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Research review

Compulsive overeating as an addiction disorder. A review of theory and evidence

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ABSTRACT

In this paper we argue that compulsive overeating has compelling similarities to conventional drug addiction. Our case is based on their comparable clinical features, the biological mechanisms they have in common, and on evidence that the two disorders have a shared diathesis. In making the argument for overeating as an addictive behaviour, it is clearly not appropriate to include all cases of excessive food consumption in this taxon. Nor are we claiming that obesity and addiction are one and the same. However, it is proposed that Binge Eating Disorder (BED) is a phenotype particularly well-suited to such a conceptualization, and that sound clinical and scientific evidence exists to support this viewpoint. We have provided some recommendations for treatment modifications that recognize the similarities between treating drug dependence and compulsive overeating.

Contents

Our aim in writing this paper is to propose, with supporting theoretical and empirical evidence, that compulsive overeating is sufficiently similar to conventional substance dependence to justify its inclusion as an addiction disorder. It is also our belief that researchers and clinicians would profit from considering this perspective when investigating the causes of excessive consumption, when trying to understand its phenomenology, and when considering the factors that increase individual vulnerability. In presenting our case, we will consider clinical and behavioural parallels between food and drug abuse, and the similarities in their biological underpinnings. We will also discuss overlapping factors in their risk of occurrence. Finally, we will briefly consider the implications of an addiction perspective for therapeutic outcomes.

History and background

Although people had written about the enslaving properties of opium and alcohol for centuries, it was not until the 1800s that the

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notion of drug abuse as a disease entity – rather than an issue of moral culpability – entered the general parlance of medical professionals (see London, 2005). The original (17th century) use of the word *addicted* meant "to give over...to someone or some practice", however, its first appearance, with specific reference to narcotics, only occurred in the early 20th century (Berridge & Mars, 2004), and for most of that time was largely confined to the misuse of alcohol and the opiates. With the rise in popularity of psychiatry after World War II, and the 'rediscovery of addiction' (Berridge, 1997), other substances such as cocaine, amphetamine, and nicotine were added to the list of addictive drugs.[1]

In recent years, there has been an interesting clinical and scientific shift in perspective with many believing that addiction should encompass the compulsive engagement in activities such as gaming, Internet use, and shopping, in addition to its conventional relation with pharmacologic rewards (Grant, Brewer, & Potenza, 2006; Holden, 2001; Orford, 2001a, 2001b). Current debate has even extended to the possibility that so-called 'behavioural addictions' should include the abuse of *natural rewards* – that is, behaviours that are intrinsically necessary for our survival, and in which we freely engage with pleasure and without social sanction (Davis & Claridge, 1998; Volkow & Wise, 2005).

A few generations ago, it might have seemed heretical to suggest that food could be an addictive substance, and overeating an addictive behaviour. As testament, we were able to find only six published references to such a viewpoint from 1950 to 1970, almost all written by Randolph (e.g. 1956), the well-known founder of environmental medicine. However, a groundswell of change in perspective has occurred in the past few years as indicated, for example, by the publication of 15 academic papers, in the first two months of 2008 alone, referring to 'food addiction'.[2]

'Drugs as Food'

Conventional evolutionary 'mismatch' views of addiction propose that substance use is a relatively recent phenomenon in the history of our species, and occurs largely because of the availability of purified and synthetic drugs, and their direct routes of administration (Nesse, 1994; Smith & Tasnadi, 2007). In other words, it is the ubiquity and concentrated doses of these substances that have contributed to wide-spread human drug abuse. By contrast, Sullivan and Hagen (2002) have argued that human beings shared a co-evolutionary relationship with psychotropic plant substances in prehistory for millennia. Indeed, they frequently ate them as food because their ingestion solved a recurrent problem faced by our ancestors.

The neurotransmitters essential for normal human functioning – and most implicated in substance use – are dopamine and serotonin whose precursors must be provisioned externally from high quality nutrients like protein. During most of our history, due to famines and seasonal food shortages, these precursors were nutritionally constrained, and consequently people experienced neurotransmitter deficits with considerable regularity. Depletions of this sort tend to adversely affect critical behaviours and emotions including motor activities, cognitive abilities, and mood.

It is also generally believed that over our evolutionary history, certain plants such as Erythroxylum (coca) developed chemical defenses against mammalian predators by producing 'neurotransmitter substitutes', which had toxic effects when ingested. Sullivan and Hagen (2002) argue that in response to this threat, "behaviourally sophisticated hominids" evolved to counter-exploit the potential benefits of plant toxins. For example, because these "neurotransmitter analogues" imparted energy, prevented fatigue, diminished appetite, and increased tolerance for hunger, they helped to avoid the maladaptive "behavioural sequelae of stress" in the absence of adequate food sources. In other words, plant substances served the dual purpose of substituting for more costly energy, and buffering against the biological ravages of prolonged stress.

As history progressed, human beings developed the ability to purify psychoactive plant substances and use routes of administration that were more direct than ingestion. These developments created a more potent stimulation of brain mechanisms that regulate pleasure, and have contributed to the propensity to overuse and abuse these substances in contemporary times (Nesse, 1994). And so it seems reasonable and sensible to conclude that organic compounds in our environment can only be categorized as *beneficial* or *harmful* when we take account of 'dosage' and the relevant characteristics of those who partake of them (Gerber, Williams, & Gray, 1999).

'Food as Drugs'

This last point is particularly relevant when considering the typical macronutrient intake in current Western societies. Diets high in concentrated fats and sugars are not only dense in calories, but metabolically efficient because a large proportion of their energy is provided to the consumer. They also tend to elevate mood by releasing neuropeptides which reinforce their selective preference (Kelley, Baldo, Pratt, & Will, 2005). Evolutionary biologists believe that 'cravings' for sugar and fat evolved to enhance human energy intake in unpredictable nutritional environments, which were universally the norm until relatively recently (Gerber et al., 1999). However, in the quantities that many people ingest them today, they have an abuse potential rivaling popular addictive drugs (Spring et al., 2008).

The food industry has become especially savvy in exploiting our natural human desire for sugar and fat by increasing many-fold their 'dose' in much of our daily foods. For instance, there was a 42% *per capita* increase in the consumption of added fats and a 162% increase in cheese relative to only a 20% increase in fruits and vegetables between 1970 and 2000 (Frazao & Allshore, 2003). The sharply reduced cost of sugar and vegetable oils worldwide has greatly contributed to the production of highly palatable processed foods (Drewnowski, 2000). The incidence of snacking, especially in the form of carbohydrates, has also increased over the past 25 years, in tandem with the increase in daily energy intake (Sebastian, Cleveland, & Goldman, 2008).

In the case of conventional drugs, greater potency tends to increase their addictive potential. Directly parallel to the notion of 'drug dosage' is the size of the meals we are presently served. Wansink and Van Ittersum (2007) describe the "portion-distorted embarrassment of food" in today's supermarkets and restaurants. To illustrate, a fast-food restaurant meal – a burger, fries, soft drink, and dessert – can provide almost all of one's daily caloric requirements in a single serving. Moreover, the annual growth rate of 'fast food' dining has increased 3-fold in the past generation compared to that of at-home consumption (Richards, Patterson, & Hamilton, 2007). The size of plates, bowls, and glasses in our homes has also steadily increased over the years, and the serving size of some entrees has virtually doubled in recipe books since the 1930s (Wansink & Van Ittersum, 2007).

In summary, just as different drugs promote different degrees of dependence, foods also differ in their capacity to promote abuse (Volkow & Wise, 2005). Experts are now confident in claiming that the nutrients comprising fast foods are inherently addictive because of their concentration and high volume of fats and sugars.

[1] Although the *Diagnostic and Statistical Manual for Mental Disorders* IV [DSM-IV] (American Psychiatric Association, 1994) nomenclature does not include the word "addiction", its "dependence" terminology is closest to this condition.

[2] This data base search was carried out using Web of Science.

And, like drugs of abuse, they have the ability to alter brain mechanisms in ways that contribute to their increasingly compulsive use (see Grigson, 2002; Del Parigi, Chen, Salbe, Reimna, & Tataranni, 2003; Spring et al., 2008).

Clinical and behavioural parallels

In making the argument for overeating as an addictive behaviour, it is clearly not appropriate to include all cases of excessive food consumption in this taxon. For some individuals, overeating is a relatively *passive* event that occurs almost without awareness, in the form of liberal snacking and large portion sizes (Drewnowski & Darmon, 2005). For others, however, it can be *compulsive* and excessively driven. We believe that Binge Eating Disorder (BED) is a phenotype particularly well-suited to an addiction conceptualization, and that sound clinical and scientific evidence exists to support this viewpoint. Cassin and von Ranson (2007) found, for example, that 94% of their adult BED sample described themselves as "food addicts" or "compulsive overeaters" and met criteria for substance-dependence disorder in DSM-IV when the term "substance" referred to "binge eating".

While there are strong links between BED and obesity (Bean, Stewart, & Olbrisch, 2008), we are also not claiming that obesity and addiction are one and the same. In a recent study we found that individuals with BED are a specific subtype of obesity and that their proneness to binge eating may be influenced by a biologically based hyper-reactivity to the hedonic properties of food, coupled with an enhanced motivation to engage in appetitive behaviours (Davis et al., in press). This predisposition can be easily exploited in our current environment with its highly visible and easily accessible surfeit of sweet and fatty things to eat.

Finally, it is important to acknowledge that binge eating is also a key diagnostic criterion of bulimia nervosa (BN), and that arguments have previously been made for the addictive nature of this disorder (e.g. Davis & Claridge, 1998; Goodman, 2008; Orford, 2001a, 2001b). However, it is our view that because BN is typically followed, and/or preceded, by severe compensatory behaviours such as vomiting or excessive physical activity, there may be differences in the triggers for, and the phenomenology of, the binge eating in these two disorders (e.g. Schienle, Schafer, Hermann, & Vaitl, 2009). A discussion of these issues is beyond the scope of the present review.

Loss of control

While there is not complete consensus on the common elements of all addictions, most would agree that a core set of defining characteristics is necessary for inclusion. Perhaps the most obvious feature is the increasingly compulsive use and abuse of the addictive behaviour, even in the face of detrimental consequences to health, safety, social relationships, and financial stability. BED is characterized by repetitive and aboulic episodes of overeating, not typically driven by hunger nor followed by any compensatory behaviours such as purging, fasting, or excessive exercising. It also appears to be a chronic and stable condition (Pope et al., 2006) with strong links to obesity (see Devlin, 2007). Although initially believed to be a disorder of adulthood, there is a growing awareness that BED also occurs in children and adolescents (Bulik, Brownley, & Shapiro, 2007). In addition to clinical research, there are good experimental paradigms whereby rats fed an intermittent diet of sugar develop a pattern of copious consumption resembling human cases of binge eating (e.g. Avena & Hoebel, 2003).

BED sufferers typically report distress and guilt about their eating habits, and have great difficulty controlling these behaviours despite unhealthy weight gain and ensuing medical problems such as diabetes and hypertension (Colles, Dixon, & O'Brien, 2008). With ubiquitous warnings from the medical profession and public health educators, it is fair to assume that members of our society are generally informed and relatively knowledgeable about the negative consequences of poor nutrition and obesity, and are sentient of dietary recommendations for good health (see Kessler, 1995). We can therefore conclude with some confidence that binge eating exists despite an awareness of its poor-health outcomes.

Tolerance and withdrawal

In the most general sense, tolerance occurs when a stimulus of a particular magnitude elicits an increasingly diminished response with each repeated exposure. This phenomenon is a key characteristic of all drug addictions – and one of the factors that fosters the escalation of intake. Animal studies have demonstrated that a sugar-enhanced diet increases daily food intake over time (Avena, Rada, & Hoebel, 2008). While this evidence is indirect, it has considerable relevance to BED since binge foods are typically high in sugar content (Yanovski, 2003). Direct evidence of tolerance in the human condition arises largely from anecdotal clinical reports of individuals consuming more food in each binge as the disorder becomes more chronic. The finding that higher body weight correlates with the frequency and severity of binge eating episodes also provides indirect evidence of tolerance effects (Picot & Lilenfeld, 2003). As well, a high proportion of BED adults reported being overweight before the onset of their disordered eating, suggesting that over time high calorie diets prompt greater intake and may contribute to binge eating (Reas & Grilo, 2007).

The impact of tolerance on the progression of addictive behaviours is made more poignant by its synergy with the debilitating symptoms of withdrawal. Certain foods – particularly sugar – can cause pronounced withdrawal symptoms when removed from the diet, and these effects most clearly resemble the physicals signs of distress seen in opiate withdrawal (Avena et al., 2008). The most compelling evidence comes from animal research in which rats are initially maintained on a 25% glucose solution (e.g. Avena et al., 2008; Wideman, Nadzam, & Murphy, 2005). Following its removal, they show aggression, anxiety, a drop in body temperature, teeth chattering, forepaw tremor, and head-shaking – all symptoms associated with withdrawal from drugs like heroin. While there is also human evidence of 'sugar withdrawal', it comes mostly from clinical observation, self-help books, and Internet websites (e.g. http://www.wisegeek.com/what-are-symptoms-of-sugar-withdrawal.htm). They are, however, uniform in describing headaches, irritability, and flu-like symptoms among heavy sugar consumers who become abstinent. Nevertheless, it is important to emphasize that this information is largely anecdotal and has not been subjected to the scrutiny of controlled scientific investigation.

Cravings and relapse

One of the most distinguishing features of drug abuse is the pronounced sense of craving reported by addicts, and their dismal and repeated failures at giving up the habit. Addiction is rarely an acute illness. A decade ago, Leshner (1997) coined the term "chronic relapsing disorder" to describe these disorders because total and permanent abstinence seldom occurs after a single treatment episode. For most individuals, there are repeated cycles of cessation and relapse. Human weight cycling is, almost by definition, a sign of repeated defeat in one's effort to curb overeating, and is found to be a significant risk factor for binge eating (Petroni et al., 2007).

The addict's powerful cravings that can be elicited from even a small 'dose' – as well as from the many conditioned environmental cues – are thought to contribute to poor long-term treatment outcome. Studies have also demonstrated that food cravings are significantly higher in adults with BED than in their non-bingeing counterparts of comparable weight (Davis et al., in press; Mussell et al., 1996). This meshes with other evidence that those with BED show enhanced preference for sweet and fatty foods compared to other obese individuals (Yanovski, 2003). Craving in animals can only be inferred from their behaviour, and is typically defined as the enhanced motivation to procure an addictive drug by operant responding (Koob & Le Moal, 2005). Such behaviour has also been observed in rats who lever pressed for 23% more glucose than at baseline, after a 4-week period of a sugar-enhanced diet (Avena et al., 2008).

Neurobiological parallels

Brain reward circuitry almost certainly evolved to foster our selective engagement in activities like eating, sex, and maternal behaviour, which are the essence of our survival as a species. This otherwise highly adaptive neuro-anatomical mechanism is also at the heart of all dependence disorders. Abused substances have psychomotor stimulant properties, which activate the same brain reward pathways as life's natural pleasures. In other words, there is a shared substrate for food and drug reward (Pelchat, 2002). Due, however, to the potency of most addictive substances, and to their direct route of administration, the claim that drugs "hijack" the brain has become a popular idiom to describe the downwardly spiralling pattern of drug abuse.

Dopamine neurons in the ventral tegmental area (VTA) send subcortical projections to striatal regions of the basal ganglia – importantly, the nucleus accumbens – and to various limbic structures such as the amygdala. VTA projections also extend to the prefrontal cortex. This mesocorticolimbic neural network is fundamentally complex and cleverly 'designed' to regulate the many emotional, motivational, and cognitive processes involved in reward. For example, our engagement in these behaviours increases our sense of pleasure and well-being – events that galvanize our attention in preference to more neutral, and less essential, activities. Secondly, we have the desire to repeat these behaviours even in the face of distracting stimuli. They also nurture a strong positive memory, which increases their salience and enhances our appetitive motivation in their direction. Thirdly, we quickly learn the cues in our environment which signal the approach or availability of these rewarding behaviours. And finally, we become resistant (temporarily) to their rewarding properties in order to move on to other activities.

The exact role of dopamine in the various aspects of reward has been vigorously debated in recent years. Some, like Berridge (2007) have argued convincingly that mesocorticolimbic dopamine fosters the motivation to engage in rewarding behaviours – what he calls the 'wanting' of reward – more than the 'liking' of reward. In other words, dopamine may not be *central* to the normal pleasure or hedonic reactions to food or drugs. However, this position is still moot and not easily resolved since reward involves a myriad of emotions including anticipation, expectation, pleasure, and memory that are difficult to separate experimentally.

Other neurotransmitter systems such as GABA, the opioids, and serotonin are also integral to the reward process (Wang, Volkow, Thanos, & Fowler, 2004). With respect to food, for instance, dopamine release in the nucleus accumbens is generally associated with its reinforcing effects, while opioid signalling in this area regulates its palatability and hedonic properties (Cota, Tschop, Horwath, & Levine, 2006; Esch & Stefano, 2004).

Until relatively recently, the neurobiology of overeating was largely focused on the hypothalamus, while drug addiction research, by contrast, channelled its attention to the mesocorticolimbic pathways (Trinko, Sears, Guarnieri, & DiLeone, 2007). With the increasing sophistication of brain imaging techniques we are now able to witness the activation of brain reward sites in response to palatable food (Beaver et al., 2006; Small, Zatore, Dagher, Evans, & Jones-Gotman, 2001). Prefrontal systems also play a prominent role in eating and appetite. For instance, increased "dysexecutive" traits have been associated with binge eating and food cravings as they are with drug abuse (Spinella & Lyke, 2004).

Experts are now generally agreed that the reinforcing effects of addictive drugs and palatable foods are regulated, in large part, by the same dopamine pathways (Corwin, 2006; Kelley et al., 2005; Wang et al., 2004). While addictive drugs share with food the property of increasing dopamine in brain reward pathways, they also bypass the adaptive mechanisms of normal reward, such as the habituation that constrains the responsiveness of the brain to food (Di Chiara & Bassareo, 2007). Potent drugs abnormally facilitate Pavlovian incentive learning to drug-conditioned stimuli, and sensitize the individual to cravings for the substance (Koob, 2006). Drugs also cause a tolerance to their rewarding properties by the downregulation of dopamine receptors in the striatum (Volkow, Fowler, & Wang, 2002), and a decrease in the function of the extended amygdala reward system, which produces the negative affect and anxiety associated with abstinence (Koob, 2006). There is now compelling evidence that highly palatable foods eaten in abundance have the potential to cause these same neuroadaptations – alterations which increase compulsive use, foster strong cravings, and contribute to the symptoms of withdrawal (Grigson, 2002; Hajnal, Margas, & Covasa, 2008; Pelchat, 2002). In other words, excessive exposure to natural rewards can induce biobehavioural responses that mimic those seen in the 'addiction' responses to drugs – viz. downregulation, sensitization, and withdrawal (Grigson, 2002). The chronic overconsumption of highly palatable foods that is characteristic of BED, makes it reasonable, therefore, to conclude that such neuroadaptations are likely to occur in individuals with this condition.

Risk-factor similarities

A recent study found significantly higher rates of psychopathology, including substance abuse, in 1st degree relatives of women with BED compared to relatives of control women (Lilenfeld, Ringham, Kalarchian, & Marcus, 2008). Importantly, however, all the disorders elevated in BED relatives followed a pattern of independent transmission from BED, except substance use disorder, whose transmission pattern indicated a shared etiology.[3] While the vulnerability for both disorders has many social and cultural parallels – such as availability and cost contributing to their consumption – this review only targets the prominent *psychobiological risks* they have in common.

Reward sensitivity

The sensitivity or reactivity of the 'common reward pathway' is affected by several biological factors such as the density of dopamine receptors, the amount of dopamine released into the synapse, and the rapidity of its transport back into the cell by the

[3] While these findings seem discordant with previous family studies (e.g. Schuckit et al., 1996; von Ranson, McGue, & Iacono, 2003), these earlier reports either included a broad range of eating disorders, or only focused on anorexia nervosa and bulimia nervosa. Moreover, the study by Schuckit and colleagues only considered alcoholism.

re-uptake protein. Individual differences in *reward sensitivity* have been strongly implicated in the risk for drug addictions (Bowirrat & Oscar-Berman, 2005; Kreek, Nielson, Butelman, & LaForge, 2005; Loxton & Dawe, 2006; Nader & Czoty, 2005) as well as compulsive overeating (Davis & Woodside, 2002; Davis et al., 2008). The research is divided, however, about the causal direction of this association.

One argument favours the view that *hypo*-dopaminergic functioning – what has been called a *Reward Deficiency Syndrome* (RDS) – is a key factor in the development of addiction disorders (Blum et al., 2000; Jimenez-Arriero et al., 2006). The premise is that substances (like addictive drugs and palatable food) are used as a form of 'self-medication' to boost a sluggish dopamine system and increase hedonic capacity. The counter argument is that *hyper*-sensitivity to reward contributes to increased risk for addictive behaviours due to an enhanced motivation to engage in pleasurable activities. For instance, in several studies, heightened reward sensitivity was associated with emotional overeating, preference for high fat food, binge eating, and food cravings, as well as with hazardous alcohol consumption (Davis et al., 2007; Franken & Muris, 2005; Loxton & Dawe, 2006). One explanation for the apparent disaccord between the two bodies of research may be a *dual vulnerability* to addictions whereby both paths can confer risk, albeit in different individuals and perhaps with different levels of severity.

Impulsivity and decision-making deficits

Poor decision-making is a core symptom of certain mental health problems – but is most prominently seen in drug dependence (Bechara & Damasio, 2002; Bechara & Martin, 2004). Addicts tend to choose actions that bring immediate reward, even when this leads to a deleterious outcome at some later date. The human ability to choose *present* options, which favourably influence *future* outcomes, depends crucially on an accumulated 'emotional memory' of the consequences of our *past* interactions with similar events (Damasio, 1994). In other words, we form a probabilistic impression of how a particular action will turn out in the future from an emotionally biasing 'gut' feeling, which was generated when that action caused either a positive or a negative reaction in the past. The orbitofrontal cortex, in particular, is critical for activating feelings or emotional states from "thoughts" about rewarding or punishing events that are not currently present in our environment (Bechara, 2004; Bechara, Damasio, & Damasio, 2003).

Much of the early research on decision-making came from studying the social impairments of patients with ventromedial prefrontal cortical (VMPFC) lesions, and the observation that their behavioural deficits are typically caused by an inability to advantageously assess future consequences (Anderson, Bechara, Tranel, Damasio, & Damasio, 1994; Sanfey, Hastie, Colvin, & Grafman, 2003). A plethora of research using neuropsychological tests of decision-making ability has shown impairment in those dependent on a variety of addictive substances (e.g. Vassileva et al., 2007; Verdejo-Garcia et al., 2007). To date, there have been no systematic studies of decision-making deficits in BED, although these impairments have been found in bulimia nervosa patients, and in obese women (Boeka & Lokken, 2006; Brand, Franke-Sievert, Jacoby, Markowitsch, & Tuschen-Caffier, 2007; Davis, Levitan, Muglia, Bewell, & Kennedy, 2004).

Relatedly, *impulsivity* – a personality trait characterized by the diminished ability to inhibit behaviour when restraint is the most advantageous and appropriate response in a particular situation – is a key component of decision-making deficits. Impulsive individuals also show pronounced weaknesses in learning appropriate associations between reward and punishment, which is essential to making advantageous choices (Franken, van Strien, Nijs, & Muris, 2008). High expression of this endophenotype – including both response inhibition and reward-driven impulsivity – has strong links both with BED (Galanti, Gluck, & Geliebter, 2007; Steiger & Bruce, 2007) and with drug abuse (Nigg et al., 2006; Vassileva et al., 2007; Verdejo-Garcia et al., 2007). In a recent study of obese children with and without binge eating, it was also confirmed that the binge eaters were more impulsive on a reward-based measure of this personality trait (Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006). Due, however, to the difficulties of doing prospective risk-factor research in these areas, it is not clear whether impulsive responding is a precursor to addiction disorders or whether it only occurs because of the brain alterations caused by excessive use.

A recent and interesting study has demonstrated how reward-based impulsivity may be especially problematic in our current food environment with its superfluity of highly palatable and varied sources of energy. Using a bogus taste test in either a monotonous food condition or a varied food condition, Guerrieri, Nederkoorn, and Jansen (2008) found a significant group (high vs. low impulsivity) $\times$ food condition interaction, but **only** for the reward-based measure of impulsivity. When the food was monotonous there were no impulsivity differences. However, when variety was present, the high impulsive group consumed significant more calories than their low-impulsive counterparts. Although these results are preliminary, the authors suggest that while impulsivity appears to be a risk for over-consumption in our present food environment, this trait may have played little role in food intake in earlier times in our history.

Treatment implications

While there is strong empirical support for the use of cognitive behavioural therapy (CBT) in the treatment of both BED and drug addiction (Carroll & Onken, 2005; Wilson & Fairburn, 2007), a substantial proportion of cases are non-responders, and the rate of relapse is significant. We believe that the clear parallels between drug addiction and compulsive overeating, outlined earlier in this paper, can provide a helpful framework for developing more effective treatments for BED. Importantly, however, a major limitation for compulsive overeaters is the impossibility of completely abstaining from the 'addictive substance' as is often recommended for drug addiction. Moreover, some treatment approaches, such as Overeaters Anonymous, view compulsive overeating as a disease that sufferers are powerless to overcome and recommend "surrendering oneself to a higher power". We wish to emphasize that this approach is quite different from the treatment strategies for BED being suggested in the current paper, and that we are unaware of any controlled studies of its effectiveness.

Presenting BED patients with an addiction model of compulsive overeating – with the implicit message that they may be fighting a strong neurobiological drive to overeat in an environment that exploits these urges – may help foster a therapeutic sense of self-empathy as well as an understanding that treatment is likely to involve learning effective strategies and enduring life-long efforts to resist overeating and prevent relapse.

All addictive behaviours may be used to increase positive feelings or sensations. For compulsive overeaters, the rewards may include the pleasant taste, the increased energy from rises in blood glucose, and an improved mood. Recognition of these effects points to the importance of helping BED patients find alternative sources of reward and pleasure in their lives besides food and eating. Clinical experience suggests that BED patients often report an overemphasis on food as the only source of enjoyment in their lives. On the other hand, negative emotions are also major triggers

for both binge eating and substance use, since food and drugs can dull or distract from emotional distress. Overcoming an addiction requires an individual to learn to tolerate negative emotional states, including cravings and urges, without acting on them (Dingemans, Martijn, Jansen, & van Furth, 2009; Macht & Mueller, 2007). That is why the development of emotion regulation and distress tolerance skills could be central to the treatment of both BED and drug addiction.

Drug addiction and compulsive overeating also require the interruption of learned habits and stimulus preferences that are maintained by the reinforcing properties of powerful rewards. Both behaviours are conditioned responses to certain triggers and are generally followed by highly reinforcing consequences. Use of stimulus control strategies such as limiting the availability of "binge foods" may be helpful. Another strategy – adopted from drug abuse therapy – which may be useful is cue exposure with response prevention [CERP] (Jansen, 1998). This approach is based on the evidence that in each individual's environment there exist many conditioned cues that can act as disinhibitors and foster binge eating. These can take the form of visual images of food, or the pleasant smell of food cooking. The CERP therapeutic model predicts that cue-reactivity will be extinguished when the bond between the conditioned stimuli (food cues) and the unconditioned stimuli (eating) is broken. Treatment consists of repeated presentations of the relevant food cues, but eating (the response) is prevented. To date, the evidence for success of this treatment in reducing cravings and urges has been well established in various small-scale studies with binge eaters (see Jansen, 1998) and with substance abusers (Havermans, Mulkens, Nederkoorn, & Jansen, 2007).

As in drug addiction, it is possible that the consumption of binge foods, typically high calorie, high fat foods, has a priming effect that can trigger compulsive overeating (Volkow & Wise, 2005). Whereas the CBT model recommends incorporating binge foods into one's daily meal plan, from an addiction perspective, avoiding trigger foods is the recommended course of action. As impulsivity is strongly associated with compulsive overeating and BED (see earlier review), effective treatments also need to address problem-solving and decision-making skills to improve impulse control.

There is also now a range of available medications for each of the major classes of addictive drugs which, in combination with psychotherapy or counseling, have been effective in reducing the likelihood of relapse (see O'Brien, 2005). One mechanism for the success of these drugs seems to be their ability to diminish the strong urges and cravings associated addiction disorders. A good example of an anticraving medication is Naltrexone – which blocks the opiate receptors that regulate the release of dopamine in reward pathways (Gonzales and Weiss, 1998) – and which has been used successfully in the treatment of opiate addiction and alcoholism. Because the endogenous opioid system is also involved in the regulation of food intake, some have suggested that Naltrexone may be a promising treatment for Binge Eating Disorder (e.g. Neumeister, Winkler, & Wober-Bingol, 1999). To date, however, its efficacy for BED lacks the appropriate randomized trials. The science of pharmacogenetics offers a promising new approach to drug treatments for addictions in light of the heterogeneous of these conditions and their highly variable response to medication. The identification of functional polymorphisms of genes implicated in the metabolism of specific drug treatments could, in the future, help clinicians choose the most appropriate medication for patients with addictions (Ramoz, Versini, & Gorwood, 2007).

Summary

In this paper we have presented the view that compulsive overeating has compelling similarities to conventional drug addiction. Our case is based on their comparable clinical features, the biological mechanisms they have in common, and on evidence that the two disorders have a shared diathesis. However, compulsive overeating is unique in the area of addictions as it straddles the divide between substance dependence and behavioural addictions, encompassing characteristics of both. On the one hand, highly palatable food is a dopamine-activating *substance* that acts on brain reward mechanisms in a similar way to other addictive drugs. On the other hand, eating is analogous to other addictions like gambling or shopping in that the *activity* itself is rewarding and reinforcing. One of the many challenges for future research, however, is to understand what factors influence why some vulnerable people abuse drugs or gamble compulsively while others excessively over-indulge in food. The answer, in large part, must lie with social and cultural factors as well as gender, age, socioeconomic status, and relevant personality variables.

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