Taste, Neuronal Activation and Hormonal Release

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While it is well recognized that the flavor of food plays an important role in our choice and selection of the foods we eat, few people realize that taste also plays a role in regulating the hormones that help us metabolize the nutrients provided by that food. At every meal, the food we eat is broken down by digestive enzymes in the stomach. The nutrients from the food are absorbed in the intestine and then as levels of these nutrients rise in the blood, this stimulates the release of hormones from the pancreas and intestine to promote the storage of nutrients. A typical example occurs during the ingestion of a carbohydrate such as rice, which is hydrolyzed in the stomach and absorbed into the blood in the form of glucose. The increase in blood glucose stimulates the release of the essential hormone insulin from the pancreas. Insulin then promotes glucose uptake and storage into muscle, liver and adipose tissue. However, studies in animals and humans demonstrate that just the taste of food, prior to swallowing or even nutrient absorption, elicits the release of hormones, including insulin.

Insulin release which occurs prior to nutrient absorption is often termed cephalic (meaning brain) phase insulin release (CPIR). CPIR occurs prior to and at the onset of food ingestion in response to the sensory aspects of food. The rise in plasma insulin levels occurs independently of increases in blood glucose and typically peaks within 4 minutes after food enters the oral cavity and returns to baseline levels by 8-10 minutes. When receptors in the oral cavity are activated by flavor, this initiates a neural relay that sends a message to the brain and subsequently activates the vagus nerve, the primary nerve fiber which connects the brain to all organs involved in nutrient metabolism, including the stomach, intestine, pancreas and liver. Activation of the vagus nerve has profound effects on how nutrients are digested, absorbed and metabolized. The vagal nerve fibers which terminate on the pancreas stimulates hormones which are involved in the regulation of blood glucose levels.

To measure CPIR in humans, subjects are requested to perform a modified sham-feed in which food is tasted, chewed and then spit out so that none of the food is swallowed and digested. This allows the investigators to differentiate hormonal release that is elicited just by the taste of food from hormones released in response to elevated levels of nutrients in the blood (post-prandial stimulation). Experimental paradigms such as these have revealed some of the necessary conditions for CPIR as well as species differences. For example, while sweet tasting solutions consistently and reliably elicit CPIR in animals, our laboratory has shown that in humans, neither sucrose, aspartame nor saccharine solutions are effective stimuli. In contrast, mixed nutrient solid foods, particularly those containing fat are effective stimuli. Flavor and palatability may also influence the magnitude of CPIR. Sweet food is a more potent stimulus of CPIR release than salty foods and foods rated as unpalatable elicit smaller or no responses compared to palatable foods.

In addition to the studies described above, our laboratory at Monell has shown that in the absence of oral sensory stimulation of food, blood glucose levels are higher and less well controlled compared to when oral sensory stimulation is provided by the taste of food. Conversely, if CPIR is enhanced in obese human subjects, their blood glucose levels after glucose ingestion are lowered, improving their glucose tolerance. These and other studies demonstrate that an increase in insulin during the pre-absorptive period in response to the taste of food is essential for normal glucose tolerance. Thus, the taste of food plays a critical role in human health and metabolism.

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