of genes related to inflammation and plasticity was determined via reverse transcription polymerase chain reaction (RT-PCR) and the fecal microbiota was quantified via high-throughput sequencing of the 16S ribosomal RNA. Weight gain and energy intake were comparable across the diets. Rats consuming the SFA and Sugar diets were impaired on hippocampal-dependent place recognition memory compared to Controls and PUFA rats. All rats performed comparably on the perirhinal-dependent object recognition task. Hippocampal and hypothalamic inflammatory and neuroplasticity genes were not substantially affected, but each of the diets significantly altered the microbial composition in distinct ways. Specifically, the relative abundance of 89 taxa differed between groups with the majority of changes accounted for by the Clostridiales order and within that, *Lachnospiraceae* and *Ruminococcaceae*. These taxa showed a range of macronutrient specific correlations with place memory. In addition, Distance based Linear Models found relationships between memory, a cluster of hippocampal inflammation-related genes and gut microbiota composition. In conclusion, our study shows that even in the short-term the macronutrient profile of the diet is crucial for diet-induced memory deficits and suggests a possible link between diet, gut microbiota and hippocampal inflammatory genes. Longer term studies are warranted.

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AMPK-ACC signalling is required for increasing appetite under conditions of metabolic stress

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Activation of AMP-activated protein kinase (AMPK) during increased energy demand promotes food intake and reduces brown fat thermogenesis to shift the organism to neutral energy balance. The underlying molecular interactions are not entirely understood.

The acute effects of AMPK on lipid metabolism are mediated by phosphorylation of acetyl-CoA carboxylase (ACC) 1 at Ser79 and ACC2 at Ser212, thereby inhibiting fatty acid synthesis and promoting fatty acid oxidation. To investigate the physiological impact of this regulation on whole body energy balance, we generated mice with Ser79Ala/Ser212Ala knock-in mutations (ACC double knock-in, ACC DKI). ACC DKI mice have increased ACC1/2 activity in peripheral tissues and a propensity for increased lipid synthesis. Despite deregulated lipid metabolism, ACC DKI mice do not gain more weight when compared to wild type control mice and, in contrast, show a tendency for reduced body weight from 15 weeks of age.

Food intake measurements showed that ACC DKI mice have reduced appetite in response to metabolic stress, such as overnight fasting or cold exposure. Furthermore, while ACC DKI mice are able to maintain normal body temperature under cold stress, they compensate for reduced energy intake by utilising lipids as preferred energy source. Cold exposure and overnight fasting are accompanied by increased plasma levels of the orexigenic hormone ghrelin in ACC DKI mice. Importantly, we demonstrate that feeding in response to ghrelin is attenuated and ghrelin-induced expression of the orexigenic neuropeptides NPY and AgRP is inhibited, indicating that the anorexic phenotype of ACC DKI mice may be due to ghrelin insensitivity.

These results show that AMPK regulation of ACC is an important physiological mechanism in the control of body weight regulation, whereby the lipid accumulating effects in the periphery are outweighed by anorexic effects in the hypothalamus.

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Invited talk: Executive dysfunction in obese individuals

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Research has indicated that individuals with obesity have neurocognitive deficits, especially in executive function, which may in turn impact on weight loss and maintenance. In this talk I will review the evidence of this relationship, highlighting some of the mechanisms, and limitations of the literature. I will then present data on our latest randomised controlled trial which examined efficacy of a manualised cognitive remediation therapy for obesity (CRT-O) in terms of improving executive function, reducing binge eating behaviour and helping with weight loss. 80 adults with obesity (body