International Conference on

CARDIOLOGY AND CARDIAC NURSING

July 12-13, 2019 | Zurich, Switzerland

Hypertriglyceridemia in Type 2 diabetes-ominous new pathways

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Hypertriglyceridemia is a major component associated with atherosclerosis as well as type II diabetes. It is generally believed that increased lipid synthesis as well as decreased catabolism may account for the increased TG. In our studies, we noticed that mitochondria in diabetic state and from diabetic animals showed poor functionality. We also noted that Pyruvic acid; an energy substrate was degraded extra-mitochondrially yielding acetate from a non-energy producing mechanism. Furthermore, malondialdehyde (MDA), an end product of lipid peroxidation was converted to malonic acid and entered the saturated fatty acid synthetic pathway. Based on these, we propose a new paradigm and two novel pathways that would link oxidative stress to the increased generation of precursors for FA/TG synthesis. We propose that a) Pyruvate (PYR) in the cytoplasm would be non-enzymatically converted to acetate by lipid peroxides (LOOH) and hydrogen peroxide ($H2O_2$), thus increasing the acetate levels. Acetate, via acetyl coa would be used for both FA as well as for cholesterol synthesis. B) We also propose that malondialdehyde derived from the breakdown of lipid peroxides will be oxidized to malonic acid, which via malonyl coa will enter the FA synthetic pathway. As this bypasses the rate limiting acetyl coa carboxylase step, the process would not only favor FA accelerated synthesis but also could result in the depletion of polyunsaturated fat (via lipid peroxidation) and synthesis of saturated de novo fatty acid synthesis.

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