

The very low-density lipoprotein-triacylglycerol pathway and metabolic heterogeneity in familial combined hyperlipidemia

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Stellingen behorend bij het proefschrift:

“The Very low-density triacylglycerol pathway and metabolic heterogeneity in familial combined hyperlipidemia”

1. “Cytosolic triglyceride accumulation in nonadipose tissues such as muscle and liver is linked to the development of insulin resistance as these tissues also attempt to protect themselves from energy overload.” (GF Lewis et al., Endocrine Reviews, 2002; 23:201-229)
2. Glucometabolic insulin resistance of the liver can be secondary to full sensitivity to insulin-stimulated lipogenesis.
3. Hepatic steatosis is an integral part of familial combined hyperlipidemia, specifically the hypertriglyceridemic phenotype. (dit proefschrift)
4. “There is no threshold insulin cut-off value that marks the onset of insulin resistance in a given individual.” (M. Haque et al., Best Practice and Research, 2002; 16:709-731).
5. Insulin resistance may precede the dyslipidemia in familial combined hyperlipidemia.
6. The mechanism of hepatic secretion of heterogeneous very low-density lipoprotein subspecies is relevant to understand the metabolism of lipoproteins in familial combined hyperlipidemia and insulin resistance. (dit proefschrift)
7. The very low-density lipoprotein-triacylglycerol metabolic pathway is an important determinant of the phenotypic heterogeneity of familial combined hyperlipidemia, as characterized by multiple Fredrickson type hyperlipidemia within a family. (dit proefschrift).
8. Familial combined hyperlipidemia can be regarded as a genetic form of the metabolic syndrome.
9. The increased risk of coronary artery disease in the metabolic syndrome is associated with clustering of metabolic risk factors, rather than a single factor.
10. The light at the end of the tunnel can be a train (Murphy’s Law).
11. The best way to counteract weight gain and insulin resistance can be to limit your total calories, not just the fat intake.