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Brain insulin signaling in metabolic homeostasis and disease

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Insulin signalling in the central nervous system regulates energy homeostasis by controlling metabolism in several organs and by coordinating organ crosstalk. Studies performed in rodents, non-human primates and humans over more than five decades using intracerebroventricular, direct hypothalamic or intranasal application of insulin provide evidence that brain insulin action might reduce food intake and, more importantly, regulates energy homeostasis by orchestrating nutrient partitioning. In my presentation I will discusses the metabolic pathways that are under the control of brain insulin action and explains how brain insulin resistance contributes to metabolic disease in obesity, the metabolic syndrome and type 2 diabetes mellitus.

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The adult weight management program for the prevention of Type 2 Diabetes

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Diabetes is a non-communicable disease when the body doesn't make enough insulin or cells stop responding to insulin causing too much glucose to remain in your bloodstream over time that can cause serious health implications such as heart disease, vision loss and kidney diseases. More than 4.9 million people in the UK have diabetes and the numbers are rising. The people mostly at risk for type 2 diabetes are those who are 45 years or older, overweight, people who have a family history of diabetes, as well as those who are physically inactive and much more to consider such as race. Gillian is helping to tackle this major issue, by promoting social prescribing in the UK to reduce health inequalities. The Adult weight management is a 12-week behavioural change programme aimed for adults located in the most deprived demographic areas, who are overweight before they are diagnosed with type 2 diabetes. The focus is providing information and practical tasks to implement, to support physical and mental health reducing the participants' risk of developing diabetes. This is an evolutionary process, increasing the participants awareness of healthy eating and physical activity which creating self-sustainable habits for life, reducing the dependency on medications. Topics to cover are understanding carbohydrates, meal planning, preparing, portion sizing, recipe adaptions as well as the psychological links to food and emotions.

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Pu-erh tea and theabrownin ameliorate metabolic syndrome in mice via potential microbiota-gut-liver-brain interactions

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Metabolic Syndrome (MS) is a common metabolic disorder characterized by obesity, insulin resistance, cardiovascular disease and gut microbiota dysbiosis. Pu-erh tea and its ingredient theabrownin have known functions on the reduction of body weight gain and fat accumulation. However, few studies systematically analyze the different contributions and mechanisms of their anti-metabolic syndrome functions through multi-omics combination analysis. We used meta-genomics, transcriptomics and metabolomics technology to investigate the anti-metabolic syndrome mechanism of Pu-erh tea and theabrownin in MS mice. Our results suggested that Pu-erh tea and theabrownin interventions could improve the physiological functions of liver, jejunum and adipose tissues in MS mice. Hepatic transcriptome revealed that both interventions could regulate the circadian rhythm pathway. Glycerophospholipid and linoleic acid metabolism were also modulated by both interventions through serum and brain metabolome analysis. Faecal metagenome demonstrated that both interventions could increase the relative abundance of *Clostridiales bacterium 42_27*, *Blautia coccoides* and *Firmicutes bacterium* ASF500, but decrease the relative abundance of *Brevundimonas vesicularis*. Otherwise, compared with Pu-erh tea, theabrownin markedly upregulated the levels of hepatic antioxidants (i.e., SOD, GSH), prominently down-regulated hepatic inflammatory factors (i.e., IL-1, IL-6, TNF-α) and malondialdehyde oxidant, but modestly reduced obesity associated short-chain fatty acids in faeces in MS mice. Taken together, our data provided insights into the homogeneous and heterogeneous natural biological functions of theabrownin and Pu-erh tea in the treatment of metabolic syndrome.

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Dynamics of the functional state of the kidneys in patients with Diabetes mellitus in the process of PCI with a high risk of CI-AKI in the background of the use of preventive measures

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Aim: Analysis of the pathogenetic mechanisms of the development of contrast nephropathy in patients with coronary artery disease on the background of type 2 diabetes mellitus and the effectiveness of the proposed preventive measures.

Materials and methods: The first stage, a retrospective one, included a comparative analysis of two groups of diabetic patients who underwent an endovascular radiopaque procedure (EVRCP): 29 patients who developed CI-AKI (CI-AKI+ group) and 27 patients in whom the post-procedure period was uneventful. CI-AKI was defined as an increase in venous creatinine concentration of more than 25% by the end of 48 hours after EVRD.

Results of the study: we developed a risk scale for the development of CI-AKI in patients with DM. In the process of compiling the scale, the value of RR for the development of CI-AKI in the presence of the corresponding factor, rounded to the nearest whole number, was used as risk coefficients. According to the developed scale, in the presence of a risk factor, the patient is assigned a certain score. The score determines the risk of CI-AKI.

Conclusion: A risk score of 28 points or more demonstrates a predictive sensitivity in terms of the development of CI-AKI of 96.55% ($p<0.001$).

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The G protein-coupled receptor 30 (GPR30): A novel candidate for Diabetic cardiomyopathy in postmenopausal women

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Diabetic Cardiomyopathy is characterized by significant changes in cardiac metabolism and are increased in postmenopausal women, which emphasize the role of 17β-Estradiol (E2). Despite this, there are few safe pharmacological treatments for these disorders. The G Protein-coupled Receptor 30 (GPR30), which mediates the non-genomic effects of E2, has beneficial cardiac effects in both Type 2 Diabetes (T2D) and menopause. Based on pharmacological approaches, a growing body of experimental evidence highlights the role of GPR30, in particular, the adjustment of cardiometabolic function, but its exact mechanism is not fully understood. Generally, our results showed that T2D leads to cardiovascular dysfunction, possibly due to a change in the cardiac CD36, Peroxisome Proliferator-Activated Receptor α (PPARα), Hexokinase 2 enzyme (HK2), Glucose Transport 4 (GLUT4) and inflammatory and anti-inflammatory cytokines. Furthermore, induction of the menopausal model can worsen the effects of diabetes. However, our results showed that the cardiovascular protective mechanism of the GPR30 in addition to correcting lipid and glycaemic profiles, reducing insulin resistance and increase in GPR30 level protein also reduces inflammatory cytokines and increases anti-inflammatory cytokines. Although all inflammatory cytokines are not affected by stimulation or inhibition of GPR30, the ratio of inflammatory to anti-inflammatory cytokines decreases. In relation to changes in cardiac metabolism, our results showed that the stimulation of GPR30 by increasing the expression of PPARα and HK2 leads to a decrease in cardiac content of glycogen, glucose, free fatty acids and lipids. We conclude that G-1 as a GPR30 agonist is a prototype candidate drug for potential translation into clinical applications. It is suggested that in future studies, firstly the role of other estrogen receptors in the cardiovascular protective action of this sex steroid in postmenopausal diabetic animals should be investigated and secondly, the intracellular signalling pathway of the membrane receptor in diabetes should also be investigated.

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