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Supplementation with high esterified pectins decreases energy efficiency and adiposity in an obesity-prone rat model, activating AMPK and inhibiting ACC enzymes in liver by phosphorylation

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The composition of gut microbiota, which can be modulated by prebiotics, has emerged as an important factor related with the control of energy metabolism.

We aimed to study the effects of supplementation with prebiotics, high esterified pectins (HEP), in the obesity-prone rat model CR (offspring of moderate caloric-restricted dams).

After weaning, male offspring of 20% caloric-restricted dams (pregnancy days 1–12), were fed with a standard diet only (CR) or supplemented with 10% HEP (CR-P) until 4-month age, then half of each group was also supplemented with 30% sucrose (CR-S/CR-P-S). Body weight and adiposity (by NMR) were followed and, at 5-month age, the balance between energy intake and its loss by faeces was determined (to determine actual calories absorbed). The ratio of phosphorylated/non phosphorylated AMP activated protein kinase (AMPK) and Acyl-CoA Carboxylase (ACC) was determined by Western blot in liver at 6-month age, and mRNA levels were analysed by RT-qPCR.

HEP-supplemented rats showed significantly lower body weight and adiposity than their controls, even under a high sucrose diet. Energy efficiency (estimated as the increase of weight gain per calories absorbed) was significantly lower in the HEP animals. mRNA levels of ACC1 were increased under sucrose diet both in pectin and non-pectin groups but the pectin supplemented animals were the only able to increase AMPK mRNA expression under the sucrose stimulus. The ratio of phosphorylated/total protein was increased both for AMPK and ACC under sucrose supplementation only in the pectin group, indicating an activation of AMPK and an inhibition of ACC.

In conclusion, HEP can help to decrease adiposity in the obesity-prone model of CR rats by decreasing energy efficiency, which can depend, at least in part, on higher capacity to activate oxidative metabolism and to inhibit fatty acid synthesis under an obesogenic stimulus by activating AMPK and inhibiting ACC in liver.