Elevated triglycerides and rise in HDL cholesterol increase cardiovascular risk and diabetic dyslipidemia.

Peter Clay*

Division of Nutrition and Chronic Diseases, Pennington Biomedical Research Center, LSU System, 6400 Perkins Road, Baton Rouge, LA 70808, USA

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Introduction

Elevated triglyceride levels are a common dyslipidemic feature of type 2 diabetes and pre-diabetic diseases. A fasting triglyceride level of 150 mg/dl (1.70 mmol/l) is one of five accepted criteria for identifying those at high risk for cardiovascular disease and type 2 diabetes, termed the "metabolic syndrome" by some. According to some study, fasting triglyceride levels may help predict type 2 diabetes in the future. This was largely demonstrated when triglyceride levels were connected to other clinical indicators such as BMI, blood pressure, and other traditional cardiovascular disease risk factors, or with "high-normal" fasting plasma glucose levels.

The level of circulating triglycerides is influenced by the fed-fasted state, insulin sensitivity, and lifestyle factors such as nutrition and physical exercise [1]. These characteristics make triglyceride levels a very sensitive lifestyle biomarker at a given time point, but they also suggest that a single triglyceride measurement may not adequately reflect long-term triglyceridemia, particularly if lifestyle modifications were made during the follow-up period. It's uncertain whether measuring triglycerides at many time periods improves the link between triglycerides and diabetes.

The association between low HDL cholesterol and an increased risk of heart disease is widely proven, regardless of TG levels or other risk factors. In fact, "low HDL cholesterol" or "hypoalpha" is the most prevalent lipoprotein anomaly in coronary patients. According to intravascular ultrasonography studies, patients with low HDL cholesterol and high TG levels have more extensive coronary atheromas than those with a single increase in LDL cholesterol. Patients with low HDL cholesterol levels have similar intima-media thicknesses to those with familial hypercholesterolemia, but those with preexisting carotid atherosclerosis had plaque development reduced by high HDL cholesterol levels [2].

Triglyceride metabolism and diabetic dyslipidemia

Fatty acids and glucose both play key functions in delivering energy to physiological tissues during eating and fasting cycles. In addition to energy storage in adipocytes and other cells, triglycerides enable bulk transit of esterified fatty acids in circulating chylomicrons, very low-density lipoproteins (VLDL), and their remnants. Lipoproteins that are high in triglycerides are known as triglyceride-rich lipoproteins (TRL) [3].

Intestinal mucosal cells convert dietary fatty acids to triglycerides, which are then released as chylomicrons, which bypass the liver and enter the systemic circulation via the thoracic duct via intestinal lymph. Lipoprotein lipase (LPL), which hydrolyzes chylomicron triglycerides to liberate free fatty acids while also producing chylomicron remnants, subsequently transports dietary fatty acids to peripheral tissues via chylomicrons.

The liver receives some fatty acids from further lipolysis and absorption of remaining lipoproteins. Two further main sources of hepatic fatty acids are de novo hepatic lipogenesis and absorption of nonesterified fatty acids (NEFA) that circulate in plasma linked to albumin. Adipocyte triglyceride lipase (ATGL) and hormone sensitive lipase (HSL) are enzymes that release NEFA from adipocytes. Insulin prevents these intracellular enzymes from accessing adipocyte triglycerides, but when insulin levels are very low, they become active. Excessive NEFA synthesis by adipocytes in the context of insulin resistance and/or insufficiency appears to be a major contributor to dyslipidemia in diabetes and insulin-resistant conditions such as obesity [4]. LPL in peripheral tissues gradually degrades circulating VLDL, releasing fatty acids for use by muscle and other tissues, as well as storage as triglycerides in adipocytes. LPL activity also produces VLDL residual particles, commonly known as intermediate-density lipoproteins (IDL). IDL return to the liver, where they are partially internalised and partially processed at the cell surface by hepatic lipase, resulting in LDL. Insulin stimulates LPL activity, hence insulin resistance causes VLDL particle metabolism to be poor.

Hypertriglyceridemia can be caused by excessive VLDL production and/or insufficient lipolysis. TRL is involved in the cholesteryl ester transfer protein-mediated heteroexchange of neutral lipids (triglycerides and cholesteryl esters) with LDL and HDL, resulting in triglyceride enrichment of LDL and HDL particles in either scenario. Hepatic lipase activity causes LDL particles to become smaller, denser, and more atherogenic. As a result of similar lipolysis, HDL particles lose some of their apolipoproteins, which desorb from the

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decreasing HDL surface and are catabolized in the kidney.

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***Correspondence to:**

Peter Clay
Division of Nutrition and Chronic Diseases,
Pennington Biomedical Research Center,
Baton Rouge, USA
E-mail: claypeter@pbrc.edu

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