

***In vitro* Studies of Low-density Lipoprotein (LDL) Particles Modified by very Low-density Lipoprotein (VLDL) Particles Isolated from Diabetic and Non Diabetic Donors.**

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Hypertriglyceridemia (HTG) is a common complication in type II diabetes (DM) resulted from very low-density lipoprotein (VLDL) overproduction by the liver. Furthermore, VLDL particles from HTG patients carry more TG than VLDL particles isolated from normotriglyceridemia, leading to an accelerating transfer of triglyceride (TG) from VLDL core to LDLs. Triglyceride-rich low-density lipoproteins (LDLs) undergo TG core hydrolysis by lipoprotein lipase to become a smaller and denser particles called “small, dense LDL (sd-LDL)”. Such particles are more atherogenic than ordinary LDLs. This study was set up to study the influence of VLDLs isolated from different hypertriglyceridemic states of non DM and DM donors. VLDL particles were collected from DM and non DM donors which categorized by plasma TG; i.e., normotriglyceridemia (nTG, plasma TG<150 mg/dL), moderate hypertriglyceridemia (mHTG, plasma TG between 150-300 mg/dL) and severe hypertriglyceridemia (sHTG, plasma TG>300 mg/dL). LDLs were incubated with different VLDL particles in physiological condition. After 24-h incubation, modified LDLs (m-LDLs) became TG-rich and total cholesterol (TC) poor particles. Such lipid profiles which found in modified LDLs were similar to LDLs isolated from HTG donors. Both VLDLs isolated from HTG donors with DM and non DM altered LDL lipid compositions in a similar manner, whereas VLDLs isolated from nTG, DM donors yielded similar atherogenic m-LDLs. This study demonstrated that LDLs were able to be more atherogenic in both HTG and type II DM states, despite the latter being in nTG states.

Keywords: type II diabetes, hypertriglyceridemia, small, dense LDL