

others, are promising. Intervention studies in large cohorts and other disorders like schizophrenia, bipolar and anxiety disorders are a key next step.

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Early life stress and subsequent obesity in women



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A history of childhood trauma, defined as events of abuse (physical, emotional or sexual) or neglect (physical or emotional) occurring before the age of 18 years is overwhelmingly present in individuals with Borderline Personality Disorder (BPD). This has been demonstrated repeatedly within the research, with reported figures of up to 84% of BPD patients having experienced some form of abuse or neglect and usually sexual and physical abuse. As such, childhood trauma is considered one of the most important factors in the aetiology of BPD. Childhood maltreatment coupled with genetic vulnerability evokes a stress response that can promote pathophysiological processes thus predisposing an individual to BPD. Chronic stress results from prolonged early life trauma, and also when the stressor itself is short in duration but is *perceived* to be threatening for much longer. As such, individuals with a history of childhood trauma often have altered hypothalamic–pituitary– adrenal (HPA) axis activity. The hyperactivation of the HPA axis due to chronic stress can increase the level of androgens in the body and result in clinical hyperandrogenism, which is a key feature in the diagnosis of polycystic ovarian syndrome (PCOS). Obesity is commonly associated with PCOS and also in women with early life trauma. In this presentation, the underpinning psychoneuroendocrine causal factors as well as the mental health consequences of obesity, PCOS and Borderline Personality Disorder will be discussed as well as novel treatment approaches.

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Facilitating change in retail settings covering government, small stores and supermarkets



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The facilitators and barriers to implementation from the experience of government in settings under their control



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What else can be implemented to enhance policy outcomes including pricing, labelling, placement and product



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The experience of Wester Leisure Services in improving the nutritional value of the food supply from a business perspective



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Engaging food businesses in healthy eating initiatives: lessons from the UK



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Introduction: Local governments play a large role in the health of their communities, and never more so than in the UK following the decentralisation of public health to local boroughs in 2012. Following this, the Healthier Catering Commitment (HCC) award was created as a response to high childhood obesity rates and the proliferation of fast food in London boroughs. HCC is a voluntary, local government–delivered initiative that engages food businesses (restaurants and takeaways) to improve the healthiness of their offerings (e.g. switching to healthier frying methods). Understanding the successful engagement strategies, challenges, and how to overcome them, will provide valuable insight into how local governments can influence their local food environments.

Methods: Key informant, semi-structured interviews were conducted with local government staff involved in HCC delivery, exploring their experiences and challenges of engaging food businesses. A thematic analysis approach was used.

Results: Participants drew on a variety of strategies to engage businesses, highlighting incentives, the ease of joining and the potential benefits both to their business and the health of their customers and community. The main barriers to joining were a fear of loss of business, as well as practical challenges to implementing and maintaining the award. HCC officers were also impeded by limited resourcing, balancing this by drawing heavily on shared resources and in-kind support from within and across councils. The ease of understanding and implementing the HCC, and its flexibility in delivery were seen as key strengths. Participants discussed

the potential of HCC to have wide-spread impact by changing the expectations around healthy food provision.

Conclusion: This study shows engagement methods and potential challenges of the HCC, and provides lessons for other local governments in the UK and abroad considering similar actions to address the healthiness of food businesses in their community.

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Cellular energy sensing and metabolism: implications for treating obesity



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The survival of all cells is dependent on the constant challenge to match energetic demands with nutrient availability, a task which is mediated through a highly conserved network of metabolic fuel sensors that orchestrate both cellular and whole organism energy balance. A mismatch between cellular energy demand and nutrient availability is a key factor contributing to the development of obesity, thus understanding the fundamental mechanisms by which cells sense nutrient availability and demand may lead to the development of new treatments. Glucose lowering therapies such as caloric restriction, exercise, metformin and cold all induce an energetic challenge that results in the activation of the cellular energy sensor AMP-activated protein kinase (AMPK). Activation of AMPK in turn suppresses lipid synthesis and inflammation while increasing glucose uptake, fatty acid oxidation and mitochondrial function. In contrast, high levels of nutrient availability, suppress AMPK activity while also increasing the production of peripheral serotonin, a gut-derived endocrine factor which suppresses beta-adrenergic-induced activation of brown adipose tissue. Identifying new ways to manipulate these two ancient fuel gauges, by activating AMPK and inhibiting peripheral serotonin, may lead to the development of new therapies for treating obesity.

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What should we over-eat?

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The diverse obesity phenotypes – Implication for treatment



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Over the past 30 years researchers described varied metabolic phenotypes in overweight and obesity with diverse disease outcomes. Well-conducted randomized large cohort clinical trials in individuals with overweight and prediabetes or type 2 diabetes suggest mixed response to caloric restriction or metformin, the

first-line medication in type 2 diabetes [1]. In the Diabetes Prevention Program, 21% of individuals with prediabetes treated with metformin progressed to diabetes at 3 years [2]. Similarly, 55% of individuals with prediabetes in the Tubingen Lifestyle Intervention Program did not revert to normal glucose tolerance in response to energy restriction and moderate exercise intervention [3]. Using gold-standard phenotyping tools of hepatic and peripheral glucose regulation, we find different levels of insulin resistance in liver and muscle in individuals with obesity. Using latest-generation plasma metabolomic and lipidomic analyses combined with machine learning, random forest-based feature selection and classification we identified three plasma lipids that can classify subcohorts of liver versus muscle insulin resistance in obesity with remarkable accuracy. We propose that therapy guided by plasma biomarkers will be more therapeutically effective, have less side-effects, and be more cost-effective.

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Disruption of AMPK-glycogen binding in vivo reveals novel roles in whole-body and tissue metabolism



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The AMP-activated protein kinase (AMPK) and glycogen are essential for maintaining metabolic homeostasis. The energy-sensing AMPK heterotrimer contains a regulatory β subunit with a carbohydrate-binding module (CBM) known to bind glycogen. However, the physiological roles of AMPK-glycogen binding in metabolism *in vivo* are unknown. To determine the effects of disrupting AMPK-glycogen binding, two whole-body knock-in (KI) mouse lines were generated targeting tryptophan residues known to mediate glycogen binding in either the AMPK $\beta 1$ (W100A KI) or $\beta 2$ (W98A KI) subunit, predominantly expressed in liver and skeletal muscle, respectively. Whole-body, serum and tissue analyses were performed in male KI and wild type (WT) litter mate mice maintained on an *ad libitum* chow diet. Intraperitoneal glucose tolerance testing revealed normal glucose tolerance in W100A mice but impaired glucose handling in W98A mice (56% increase in AUC; $P < 0.05$) compared to WT, with no differences observed in fasting serum insulin. Body composition (determined from EchoMRI) showed normal whole-body fat and lean mass in W100A mice. Strikingly, W98A mice displayed a 42% increase in fat mass ($P < 0.05$) and 5% decrease in lean mass ($P < 0.05$) relative to WT. Metabolic caging demonstrated no changes in cumulative food intake, O_2