CORREPONDENCE



Human hypocretin-deficient narcolepsy aberrant food choice due to impaired taste?

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ABSTRACT

Authors demonstrate that patients with narcolepsy type 1 (N1) have more tendency of eat salty snacks after satiety than health volunteers. A few mechanisms to explain the weight gain have been discussed in narcolepsy. The hypocretin-1 deficiency can influence the olfactory system. The olfactory system should be modulated through hypocretin-1 via connections from the hypothalamic to other brain regions. Likewise, hypocretin-1 can be synthesized locally in our olfactory mucosa with possible private role modulating the olfactory. In experimental studies, different kinds of smell influence the preference for type of diet. Olfactory and taste sensations help control of appetite and regulate the quantity and quality of foods that will be chosen. N1 patients have lower levels of hypocretin-1 and consequent inferior olfactory threshold, less olfactory discrimination, and these findings improved after nasal hypocretin-1 administration. It is possible that the hyposmia influenced the quality and quantity of food by narcoleptic patients. We suggest that a complementary analysis of olfactory function should be done concomitant with food preferences to compare narcoleptic patients with and without hypocretin-1 deficiency.

Keywords: Narcolepsy; Hypocretin-1; Obesity; Olfaction.

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Recently, authors demonstrate that patients with narcolepsy type 1 have more tendency of eat salty snacks after satiety than health volunteers¹. These findings are very promisor to help us to understand the frequent overweight of narcolepsy patients. A few mechanisms to explain the weight gain have been discussed in narcolepsy such as impairment of oxidative metabolism, hypothalamus dysfunction, and eating disorders².

The hypocretin-1 deficiency in narcolepsy type 1 patients drives daytime sleepiness, cataplexy, sleep palsy, sleep fragmentation, hallucinations, and autonomic changes³⁻⁵. However, hypocretin-1 can also influence the olfactory system for two different pathways. The hypocretin-1 projections can be detected in olfactory nuclei, brain cortex, tonsils, and nasal mucosa⁶. The olfactory system should be modulated through hypocretin-1 via connections from the hypothalamus to other brain regions. Likewise, hypocretin-1 can be synthesized locally in our olfactory mucosa with possible private role modulating the olfactory system^{7,8}.

The olfaction is fundamental for regulating appetite. In experimental studies, different kinds of smell influence the preference for type of diet. Olfactory and taste sensations help control of appetite and regulate the quantity and quality of foods that will be chosen⁹.

Patients with narcolepsy type 1 have lower levels of hypocretin-1 and consequent inferior olfactory threshold, less olfactory discrimination, and these findings improved after nasal hypocretin-1 administration¹⁰. In recent and interesting paper published by van Holst et al.¹, all patients with gustatory and hyposmia complains were excluded from original study. Thus, it is possible that the hyposmia influenced the quality and quantity of food sought by narcoleptic patients in this study, since olfactory conditions are directly related to changes in tasting. Indeed, a previous study in subjects without narcolepsy demonstrated higher occurrence of olfactory dysfunctions in obese patients¹¹. We suggest that a complementary analysis of olfactory function should be done concomitant with food preferences to compare narcoleptic patients with and without hypocretin deficiency.

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