Chapter 8
Binge Eating, Disinhibition and Obesity

Prof. Stanley Ulijaszek, Ph.D. and Eleanor Bryant, Ph.D.

Lay summary Obese people are more likely to eat at every opportunity (display disinhibition) and often binge on food. We argue in this chapter that binge eating and disinhibition are evolved mechanisms for dealing with one of the most fundamental of insecurities, that of food, especially in seasonal and unpredictable environments. It is only in recent decades, with improved food security in industrialized nations and the emergence of obesity at the population level, that they have become deleterious for health and have been medically pathologized. Binge eating and disinhibition are no longer responses to uncertainty in food availability as they would have been across evolutionary history. Rather, uncertainty and insecurity in everyday life in present-day society are likely to lead to disinhibition, binge eating and obesity, through the linked physiology of stress and appetite.

8.1 Introduction

Obesity is new in human evolutionary history. It has become possible at the population level since the increase in food security and has risen in most countries on most continents since the 1980s [1]. While prevalence rates of obesity have since levelled off in a small number of regions [2], they have risen steadily in most

S. Ulijaszek (✉)
Unit for Biocultural Variation and Obesity, School of Anthropology, University of Oxford, Oxford, UK
e-mail: stanley.ulijaszek@anthro.ox.ac.uk

E. Bryant
Division of Psychology, Richmond Building, University of Bradford, Bradford, UK

A. Alvergne et al. (eds.), *Evolutionary Thinking in Medicine*, Advances in the Evolutionary Analysis of Human Behaviour,
DOI 10.1007/978-3-319-29716-3_8

industrialized countries. In the USA, the prevalence of obesity (body mass index greater than 30 kg/m^2) and extreme obesity (body mass index greater than 40 kg/m^2) has shown rapid increases from the 1980s to the 2000s. In 2003–2004, the national prevalence of obesity was 30 %, while that of extreme obesity was 5 % [3, 4], rising to 34 % (obesity) and 6 % (extreme obesity) in 2008 [5, 6]. In the UK, the prevalence rates of obesity and extreme obesity have been much lower than in the USA, but have followed similar trajectories [7, 8].

There are many pathways, still poorly understood, to the positive energy balance (due either to high dietary energy intake, low energy expenditure or both) that leads to obesity. Among the eating disorders associated with obesity (and extreme obesity especially), binge eating disorder (BED) is particularly important. This was first described by Stunkard [9] and linked to obesity by Spitzer et al. [10, 11]. Studies of obese individuals who sought treatment identified subgroups that experienced distress and dysfunction due to binge eating [12]. The diagnostic and statistical manual of mental disorders 4 (DSM4) [13] defines binge eating as a series of recurrent binge episodes in which each episode is defined as eating a larger amount of food than normal during a short period of time (usually within any two-hour period). Characterized by economists as "high time preference", the desire to binge-eat might reflect the desire for palatable food now, rather than have a slim body, or the health that can go with it, later [14]. Binge eating behaviour is not always associated with obesity and does not necessarily have to be practised at extreme levels to be of importance for the causation of obesity.

The vast majority of binge eating remains undiagnosed or outside of clinical parameters [15], for a range of reasons. These include the lack of valid and reliable instruments for its measurement, and the likelihood that many people who experience binge eating does not come for help, often because of embarrassment, or because they do not see their behaviour as problematic. Binge eating disorder is usually identified when people seek help, as for example when obese subjects put themselves forward for bariatric surgery. Regular overeating within meals and snacking between meals are behaviours that are strongly linked with both obesity and subsequent health issues, and have similar underlying mechanisms to BED. Such behaviours are well captured by the psychological measure of disinhibition, as assessed by the Three-Factor Eating Questionnaire [16]. An individual scoring highly on the disinhibition scale of this measure is characterized as having very opportunistic eating behaviours and expressing a readiness to eat [17], both being associated with BED.

Binge eating (whether associated with obesity or not) can be thought of as a response to insecurity with deep evolutionary roots. The greatest cause of insecurity among past populations was that related to food and its provisioning and of climatic seasonality and variation in which food availability could not be guaranteed across the year, or sometimes from day-to-day [18]. Across evolutionary time, environmental variability has been the norm, and modern humans are very capable of responding physiologically, behaviourally and culturally to such variability [19, 20].

Such variability would have led to variation in dietary possibilities, food intake and uncertainty in food availability. We argue that binge eating and disinhibition are mechanisms that have been selected for by natural selection as they enabled individuals dealing with one of the most fundamental of insecurities, that of food, especially in the seasonal and unpredictable environments during the evolution of early Homo, from about 2–4 million years ago [21]. High reactivity to external cues such as colour, taste and smell [21] and subsequent disinhibited eating under conditions of food scarcity and/or high levels of competition would have favoured survivorship for most people and populations until fairly recent times. Only with improved food security in industrialized nations, the decline in price of energy dense foods and the emergence of obesity at the population level that disinhibited eating and binge eating would have become deleterious in terms of health outcomes. The chapter begins with descriptions of binge eating disorder and disinhibited eating and then considers the evolutionary importance of rapidly consuming large quantities of food in seasonal environments. It concludes by examining the evolutionary significance of binge eating and disinhibition and their implications for medicine in an age of obesity.

8.2 Research Findings

8.2.1 Binge Eating Disorder and Disinhibition

According to the DSM4 [13], binge eating episodes are associated with 3 or more of the following presentations: (1) eating until feeling uncomfortably full, (2) eating large amounts of food when not physically hungry, (3) eating much more rapidly than normal, (4) eating alone because of embarrassment about how much is eaten in a single eating episode, (5) feeling disgusted, depressed or guilty after overeating or (6) marked distress or anxiety regarding eating in such a way. Aside from those who are clinically diagnosed as having binge eating disorder, there are far more people who often binge on food, but perhaps not regularly enough to warrant a clinical diagnosis [15]. In line with this, disinhibition describes similar eating behaviour patterns as BED, although it is not used to diagnose clinical symptomatology; it is related to eating disorder severity [22]. Disinhibition is an eating behaviour trait measured by the Three-Factor Eating Questionnaire [16] which has been validated among a number of populations in Europe, the USA and Australia. It measures enduring characteristics of an individual's behaviour towards food. Disinhibition is defined by a readiness to eat [17] and is associated with frequent overeating and/or binging [23], frequent snacking [24], a high liking of all food groups with a particular preference for high-fat sweet foods [22], a tendency to gain weight and regain weight quickly following weight loss [25] and a tendency to be engaged in sedentary behaviour [26].

Although there is no clear line dividing eating a large meal and a pathological binge, people usually binge on highly palatable energy-rich food, typically high in fats, sugars or both [15]. It is impossible to know whether prehistoric foragers had cuisine, but they are certain to have had food preferences, if only on the basis of energy density, sweetness or fattiness of foods [18]. Individuals that binge on sweet foods tend to do so more frequently than those binging on other types of foods, and there is the potential for abuse of carbohydrates among individuals who claim to have cravings for them [27]. Similar addictive-like states can be created for fat consumption in animal models [28, 29], which also engage the dopaminergic reward system [30]. This system has been invoked for both addictions and obesity [31], and binge eating and disinhibition may be the link. From an evolutionary perspective, it is reasonable to think of addiction to palatable energy dense foods as being an evolved predisposition, if binge eating, by maximizing energy intake, also maximized reproductive success.

8.2.2 The Evolutionary Importance of Binge Eating and Disinhibition in Seasonal Environments

If the environment is uncertain or unstable, being able to eat a lot of energy dense foods very quickly when they are available is expected to have favoured survivorship and, by extension, reproduction. Although many foraged foods may not deteriorate very quickly, competition for food in uncertain or unstable environments would drive the need for fast eating. There is a physiological ceiling on how much protein an individual can eat, because of the finite ability of the liver to up-regulate enzymes necessary for urea synthesis in the face of increasing dietary protein intake [32]. According to Cordain et al. [32], hunter-gatherers would have had several options to circumvent the dietary protein ceiling. They could have eaten proportionately more plant food energy; hunted larger animals because percentage body fat increases with increasing body size; hunted smaller animals in seasons of plentiful forage, when body fat is maximized; selectively eaten only the fattier portions of the carcass; and/or increased their intake of concentrated sources of carbohydrate such as honey. Human feeding adaptations are likely to have arisen in the context of resource seasonality in which diet choice for energy dense and palatable foods would have been selected for by way of foraging strategies that maximized energy intake [21]. This may have been facilitated by heightened responses to visual cues associated with foods of high hedonic value when hungry [33]. These visual cues may have included aspects of colour, shape and size that reflect the relative ripeness, palatability and gustatory satisfaction to be gained from fruits and vegetables, for example, and would have been learned from previous exposure to, and consumption of, such foods [33]. Although primates respond to visual cues of food palatability much as do humans [28], there is no evidence that they have heightened responses to them when hungry.

Evidence that BED is a physiological feeding adaptation to food uncertainty comes from a mix of psychological and physiological studies of human feeding. Food uncertainty may have benefitted those able to consume great amounts of food in one go, potentially resulting in natural selection for this ability. Such selection would have been in addition to the strategies for protecting energy reserves shared by all human beings by being more sedentary, by readily storing body fat, being resistant to weight loss and quickly regaining weight lost, and the ability to binge on large volumes of food by some individuals points to this combination of characteristics being one type of thrifty phenotype [17]. Physiological differences in the appetite systems of individuals with a high disinhibition score and among binge eaters could also point to adaptations that enable consumption of greater amounts of food. For example, obese individuals with high disinhibition have high serum ghrelin and insulin levels [34]. These hormones regulate and initiate hunger, respectively. Furthermore, the cholecystokinin response of the gut, which promotes satiety in response to having eaten, is blunted in people who score highly on the tests of disinhibition [35]. On the other hand, serum ghrelin levels have been found to be lower in individuals with BED [36], suggesting a down-regulation of hunger in response to binge eating behaviour, much as obese people experience physiological down-regulation of hunger. Binge eating can take place in the absence of a hunger stimulus from the gut, especially of highly palatable foods for pleasure [37]. Food consumption under such conditions has been termed hedonic hunger [38].

Differences between those with BED and high disinhibition and those without have also been found in neurophysiology. For example, increased activity in the insular cortex (an area involved in emotion, motor function and homeostasis; [39]) and the prefrontal cortex (an area associated with executive function such as emotion regulation and general processing) when full [40] has been observed in individuals with high disinhibition scores in response to ingestion of high-fat food. Furthermore, those with BED show a higher activation in the orbitofrontal cortex (an area involved in sensory integration, emotion and decision-making [41]) in response to images of food. Those neurological differences could lend at least a partial explanation to individual response to food and ability to overeat, although the direction of the relationships between behaviours and imaged patterns of brain activity is not resolved (brain image patterns may be either the cause or consequence of food-related behaviours). While the mechanisms that these findings represent are not clear, they indicate a very strong emotional involvement with food and a higher degree of integration of sensory information associated with seeing, eating and digesting food among those with BED and high disinhibition scores than among those who do not display these feeding traits. The evidence for food uncertainty among prehistoric humans is indirect, but compelling [18]. Dispersal of anatomically modern human populations out of Africa into Europe and Asia would have exposed them to new stresses, physical, biological and social, which would have varied over time and place. In all places, tropical and temperate, humans may have experienced a double burden of resource fluctuation through seasonality as well as shifts in their environment caused by abrupt climate change [18]. At points in the Pleistocene, especially during the short-lived warming events that occurred

within glacial periods, climatic transitions that potentially affected food security and resource distribution may have taken place over timescales of decades rather than many generations [42]. With less time to adapt, it is easy to imagine the starvation- and cold-related mortality that resulted and the food uncertainty that would have been a daily reality, especially among those that colonized the higher latitudes in temperate regions where extreme temperature and precipitation seasonality would have been the norm. Binge eating when food was available would have been an advantageous trait under such circumstances. According to Wisman and Capehart [43: 24]

> putting on weight -banking calories- is a 'natural' response to insecurity, a genetic trigger selected during the course of evolution. When sustenance, life-and-limb, or social positions were threatened, favouring fat-rich morsels and banking energy in the form of fat reserves would have been adaptive.

The amount of weight banking among contemporary foraging and agricultural societies can be as high as 5.9 kg body weight, equivalent to over 40,000 kcals dietary energy [44]. This would meet daily energy needs under complete starvation for at least 16 days if there were no physiological adaptation to starvation. However, such adaptation, as down-regulation of energy metabolism and reduced physical activity, is well documented [45], and weight banking to this extent would more likely cover the energy cost of complete starvation for double this time, or just over a month. Total food shortages among societies practicing foraging or traditional agriculture are rare, however, and putting on weight when food was plentiful would have provided a reliable fallback to subsequent food shortages in most years.

Eating, dietary manners and restraint are unlikely to have been favoured under such conditions. Common to many mammalian species, gorging and binging among early humans and their hominin ancestors when the circumstances allowed would have been the norm:

> humans are evolved to over indulge when surpluses are available. Because surpluses of this kind are rare in the natural environment, such a strategy does not commonly lead to obesity in traditional societies. However, in such conditions, it pays to be attracted to sweet, sugary foods and to carbohydrates, and to gorge on them whenever they happen to be available, since these provide the primary sources of free energy. In the 'natural state', this is not a problem, since the feast-times do not occur all that often. It is only in modern post-industrial economies, where we live in a state of permanent feast, that it becomes an issue [46: 56].

Seasonality continues to influence activity and behaviour, even though humans, particularly in the industrialized world, are buffered against many aspects of environmental seasonality. This is especially so with respect to the availability of cheap, high-energy-density foods based on refined carbohydrates and fats, upon which most of the nutrition transition has been based [47] and which dominates diet in the industrialized and post-industrial world.

8.3 Implications for Policy and Practice

In contemporary society, the structures of food and of human feeding are at least partly incompatible and, at the extreme, pathological. In considering binge eating and disinhibition, it is important to think about the structures that human societies, and individuals in them, have in place to regulate eating, be it enough, too little, or too much. It is often not clear what the distinction is between eating a large meal and binge eating [15], but it might be that the structure of eating, social and individual, is a defining principle often missed by biomedicine. There are many social conventions surrounding food and its consumption in all societies [48, 49], mediating the physiological drive to eat, whether for hunger or pleasure (or both), and mediating individual responses to the dopaminergic reward system. Eating while listening to the radio or watching television increases the amount of food consumed [50], and the presence of familiar others is particularly potent at increasing food intake [51]. Both allow diversion of attention from food consumption and reduced self-monitoring of intake, allowing eating beyond immediate dietary energy needs [52]. Engaging in other tasks, such as working at the computer, also reduces self-monitoring of food intake. Modern humans in industrialized and post-industrial societies consume high-energy-density diets, often in unstructured ways [21], often under conditions of distraction. Modern life and cheap palatable foods make disordered, disregulated and distracted eating easier to practice, and extreme bodily phenotypes signal disordered eating to others, even in societies where generosity and plenty are valued, and large bodies are traditionally seen as carrying prestige [53].

The processing of agricultural raw materials by the food industry has produced a huge range of food products with reduced micro-nutrient content and high energy density. It has also resulted in the production of foods with altered physical matrices and new combinations of ingredients that make them more pleasurable to eat [18]. Food technology has been used to both respond to existing markets for products and create new market niches. This has involved the production of cheaper existing ingredients, completely new ingredients, new food types with existing ingredients, existing food types with proportions of new ingredients and new food types with new ingredients. Because food products are placed in competitive marketplaces in most countries, they must maximize some combination of novelty, status, price and palatability that will ensure their economic success. The cheaper raw ingredients are overwhelmingly cereal-based commodities and fats, and it is unsurprising that the majority of food products on the market involve some palatable combination of these ingredients [18]. Where palatability leads, increased consumption follows. The drive to create new, highly palatable high-fat and/or high sugar food products to develop and maintain market share by transnational companies has had profound effects on human food consumption, including ambivalence about eating [54] and possibly disordered eating [55]. Obesity may be a result of incremental overeating across many years or of periodic or sporadic binging, dieting, restriction, loss of

control in response to guilt and ambivalence about foods and food types and distorted self-perception of body weight.

Binge eating, BED, high disinhibition and obesity all approach or fall into the category of biomedical pathology. But from an evolutionary perspective, binge eating and disinhibition are evolved predispositions. Among contemporary industrial and post-industrial society, they are deleterious, largely because of the plentiful nature of, and lack of seasonality in, the foods that promote gorging, however categorized. The sheer abundance of high-energy-density foods in industrial and post-industrial societies means that it is almost inevitable that people experience binge eating to some extent and at some time or another. It is social convention that categorizes a food consumption binge as taking place when it is solitary and involves high-energy-density foods, for example, and not taking place when a substantial symbolic meal like Christmas or thanksgiving dinner is consumed with family and friends. Feasting across Christmas and thanksgiving has been shown to be associated with significant weight gain [56, 57], but there have been no serious moves anywhere to pathologize either of these meals. Binge eating and disinhibition are no longer a response to uncertainty in food availability, as they are likely to have been across evolutionary history. Rather, there may be other types of uncertainty and insecurity that lead to disinhibition, binge eating and obesity. Stress-related chronic stimulation of the hypothalamic–pituitary–adrenal axis and resulting excess glucocorticoid exposure is tightly intertwined with the endocrine regulation of appetite, as well as orchestrating appropriate physiological response to stress [58]. This is hardly surprising given the importance of the regulation of energy and food intake under stress for survival [58].

Stress for survival takes on a different meaning in the contemporary world, but the mechanism for dealing with it is the same. Overeating is often a personal response to chronic life stress, and market liberalism, especially in countries such as the USA, UK and Australia, creates environments of great individual insecurity that cuts across socio-economic position, and this is the source of stress that drives higher levels of obesity in such countries [14]. The structures that neoliberal societies put in place promote insecurity and inequality, while work-related insecurity, including low income, poor job mobility and absence of union protection, raises the likelihood of stress and ill health. Responses to stress, in turn, include overeating and preferences for high-energy-density foods, both of which are implicated in the causation of obesity. Not everyone becomes obese, however, and the mechanism whereby insecurity and obesity are linked through overeating may well be binge eating, which at its extreme has been classified as the pathology of BED. Although it is not usually the place of clinical practice to question the ways in which society is run, it can at least engage with some of the health consequences of living in a neoliberal world. Stresses at work and in everyday life are both higher-level factors that structure health and illness of patients and communities, and downstream consequences of living insecure lives.

8.4 Future Directions

An important future direction would involve researchers and clinicians identifying the extent of stress-related binge eating. General practice researchers in the UK have made a strong case for brief interventions in primary care settings for weight management [59]. Furthermore, the National Obesity Observatory [60] has compiled good evidence that brief interventions can lead to at least short-term changes in weight-related behaviour and body weight if they meet a number of criteria. These are that they focus on both diet and physical activity; are delivered by practitioners trained in motivational interviewing; incorporate behavioural techniques, especially self-monitoring; are tailored to individual circumstances; and encourage the individual or patient to seek support from other people. From an evolutionary perspective, this seems strange and difficult: the major weight management task in prehistory was to keep body weight as high as possible. Binge eating, then and now, was and is a response to stress. To ask people to manage their weight, especially if the emphasis is placed on self-monitoring and individual responsibility, is to also ask them to manage their stress. It might be more effective if clinical practitioners could take the lead in linking awareness of stress and binge eating through their brief interventions.

Another future direction would be to identify how evolved predispositions to overeat could be located within frameworks that encourage increased physical activity, rather than reduced food intake and the self-control that this requires.

Glossary

Binge eating	When a larger than normal amount of food is consumed in one sitting
Disinhibition	An eating behaviour trait measuring a readiness for eating or opportunistic eating: a "see food and eat it" response. This trait is assessed through the Three-Factor Eating Questionnaire [16]
Dopamine reward system	Systems in the brain (mesolimbic pathway and mesocortical pathway in the ventral tegmental area); when these areas are stimulated, reward is perceived
Glucocorticoid	A corticoid substance which increases gluconeogenesis, increasing blood glucose levels. The main glucocorticoid is cortisol, which is responsible for regulating metabolism of protein, lipids and carbohydrates

Hedonic hunger	When an individual experiences frequent thoughts, feelings and urges towards food, while they have no physiological need for food
Hypothalamic–pituitary–adrenal axis	Feedback interactions among the hypothalamus, pituitary gland and the adrenal glands that control reactions to stress and, among other things, regulate digestion, mood, emotions and energy balance
Nutrition transition	Shifts in dietary consumption towards higher-energy-density foods, reduced physical activity and energy expenditure that have coincided with economic, demographic and epidemiological changes across the world
Psychological ambivalence	Describes a situation where an individual holds both positive and negative attitudes towards a food (e.g. I love the taste of this food, but I hate it as it makes me fat)
Three-Factor Eating Questionnaire	A psychometric tool to assess the eating behaviour traits of disinhibition (tendency to overeat and eating opportunistically), restraint (restricting intake to control body weight) and hunger (the extent to which feeling of hunger elicit eating episodes)
Thrifty Phenotype	The thrifty phenotype hypothesis suggests that early-life metabolic adaptations help in the survival of the organism by selecting an appropriate trajectory of growth in response to environmental cues (See also Chap. 6)

References

1. Stevens GA, Singh GM, Lu Y, Danaei G, Lin JK, Finucane MM, Bahalim AN, McIntire RK, Gutierrez HR, Cowan M, Paciorek CJ, Farzadfar F, Riley L, Ezzati M (2012) National, regional, and global trends in adult overweight and obesity prevalences. Popul Health Metrics 10:22
2. Rokholm B, Baker JL, Sorensen TIA (2010) The levelling off of the obesity epidemic since the year 1999—a review of evidence and perspectives. Obes Rev 11:835–846
3. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM (2006) Prevalence of overweight and obesity in the United States, 1999–2004. J Am Med Assoc 295:1549–1555
4. Wang Y, Beydoun MA (2007) The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. Epidemiol Rev 29:6–28
5. Ogden CL, Carroll MD (2010) Prevalence of overweight, obesity, and extreme obesity among adults: United States, trends 1960–1962 through 2007–2008. Centers of Disease Control: NCHS Health and Stats

6. Friedman DS (2011) Obesity—United States, 1988–2008. Centers for Disease Control and Prevention Morbidity and Mortality Weekly Report Supplements 60:73–77
7. Rennie KL, Jebb SA (2005) Prevalence of obesity in Great Britain. Obes Rev 6:11–12
8. Zaninotto P, Head J, Stamatakis E, Wardle H, Mindell J (2009) Trends in obesity among adults in England from 1993 to 2004 by age and social class and projections of prevalence to 2012. J Epidemiol Community Health 63:140–146
9. Stunkard AJ (1959) Eating patterns and obesity. Psychol Bull 33:284–294
10. Spitzer RL, Devlin M, Walsh BT, Hasin D, Wing R, Marcus M, Stunkard A, Wadden T, Yanovski S, Agrass S, Mitchell J, Nonas C (1992) Binge eating disorder: a multisite field trial of the diagnostic criteria. Int J Eat Disord 11:191–203
11. Spitzer RL, Yanovski S, Wadden T, Wing R, Marcus MD, Stunkard A, Devlin M, Mitchell J, Hasin D, Horne RL (1993) Binge eating disorder: Its further validation in a multisite study. Int J Eat Disord 13:137–153
12. Yanovski SZ (2003) Binge eating disorder and obesity in 2003: could treating an eating disorder have a positive effect on the obesity epidemic? Int J Eating Dis 34(S1):S117–S120
13. American Psychiatric Association (1994) Diagnostic and statistical manual of mental disorders, 4th edn. American Psychiatric Association, Washington DC
14. Offer A, Pechey R, Ulijaszek SJ (eds) (2012) Insecurity, inequality and obesity. The Clarendon Press, Oxford
15. Avena NM, Rada P, Hoebel BG (2009) Sugar and fat bingeing have notable differences in addictive-like behavior. J Nutr 139:623–628
16. Stunkard AJ, Messick S (1985) The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res 29:71–83
17. Bryant E, King N, Blundell J (2008) Disinhibition: its effects on appetite and weight regulation. Obes Rev 9:409–419
18. Ulijaszek SJ, Mann N, Elton S (2012) Evolving human nutrition: implications for public health. Cambridge University Press, Cambridge
19. Elton S (2008) Environments, adaptation, and evolutionary medicine: Should we be eating a stone age diet? In: Elton S, O'Higgins P (eds) Medicine and evolution: current applications, future prospects. CRC Press, Boca Raton, pp 9–33
20. Elton S (2008) The environmental context of human evolutionary history in Eurasia and Africa. J Anat 212:377–393
21. Ulijaszek SJ (2002) Human eating behaviour in an evolutionary ecological context. Proc Nutr Soc 61:517–526
22. Bryant E (2006) Understanding disinhibition and its influences on eating behaviour and appetite. PhD dissertation, University of Leeds
23. Mailloux G, Bergeron S, Meilleur D, D'Antono B, Dube I (2014) Examining the associations between overeating, disinhibition and hunger in a nonclinical sample of college women. Int J Behav Med 21:375–384
24. Chaput JP, Depres JP, Bouchard C, Tremblay A (2011) The association between short sleep duration and weight gain is dependent on disinhibited eating behaviour in adults. Sleep 34:1291–1297
25. Niemeier HM, Phelan S, Fava JL, Wing RR (2007) Internal disinhibition predicts weight regain following weight loss and weight maintenance. Obesity 15:2485–2494
26. Bryant EJ, Kiezebrink K, King NA, Blundell JE (2010) Interaction between disinhibition and restraint: implications for body weight and eating disturbance. Eat Weight Disord 15:43–51
27. Spring B, Schneider K, Smith M, Kendzor D, Appelhans B, Hedeker D, Pagoto S (2008) Abuse potential of carbohydrates for overweight carbohydrate cravers. Psychopharmacology 197:637–647
28. Le Magnen J (1990) A role for opiates in food reward and food addiction. In: Capaldi PT (ed) Taste, experience, and feeding. American Psychological Association, Washington DC, pp 241–252
29. Teegarden SL, Bale TL (2007) Decreases in dietary preference produce increased emotionality and risk for dietary relapse. Biol Psychiatry 61:1021–1029

30. Arias-Carrion O, Stamelou M, Murillo-Rodriguez E, Menendez-Gonzalez M, Poppel E (2010) Dopaminergic reward system: a short integrative review. Int Arch Med 3:24
31. Volkow ND, Wang GJ, Fowler JS, Telang F (2008) Overlapping neuronal circuits in addiction and obesity: evidence of systems pathology. Philos Trans R Soc Lond B Biol Sci 363:3191–3200
32. Cordain L, Brand-Miller J, Eaton SB, Mann N, Holt SHA, Speth JD (2000) Macronutrient estimations in hunter-gatherer diets. Am J Clin Nutr 72(6):1589–1592
33. Cornier M-A, von Kaenel SS, Bessesen DH, Tregellas JR (2007) Effects of overfeeding on the neuronal response to visual food cues. Am J Clin Nutr 86:965–971
34. Bryant EJ, King NA, Falken Y, Hellstrom PM, Holst JJ, Blundell JE, Naslund E (2013) Relationships among tonic and episodic aspects of motivation to eat, gut peptides and weight before and after surgery. Surg Obes Relat Dis 9:802–808
35. Burton-Freeman BM, Kiem NL (2008) Glycemic index, cholecyctokinin, satiety and disinhibition: is there an unappreciated paradox for overweight women? Int J Obes 32:1647–1654
36. Geliebter A, Ladell T, Logan M, Schweider T, Sharafi M, Hirsch J (2006) Responsivity to food stimuli in obese and lean binge eaters using functional MRI. Appetite 46:31–35
37. Lowe MR, Butryn ML (2007) Hedonic hunger: a new dimension of appetite? Physiol Behav 91:432–439
38. Witt AA, Lowe MR (2014) Hedonic hunger and binge eating among women with eating disorders. Int J Eat Disord 47:273–280
39. Delparigi A, Chen K, Salbe AD, Reiman EM, Tataranni A (2005) Sensory experience of food and obesity: a positron emission tomography study of the brain regions affected by tasting a liquid meal after a prolonged fast. Neuroimage 24:436–443
40. Lee Y, Chong MFF, Liu JCJ, Libedinsky C, Gooley JJ, Chen S, Wu T, Tan V, Zhou M, Meaney MJ, Lee YS, Chee MWL (2013) Dietary disinhibition modulates neural valuation of food in ed and fasted states. Am J Clin Nutr 97(5):919–925
41. Schienle A, Scafer A, Hermann A, Vaitl D (2009) Binge-eating disorder: reward sensitivity and brain activation to images of food. Biol Psychiatry 65:654–661
42. Dowdeswell JA, White JWC (1995) Greenland ice core records and rapid climate change. Philos Trans Phys Sci Eng 352:359–371
43. Wisman JD, Capeheart HW (2012) Creative destruction, economic and security, stress, and epidemic obesity. In: Offer A, Pechey R, Ulijaszek SJ (eds) Insecurity, inequality and obesity. Oxford University Press, Oxford, pp 5–53
44. Ulijaszek SJ, Strickland SS (1993) Nutritional anthropology: prospects and perspectives in human nutrition. Smith-Gordon and Company Ltd, London
45. Ulijaszek SJ (1996) Energetics, adaptation, and adaptability. Am J Human Biol 8:169–182
46. Dunbar RIM (2012) Obesity: an evolutionary perspective. In: Offer A, Pechey R, Ulijaszek SJ (eds) Insecurity, inequality and obesity. Oxford University Press, Oxford, pp 5–53
47. Popkin BM (2009) The world is fat. The fads, trends, policies, and products that are fattening the human race. Avery, New York
48. Coveney JD (2000) Food, morals and meaning. The pleasure and anxiety of eating. Routledge, Abingdon
49. Lupton D (1996) Food, the body and the self. Sage Publications, London
50. Bellisle F, Dalix AM, Slama G (2004) Non food-related environmental stimuli induce increased meal intake in healthy women: comparison of television viewing versus listening to a recorded story in laboratory settings. Appetite 43:175–180
51. de Castro JM (1994) Family and friends produce greater social facilitation of food intake than other companions. Physiol Behav 56:445–455
52. Hetherington MM, Anderson AS, Norton GNM, Newson L (2006) Situational effects on meal intake: a comparison of eating alone and eating with others. Physiol Behav 88:498–505
53. de Garine I, Pollock N (eds) (1995) Social aspects of obesity and fatness. Gordon and Breach, New York

54. Maio GR, Haddock GG, Jarman HL (2007) Social psychological factors in tackling obesity. Obes Rev 8(Supplement 1):123–125
55. Ifland JR, Preuss HG, Marcus MT, Rourke KM, Taylor WC, Burau K, Jacobs WS, Kadish WM, Manso G (2009) Refined food addiction: a classic substance use disorder. Med Hypotheses 72:518–526
56. Reid R, Hackett AF (1999) Changes in nutritional status in adults over Christmas 1998. J Hum Nutr Diet 12:513–516
57. Hull HR, Radley D, Dinger MK, Fields DA (2006) The effect of the thanksgiving holiday on weight gain. Nutr J 5:29
58. Adam TC, Epel ES (2007) Stress, eating and the reward system. Physiol Behav 91:449–458
59. Lewis A, Jolly K, Adab P, Daley A, Farley A, Jebb S, Lycett D, Clarke S, Christian A, Jin J, Thompson B, Aveyard P (2013) A brief intervention for weight management in primary care: study protocol for a randomized controlled trial. Trials 14:393
60. Cavill N, Hillsdon M, Anstiss T (2011) Brief interventions for weight management. National Obesity Observatory, Oxford