
The Modern Obesity Epidemic, Ancestral Hunter-Gatherers, and the Sensory/Reward Control of Food Intake

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Obesity has become a true pandemic. In the United States, over two thirds of adults are obese or overweight. The prevalence of obesity has doubled since 1980. The increase in the prevalence of obese and overweight individuals has happened too rapidly for it to be due to an alteration in the genome. The gastrointestinal, sensory (taste and olfaction), and brain feeding mechanisms that developed during the past 2 million years were highly adaptive for ancestral hunter-gatherers living in an environment with limited high-density foods and periods of food deprivation. Today, however, humans in industrialized countries live in what has been called an "obesogenic environment." The nonhomeostatic brain reward circuitry that was acquired during evolution to seek out and eat as many nutritionally high-dense foods as possible is able to overrule the physiological inhibitory mechanisms that were designed to limit meal size and weight gain.

Keywords: obesity, hunter-gatherers, food intake, taste, brain reward circuitry

Obesity is a widespread problem. Among industrialized countries, it is a true pandemic (Swinburn et al., 2011). If obesity is defined as having a body mass index (BMI) greater than 30 kg/m², 502 million people worldwide are obese, with another 1 billion being overweight (BMI > 25 kg/m²). This obesity figure includes 170 million children, 42 million under the age of five years (de Onis, Blössner, & Borghi, 2010). BMI is correlated very highly with percentage body fat (Flegal et al., 2009).

In the United States, 35.7% of adults (35.5% of men and 35.8% of women) are obese and another 33.1% are overweight according to a 2010 nationally representative sample (Flegal, Carroll, Kit, & Ogden, 2012). Over 6% are in the category of "extreme obesity" (BMI > 40 kg/m²). Close to 17% of children and adolescents (12.5 million) have a high BMI (Ogden, Carroll, Kit, & Flegal, 2012).

Widespread obesity is a recent phenomenon. In the United States, the prevalence of obesity among adults was moderate in 1960, about 1 in 5 individuals was "overweight" then (U.S. Public Health Service, n.d.), and the prevalence of obesity remained stable until about 1980. However, by the early 1990s, the prevalence of obesity had increased by 8 percentage points, and it had increased even

further by 2000 (Flegal, Carroll, Ogden, & Johnson, 2002). Overall, the prevalence of obesity in the United States has leveled off somewhat since 2000 (Flegal et al., 2012), but a recent study estimated that by 2030, 42% of Americans will be obese and 11% will be extremely obese (Finkelstein et al., 2012).

The increase in the prevalence of obesity is even more dramatic among U.S. children (defined as having a BMI > 95th percentile for age), with rates tripling since 1980 (Ogden et al., 2012). Similar trends have been observed in many other countries (see Swinburn et al., 2011). It is estimated that worldwide, the prevalence of childhood overweight and obesity will further increase from 6.7% to 9.1% by 2020 (de Onis et al., 2010).

Public health measures have been effective in combating preventable deaths caused by such things as infectious diseases and the use of tobacco, but no country has yet found a means to reduce the prevalence of obesity (Swinburn et al., 2011). The difficulty of treating obesity was recognized at least as far back as 1958:

Most obese patients will not remain in treatment. Of those who do remain in treatment, most will not lose significant poundage, and of those who do lose weight, most would regain it promptly." (Cornell Conferences on Therapy, 1958, p. 87)

Little has changed in the last 50 years. Behavioral interventions, even when intensive, generally result in modest weight loss, and only a few individuals maintain the loss long term (e.g., Christiansen, Bruun, Madsen, & Richelsen, 2007; LeBlanc, O'Connor, Whitlock, Patnode, & Kapka, 2011). In order to effectively deal with obesity, we must understand its etiology.

Inhibitory (Satiety) Feeding Mechanisms

At first glance, it would appear that the brain is replete with mechanisms to inhibit food intake after initiation of a meal and/or weight gain. In the last several decades, there have been over 10,000 published papers on brain nuclei, path-

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ways, hormones, and neurotransmitters involved in the satiation of feeding. Damage to specific brain nuclei results in overeating and obesity, suggesting that they play a key role in limiting food intake and body weight. These include the hypothalamic paraventricular nucleus (Leibowitz, Hammer, & Chang, 1981), the ventromedial hypothalamus (King, 2006b), and the posterodorsal amygdala (King, 2006a). Transection of the ventral noradrenergic bundle, the stria terminalis, or the fibers projecting from the arcuate nucleus to the paraventricular nucleus has similar effects (Ahlskog & Hoebel, 1973; Bell, Bhatnagar, Akana, Choi, & Dallman, 2000; Rollins, Stines, & King, 2006).

There are both short- and long-term mechanisms to limit food intake. In addition to activating mechanoreceptors in the stomach and small intestine that limit meal size by responding to the weight or volume of food (de Graaf, Blom, Smeets, Stafleu, & Hendriks, 2004), ingested food (particularly fats) causes release of cholecystokinin, a peptide hormone that is detected by duodenal receptors and inhibits food intake by restricting gastric emptying and also by signaling the brain via the vagus nerve (Smith & Gibbs, 1992). Other gut peptides, such as glucagon-like peptide 1, are purported to have similar effects (de Graaf et al., 2004; Woods, Schwartz, Baskin, & Seeley, 2000). Long-term mechanisms respond to changes in body weight. Insulin is released in proportion to body fat content and reduces feeding behavior by acting on the arcuate nucleus and midbrain dopaminergic cells (Könner, Klöckener, & Brüning, 2009; Woods et al., 2000). Leptin is a hormone secreted by adipocytes in direct proportion to body fat levels, and it inhibits food intake by acting on the arcuate nucleus (and inhibiting neuropeptide Y transmission to the paraventricular nucleus; Könner et al., 2009).

Other brain appetite-suppressing chemicals (anorexigens) include pro-opiomelanocortin (POMC) and α -mela-

nocyte-stimulating hormone (α -MSH), both of which play critical roles within the ventromedial hypothalamus, and cocaine- and amphetamine-regulated transcript, or CART (Cameron & Doucet, 2007; King, 2006b; Woods et al., 2000).

When studied in laboratory animals maintained on standard lab chow, these inhibitory mechanisms appear to be highly effective in limiting food intake. In fact, for some species, opposing excitatory–inhibitory mechanisms dominate food intake. For example, if the physiological inhibitory mechanisms in a blow fly are eliminated, it will eat until its stomach bursts (Dethier, 1967). Research findings such as these supported the concepts of homeostasis and set points (see Berridge, 2004). According to these theories, body weight is regulated around a stable set point and deviations from this body weight (such as excessive weight gain) result in corrective measures by the many physiological mechanisms previously listed.

However, set point theory cannot explain the recent alarming increase in overweight and obesity that has affected a larger and larger proportion of the human population worldwide. Pharmacological therapies that are intended to augment the normal homeostatic feeding mechanisms, including leptin therapy and even gastrointestinal surgery for extreme obesity, often have only small to modest long-term effects in obese individuals (Christou, Look, & Maclean, 2006; Heymsfield et al., 1999; Li et al., 2005; Rucker, Padwal, Li, Curioni, & Lau, 2007). In fact, research by de Castro and colleagues (see de Castro, 2010, for a review) on multiple factors affecting feeding behavior in humans (including meal size and density, intermeal intervals, as well as social and environmental variables) has determined that physiological homeostatic mechanisms can account for only about 14% of the variance in daily food intake of freely living humans, and that environmental factors account for over 86% of the variance. It is not uncommon for people to gain excessive weight when they move from one area of the world to another in which highly palatable foods are readily available (Levitsky, 2005). The present review proposes that humans are genetically predisposed to consume high-fat and high-caloric foods and to gain excess body weight. To understand this, we must begin with our early ancestors.

Ancestral Hunter-Gatherers

Our prehuman hominid ancestors (australopithecines) existed at least 4 million years ago. The genus *Homo* originated during the early Pleistocene period (*Homo habilis*), about 2 million years ago. *Homo erectus* first appeared about 1.6 million years ago, but anatomically modern humans (*Homo sapiens*) have been in existence for only 200,000–400,000 years, and modern man (*Homo sapiens sapiens*) for only about 50,000–60,000 years (Mellars, 2011).

To survive, organisms must have an innate drive to seek out and consume food, and thus genetic adaptation responds to the availability of food. It is believed that humans' genetic adaptation to the food supply was estab-

lished by the time *Homo sapiens sapiens* appeared, with only minor changes having occurred since then (Eaton & Konner, 1985). This means, therefore, that to fully understand modern humans' genetic basis for feeding behavior, we must know the eating habits of our ancestors 2 million to 50,000 years ago.

Our hominid ancestors, like modern humans, were omnivores. Plant life was probably a major component of the diet of *Homo erectus* (Howell & Clark, 1963; Isaac & Crader, 1981). However, the archeological record shows that stone tools were used by *Homo habilis* to process meat as early as 2 million years ago (Bunn, 1981; Clark, 1984). Some believe that the meat intake of *Homo habilis* and early *Homo erectus* was obtained primarily by scavenging (Outram, 2007; Shipman, 1983). Nevertheless, there is clear evidence that *Homo erectus* were hunting with wooden spears as far back as 500,000 years ago (Thieme, 1997) and that *Homo sapiens* were highly effective at hunting large game (mostly ungulates) as long ago as 250,000 years (Kuhn & Stiner, 2001). *Homo sapiens* were such skilled hunters that they contributed to the extinction of many species of large mammals (Lorenzen et al., 2011).

It has been estimated that over half of the diet of *Homo sapiens* consisted of meat (Eaton & Konner, 1985; Kuhn & Stiner, 2001). This was true regardless of latitude (Kuhn & Stiner, 2001). Compared to other primates and mammals with diets high in vegetation, modern humans have longer small intestines and shorter colons (Armelagos, 2010), a genetic adaptation to higher intake of meat and other nutritionally high-dense foods (Milton, 1999). A diet high in meat helped to satisfy the energy required by a much larger brain, which doubled in size in the last 2 million years and now accounts for 20% of our energy requirement (Halloway, 1996).

The archeological record reveals few tools for processing vegetation until about 10,000 years ago (Kraybill, 1977). There is also little evidence that prehistoric humans stored plant foods other than nuts and seeds (Kuhn & Stiner, 2001). Plant foods (e.g., berries, fruit, and vegetable foods) were gathered when in season and consumed rather quickly. Thus, if we begin with *Homo habilis*, during well over 99% of the last 2 million years our ancestors lived as hunter-gatherers (Lee & Devore, 1968). Half the world's population still lived as hunter-gatherers at the time of Christ (Devore, 1984).

Prehistoric hunter-gatherer groups are believed to have been quite small, sometimes numbering no more than 25 individuals (Kuhn & Stiner, 2001). They were generally nomadic, moving from place to place as the seasons (and availability of food) changed (e.g., Lieberman, 1993). As nearby animals were killed, hunters had to travel greater and greater distances from a site to find new game. This travel, along with persistence hunting (chasing animals to exhaustion), would have required a great deal of energy (Bramble & Lieberman, 2004; Liebenberg, 2006). Thus, prehistoric hunters would have had to weigh food sources in terms of energy expended and nutritional value gained. The archeological record shows that large game was highly

preferred to small animals as a source of protein (Kuhn & Stiner, 2001).

Wild game has a very low fat content (Eaton & Konner, 1985), and a diet high in lean meat has severe health consequences (e.g., liver and kidney damage) and cannot be tolerated for long (Cordain, Brand-Miller, Eaton, Holt, & Speth, 2000; Outram, 2007). Modern hunter-gatherers and pioneers of the American West are known to have abandoned killed game that they found to be very high in lean meat and low in fat (or to have eaten only the fattier portions of the animal) and to have actively pursued animals that they perceived to be higher in fat content (Cordain et al., 2000; Outram, 2007). In an extreme example, today's Alaskan Arctic Eskimos have a diet that is 50% fat, 30%–35% protein, and only 15%–20% carbohydrates (Ho, Mikkelsen, Lewis, Feldman, & Taylor, 1972). The archeological record shows that prehistoric hunter-gatherers targeted bone marrow, which is very high in fat (Outram, 2007). When plant foods are in short supply, animal fat can serve as a source of essential vitamins (Speth, 1983).

With seasonal variations in the availability of food, particularly plant foods, and in the absence of farming, hunter-gatherers would have experienced both periods of plentiful food and periods of food shortage (Devore, 1984; Eaton & Konner, 1985; Lieberman, 1993). The periods of shortage may have been brief (e.g., resulting from an unsuccessful hunt) and/or prolonged (e.g., due to weather conditions). Individuals who ate only when experiencing a short-term intrameal deficit in energy would not have been prepared for longer periods of deprivation (Pinel, Asanand, & Lehman, 2000). Some researchers have proposed that in the course of evolution, prehistoric humans acquired a "thrifty gene"—those who were best able to store energy as fat had a greater chance of survival during times of food shortage (Neel, 1962). In short, for ancestral hunter-gatherers, a moderately overweight phenotype was highly adaptive for their natural environment (Pinel et al., 2000). As part of the survival mechanism, prehistoric humans almost certainly would have consumed as much animal fat and carbohydrates as possible when those foods were available (Outram, 2007). Modern hunter-gatherers have been observed to consume food in this manner (Speth & Spielmann, 1983).

The consumption of nutritionally high-dense foods (to be converted into stored fat) would require that the ancestral brain have evolved to signal the need, or desire, for such foods. In addition to hypothalamic nuclei common to all mammals, researchers believe that *Homo sapiens* developed unique cortico-limbic pathways that are essential in feeding behavior, particularly selection of foods (see later section). Regardless of which areas of the brain evolved to signal preference for high-fat and high-carbohydrate foods, modern man approached 8,000 B.C. with both a gastrointestinal system and brain circuitry that favored the intake of nutritionally high-dense foods.

The archeological record shows that farming first developed about 10,000 years ago (Cohen, 2009; Lichtenstein, 1999). During the ensuing "agricultural revolution," there was a dramatic shift in the diet of *Homo sapiens*

sapiens, from a diet heavy in meat to one that was 90% plant-based (Eaton, Eaton, Konner, & Shostak, 1996). This revolution initially focused on the farming of wheat, barley, rice, and beans but now includes the farming of animals high in fat content (see Armelagos, 2010). The last century witnessed the industrialization of the food supply, that is, large changes in the processing and distribution of food (Grey, 2000), so that today, humans in industrialized countries need to expend few calories to obtain a meal high in both fat and carbohydrates. They no longer have to weigh a food source in terms of the energy expended to obtain it and the nutritional value gained.

The negative effects of the agricultural and industrial revolutions and a carbohydrate-rich and high-fat-rich diet on health are well documented (e.g., Armelagos, 2010; Eaton & Konner, 1985; Pinel et al., 2000), but that is not the focus of this review. Ten thousand years is too short a time period for significant genetic adaptation to have occurred. Humans today react to the modern food supply in a manner that was genetically adapted for the hunter-gatherers who existed prior to the agricultural revolution and the industrialization of the food supply.

Not everyone becomes obese when exposed to an obesogenic environment. If 68.8% of American adults are overweight or obese, then 31.2% are not (Flegal et al., 2012). Thus, any genetic predisposition for obesity must not be equally distributed in the population; some individuals are more obesity-prone than others (Blundell et al., 2005; Carnell & Wardle, 2008). This should not be surprising, as diversity in genetic make-up within the population helps to ensure survivability of the species in the face of changing environmental conditions. (An “anti-obesity” genotype might have been a disadvantage 1 million years ago but may well provide a cardiovascular benefit in today’s environment.)

To date, genome-wide association studies have revealed 32 genetic loci associated with BMI (Speliotes et al., 2010). Studies with monozygotic and dizygotic twins have concluded that 55% to 85% of the variance in BMI is due to heritable genetic differences (e.g., Hewitt, 1997; Maes, Neale, & Eaves, 1997; Wardle, Carnell, Haworth, & Plomin, 2008). If it is also true that 86% of the variance in the daily food intake of freely living humans is attributable to environmental factors, then it logically follows that many of the individual differences in body weight are due to inherited behavioral/psychological differences in response to an obesogenic environment (de Castro, 2010).

Sensory Control of Food Intake

If humans are genetically predisposed to eat high-density foods, the question remains as to what the anatomical products of these genes are that lead to consumption of such foods. Two likely candidates are our senses of taste and smell. The tongue has receptors for five basic tastes: sweet at the tip of the tongue, sour at the sides, bitter at the back, salty elsewhere, and umami (Bachmanov & Beauchamp, 2007). These tastes, along with olfactory input, are processed at higher levels of the taste pathway to represent

flavors (E. T. Rolls, 2005). Olfactory input plays a major role in determining the palatability (not to be confounded with taste) of a food (Le Magnen, 1971). Positron emission tomography (PET) scans and functional magnetic resonance imaging (fMRI) studies in humans reveal that the amygdala, insula, and orbito-frontal cortex are particularly involved in the evaluation of pleasantness of taste and smell (de Graaf et al., 2004). The primary sensory cue for fat in the mouth is texture (Mattes, 2005), and neurons in the primate orbitofrontal cortex also respond to this sensory property of fatty foods in the mouth (E. T. Rolls, Critchley, Browning, Hernadi, & Lenard, 1999).

Can it just be a coincidence that in a species that evolved with a gastrointestinal system designed to process high-density foods that taste receptors for sweetness are at the very tip of the tongue? Studies with newborn animals show that “sweet substances are congenitally treated . . . as a basis of responses to a caloric source” (Le Magnen, 1971, p. 247; Pfaffmann, 1936). One can be certain that for most of the year, foods high in sugar (e.g., ripe fruits) were relatively uncommon for ancient hunter-gatherer societies, and because of their energy benefit, it was adaptive to detect them easily. Taste receptors for bitterness enabled humans to avoid foods such as green fruits. Refined sugar was once a luxury item and did not become readily available worldwide until the 1500s (Armelagos, 2010). Today, the average American’s daily diet includes 22.2 teaspoons (355 calories) of added sugar (R. K. Johnson et al., 2009).

When rats are given a choice between drinking water and a glucose solution, at some concentrations they highly prefer the glucose solution (Richter & Campbell, 1940). The preference is driven primarily by taste, not post-ingestive factors, as the same results are found with water versus saccharin solutions (Sheffield, 1966). These results are affected by physiological need; that is, hunger-deprived animals prefer the sweet solutions even more than do nondeprived animals (Bacon, Snyder, & Hulse, 1962; Jacobs & Sharma, 1969). The same results are found with standard lab chow versus chow with added sweetener or fat (Jacobs & Sharma, 1969).

Numerous studies with humans have also demonstrated that palatability of the diet is positively correlated with the amount of food consumed (see de Graaf, de Jong, & Lambers, 1999). This is true under identical premeal deprivation conditions (Le Magnen, 1969). Nevertheless, as more and more of a single palatable food is consumed during a meal, the subjective pleasurable ratings of that food’s taste, texture, and smell decline. This is called sensory-specific satiety because the satiation effect is limited to that one food or very similar foods (Hetherington, 1996; B. J. Rolls, 1986). Again, neurons within the human orbitofrontal cortex respond to the changing subjective pleasantness of that particular food (E. T. Rolls, 2005; E. T. Rolls, Sienkiewicz, & Yaxley, 1989). If an individual is presented with another highly palatable food after satiating on the first, he or she will consume the second food until reaching sensory-specific satiety, and this may continue for several courses (B. J. Rolls, van Duijvenvoorde, & Rolls, 1984). When offered a variety of highly preferred foods,

many people will eat until the point of feeling ill (Pinel et al., 2000).

Sensory-specific satiety depends not on postingestive factors but instead on sensory factors (e.g., taste). The same results are observed when human subjects are allowed to chew (for as long as they normally would during a meal), but not swallow, a food (Drewnowski, Grinker, & Hirsch, 1982; E. T. Rolls & Rolls, 1997). Partial sensory-specific satiety can be obtained by allowing subjects to just smell the food (E. T. Rolls & Rolls, 1997). Sensory-specific satiety also does not depend on explicit memory of eating a particular food, for it is observed in amnesiac patients who do not remember which food they have just consumed (Higgs, Williamson, Rotshtein, & Humphreys, 2008).

When continuously presented with a large variety of highly palatable foods (commonly called a “cafeteria diet”), animals eat more (because the satiating effect of each food is sensory specific) and gain substantially more body fat than when presented with just standard lab chow (B. J. Rolls & Hetherington, 1989). Humans also consume more food and gain more weight over time when offered a large variety of highly palatable foods (Raynor & Epstein, 2001). Thus, the evidence indicates that it is the palatability of foods rather than homeostatic physiological hunger signals that drives many humans to seek out and consume foods.

In their studies of free-living humans, de Castro and colleagues calculated that density of the diet and palatability of the food accounted for only about a modest 10% and 5% of the variance in meal size, respectively (de Castro, 2004, 2010; de Castro, Bellisle, Dalix, & Pearcey, 2000). However, they acknowledged the real possibility of a statistical ceiling effect that was due to the subjects self-selecting their own diets. Correlation is highly affected by the variability in data of the measured variables (King, Rosopa, & Minium, 2011), and in a society in which there is already an abundance of high-density and palatable foods, the range of choices would be restricted. Nevertheless, it is important to note that even with this restricted range of observations, by comparing the eating habits of identical and fraternal twin pairs the researchers found that, in addition to the environment (the major factor), choices with regard to both density of the diet and palatability of the food were significantly influenced by heredity (de Castro, 2010).

The question arises as to what differentiates those individuals who become obese from those who do not. If the chemosensory systems alone were responsible for obesity, then loss of taste and/or smell should eliminate excess weight gain. Loss of olfaction sometimes results in decreased food intake and weight loss, but usually there is no change in body weight, and initial weight gains in newly anosmic individuals are not unusual (Aschenbrenner et al., 2008; Ferris & Duffy, 1989; Mattes & Cowart, 1994; Mattes et al., 1990). Weight loss is more common the longer the history of anosmia (Aschenbrenner et al., 2008; Ferris & Duffy, 1989), and individuals with congenital anosmia are less likely than normal adults to have an elevated BMI. Nevertheless, some congenitally anosmic

individuals do become overweight (Aschenbrenner et al., 2008). Similarly, loss of taste, independent of anosmia, is sometimes associated with weight loss, the more so the greater the loss, but many who lose the sense of taste do not lose weight (Mattes & Cowart, 1994; Mattes et al., 1990; Mattes-Kulig & Henkin, 1985), leading to the conclusion that it is not the senses of taste and smell alone that lead to obesity.

Brain Reward Systems

What clearly differentiates obese individuals from lean persons is not the chemosensory systems or the environment (availability of highly palatable foods) per se but obese individuals’ interaction with the environment, that is, their feeding responses to food-related stimuli of all types—the taste, smell, texture, and sight of food and watching others eat. It has long been known that obese individuals are much more responsive to these external feeding cues than are normal-weight individuals (Schachter, 1968). In fact, obese individuals are much more responsive to external cues than to biological mechanisms (Blundell et al., 2005; Carnell & Wardle, 2008; Levitsky, 2005; Schachter, 1968). For example, unlike normal-weight children, overweight children do not compensate by eating less after a preload of food (Jansen et al., 2003; S. L. Johnson & Birch, 1994). Obese individuals have been described as “stimulus-bound” (Schachter, 1971). Food-cue responsiveness has been found to be a strongly heritable trait (Carnell & Wardle, 2008).

Some researchers have argued that the sensory pleasure of food can be regarded as a reward or reinforcement (Pfaffmann, 1960; see Berridge, 2004):

Orosensory stimulations inasmuch as they are the bases of palatability responses are by themselves reinforcing of other behavioral responses or of the feeding response itself . . . the pleasure of sensation reinforces the repetition of the consummatory response which provides this pleasure and conversely. (Le Magnen, 1971, p. 252)

Neuroimaging studies reveal that the brain responses of obese and lean individuals to the taste and smell of food differ (Bragulat et al., 2010; Del Parigi, Chen, Salbe, Reiman, & Tataranni, 2005). As the taste, smell, and texture of food are processed along the higher order taste and feeding pathways, very pleasant stimuli also activate the brain “reward” systems. These are cortico-limbic-striatal structures and circuits that respond to, and drive the individual to seek out, positive reinforcers of all types, not just food. Unlike the hypothalamic homeostatic feeding mechanisms, the reward circuits are not homeostatic (Zhang, von Deneen, Tian, Gold, & Liu, 2011). Using fMRI with humans choosing monetary rewards, McClure, Laibson, Loewenstein, and Cohen (2004) identified circuits within this system that respond to immediately available rewards (e.g., midbrain dopamine system, paralimbic cortex) and other circuits that respond to delayed rewards (e.g., lateral prefrontal cortex, posterior parietal cortex).

Behaviorally, addicts of various types have a strong tendency to discount delayed large rewards in favor of

smaller immediate rewards. For example, drug addicts have a high preference for immediate reward; that is, they discount delayed rewards much more than do nonaddicted controls (Coffey, Gudleski, Saladin, & Brady, 2003; Kirby, Petry, & Bickel, 1999). The same is true of cigarette smokers (Baker, Johnson, & Bickel, 2003) and alcoholics (MacKillop et al., 2010; Petry, 2001). Obese individuals display the same preference for immediate rewards (e.g., enjoyment of the taste of foods), discounting delayed rewards (e.g., health consequences; Bonato & Boland, 1983; Davis, Patte, Curtis, & Reid, 2010; Geller, Keane, & Scheirer, 1981; W. G. Johnson, Parry, & Drabman, 1978; Weller, Cook, Avsar, & Cox, 2008).

Studies using fMRI have observed abnormal activity in the immediate reward circuits of adult obese subjects compared with normal-weight subjects when shown stimuli such as high-calorie foods (e.g., Rothmund et al., 2007; Stoeckel et al., 2008, 2009). Others have similarly observed greater activation of brain reward circuitry in obese adults (compared with normal-weight controls) in response to pleasant food stimuli (Demos, Kelley, & Heatherton, 2011; Ng, Stice, Yokum, & Bohon, 2011; see Carnell, Gibson, Benson, Ochner, & Geliebter, 2012, for a review).

Obese children also display hyperactivation of brain reward areas when shown images of food (Bruce et al., 2010). Obesity is associated with polymorphism of the TaqI A1 allele of the DRD₂ dopamine receptor gene (Blum et al., 1996), and neuroimaging studies in children susceptible to future obesity because of variations in this gene show hyperactivity in the brain reward circuitry (Stice, Yokum, Bohon, Marti, & Smolen, 2010; Stice, Yokum, Burger, Epstein, & Small, 2011).

Conclusions

This review is not intended as an all-encompassing explanation of obesity and is limited to heritable behavioral/psychological characteristics contributing to overeating. It is likely that as the brain doubled in size since the appearance of *Homo erectus* (Halloway, 1996), it was the reward circuits that evolved to work with hypothalamic homeostatic feeding mechanisms. It would have been adaptive in the time of ancestral hunter-gatherers for the brain circuits mediating preference for immediate rewards to steer humans to consumption of very palatable foods. However, there is increasing evidence that in an obesogenic environment it is these same nonhomeostatic brain reward areas that override the homeostatic controls and lead many humans to overconsume when presented with an almost unlimited variety of pleasant tasting foods and drinks (Cameron & Doucet, 2007; Carnell et al., 2012; Levin, 2005).

In the treatment of obesity, one should not underestimate the strength of the drive to experience pleasant tasting stimuli. Hyperactivity in the brain reward circuitry in response to food stimuli was still observed in previously obese individuals who had maintained their reduced weight for at least three months (Del Parigi et al., 2004). To lose weight and maintain the loss will take considerable cognitive restraint, but this too is a strongly heritable character-

istic on which some people do not score high (de Castro, 2010; de Castro & Lilenfeld, 2005). It is not surprising, therefore, that weight-loss strategies that emphasize changes in diet have proven unsuccessful long term (LeBlanc et al., 2011). We must also recognize the origins and strength of people's desire for palatable foods and work to modify the environment and people's interaction with their environment.

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