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Effects of Intermittent Fasting on Taste Modulation and Perception

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Intermittent fasting has remarkable documented positive effects on human health and aging. These effects are considered to be the result of a variety of fasting-induced physiological changes that include reduced production of oxygen free radicals, activation of processes to repair or remove damaged molecules, reduced inflammation, and optimized tissue metabolism. The effects of intermittent fasting on the perception of odor and taste have also been debated for years. Although sufficient literature is available on the effects of intermittent fasting on odor perception, similar data on gustatory perception is strikingly lacking.

Firstly, the sensitivity of the gustatory system is modulated by multiple factors such as age, gender, body mass index, comorbid conditions, smoking, stress, poor oral hygiene, excessive alcohol drinking, and nutritional deficiency. Females generally have higher gustatory sensitivity than males. This sensitivity further varies during different phases of the menstrual cycle with increased sensitivity at mid-cycle.

Secondly, the gustatory pathway is a complex pathway that involves peripheral nerves (lingual, glossopharyngeal, and vagus nerves) and their brainstem nuclei, thalamus, gustatory cortices (anterior insular and frontal opercular cortex), and the limbic system.

Thirdly, it is difficult to assess different taste variables (e.g. sweet, sour, bitter, and salty) accurately and repetitively.

Fourthly, a gustatory response is linked with a combination of other external food-related cues such as odor, temperature, and appearance of food.

Lastly, the residual stimuli from prior testing also affect the measurements of sensitivity, intensity, and quality of the gustatory response.

In 1959, Rose Marie Pangborn published one of the earliest studies on the changes in taste perception and concluded no significant difference in taste perception between intermittent fasting and satiety states. A similar result was reported by Pasquet et al. These results are, however, contradicted by other studies. Zverev et al in 2004, Hanci et al. in 2016, and Khobragade et al in 2018 reported an increase in taste sensitivity to sweet and salty tastants in the intermittent fasting state as compared to satiety state but the sensitivity to bitter tastant was either similar in both groups or increased in the satiety state. These results can be explained by the physiological feedback response of our body and biological importance of certain foods. The sweet and salty foods are preferred in the fasting state to replenish the depleted energy and electrolyte stores, respectively, while bitter tastes are related to the poisons and foods that are not suitable for consumption that should be avoided in both fasting and satiety states.

Several mechanisms have been proposed for fasting-induced taste modification. The caloric-deficient state or food motivation may alter the sensitivity of central structures involved in the taste perception as well as the areas involved in motivation and reinforcement. Functional brain imaging including positron emission tomography (PET) measurements of regional cerebral blood flow changes has been used to study the central responses to taste perception. The anterior temporal cortex, thalamus, hippocampal formation, cingulate cortex, and caudate nucleus are preferentially involved in the taste perception. Gautier et al further studied the central responses during intermittent fasting and satiety states. They reported that additional areas of the medial prefrontal cortex, supplementary motor area, supramarginal gyrus, inferior parietal lobule, midbrain, occipital cortex, and cerebellum are activated during the fasting state and may be partly responsible for central modulation of taste perception. A similar difference in gustatory sensitivity and dietary disinhibition has been noted in lean and obese individuals. Persons with high dietary inhibition tend to eat regardless of prior food intake and are more likely to gain weight. They also showed increased activity in insular and medial prefrontal regions, similar to the intermittent fasting state.
Fasting-induced modulation of the sensitivity of peripheral gustatory receptors is another suggested mechanism. Gastric mechanoreceptors and osmoreceptors may affect the gustatory receptors through modulating the vagus and the glossopharyngeal nerve responses. Alteration of the autonomic nervous system activity during intermittent fasting state may also contribute to the taste modulation. In lower mammals, the neural circuits for hunger have been extensively studied. Agouti-related peptide (AgRP)-expressing neurons are key components of this circuitry. The AgRP neurons are localized in the arcuate nucleus of the hypothalamus. These neurons activate during the fasting state and trigger acute food intake. The AgRP neurons will induce feeding behavior even if artificially activated during the satiety state.

In conclusion, taste modulation and increased sensitivity to sweet and salty food is a normal physiological gustatory feedback response during fasting to promote eating behavior. Unlike peripheral and central mechanisms, the psychological effects of intermittent fasting on the taste perception have not been widely addressed. Further large scale studies are needed to validate these findings and to limit the confounding agents. The underlying central mechanisms responsible for the taste modulation should also be further elucidated. Since similar central mechanisms may play a key part in dietary disinhibition seen in obese individuals and during fasting, we may get further insight into the central pathogenesis of obesity and halt the currently growing prevalence of obesity worldwide.
References:


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Sajid Hameed: data collection, data analysis, manuscript writing, manuscript review
Mohammad Wasay: concept, data analysis, manuscript writing, manuscript review