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Changes in brain activity after a diet induced obesity

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Compared to lean subjects, obese men have less activation in the dorsolateral prefrontal cortex, a brain area involved in the inhibition of inappropriate behavior, satiety and meal termination. Whether this deficit precedes weight gain or is an acquired feature of obesity remains unknown. An adult animal model of obesity may provide insight to this question since brain imaging can be performed in lean versus obese conditions in a controlled study. Seven diet-induced obese adult minipigs were compared to nine lean adult minipigs housed in the same conditions. Brain activation after an overnight fasting was mapped in lean and obese subjects by single photon emission computed tomography (SPECT). Cerebral blood flow (CBF), a marker of brain activity, was measured in isoflurane-anesthetized animals after the IV injection of ^{99m}Tc -HMPAO (750 MBq). Statistical analysis was performed using SPM software and CBF differences were determined using co-registered T1 MRI and histological atlases. Deactivations were observed in the vicinity of several areas of the prefrontal cortex for obese versus lean subjects. They were also observed in the frontal cortex, pons, ventral tegmental area, anterior hypothalamic area and nucleus accumbens. On the contrary, activations were found in several areas of the occipital and the parietal cortices, as well as in the thalamus. Moreover, the activity of several regions of the prefrontal cortex was negatively associated with the body weight. We suggested that the reduced activation of prefrontal cortex observed in obese men is probably an acquired feature of obesity since it is also found in minipigs with a diet-induced obesity. This abnormality was related to the nutritional context and associated with the deactivation of some regions of the reward circuit, but its severity was dependent on the body weight.