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JOINT EVENT

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Obesity and GLP-1: Obesity pathophysiology and GLP-1 treatment potential

besity impairs almost all aspects of health and is a global challenge to our healthcare system as the prevalence reaches to 1 billion humans. Therefore, there is an acute need for better prevention and treatment strategies. Glucagon-like-peptide-1 (GLP-1), secreted from endocrine cells in the intestine upon meal intake, reduces food intake. We have previously shown that: Obese people have low endogenous GLP-1 response; weight loss induces a marked increase in GLP-1 response and; treatment with GLP-1 analogues facilitates long term weight loss maintenance (12 kg) accompanied by substantial improvement in metabolic health, compared to diet-induced weight loss maintenance. Chronic inflammation is an established part of the pathogenesis of obesity and activation of macrophages and T-cells in the expanded adipose tissue is coupled to the development of a pro-inflammatory state and insulin resistance. Interestingly, emerging evidence identifies GLP-1 as a potentially important immuno-modulator. GLP-1 decreases inflammation-associated gene and protein expression in macrophages and exerts antiinflammatory actions in adipocytes and endothelial cells as well as potent anti-inflammatory effects in humans.

Biography

Signe Torekov is currently an Associate Professor at University of Copenhagen, Denmark. She has a strong background in metabolic translational research. She has authored 30 original peer-reviewed papers; much of this work has been published in high-ranking journals in the field. In 2016, she has received the prestigious Novo Nordisk Foundation Excellence Fellowship. In 2015, she formed an international alliance in immuno-metabolism with top researchers at Oxford and Karolinska University.

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