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The Gravity of Weight

"Sufficient Unto the Day:" The Complexities of Satiety

"Give us this day our day bread"--and a sense of fullness as well

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Many years ago, Rockefeller University researcher Jules Hirsch noted that over a lifetime, a person may consume about 70 million calories or 14 tons of food that the body must process, and considering all the variables, does so remarkably well. There are many complex physiological measures involved in how the body prepares itself for the ingestion, digestion, and metabolism of our food. These anticipatory responses

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are called cephalic-phase responses and they can be physical (e.g. motility of our gastrointestinal system), secretory (e.g. release of enzymes and hormones), or even metabolic (e.g. process of thermogenesis.) The process of eating, as Power and Schulkin note in their 2009 book *The Evolution of Obesity*, is ironically both required to maintain homeostasis and simultaneously a threat to homeostasis. Initiating a meal is “largely opportunistic,” according to Chambers et al in a 2013 issue of the journal *Current Biology* and involves cognitive assessments, such as food availability, time of day, palatability, and learning. What determines, though, how the body prepares itself to stop eating? Determining the end of a meal, in fact, involves literally a “cascade” of physical and biochemical processes, including the release of multiple hormones, that enable us to stop eating. The intestinal hormone CCK, released preferentially in response to a fatty meal, for example, delays the emptying from the stomach and reduces food intake and meal size and hence is one factor that leads to satiety. Other hormones that inhibit food intake are the intestinal GLP-1 (glucagon-like peptide 1), and the

pancreatic hormones Peptide YY (PYY) and amylin. Many of these hormones are mediated through the vagus nerve.

There are separate physiological processes involved in short-term *satiation* (i.e., fullness and decrease in hunger during a meal) that leads to termination of that particular eating episode and long-term *satiety* (i.e., involving food intake over the entire day or longer and food intake frequency from one meal to another.) There is also *sensory specific satiety* that occurs after eating a specific food, such that we lose our taste for or interest in it. Even Shakespeare knew that when he said, “A surfeit of the sweetest things /The deepest loathing to the stomach brings.” (*A Midsummer-Night’s Dream*, ii, 2, 137). The more variety of food choices to which we are exposed (e.g. a buffet), the more we tend to eat.

Ivan Pavlov famously experimented with sham feeding.

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The Russian scientist Ivan Pavlov, at the turn of last century, performed some of the early experiments with dogs with what he called “fictional feeding”--or “sham feeding,” in which ingested food did not reach the stomach but drained out of an artificially created fistula. These experiments with dogs and later ones by others with rats demonstrated that when food does not reach the stomach, animals eat more. In other words, food in the mouth is not enough to elicit satiation or satiety. Gastric distention from food in the stomach--i.e., that sense of fullness we get when we eat, seems to be required to stop eating. Chambers et al note that there is “no question that gastric volume is a rate-limiting factor in meal size.” Incidentally, the prophet Mohammed seemed to know that intuitively: it is reported that when he was hungry, he tied a stone to his belly to ward off the feelings of hunger. In recent years, we use horizontal or vertical lap band bariatric surgery to make the stomach capacity literally much smaller.

Sometimes, though, when the mechanical and biochemical safety brakes fail, people eat beyond that sense of fullness to the point of discomfort in what we call excessive eating (i.e. gluttony.) Gluttony, described by Pope Gregory in the Sixth

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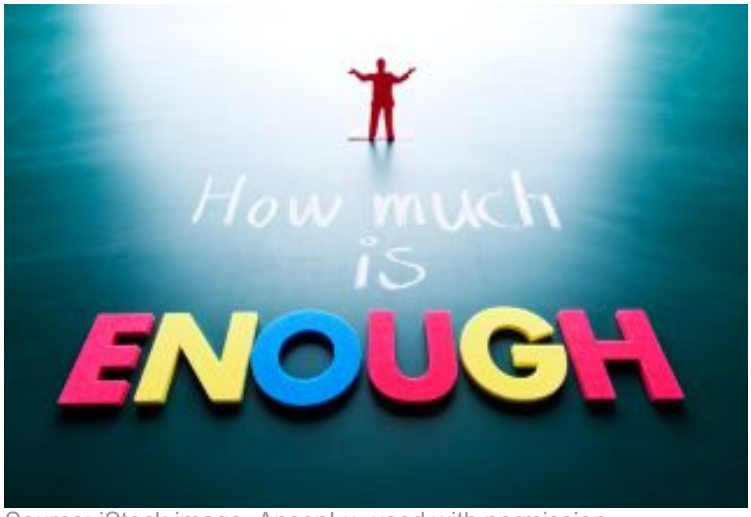
Century, was designated one of *The Seven Deadly Sins* in early Catholic teaching. Saint Thomas Aquinas, philosopher and theologian in the 13th century, elaborated on the sin of gluttony and described several ways that people commit this sin: eating excessively; eating too expensively or luxuriously; eating too eagerly; eating too daintily or too elaborately; and eating at an inappropriate time.

Jules Hirsch reported on experiments whereby lesions of the lateral hypothalamus produced decrease eating (hypophagia), profound weight loss, and even death by starvation, whereas lesions of the ventromedial hypothalamus produced hyperphagia, massive obesity, and even examples when hyperphagia led to stomach rupture in rats. The ventromedial hypothalamus then became known as the satiety center. In the psychological thriller *Se7en*, a serial killer uses The Seven Deadly Sins as a template for his murdering spree. The glutton dies by being forced to eat himself to death, ostensibly rupturing his stomach by his excessive consumption.

Williams, writing in the journal *Physiology and Behavior* (2014) notes that for years, homeostatic eating (i.e. for maintenance of energy balance and providing negative feedback on eating) was seen as distinct and as operating in opposition to hedonic eating (i.e. eating for pleasure and the system that can override the homeostatic system and lead to overeating.) For Williams, these two systems overlap and it is “inaccurate” to continue to think of them as two separate systems. Williams believes that the hormone orexin A, found predominantly in the lateral hypothalamus, is involved in increasing our motivation to obtain and continue to eat highly rewarding food at the expense of our sense of satiation.

Satiety, of course, is particularly important to dieters. Rebello et al, writing in a 2013 issue of *Advances in Food and Nutrition Research,* emphasize that success of a weight-loss dietary regimen is closely related to compliance, which, in turn, is “largely dependent on hunger, appetite, and satiety.” For example, certain, but not all, dietary protein is the most satiating of the food groups for some people. Not only does it take more energy to metabolize protein, but there is also speculation that satiety due to the ingestion of protein is related to increases in two hormones that decrease eating, GLP-1 and PYY, and to a decrease in ghrelin, the hormone that leads to an increase in eating.

Bottom line: At one time, we thought that satiety was related to specific regions of the hypothalamus. We now know that many complex endocrine, cognitive, and neural systems are also involved, with multiple and "redundant" protective mechanisms in place. There are many biological factors involved in the control of how much we eat that have yet to be elucidated. With all of these systems, we may, unfortunately, have much less conscious control over eating than we think we have.



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About the Author

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In Print: *The Gravity of Weight: A Clinical Guide to Weight Loss and Maintenance*

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