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Emotions and Eating Behaviour: Implications for the Current Obesity Epidemic

ABSTRACT

Developed countries around the world are experiencing an epidemic of overeating and obesity with significant costs at a personal, familial, and societal level. While most research on obesity has focused on metabolic factors, this paper considers how emotional factors might contribute to this problem. Two examples we address are the use of food to modify negative mood states, also called emotional eating, and food intake as an addiction. Our central question is what makes some individuals prone to emotional eating and/or food addiction, while others are clearly less vulnerable in this regard. Ultimately, we suggest how obesity research, prevention, and treatment might address the emotional underpinnings of the current overeating epidemic.

Keywords: obesity, binge eating, comfort food, emotional regulation, addictive models of overeating

INTRODUCTION: CASE STUDY

One of Jane's poignant childhood memories is sneaking more food than was offered when her mother wasn't looking. 'I would ask my mom if I could have a cookie and then take three or four and hide them in a little spot under the couch.' When she got a bit older and had a part-time job, she spent all her money on bags of junk food. Jane was already obese at the age of twelve. By the time she was in her twenties she was consuming huge amounts of food, especially pizza, whenever she was home alone. Now she is twenty-eight and reports typical evening consumption: 'When it gets dark, I am ravenously hungry. In the winter, I typically eat my first dinner around six o'clock. Then, at eight-thirty or nine, I am hungry again and I will eat another whole other dinner. It's like I did not even eat that other meal.' Not only is this a very expensive activity but she has begun to worry about people coming over to her house because of all the food she keeps in her fridge and all the empty pizza boxes around her apartment.

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Jane has tried many diets but they never lasted long and they all failed. Efforts to restrict her intake seemed only to foster a kind of 'obsession' about food. Jane also reports that almost all negative emotions like frustration, aggravation, fatigue, and boredom will trigger a bout of overeating. When Jane was younger she loved to go out in the evening with friends, but now all she wants to do is stay home, watch television, and eat: 'I just like quiet; I like eating, so I have to be somewhere where I can eat a lot, which is home.'

In almost every way, Jane has a very emotional relationship with food. 'Eating for me starts off with thinking about how good the food will taste, but after that I'm just on autopilot. I feel great while I am eating. The minute it is gone, it is like taking a needle off a record when it is playing. Once there is no more, the enjoyment is gone. There is no lasting sugar rush of happiness. When it's gone I just feel very down.' Jane also talks about the comfort she gets from the rituals involved in eating. 'There are things that I eat in very specific ways. For example, when I eat a pizza, I take all the toppings off each piece and I eat the crusts first. I leave all the topping for the end and put them all on the last piece. I *always* do that. I don't like to order pizza in front of people, because that is how I like to eat it, and I know it looks weird!'

Jane has likened her inability to eat moderately to an addict who is hooked on drugs. 'I don't think I could ever stop gorging on food. If I talk to someone who wants to quit smoking, I want to say, "Well, just don't buy cigarettes anymore." But, if someone told me, "Just don't buy all this stuff," I would definitely have to say, "I can't stop what I'm doing. That is not a solution for me."' Jane sums up all her emotions about food by saying, 'I think I am just a hedonist and a glutton. I like things a lot. If I'm shopping and I see something I like, then I want several of them. I especially love food and sleeping in – all those sort of pleasurable things. In fact, I think I go to work just so I can eat a lot and sleep at night.'

OVERVIEW: EMOTIONS AND EATING BEHAVIOUR

Jane's experience with food, which enables her temporarily to suppress negative emotions on the one hand, while having an addictive quality on the other, is shared with tens of millions of people in developed countries around the world. As is the case for Jane, this emotional relationship with food from an early age brings with it important long-term consequences, most commonly obesity, which has significant costs at personal, familial, and societal levels (Hedley et al.; Wang et al.). For example, obesity is associated with a host of medical complications including diabetes, cardiovascular disease, and a shortened life

expectancy. Obesity is also associated with negative mental health outcomes that markedly decrease one's quality of life.

Unfortunately, despite significant efforts, the prevention and treatment of obesity across the lifespan have fallen well short of established goals. One possible explanation is that most work has focused on metabolic and hormonal factors such as insulin and leptin, while proportionally little research and prevention have looked at the role of eating behaviour on weight regulation over the lifespan. Indeed, increased consumption of high-caloric, highly palatable foods, as opposed to purely metabolic factors or decreased physical activity, may best explain the very rapid rise in obesity in recent decades (Blundell and Cooling; Bray). While Jane identifies several emotional triggers for her overeating, chances are that most people will tell her simply to 'exercise more and eat healthier foods.' But like all of us, Jane faces a rather daunting reality: it may also be that current strategies to prevent and treat obesity are simply unable to override the basic evolutionary processes that have protected humans from starvation over millions of years by making high-caloric foods a rich source of pleasure and reward (Mietus-Snyder and Lustig). Indeed, some forms of overeating and obesity are likely driven by an excessive motivational drive for food, similar to what occurs for addictive behaviours in general (Davis and Carter; Volkow and O'Brien).

The goal of this paper is to consider the role of emotions in eating behaviour and in the current obesity epidemic, focusing on two common phenomena described by Jane in her personal account. The first of these is *stress-related/emotional eating*, which describes the use of high-caloric, highly palatable foods to deal with negative emotions. As described by Jane, a variety of negative emotions, including frustration, aggravation, fatigue, and boredom, can all trigger a bout of overeating in vulnerable individuals. In some cases, anxiety and depression are the primary drivers. The second phenomenon is *overeating as a form of addiction*, as exemplified by Jane's comparison of her inability to eat moderately to the experience of an addict hooked on drugs. What makes some individuals prone to emotional eating and/or food addiction, while others are clearly less vulnerable in this regard, is a critical question that has particularly important implications for prevention and treatment. We consider this in terms of both nature and nurture, based on the assumption that both our genetic makeup and early life experiences play critical roles in emotional regulation and food reward. The possible role of insecure attachment experiences, and genetic variants tied to the brain's reward system, will be emphasized in this regard. We will end our discussion with a consideration of how obesity research, prevention, and treatment might be developed to address the emotional underpinnings of the current overeating epidemic.

STRESS, EMOTIONS, AND EATING BEHAVIOUR

The facts that emotions have a powerful influence on food choices and eating behaviour, and that eating behaviour can have a powerful effect on emotions, have been well known for many decades. Recent evidence suggests that such relationships can be quite complex and require careful consideration of mediating and moderating factors. One strategy to study appetitive behaviour and emotions is to perform laboratory-based studies of food intake following a mild stress. In one such study, Grunberg and Straub exposed healthy men and women to both stressful and positive experimental stimuli and then allowed them access to sweet, salty, and bland snack food. Results indicated a significant interaction between the sex of the subject and the stress manipulation on food intake: stress significantly decreased all food consumption in men but doubled the consumption of sweet food in women relative to the non-stressful condition. This suggests that the relationship between stress and eating depends on the sex of an individual and the type of food available (Grunberg and Straub). Oliver, Wardle, and Gibson studied anticipatory stress and food intake in a sample of healthy men and women. While no main effect of stress on food intake was found, stressed emotional eaters as assessed using the Dutch Eating Behaviour Questionnaire (DEBQ; Van Strien et al.) did consume more sweet, high-fat foods than did non-stressed and non-emotional eaters. Surprisingly, dietary restraint did not alter appetitive responses to stress in this study in contrast to other work (Cools, Schotte, and McNally; Herman et al.; Rutledge and Linden; Wardle et al.). These studies exemplify the importance of considering modifying effects when performing studies of this type.

Adolescence is a critical time for the development of weight gain and eating. It is also a time of significant stress and thus constitutes an important developmental window for early intervention and prevention. Nguyen-Rodriguez, Unger, and Spruijt-Metz looked at associations between a variety of negative emotions and emotional eating in a large sample of middle-school students of diverse backgrounds in Los Angeles County. They found that perceived stress was by far the strongest trigger for emotional eating in girls. While the proportion of boys reporting emotional eating was the same as for girls, none of the stress-related variables was associated with emotional eating in boys, who appeared to eat in response to non-specific emotional factors. Whether this reflects a fundamental difference in the emotional drivers of eating behaviour in the two genders, or simply less awareness of specific emotional states in boys, is a question for future research. It does suggest, however, that early intervention strategies, for girls in particular, need to address stress reduction, more adaptive coping mechanisms for a variety of anxiety states, and the promotion of positive mood wherever possible.

Mindfulness-based meditation to reduce stress in overeating populations have shown preliminary success in this regard (Proulx; Singh et al.).

While adolescence is an important period for the early prevention and treatment of obesity, it may be that the roots of emotional eating are established at much earlier stages of human development. Indeed, school-age children now represent a significant population at risk, not only for obesity, but for the chronic medical complications that emanate from it, such as type-2 diabetes mellitus. While the early origins of emotional eating are poorly understood at this time, several different mechanisms have been proposed. Some suggest that emotional eating emanates from basic reinforcement processes tied to the repeated experience of palatable food consumption during periods of distress (Gibson and Desmond; Mercer and Holder). Other evidence suggests that personality and temperament also play a role in this regard. For example, Markus et al. found that subjects with high neuroticism (a longstanding tendency to experience negative emotional states), but not their less anxious counterparts, were protected from depressed moods following a stressor if they ate carbohydrate-rich/low-protein meals. As this specific meal type is the most likely to increase brain serotonin levels (Wurtman; Wurtman and Wurtman), it may be that neurotic individuals are more prone to 'self-medicate' with food because of their increased sensitivity to brain serotonin levels and its effects on mood state. Another system that likely plays a role in stress-induced preference for sweet fatty foods is the endogenous opioid system, which is both critical for reinforcement and activated by highly palatable foods in response to stressful conditions (Mercer and Holder).

Dallman et al. have studied the effect of palatable foods on stress-system physiology. As highlighted by these authors, chronically depressed individuals who overeat tend to have low functioning of basic stress systems, including the hypothalamic-adrenal-pituitary (HPA-) axis and central corticotrophic-releasing factor (CRF), while other depressed individuals tend to have elevated activity in these stress systems. In animals, the ingestion of comfort foods decreases the expression of CRF in the hypothalamus. Dallman et al. propose that overeaters with depression or other chronically stressed states use comfort food to decrease activity in the stress-response network, with short-term benefits in decreased negative affect – a hallmark of emotional eating. Unfortunately there is a cost to this behavioural strategy in abdominal obesity, diabetes, and myriad medical problems that follow, such as cardiovascular disease and stroke.

One potential developmental approach to the study of emotional eating, stress reactivity, and obesity that has received surprisingly little attention is attachment theory. John Bowlby first proposed the existence of an innate attachment system, described as a set of behaviours intended to keep caregivers in close proximity, and triggered when an infant

perceives a threat or needs help (Bowlby, vols 1–3). Using careful observation of mother–infant dyads, Ainsworth et al. established three distinct patterns of attachment, labelled secure, insecure-ambivalent, and insecure-avoidant. Each of these is thought to be a potentially adaptive strategy depending on the circumstances and particular mother–infant pairing. *Security* in attachment develops from early relationships with nurturing adults who are sensitive and responsive to signals of distress. In response to brief separation from caregivers, *securely* attached infants are mildly distressed during the caregivers' absence, seek contact at reunion, and are easily soothed, promoting a rapid decrease in distress. *Insecure-ambivalent* infants also show separation distress and seek contact upon reunion, but are more irritable and difficult to soothe. This is thought to emanate from a parenting style that is unreliably sensitive to the infant's emotional state. *Insecure-avoidant* infants show the opposite pattern, with minimal crying during separation and avoidance of contact following reunion. Avoidant attachment is thought to evolve from early interactions with caregivers who are consistently unavailable and unresponsive. Main and Solomon added a fourth attachment classification labelled *disorganized*, which represents the collapse of any organized attachment strategy in the face of an acute challenge.

Attachment theory may be particularly helpful in understanding stress and emotional eating in that the bond that develops between child and caregiver plays a critical longer-term role in emotional development. This is established through 'internal working models' – a set of internalized scripts containing beliefs and expectations about whether caregivers are responsive and caring, and whether the self is perceived to be worthy of care and attention. Bowlby's theory emphasized the fundamental role of the attachment system in the regulation of emotions and the establishment of self-esteem (vol 1). Maunder and Hunter have suggested that various behaviours, including overeating, can take on the function of regulating affect when more adaptive, attachment–based strategies are not available. One psychiatric phenotype that demonstrates this well is atypical depression, a disorder characterized by mood dysregulation and neuroticism (Chopra et al.), as well as food cravings and emotional eating (Gibson). Levitan, Atkinson, et al. have recently demonstrated that depressed patients with atypical features have particularly high levels of insecure and anxious attachment. Taking these findings into consideration, the notion that atypically depressed patients may consume palatable foods to compensate for an insecure attachment system merits consideration in future work and as a contributor to obesity in many cases. If particular early attachment styles are found to underlie emotional eating in future work, focused attachment-based psychotherapies that address these vulnerabilities in adults or adolescents may gain prominence in the obesity field. Even more interesting is the possibility of

addressing and reversing putative obesogenic attachment disturbances in early childhood. In support of this approach, toddler–parent attachment-based psychotherapy has proven successful in reorganizing maladaptive attachment styles in high-risk children (Toth et al.).

ANOTHER VIEW OF COMFORT FOODS: PROMOTING POSITIVE AFFECTS

While most work on comfort foods and emotions has been based on a model of negative reinforcement – using palatable foods to decrease negative emotions – Dubé, LeBel, and Lu point out that some individuals consume foods in order to induce positive emotions. This distinction is important, as positive and negative affects are mediated by distinct neuro-behavioural mechanisms (Davidson). Dubé, LeBel, and Lu use the term *affect asymmetry* to describe this important distinction and have examined demographic and dietary factors underlying it. For example, men may be more likely to eat comfort foods to maintain or enhance *positive* emotions, whereas emotional eating in women tends to be driven by *negative* mood states. Several factors might contribute to this gender difference. It might simply be that men are less conscious of negative emotions in general, or use other strategies to deal with them (Dubé, LeBel, and Lu). It might also be that the ability of comfort foods to impinge on biological systems that control moods is different in the two sexes. The serotonin system, which plays a major role in eating behaviour and mood, is known to be strongly influenced by sex hormonal effects (Maki and Dumas). The possibility that different types of foods are consumed to achieve negative vs. positive reinforcement also merits consideration.

ADDICTIVE MODELS OF OVEREATING AND OBESITY

As noted above, while consumption of high-caloric, highly palatable foods can develop as a behavioural strategy to eliminate negative mood states, particularly under stressful conditions, a second factor that plays a major role in overeating is the hedonic quality of many foods. The combination of a biology developed over millions of years to prevent starvation, and the widespread availability of fatty and sugary foods designed to induce short-term reward, is a potent combination that promotes overeating and obesity in a large proportion of the population.

The notion that high intake of sweet and fatty foods is a form of addictive behaviour has been considered in several recent reviews (e.g., Davis and Carter; Mietus-Snyder and Lustig; Volkow and O'Brien). Indeed, recent evidence suggests that brain reward circuits can be activated with natural rewards like food, just as with drugs of abuse (Grigson; Holden; Volkow and Wise). The evidence of a common substrate

mediating the rewarding properties of addictive drugs *and* natural rewards supports the argument that highly palatable food can be addictive, can be used for mood altering or emotionally driven purposes even in excess of basic energy requirements, and can have adverse consequences similar to those associated with drug abuse (Kelley et al.). If food is conceptualized as a 'drug,' then it follows that food, like other addictive substances, may be used for comfort or to improve mood. Indeed, there is considerable evidence that many people 'self-medicate' with food when depressed or anxious (Arnow, Kenardy, and Agras). A substantial body of research shows that palatable foods can produce significant analgesic effects (Pelchat). Also supporting the perspective that food can be a substance of abuse is evidence that excessive use of high-caloric foods can perturb the brain in a manner similar to addictive drugs (e.g., Di Chiara et al.; Schroeder, Binzak, and Kelley; Wise, 'Drug-Activation'). Thus, excessive exposure to natural rewards can induce behavioural responses that mimic those seen in the 'addiction' responses to drugs –down-regulation or sensitization of the reward system and withdrawal (Grigson). For example, repeated and high intake of sugar causes behavioural and neurochemical signs of withdrawal in rats when availability is restricted (Colantuoni et al.). The fact that binge-eating is often triggered by the ingestion of small amounts of a highly palatable food parallels the 'priming' effect of drugs in addicts whereby the initial ingestion of the drug tends to elicit a strong 'craving' or compulsion for further use. Finally, abstinent drug addicts show a heightened preference for, and intake of, sweet foods (Carr; Pelchat).

DOPAMINE-MEDIATED REWARD AND ITS ROLE IN FEEDING

Brain dopamine (DA) activity is implicated in motivated behaviours such as feeding (Barsh and Schwartz; Schultz; Wise, 'Brain Reward Circuitry'). The major system implicated in reward is the mesocorticolimbic pathway, which includes dopamine cell bodies in the ventral tegmental area (VTA) and projections to both the striatum (nucleus accumbens [NAc]) and neocortex (including the pre-frontal cortex). Most natural incentives like food reach the central circuitry of motivation via the five senses, while pharmacologic rewards, like drug injections of cocaine or heroin, activate the same mesolimbic circuitry, but more directly and with more dramatic effect (for reviews, see Leshner and Koob; Noble).

VTA–NAc circuits appear crucial in promoting the intake of palatable foods as well as regulating the duration of meal consumption (Meguid et al.; Palmiter; Saper, Chou, and Elmquist; Shimura, Kamada, and Yamamoto). Sweet and fatty foods potentiate the release of DA, induce more pleasurable subjective feelings, and therefore are more rewarding (e.g., Grigson; Martel and Fantino). Almost any mammal, including

humans, will eat beyond its homeostatic caloric needs if presented with highly palatable food (Saper, Chou, and Elmquist).

Neuroimaging studies strongly support a role for central dopamine in food reward processes. Studies in humans reveal that

- food-related cues significantly activate areas of the brain associated with the processing of information related to the pleasurable features of stimuli (the brain reward system), such as the VTA and substantia nigra, amygdala, and orbitoprefrontal cortex (Killgore et al.; Rothemund et al.), which are areas either involved in the synthesis and release of dopamine or are targets for dopamine projections,
- activity in the amygdala and orbitoprefrontal cortex is proportional to the subjective pleasure associated with food (Beaver et al.),
- ghrelin, a hormone known to stimulate food intake, increases the response of these brain regions to food-related stimuli (Malik et al.),
- leptin, an adipose-derived hormone that produces satiation at higher levels, is associated with a decrease in the response of the NAc to food cues (Farooqi et al.); this area is a primary target for dopamine innervation and is highly sensitive to the rewarding properties of pleasurable stimuli (Menon and Levitin), and
- obese individuals show greater responses in the orbitoprefrontal cortex to food-related stimuli and report more cravings (Rothemund et al.).

DOPAMINE SYSTEM GENES, FEEDING BEHAVIOUR, AND OBESITY

It is well established that genetic factors play a major role in obesity over the lifespan. While much initial work on the genetics of obesity was focused on metabolic processes, the notion that genes might cause obesity through their influence on eating behaviour has also gained interest in recent years. Indeed, genetic variation is one potential reason why only some individuals are susceptible to overeating and obesity in the current obesogenic environment. The most popular methodology to study genetic effects on various behaviours including overeating is to perform a *genetic association study* using a *candidate gene approach*. Using this methodology, one first establishes a single hypothesis or set of hypotheses that link a particular behaviour or outcome to a particular biological system. For example, on the basis of the sections reviewed above, it is very reasonable to hypothesize that variation in dopamine system genes plays a role in shaping one's sensitivity to the rewarding aspects of food. The next step is to identify genetic variants in the relevant system that are well suited to an association approach. Variants that are reasonably common and have known functionality are particularly helpful in this regard. For example, the dopamine-4 receptor gene

(DRD4) has a particular variant called the '7-repeat' variant that is associated with decreased sensitivity to dopamine. If individuals who carry this 7-repeat variant also show an increased risk for overeating, this constitutes a *positive association* between this gene variant and this outcome.

Indeed, several associations between dopamine system genes and various aspects of overeating and weight gain are now emerging. One example of this is the dopamine-4 receptor gene (DRD4) described above. Among women with seasonal affective disorder who usually experience both carbohydrate craving and weight gain during winter depressive episodes, the 7-repeat allele of DRD4 is associated with dysphoria, binge eating, and obesity (Levitan, Masellis, Basile et al.; Levitan, Masellis, Lam, Muglia, et al.; Levitan, Masellis, Lam, Kaplan et al.). The 7-repeat/obesity link has also been replicated in a separate cohort of women with bulimia nervosa, another group of overeaters with high rates of mood disturbance (Kaplan et al.). These studies suggest that carriers of the 7-repeat allele may increase consumption of highly palatable foods to compensate for low mood mediated by a sluggish dopamine system. Sobik, Hutchinson, and Craighead further described a role for DRD4 variants in cue-elicited food craving.

Individuals who carry the TaqA1 variant of the ANKK1 gene, which is in close proximity to the dopamine-2 receptor gene (DRD2), have a thirty to forty per cent reduction in D2 receptor density in the striatal region of the brain, an area known to play a major role in brain reward processes (Jönsson et al.). The TaqA1 variant associates with a reduced sensitivity to reward (Kirsch et al.). Furthermore, food reinforcement is greater in obese than in non-obese individuals, particularly if they also carry the TaqI A1 variant (Epstein, Temple et al.). This suggests that the DRD2 genotype interacts with food reinforcement processes to influence energy intake. Likewise, subjects with either binge-eating disorder or obesity report higher reward sensitivity than do normal weight controls, but only if they carry the Taq A1 allele (Davis, Levitan, Kaplan, Carter, Reid, Curtis, Patte, Hwang, and Kennedy). Similar associations between other dopamine system genes and various aspects of eating behaviour and obesity have also been found (e.g., Davis, Levitan, Kaplan, Carter, Reid, Curtis, Patte, Hwang, and Kennedy; Figlewicz, Patterson, Johnson et al.; Müller and Kennedy; Shinohara et al.; Stice et al.; Thiesen et al.).

Taken together, these studies provide significant support for the general working hypothesis that dopamine system genes play an important role in feeding behaviour and obesity. It should be noted that while most of these studies are consistent with a model of increased eating mediated by a hypofunctioning central dopamine system, other studies link *hyperactivity* of the dopamine system with manifestations of increased eating (Davis, Levitan, Kaplan, Carter, Reid, Curtis, Patte, and Kennedy; Franken and Muris; Loxton and Dawe). It may be that both *decreased* and *increased* activity of

brain dopamine can confer sensitivity to the rewarding aspects of food, albeit in different individuals. In either case, identification of genetic variants that confer risk for overeating and obesity has both practical and theoretical relevance. At a practical level, such genetic association studies suggest one means of identifying high-risk individuals most likely to benefit from early prevention. At a theoretical level, such studies can greatly improve our understanding of the basic vulnerabilities contributing to the obesity epidemic in the population at large.

SUMMARY AND FUTURE DIRECTIONS

Despite significant efforts, the obesity epidemic continues largely unabated, with profound negative implications at personal, familial, and societal levels. As reviewed above, there is substantial evidence that emotional regulation plays a critical role in food consumption. Of particular concern is the fact that high-caloric, highly palatable foods, which are the most problematic in terms of weight gain and obesity, also have the strongest effect on alleviating negative mood states in most contexts. They also have the strongest effect on brain systems mediating addictive behaviours. Several factors moderate these effects, including gender, personality and temperament, restrained eating, and genetics.

On the basis of the above arguments, it may be that addressing the obesity epidemic with a greater focus on emotional processes will be necessary if significant progress is to be made. Given that overeating and obesity are becoming increasingly common in school-age children, understanding the earliest origins of emotional eating and developing early intervention strategies based on this understanding, are priorities in this regard. Attachment theory, which focuses on normal and abnormal emotional regulation across the lifespan that may be modifiable early in life, is one new approach to consider.

Several lines of research suggest that perceived stress and anxiety are strong contributors to emotional eating and must be addressed if long-term improvements in eating behaviour are to occur. Newer models of overeating based on addiction science are also of great interest and may point the way to other highly novel treatment and prevention strategies. Ultimately, several factors are likely to contribute to emotional eating in the population at large, suggesting that multiple treatment approaches used in combination may be necessary for major progress to occur.

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