Food intake, feelings, and gastrointestinal physiology

Harry R. Kissileff
St. Luke’s/Roosevelt Hospital, New York 10025; and Departments of Psychiatry and Medicine, Columbia University School of Medicine, New York, New York 10032

The classical methods for studying the inhibitory controls of food intake involve the manipulation of tissues hypothesized to signal sites in the brain which in turn remove or reduce excitatory influences on the muscles involved in eating (4). It is not yet possible to study all the links in this chain, from the stimuli in the periphery through to the central nervous system and back down to the muscles. Instead, changes in the brain are inferred from reports of subjects on sensitive scales, and stimuli are provided in the form of preloads designed to stimulate specific receptors that are hypothesized to participate in the inhibitory processes (3). For example, foods that vary in nutritive composition or suspected ability to fill the stomach have been given before test meals. The change in intake across conditions is taken as an index of the treatment’s effect on the inhibitory process. Signals have typically been thought to be gastric wall tension or some function of it and intestinal chemostimulation, which either releases hormones or directly signals the brain.

In the case of fats, there has been great interest in their inhibitory effects on food intake, and the mechanism has been studied by intraduodenal infusion of fatty acids of different chain lengths. Although it is known that long-chain fatty acids are more effective in reducing intake than short chain and that CCK release is a critical stimulus for this inhibition, because the effect was blocked by the CCK antagonist loxiglumide (6), it was not known until the study of Feltin et al. (2) in this issue of the American Journal of Physiology-Regulatory, Integrative and Comparative Physiology that the critical chain length break point for the response is between C-10 and C-12. Their study shows clearly and for the first time not only where the breakpoint is, but also why it’s there, and what gastrointestinal processes are involved. By infusing C-10 (decanoic) and C-12 (lauric) fatty acids at physiological rates, they showed that C-12 was effective and C-10 was ineffective in reducing food intake. The formation of chylomicrons appears to be a critical marker for whether a fat suppresses food intake and releases CCK, although it is not clear why this particular effect is linked to CCK release. Furthermore, the mechanism responsible, at least in part, for slowing of gastric emptying has also been identified as “a substantial increase in basal pyloric pressure and the number and amplitude of IPPWs and decrease in the number of antral and duodenal pressure waves as well as the number of pressure wave sequences” (2).

Thus they propose that the mechanism for decreased intake is likely to be closely related to reduction in gastric emptying. However, it is not clear how gastric emptying itself in fact relates to inhibition of food intake. In a study that actually correlated gastric emptying rate with change in amount eaten, it was found that the change in gastric emptying only correlated with amount consumed when CCK was infused, and the amount of change in gastric emptying would not account for the change in intake (7).

Another important finding in the Feltin et al. study (2) was the measurement of sensations that are correlated with changes in the gastrointestinal signals of patterns of pressures and waves from specific loci. The identification of the source of scalable sensations from the gut is an extremely important advance over simply inflating a bag and measuring associated pressures, volumes, and sensations. However, the sensations that were found suggest the need for refinement in the process of obtaining this type of information from human subjects. Although there were no differences between intake changes in subjects who did or did not report nausea, it is not clear whether those who did not report did not experience it. People’s use of words to describe visceral sensation is typically not reported in these types of studies, and it is not clear whether subjects are trained to recognize and report specific sensations induced by particular stimuli or whether they are simply given a scale with a label. For future work in this field it is important to determine how individuals use whatever words are chosen for scales and to determine how strong the sensations reported are in relation to other experiences the subjects have had. Bartoshuk et al. (1), for example, showed that individuals vary in their ability to detect bitter tastes and that when the appropriate scaling procedures are used, it is possible to distinguish super tasters from ordinary tasters. These procedures involve making ratings on a common scale with most imagined of any sensation at the top and include ratings on a variety of sensations both real and recalled. These types of scales should be applied to scaling of sensations related to both chemical and physical manipulation of the gastrointestinal tract. Finally, the ability to measure sensations such as fullness and nausea should be coupled with actual eating behavior (5) and not simply restricted to the periods before and after eating. In conclusion, this study (2) of food intake inhibition coupled with the sensation ratings and measures of gastrointestinal pressure waves and hormone levels is a tour de force that should set the standard for studies with multiple variables in this field.

Grants

The author was supported by New York Obesity Research Center National Institutes of Health Grant AM-26687.

References


