL mass, in addition to discussing new mechanisms regulating triglyceride metabolism in adipocytes and skeletal muscle cells.

Biography

Masahiro Ohira, M.D. graduated from Toho University School of Medicine, Tokyo, Japan in 1999. He was a resident of internal medicine, Sakura Hospital, Toho University Medical Center from 1999 to 2001. He was a medical staff of internal medicine, Kashima Rosai Hospital, Ibaraki, Japan from 2001 to 2003. Since October 2003, he has been a medical staff of Center for Diabetes, Endocrinology and Metabolism, Sakura Hospital, Toho University Medical Center, Chiba, Japan. He received Ph.D. degree in 2011 from Toho University. His areas of research interest are lipid metabolism and diabetes.

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