Mitochondria, lipotoxicity and skeletal muscle metabolism : implications for type 2 diabetes mellitus

Stellingen

Behorend bij het proefschrift:

Mitochondria, lipotoxicity and skeletal muscle metabolism:
implications for type 2 diabetes mellitus

1. Diet-induced oxidative stress is not a prerequisite for the development of muscle insulin resistance. (This thesis)

2. The mitochondrial protein mitoNEET does not play a role in the regulation of skeletal muscle substrate oxidative capacity. (This thesis)

3. UCP3 does not preserve skeletal muscle mitochondrial oxidative capacity under lipid-challenged conditions. (This thesis)

4. 2,4-Dinitrophenol is not a good agent for improving glucose homeostasis in skeletal muscle. (This thesis)

5. Intramyocellular lipid content is a determinant of *in vivo* insulin resistance in humans. (Perseghin G et al, Diabetes; 48:1600-1606, 1999)

6. Despite the age-related loss of cellular functionality can have major repercussions at the level of mitochondrial redundancy, the diminished mitochondrial function with advancing age can also have an important role in the loss of overall cellular functionality. (Figueiredo P et al, Biogerontology; 9(2):67-84, 2008)

7. Appetite is governed by our thoughts, but hunger is governed by the body. (Martin CG, Low Blood Sugar: The Hidden Menace of Hypoglycemia)

8. Understanding the cellular mechanism(s) of insulin resistance in turn offers the prospect of better targeted and more effective therapeutic interventions for treatment and prevention of type 2 diabetes. (Petersen KF, Am J Med; 119(5):S10-S16, 2006)

9. We thrive not when we have done it all, but when we still have more to do. (Sarah Lewis, 2014)

10. Promoveren is net als het fietsen van de Amstel Gold Race: pieken en dalen, en de finish aan de top.

Bianca van Bree, 17 oktober 2014