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Perspective

Postprandial triglyceride-rich lipoproteins from Type-2 Diabetics

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Abstract

The role of nonfasting triglycerides, carried in VLDL and chylomicrons, as an independent risk factor for cardiovascular disease and death, has recently been convincingly demonstrated by evidence from prospective epidemiological large-scale studies. The accumulation of apoB-48-containing triglyceride-rich lipoproteins (TRLs) and the overproduction of large VLDL1 by the liver both occur prior to overt hyperglycemia, emphasizing the role of insulin resistance as an early pathogenic defect. The liver, adipose tissue, muscle, intestine and multiple insulin action site all exhibit insulin resistance. Together, these insulin action disruptions cause an excess of atherogenic VLDL and chylomicron remnant particles to be produced. Therefore, excessive cholesterol influx exposes the vascular wall endothelium, leading to endothelial dysfunction, oxidative stress, and prothrombotic condition over the course of the following meals. This overview reviews recent data on TRL secretion, intravascular processing, and removal accumulated over the past ten years and summarises current knowledge of diabetic postprandial dysmetabolism. Future possibilities and various therapeutic approaches are also covered.

Keywords: Hyperglycemia, Type 2 diabetes, Postprandial lipemia.

INTRODUCTION

In the postprandial state, patients with type 2 diabetes mellitus exhibit abnormalities in the plasma lipoprotein profile, particularly with regard to triglyceride-rich lipoproteins. Postprandial lipoprotein abnormalities may play a significant role in explaining the higher rate of cardiovascular diseases seen in these patients because they have been consistently linked to an increased risk of coronary heart disease. Fasting hypertriglyceridemia is a key component of diabetic dyslipidemia, and it is correlated with the severity of postprandial lipemia. However, we recently demonstrated that even with normal fasting triglyceridemia, patients with type 2 diabetes who had very good blood glucose control still had higher postprandial levels of large very low-density lipoprotein (VLDL) particles of both exogenous and endogenous origin (Boulet et al., 2020).

Regarding the potential contribution of hyperglycemia, it has been demonstrated that type 2 diabetes can be managed through improved glycemic control, regardless of how it is attained, which also lowers fasting triglyceride levels and postprandial lipemia.

The debates surrounding this topic are primarily caused by the complex interactions between insulin resistance, hyperinsulinemia, and hyperglycemia, which make it challenging to evaluate these variables separately. In actuality, no study has attempted to assess their distinct effects, and in particular, the function of insulin resistance per se has not been assessed. Therefore, our first goal was to assess whether insulin resistance itself played a role in the development of the postprandial lipid abnormalities typically found in type 2 diabetes patients, especially those without fasting hypertriglyceridemia (Julius, 2003).

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The development of vascular injury may begin with endothelial dysfunction. Increased oxidative stress is at least partially responsible for the dysfunction of the vascular endothelium that is brought on by both postprandial hyperglycemia and faulty TRL metabolism. An elegant study that included three meals-A high-fat meal, glucose alone and a high-fat meal plus glucose-assessed the effects of postprandial hyperglycemia and lipemia on endothelial function. In healthy subjects, a high-fat load and glucose by themselves both resulted in a decline in endothelial function and an increase in nitro tyrosine, with even more pronounced effects in diabetic subjects (Schwartz & Reaven, 2012).

CONCLUSION

Recent research, which was reviewed above, indicates that enterocyte-derived chylomicron synthesis is disturbed in Type 2 diabetes as a result of insulin signalling disturbances. Therefore, it makes sense to list the intestine alongside the liver, muscle, and adipose tissue as an organ that is insulin resistant. Additionally, similar to fatty liver, Type 2 diabetes may cause subsequent meals to have even worse effects on TRL number and composition than those seen after a single test meal. Future research is required to clarify this matter and explore the intestine as a novel therapeutic target.

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