Título: Cafeteria diet alters the expression of key genes of the brain reward system over time".
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We aim to determine the effects of Cafeteria diet (CAF), rich in palatable and energy dense foods, on the expression of key genes of the brain reward system (RW) in the short and long term. Female Wistar rats were fed chow or CAF for 4 or 11 weeks. Animals were sacrificed and 2 regions of the Accumbens Nucleus (NA ? Core, NAC; and shell, NAS), Ventral Pallidum (VP) and Ventral Tegmental Area (VTA) were dissected. Serum leptin was assessed by RIA. mRNA expression of genes of the dopaminergic and GABAergic pathway, and the leptin receptor (ObRb) was evaluated by qPCR in the nuclei. Data was statistically analyzed by two-way ANOVA followed by Tukey post-test. Four weeks of CAF increased energy intake and adiposity, not affecting circulating leptin or body weight. In VTA, 4 weeks of CAF increased the expression of the dopamine active transporter (DAT) and decreased both isoforms of the enzyme involved in the synthesis of GABA (glutamate decarboxylase, GAD 1 and 2), without altering tyrosine hydroxylase (TH) expression. CAF decreased dopamine receptor (DR) 2 expression in NAS and increased DR1 levels in NAC. Also, CAF increased GAD2 levels in VP. After 11 weeks of CAF, animals sustained the hyperenergetic intake and further increased adiposity, leading to hyperleptinemia and higher body weight, only concomitant to an increased expression of ObRb in VTA. Our results indicate that the higher energy intake of CAF animals in the short-term would respond to hedonic mechanisms, given by molecular deregulations in the RW. The palatability of the diet could lead to a hypodopaminergic state, as DAT expression increase in VTA and DR2 decrease in NAS. Besides, the increment in GAD2 expression in VP indicates an inhibitory GABAergic input to dopaminergic and GABAergic VTA neurons that may, inhibit dopamine and GABA release, in line with the low expression levels of GAD1 and GAD2. Conversely, in the long-term the hypercaloric intake could respond to an altered homeostatic control.