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Invited talk: The role of NAFLD and hepatokines in metabolism

Norbert Stefan[3,1,2]

[1] *Institute of Diabetes Research and Metabolic Diseases (IDM) of the Helmholtz Center Munich, University of Tübingen, Tübingen, Germany*
[2] *German Center for Diabetes Research (DZD, Tübingen, Germany*
[3] *University Hospital of Tübingen, Tübingen, BW, Germany*

The liver is known to be involved in the natural history of the ongoing epidemics of type 2 diabetes mellitus and cardiovascular disease. In particular, the liver has a role in increased glucose production and dysregulated lipoprotein metabolism, conditions that are often found in patients with nonalcoholic fatty liver disease. Additionally, several proteins that are exclusively or predominantly secreted from the liver are now known to directly affect glucose and lipid metabolism. In analogy to the functional proteins released from adipose tissue and skeletal muscle-adipokines and myokines-these liver-derived proteins are known as hepatokines. The first hepatokine that has been proven to have a major pathogenetic role in metabolic diseases is α2-HS-glycoprotein (fetuin-A). Production of this glycoprotein is increased in steatotic and inflamed liver, but not in expanded and dysregulated adipose tissue. Thus, research into this molecule and other hepatokines is expected to aid in differentiating between the contribution of liver and those of skeletal muscle and adipose tissue, to the pathogenesis of type 2 diabetes mellitus and cardiovascular disease.

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Invited talk: Maternal obesity: New insights

Leonie Callaway

University of Queensland, Herston, QLD, Australia

Maternal obesity remains a major public health issue and a major clinical issue in the delivery of clinical care.

It remains a key risk factor in many adverse pregnancies and neonatal outcomes and is associated with a significant economic burden.

The drivers of human obesity are fascinating — dietary advice and guidelines, the food industry, exercise, exercise guidelines, appetite regulation, industry drivers, endocrine disruptors within our food change, changes in lifestyle, medications, trauma.

New insights into this fascinating area of medicine will be discussed.

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Invited talk: Impact, impact, impact — GP pathways to successfully empower your patients and families to better health and well-being

Gary Leong

Lady Cilento Children's Hospital, Brisbane, QLD, Australia

The overweight and obese child lives within a Family and Cultural environment in which your role as their GP is critical to the Family's long-term health and well-being. Dr. Leong will highlight the many factors and barriers to Family health change and some simple strategies you can take that will empower both you and your GP team and the Families who are seeking your help. You will feel enlivened and enthused after his talk to make a small but significant difference to your clients in your practice for better health and well-being.

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Invited talk: Recognising NAFLD and what should be done?

Norbert Stefan[3,1,2]

[1] *Institute of Diabetes Research and Metabolic Diseases (IDM) of the Helmholtz Center Munich, University of Tübingen, Tübingen, Germany*
[2] *German Center for Diabetes Research (DZD, Tübingen, Germany*
[3] *University Hospital of Tübingen, Tübingen, BW, Germany*

Nonalcoholic fatty liver disease (NAFLD) has gained much attention in the recent years because of its high prevalence, amounting to more than 30% in the general population and to more than 70% in certain high risk groups, such as morbid obese individuals and patients with type 2 diabetes. NAFLD strongly associates not only with progres-

sive hepatic, but also with cardiometabolic diseases and NAFLD is thought to be involved in the pathogenesis of cardiometabolic diseases. Diagnosis of NAFLD by the gold standard method, liver biopsy, is invasive and, therefore, not feasible in routine practice. Consequently, there has been intense interest in blood markers that, alone or in combination with clinical parameters, would be able to identify patients with NAFLD. The most effective and safe treatment strategy to reduce liver fat content and improve hepatic inflammation and fibrosis in subjects with NAFLD is lifestyle intervention. However, a considerable amount of patients is not compliant with the respective recommendations or liver fat content and/or liver pathology does not improve, although weight loss can be achieved. In this respect novel studies have indicated that specific pharmacological treatment approaches may be effective and relatively safe to treat NAFLD.

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Glucose-sensing neurons of the mediobasal hypothalamus project to brown adipose tissue

Paul N. Mirabella*, Aneta Stefanidis, David C. Spanswick, Brian J. Oldfield

Monash University, Clayton, Victoria, Australia

It is well established that neural input to BAT remains a critical feature of its functional recruitment. In the case of postprandial thermogenesis, activation of BAT sympathetic nerve activity following peripheral or central glucose infusion suggests a central nutrient-sensing mechanism in the regulation of BAT activity. It is hypothesised that BAT-directed neurons in discrete hypothalamic brain regions alter their electrophysiological properties in response to increased extracellular glucose concentration.

Injection of the GFP-tagged, transsynaptic retrograde virus, pseudorabies virus (PRV), into the interscapular BAT of Sprague-Dawley rats allowed for identification of neurons with a known polysynaptic projection to BAT. Whole-cell patch clamp recordings were performed on GFP+ neurons from coronal sections of the arcuate nucleus (ARC) and retrochiasmatic area (RCh). Increasing the extracellular glucose concentration from 1 mM (''fasted'') to 5 mM (''fed'') revealed both glucose-excited (6.00 ± 0.84 mV; 0.63 ± 0.18 Hz; $n = 14$ (29%)) and glucose-inhibited (-5.34 ± 0.75 mV; -0.34 ± 0.07 Hz; $n = 18$ (37%)) BAT-directed neurons in the ARC. Similarly, there were also substantial numbers of glucose-excited (7.32 ± 2.20 mV; 0.75 ± 0.22 Hz; $n = 5$ (45%)) and glucose-inhibited (-3.12 ± 2.24 mV; -0.80 ± 0.44 Hz; $n = 4$ (36%)) neurons in the RCh that projected polysynaptically to BAT. Retrospective immunohistochemical analyses of biocytin-filled cells revealed both POMC+ ($n = 9$) and POMC− ($n = 5$) glucose-sensitive neurons in both regions.

Furthermore, in attempt to delineate the heterogeneity of glucose-sensitive neurons through their monosynaptic projections, Retrobeads (Rb) were injected into the paraventricular nucleus, lateral hypothalamus and spinal cord of rats, and the glucose sensitivity of ARC/RCh double-labelled (Rb+/PRV+) neurons was tested.

These data provide a basis for the postprandial regulation of BAT thermogenesis through glucose-sensing mechanisms in hypothalamic neurons. They also provide additional insights into the axonal trajectory of identified hypothalamic glucose-sensors, which may form the basis of the observed heterogeneity within these populations of glucose-responsive, BAT-directed neurons.

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Effect of glucocorticoid on brown adipose tissue function in humans — A randomised double-blind placebo controlled cross-over study

Moe Thuzar[1,2,3,*], W. Phillip Law[2,4], Jeyakantha Ratnasingam[1], Christina Jang[1,2], Ken K.Y. Ho[1,2]

[1] *Department of Endocrinology & Diabetes, Princess Alexandra Hospital, Brisbane, Queensland, Australia*
[2] *School of Medicine, University of Queensland, Brisbane, Queensland, Australia*
[3] *Princess Alexandra Hospital, Translational Research Institute, University of Queensland, Brisbane, QLD, Australia*
[4] *Department of Molecular Imaging, Princess Alexandra Hospital, Brisbane, Queensland, Australia*

Background: Glucocorticoid (GC) excess causes obesity. In animals, GC inhibits brown adipose tissue (BAT) function, leading to weight gain. The involvement of BAT in the development of obesity induced by GCs in humans is not known.