Título: Junk-food' differentially affects the brain reward system over time.
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We analysed the short and long-term effects of the intake of a highly palatable junk-food cafeteria diet (CAF) on the expression of key genes of the brain reward system. Female Wistar rats were fed with chow or CAF from weaning for 30 (CAF30) or 77 (CAF77) days. The Ventral Tegmental Area (VTA), two regions of the Accumbens Nucleus (Core, NAcC; and shell, NAcSh), and the Ventral Pallidum (VP) were isolated, and the expression of several genes of the dopaminergic and GABAergic pathway, and the leptin receptor (ObRb) was evaluated by qPCR. Circulating leptin was assessed by radioimmunoanalysis. Data was statistically analysed by two-way ANOVA followed by Tukey post-test. CAF30 increased energy intake and adiposity, without affecting circulating leptin or body weight. In VTA, CAF30 enhanced dopamine active transporter (DAT) expression and decreased both isoforms of glutamate decarboxylase (GAD1 and GAD2), without altering tyrosine hydroxylase (TH) levels. In NAcSh, CAF30 dopamine receptor (DR) 2 mRNA decreased. In NAcC of CAF30, increased levels of DR1 were found. Besides, CAF30 increased GAD2 levels in VP. CAF77 animals further increased energy intake and adiposity, leading to higher body weight, hyperleptinemia, and increased expression of ObRb in VTA, with no changes on the expression of key genes of the reward system. Our results indicate that, in the short-term, CAF deregulates the dopamine pathway, reflecting a reward hyposensitivity state, which promotes the excessive intake of palatable foods to compensate this status. Conversely, this is reverted in the long-term, when the hypercaloric intake could respond to an altered homeostatic control.