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## Mind & Brain / Obesity

### A New Suspect in the Obesity Epidemic: Our Brains

The urge to eat too much is wired into our heads, in several complicated and overlapping ways. Tackling obesity may require bypassing the stomach and short-circuiting our brains.

by Dan Hurley

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10:19 p.m. on a Monday evening in October, I sat in a booth at [Chevys](#) Fresh Mex in Clifton, New Jersey, reviewing the latest research into the neurobiology of hunger and obesity. While I read I ate a shrimp and crab enchilada, consuming two-thirds of it, maybe less. With all this information in front of me, I thought, I had an edge over my brain's wily efforts to thwart my months-long campaign to get under 190 pounds. But even as I was taking in a study about the powerful lure of guacamole and other salty, fatty foods, I experienced something extraordinary. That bowl of chips and salsa at the edge of the table? It was whispering to me: *Just one more. You know you want us. Aren't we delicious?* In 10 minutes, all that was left of the chips, and my willpower, were crumbs.



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I am not alone. An overabundance of chips, [Baconator Double](#) burgers, and Venti [White Chocolate Mochas](#) have aided a widespread epidemic of obesity in this country. Our waists are laying waste to our health and to our health-care economy: According to [a study published by the Centers for Disease Control and Prevention in 2010](#), nine states had an obesity rate of at least 30 percent—compared with zero states some 10 years earlier—and the cost of treatment for obesity-related conditions had [reached nearly 10 percent of total U.S. medical expenditure](#). So-called normal weight is no longer normal, with two-thirds of adults and one third of children and adolescents [now classified as overweight or obese](#). Dubbed the [“Age of Obesity and Inactivity”](#) by the *Journal of the American Medical Association*, this runaway weight gain threatens to decrease average U.S. life span, reversing gains made over the past century by lowering risk factors from smoking, hypertension, and cholesterol. We all know what we should do—eat less, exercise more—but to no avail. An estimated 25 percent of American men and 43 percent of women attempt to lose weight each year; of those who succeed in their diets, between 5 and 20 percent (and it is closer to 5 percent) manage to keep it off for the long haul.

The urgent question is, why do our bodies seem to be fighting against our own good health? According to a growing number of neurobiologists, the fault lies not in our stomachs but in our heads. No matter how convincing our conscious plans and resolutions, they pale beside the brain's power to goad us into noshing and hanging on to as much fat as we can. With that in mind, some scientists were hopeful that careful studies of the brain might uncover an all-powerful hormone that regulates food consumption or a single spot where the cortical equivalent of a neon sign blinks “Eat Heavy,” all the better to shut it off.

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After extensive research, the idea of a single, simple cure has been replaced by a much more nuanced view. The latest studies show that a multitude of systems in the brain act in concert to encourage eating. Targeting a single neuronal system is probably doomed to the same ill fate as the failed diets themselves. Because the brain has so many backup systems all geared toward the same thing—maximizing the body's intake of calories—no single silver bullet will ever work.

"I call it the 'hungry brain syndrome,'" says [Hans-Rudolf Berthoud](#), an expert in the neurobiology of nutrition at the Pennington Biomedical Research Center in Baton Rouge, Louisiana. The brain's prime directive to eat and defend against the loss of fat emerged early in evolution, because just about every creature that ever trotted, crawled, swam, or floated was beset by the uncertainty of that next meal. "The system has evolved to defend against the slightest threat of weight loss, so you have to attack it from different directions at once."

With the obesity epidemic raging, the race for countermeasures has kicked into high gear. Neuroscientists are still seeking hormones that inhibit hunger, but they have other tactics as well. One fruitful new avenue comes from the revelation that hunger, blood sugar, and weight gained per calorie consumed all ratchet up when our sleep is disrupted and our [circadian rhythms](#)—the 24-hour cycle responding to light and dark—thrown into disarray. All this is compounded by stress, which decreases [metabolism](#) while increasing the yen for high-calorie food. We might feel in sync with our high-tech world, but the obesity epidemic is a somber sign that our biology and lifestyles have diverged.

### Seeking Silver Bullets, Shooting Blanks

The path forward seemed so simple back in 1995, when three papers in *Science* suggested a panacea for the overweight: A hormone that made animals shed pounds, rapidly losing body fat until they were slim. Based on the research, it seemed that doctors might soon be able to treat obesity the way they treat diabetes, with a simple metabolic drug.

Fat cells release that "diet" hormone—today named [leptin](#), from the Greek *leptos*, meaning thin—to begin a journey across the blood-brain barrier to the [hypothalamus](#), the pea-size structure above the [pituitary gland](#). The hypothalamus serves as a kind of thermostat, setting not only body temperature but playing a key role in hunger, thirst, fatigue, and sleep cycles. Leptin signals the hypothalamus to reduce the sense of hunger so that we stop eating.

In early lab experiments, obese mice given extra leptin by injection seemed sated. They ate less, their body temperature increased, and their weight plummeted. Even normal-weight mice became skinnier when given injections of the hormone.

Once the pharmaceutical industry created a synthetic version of human leptin, clinical trials were begun. But when injected into hundreds of obese human volunteers, leptin's effect was clinically insignificant. It soon became clear why. In humans, as in mice, fat cells of the obese already produced plenty of leptin—more in fact than those of their thin counterparts, since the level of leptin was directly proportional to the amount of fat. The early studies had worked largely because the test mice were, by experimental design, leptin-deficient. Subsequent experiments showed that in normal mice—as in humans—increases in leptin made little difference to the brain, which looked to *low* leptin levels as a signal to eat more, essentially disregarding the kind of high levels that had caused deficient mice to eat less. This made leptin a good drug for maintaining weight loss but not a great candidate for getting the pounds off up front.

Despite that disappointment, the discovery of leptin unleashed a scientific gold rush to find other molecules that could talk the brain into turning hunger off. By 1999 researchers from Japan's National Cardiovascular Center Research Institute in Osaka [had announced the discovery of ghrelin](#), a kind of antileptin that is released primarily by the gut rather than by fat cells. [Ghrelin](#) signals hunger rather than satiety to the hypothalamus. Then, in 2002, a team from the University of Washington found that [ghrelin levels rise before a meal and fall immediately after](#). Ghrelin (from the Indo-European root for the word "grow") increased hunger while jamming on the metabolic brakes to promote the body's storage of fat.

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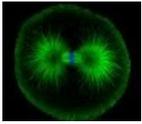
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