

## VIEW POINT

# Eating Disorders, Obesity and Addiction

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### Abstract

An addiction model of both eating disorders and obesity has received increasing attention in the popular and scientific literature. The addiction is viewed as a brain disease that must be directly targeted if treatment is to succeed. Evidence from laboratory feeding studies, epidemiology, genetic and familial research, psychopathological mechanisms, and treatment outcome research on cognitive behaviour therapy (CBT) is inconsistent with the clinical validity or utility of the addiction model of eating disorders. Neurobiological research has shown commonalities in brain reward processes between obesity and substance abuse disorders. Yet emphasis on apparent similarities overlooks important differences between obesity and drug addiction. Interest in obesity as a brain disease should not detract from a public health focus on the 'toxic food environment' that is arguably responsible for the obesity epidemic and related nutrition-based chronic disease. Copyright © 2010 John Wiley & Sons, Ltd and Eating Disorders Association.

### Keywords

eating disorders; obesity; addiction; dopamine; cognitive behaviour therapy

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Published online in Wiley Online Library (wileyonlinelibrary.com) DOI: 10.1002/erv.1048

## Introduction

The view that some foods are addictive and that eating disorders are a form of addiction is widespread among mental health professionals and laypersons alike (Benton, 2010; von Ranson & Cassin, 2007; Wilson, Perrin, Rosselli, Striegel-Moore, DeBar, & Kraemer, 2009). More recently, the notion that obesity might be a form of addiction has been gaining popularity, with the case being made that the study of drug addiction may inform our understanding of obesity (Volkow & Wise, 2005). The present paper provides a summary update of the validity and clinical utility of the addiction model of eating disorders and obesity, respectively.

## The addiction model of eating disorders

The conceptual and empirical case against the addiction model of eating disorders has been detailed in the past

(e.g. Bemis, 1985; Vandereycken, 1990; von Ranson & Cassin, 2007; Wilson, 1991; Wilson & Latner, 2001). The key findings can be summarized as follows.

### Laboratory studies of eating behaviour

If bulimia nervosa (BN) is an addiction, evidence should demonstrate that patients with this eating disorder preferentially consume a particular addicting food in laboratory studies of eating behaviour. No such evidence has been reported to date. Indeed, the pattern of macronutrient consumption of individuals with BN is similar to that of non-BN controls. The key appetitive abnormality appears to be control over the amount of food consumed during the binge, not any specific nutrient (Walsh, 1993). The same holds true for binge eating disorder (BED) (Yanovski et al., 1992). Especially noteworthy is that eating disorder patients do not

preferentially consume carbohydrates during a binge. The so-called carbohydrate craving has often been put forward as a reflection of an addictive process but is without foundation (Benton, 2010). Carbohydrates do not necessarily relieve negative affect or improve mood as is claimed as part of 'carbohydrate craving' (Jansen, van den Hout, & Griez, 1989; Toornvliet et al., 1997; Turner, Foggo, Bennie, Carroll, Dick, & Goodwin, 1991).

### **Sugar and addiction**

Recent scientific interest in this topic was spurred by the research of Hoebel and his associates (Avena, Rada, & Hoebel, 2008; Hoebel, Avena, Bocarsly, & Rada, 2009). They developed a model of sugar addiction based on the results of research in which rats were food deprived daily for 12 hours and then given access to a sugar solution and chow. After a month of this intermittent-feeding schedule the rats showed changes in their brains and 'addictive-like' behaviour comparable to that of drug addiction. For example, 'withdrawal' was demonstrated by opioid system activation reflected in elevated anxiety following sugar deprivation, and 'craving' as the consumption of excessive sugar-water when given the opportunity following deprivation. Hoebel et al. (2009) describe this eating pattern as a 'natural addiction'.

Avena et al. (2008) claimed that the correspondence of their animal model with BN and BED 'is notable' (p. 32). However, the pattern would need to be analogous either to BN or BED but not both because the two diagnoses are characterized by different eating patterns. BN patients typically restrict intake severely and then binge when they lose control. BED patients show significantly lower levels of dietary restraint than BN patients, and BED patients eat more in periods between binges than BN patients do (Wilfley, Bishop, Wilson, & Agras, 2007). Ultimately, as Avena et al. (2008) themselves note, whether this animal model has applicability to people remains to be determined. Consistent with previous literature (Haddock & Dill, 2000), Benton (2010), in a comprehensive review of the evidence, concluded that there is 'no support from the human literature for the hypothesis that sucrose may be physically addictive or that addiction to sugar plays a role in eating disorders' (p. 1).

### **Epidemiological research**

Epidemiological data are inconsistent with an addiction model. Rates of comorbid alcohol or drug dependence are significantly elevated in both clinical and com-

munity samples of individuals with BN and anorexia nervosa (AN) (Garfinkel et al., 1995). In contrast to an earlier study (Yanovski, Nelson, Dubbert, & Spitzer, 1993), recent research has shown that substance abuse is significantly associated with BED (Root, Pisetsky, Thornton, Lichtenstein, Pedersen, & Bulik, 2010). The association is not specific to eating disorders, however. Rates of substance abuse are elevated in many Axis I disorders (e.g. anxiety disorders; Kushner, Sher, & Beitman, 1990). Moreover, depression is the more common co-occurring disorder than substance abuse in both community and treatment-seeking samples of BN and BED (Bushnell, Wells, McKenzie, Hornblow, Oakley-Browne, & Joyce, 1994; Keel, Klump, Miller, McGue, & Iacono, 2005; Wilfley, Friedman, Douchis, Stein, Welch, & Ball, 2000; Wilson, Wilfley, Agras, & Bryson, 2010).

### **Genetic and familial studies**

If eating disorders and substance abuse are different forms of a common underlying addictive process, there should be a shared genetic or familial aetiology. The overall evidence does not support this important proposition. Kendler, Walters, Neale, Kessler, Heath, and Eaves (1995) showed that genetic (and familial) influences on BN are independent of those in alcohol use disorders. Lilienfeld et al. (1998) found that alcohol and drug dependence was not elevated in relatives of BN probands with BN alone. The latter results complement those from a family study of probands with alcohol use disorders (Schuckit, Tipp, Anthenelli, Bucholz, Hesselbrock, & Nurnberger, 1996) in which an increased prevalence of BN was found among probands but not among their relatives. A study of eating disorder inpatients found that it was only among those with first-degree relatives with a history of alcoholism who showed higher rates of psychopathology including substance abuse, evidence consistent with independent genetic transmission of eating disorders and substance abuse (Redgrave, Coughlin, Heinberg, & Guarda, 2007). Based on a twin study focused on individuals with either AN or BN, Keel et al. (2005) concluded that 'Across all forms of substance use disorders (alcohol use disorders, drug use disorders and nicotine dependence), we found no evidence for shared transmission with eating disorders' (p. 103).

An exception to the foregoing pattern of findings comes from a family history study of BED (Lilienfeld,

Ringham, Kalarchian, & Marcus, 2008). Whereas depression and other clinical disorders showed a pattern of independent transmission from BED, the primary exception was substance use disorders among female relatives of BED probands. Lilenfeld et al. (2008) noted that these results are consistent with a shared familial transmission, but cautioned that this 'family history study with a very modest sample size should certainly be considered tentative until replication using a direct-interview family study design with a larger, independent sample is conducted' (p. 253).

In sum, contrary to the addiction model, eating disorders and substance abuse appear to be conditions with separate aetiologies. The two disorders mutually influence each other with the impact of eating disorders on the development of alcohol use disorder being greater than the reverse pattern (Franko, Dorer, Keel, Jackson, Manzo, & Herzog, 2005). Measelle, Stice, and Hogansen (2006) found that in adolescent girls, initial eating pathology predicted future development of substance abuse, but not *vice versa*. The authors noted that this finding is consistent with evidence in adults of a prospective, unidirectional relationship between earlier eating pathology and subsequent development of substance abuse (Johnson, Cohen, Kotler, Kasen, & Brook, 2002).

### **Core psychopathology of eating disorders**

For obvious reasons, the addiction model has focused on binge eating since this behaviour is defined by loss of control over food intake. The putative addicting food is seen as analogous to the substances in forms of chemical dependency. Nevertheless, as critics of this approach have repeatedly emphasized, the core psychopathology of eating disorders is more complex than just binge eating. For example, DSM-IV criteria for AN and BN include the criterion of overvaluation of body weight or shape—the undue influence of body weight or shape on self-evaluation (American Psychiatric Association, 1994). In addition, recent research has shown that patients with BED have similar levels of overvaluation to those with BN, that its presence in BED indicates greater disturbance, and that it may predict a worse treatment outcome (Goldschmidt et al., 2010; Grilo, Hrabosky, White, Allison, Stunkard, & Masheb, 2008; Grilo et al., 2009; Wilson et al., 2010). In the well-established cognitive behavioural model of the main-

tenance of BN this overvaluation of body weight or shape drives the dysfunctional dieting that leads to binge eating (Fairburn, 2008).

Another critical difference between the reality of BN and the addiction model is that people with BN are characterized by severe and dysfunctional dietary restraint. They try to restrict food consumption and avoid certain 'trigger' foods. As Fairburn (1995) states, 'What distresses them about their binge eating is that it represents their failure to control their eating and carries the risk of weight gain. There is no equivalent phenomenon in alcohol (or drug) abuse. Individuals who abuse alcohol have no inherent drive to avoid alcohol, against which excessive drinking takes place' (p. 102).

Davis and Carter (2009) have proposed that BED is 'a phenotype particularly well-suited to an addiction conceptualization' (p. 3). They claim that drug addiction and BED share common processes in terms of loss of control, tolerance, withdrawal and craving. Loss of control is a feature of drug addiction, and it is a defining condition of binge eating (APA, 1994). Whether this similarity makes BED an addiction is dubious. Both a subset of individuals with AN and all people with BN are characterized by binge eating (loss of control). Are AN and BN then addictive disorders? As for 'tolerance' in BED, Davis and Carter (2009) state that evidence 'arises largely from anecdotal clinical reports' (p. 3). Similarly, with regard to 'withdrawal' in BED, the authors note that human evidence is anecdotal (mostly from 'self-help books and internet websites') and 'has not been subjected to the scrutiny of controlled scientific investigation' (p. 3). Finally, little evidence is offered to show that 'craving' plays comparable roles in drug addiction and BED. In contrast, Benton (2010) summarizes evidence highlighting differences between food and drug craving in humans.

In sum, despite superficial similarities as noted in previous analyses (Vandereycken, 1990; Wilson, 1991), the core psychopathology of eating disorders and drug addiction is fundamentally different.

### **Treatment outcome research**

Rigorous and extensive reviews of treatment outcome research have documented the efficacy of manual-based cognitive behaviour therapy (CBT) as the treatment of choice for BN and BED (e.g. American Psychiatric Association, 2006; National Institute for Clinical

Excellence [NICE], 2004; Wilson, 2010; Wilson, Grilo, & Vitousek, 2007). Manual-based CBT has broad based and enduring effects. It typically eliminates both binge eating and purging in roughly 30–50% of BN cases in intent-to-treat (ITT) statistical analyses. Therapeutic improvement is reasonably well maintained at 1-year follow-up. The success rate in BED (complete cessation of binge eating) is even greater, ranging from 60 to 79% with 1 and 2-year follow-ups (Wilfley et al., 2002; Wilson et al., 2010). CBT significantly decreases psychiatric comorbidity, enhances self-esteem and improves social functioning. Consistent with the theoretical model on which manual-based CBT is based, reducing dietary restraint appears to be a partial mediator of treatment efficacy in eliminating binge eating and purging in BN (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). CBT is more effective for BN and BED than treatment with antidepressant medication, an evidence-based treatment that is significantly superior to pill placebo (NICE, 2004).

Treatment derived from the addiction model is diametrically opposed to the CBT model and treatment of eating disorders. The former is based on the 12-step approach of Alcoholics Anonymous (AA) except that to simplify somewhat, the word 'food' is substituted for alcohol. Recognizing that humans have to eat, the emphasis is on avoiding particular foods deemed to be 'addicting' (e.g. sugar). Inescapably, the approach demands continual dietary restraint, avoidance of the so-called trigger foods, a heightened sense of powerlessness and the reinforcement of dichotomous thinking patterns (e.g. food is either 'good' or 'bad'; the person is either 'in control' or 'out of control'). Examples of treatment programs that embody this philosophy are Overeaters Anonymous and Food Addicts in Recovery Anonymous.

The contrasting features of evidence-based CBT and addiction-based models have been detailed elsewhere (Bemis, 1985; Fairburn, 1995; Wilson, 1991) and need not be repeated here. Suffice it to note the seminal difference by emphasizing that CBT is predicated on reducing dysfunctional dieting in favour of regular eating, featuring flexible and moderate food consumption with no forbidden foods versus the abstinence/avoidance goal of the addiction model. As Wardle (1987) observed, 'From the patient's point of view, the adoption of an addiction model, with its implicit connotations of consuming something harmful, could only delay the process of returning to normal eating' (p.

49). Furthermore, in contrast to an addiction model, CBT seeks to empower patients and build self-efficacy in coping with life's stresses.

The critical issue here is that the addiction model must predict the failure of manual-based CBT. That CBT is so effective represents a refutation of the addiction model, at least in terms of its implications for the prevention and treatment of eating disorders. In turn, evidence-based CBT predicts that the abstinence model of addiction is ineffective at best and potentially hazardous at worst because it encourages dietary restriction that is known to cause or maintain BN (Fairburn, 2008). There is no evidence from controlled outcome research on the efficacy of a 12-step, addiction-based treatment for eating disorders (von Ranson & Cassin, 2007; Wilson & Latner, 2001).

Davis and Carter (2009) have argued that binge eating is an addictive disorder but have disavowed the typical 12-step approach as summarized above in which the addict surrenders to a higher power. Nonetheless, they conclude that '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' (p. 6). They cite no evidence to support their speculation. Moreover, they fail to explain the impressive success rate of CBT with BED. The treatment recommendations Davis and Carter (2009) claim follow from the addiction model—the importance of helping patients find 'alternative sources of reward in their lives', and 'stimulus control strategies' (pp. 5–6) are not novel. Both recommendations are longstanding components of behaviour therapy in general and the Fairburn, Marcus, and Wilson (1993) treatment manual for BN and BED in particular.

In explaining their animal model of sugar addiction, Hoebel et al. (2009) state that 'it is the intermittent feeding schedule that seems to be critical for inducing bingeing and the subsequent signs of dependency' (p. 34). In a separate animal model, Bello, Guarda, Terrillion, Redgrave, Coughlin, and Moran (2009) similarly found that repeated cycles of acute caloric restriction followed by access to palatable food resulted in a 'bingelike' (p. R629) behavioural response feeding and physiological changes that might facilitate the maintenance of disordered eating. It should be noted that Bello et al. (2009) make no mention of addiction in interpreting their findings. A defining feature of manual-based CBT (Fairburn, 2008) is the early

commitment to developing a pattern of regular eating designed to replace dysfunctional dietary restraint, the component of the treatment that likely mediates reduction of binge eating (Fairburn, 2008; Shah, Passi, Bryson, & Agras, 2005; Wilson et al., 2002). The Bello et al. (2009) animal model is consistent with the evidence-based CBT approach.

## Obesity and addiction

In the food addiction literature, eating disorders and obesity are often viewed as similar manifestations of a common addiction. On the basis of their animal research, Avena et al. (2008) have proposed that sugar addiction might be a cause of obesity as well as eating disorders (see above). Benton's (2010) review of the human evidence, however, is as critical of the 'sugar addiction' thesis of obesity as it is of the eating disorders model.

The consumption of sugar-sweetened beverages has been linked to rising rates of obesity (Brownell et al., 2009; Malik, Schultz, & Hu, 2006). However, the putative mechanisms responsible for the effect of sugar-sweetened drinks may include the poor satiating properties of sugar and its consumption for social reasons (e.g. Brownell et al., 2009). Invoking the concept of addiction is not necessary.

The co-occurrence of eating disorders and substance abuse is often cited in support of an addiction model, as summarized above. It follows that if both eating disorders and obesity are a form of addiction then obesity and substance abuse should be significantly linked. The evidence suggests otherwise. In an analysis of the National Epidemiologic Survey on Alcohol and Related Conditions, Petry, Barry, Pietrzak, and Wagner, (2008) found that 'overweight and extremely obese persons did not differ from those of normal BMI in terms of alcohol dependence. Past-year alcohol use disorders did not differ across groups, nor did drug use disorders' (p. 291). Moreover, Simon et al. (2006) reported that rates of substance abuse disorder were lower in obese individuals as opposed to normal weight controls.

## Brain mechanisms and obesity

Johnson and Kenny (2010) found that the development of obesity and compulsive-like eating in obese rats was associated with deficits in brain reward mechanisms. Specifically, striatal dopamine receptors were down-regulated in obese rats as has been reported in humans

addicted to drugs. These investigators concluded that 'Common hedonic mechanisms may therefore underlie obesity and drug addiction' (p. 1).

Consistent with the Johnson and Kenny (2010) findings of downregulation of dopamine receptors Stice, Spoor, Bohon, Veldhuizen, and Small (2008) demonstrated that individuals with higher BMI showed less activation in the striatum in response to food consumption relative to those with a lower BMI. Stice et al. (2008) also found that this dopamine down-regulation can be coupled with greater activation in the gustatory cortex and somatosensory brain regions in response to anticipation and consumption of food. Both biological processes would predispose people to overeating and weight gain. Stice et al. (2008) cautioned that it is unclear whether these brain abnormalities precede or are caused by overeating.

Neurobiological research of this sort is increasing our understanding of brain mechanisms involved in obesity. Changes in brain reward systems in obesity and substance abuse appear to overlap. The question remains, however, whether these findings indicate that obesity is an addiction and should be treated as such. As Kalarchian and Marcus (2007) observed, 'even if obesity and drug addiction share common developmental vulnerability, effective treatments for obesity and addiction may differ' (p. 1268).

## Temporal course of relapse patterns

The focus on the similarities in brain mechanisms between obesity and substance abuse should not obscure important behavioural differences between the two. Leshner (1997) used the term 'chronic relapsing disorder' to describe the course of drug addiction. The concept was readily extended to obesity given the high rate of relapse that characterizes its treatment outcome (Davis & Carter, 2008). What has been overlooked, however, is that the two different disorders have uniform but strikingly different patterns of relapse over time.

A classic study by Hunt, Barnett, and Branch (1971) showed that nicotine, heroin and alcohol all produced similar rates of relapse over a 1-year period. Across all three drugs, most patients relapsed early post-treatment, and thereafter, relapse rates decelerated dramatically. In a recent quantitative review of treatment outcome studies on nicotine and alcohol, Kirshenbaum, Olsen, and Bickel (2009) demonstrated that the basic shape of the relapse curve tends to be

uniform—‘the rate of relapse decelerates after initial abstinence has been achieved, and therefore, the amount of accumulated time abstinent may be the transcending variable that operates to shape the relapse curve’ (p. 8). In short ‘abstinence begets abstinence’ (p. 9).

The temporal pattern of the typical relapse curve in behavioural treatment studies of obesity is different from that of substance abuse summarized above. As Jeffery et al. (2000) observed, the pattern is ‘remarkably consistent. The rate of initial weight loss is rapid and then slowly declines. The point of maximum weight loss is usually reached approximately 6 months after the initiation of treatment. Weight regain then begins and continues gradually until weight stabilizes somewhat below baseline levels. This temporal pattern is fairly independent of initial weight loss’ (p. 7). Efforts to prevent relapse in the treatment of obesity via aggressive maintenance strategies have been able to slow the rate of regain over time (Wadden, Butryn, & Byrne, 2004), but have failed to alter the basic accelerating function of the relapse curve.

Two conclusions follow from the contrasting relapse patterns of substance disorders and obesity. First, these findings further underscore the fundamental differences between obesity and eating disorders contrary to a key premise of most addiction models. Whereas the vast majority of patients receiving behavioural weight loss treatment relapse over time, many BN and BED patients treated with CBT show impressive maintenance of treatment-induced improvement (Wilson et al., 2007). Second, the data suggest that different mechanisms—be they biological, psychological or social—may be responsible for the contrasting patterns of maintenance of change and relapse between drug abuse and obesity.

## Treatment implications of different models of obesity

### Addiction model

Addiction is viewed as a brain disease (Leshner, 1997) and on this basis Volkow and O’Brien (2007) proposed that obesity be considered as a possible mental disorder in DSM-V. Nevertheless, unlike eating disorders, obesity is not a mental disorder. It is not included in DSM-IV and Marcus and Wildes (2009) have recommended against its inclusion in DSM-V. Obesity can be better viewed as a ‘heritable neurobehavioural disorder

that is highly sensitive to environmental conditions’ (O’Rahilly & Farooqi, 2008).

Volkow and Wise (2005) were careful to note that psychosocial factors are also involved in the development of obesity, and they do recommend ‘multimodal’ treatment including drugs. Nevertheless, their primary focus is on the brain and its dysfunctions. Leshner (1997) similarly notes that factors other than brain disease are involved in addiction, but his emphasis is clearly on the brain *per se*: ‘If the brain is the core of the problem, attending to the brain needs to be a core part of the solution’ (p. 49). It follows from these analyses that drugs might be necessary to treat the disordered brain mechanisms. For example, Wang et al. (2001) suggested that ‘strategies aimed at improving dopamine function may be beneficial in the treatment of obesity’ (p. 357). Volkow and O’Brien (2007) emphasized the need to ‘mitigate the pathologically intense drive for food consumption’ (p. 710).

The addiction model would predict that if obesity and substance abuse have common underlying neurobiological mechanisms, then a drug treatment that is effective for one of the two disorders should be effective for both. Naltrexone, for example, blocks opiate receptors that regulate the release of dopamine and has been shown to have modest albeit inconsistent effects on alcohol dependence (Gueorguieva et al., 2010; Longabaugh, Wirtz, Gulliver, & Davidson, 2009). Therefore, naltrexone should be predicted to be effective with binge eating. Yet the most comprehensive and rigorous reviews of pharmacological treatment of BED, which is closely linked with obesity, do not mention naltrexone because of the absence of suitable randomized controlled trials (RCTs) (Bodell & Devlin, 2010; Reas & Grilo, 2008). Presumably the reason is that naltrexone was applied to binge eating in obese individuals in early treatment trials that failed to show any specific benefit of the drug on frequency of binge eating (Alger, Schwalberg, Bigaouette, Howard, & Reid, 1991; Mitchell et al., 1989).

Pharmacological treatment of obesity in general has failed to produce significant, enduring weight loss (Yanovski, 2005). At best, the effects are modest and relatively short term (e.g. Wadden et al., 2005). The history of ‘phen-fen’ (phentermine combined with fenfluramine) is instructive regarding serious and unanticipated side effects of an anti-obesity drug (Yanovski, 2005). The complexity of obesity and the difficulties it poses for the development of an effective

weight loss drug remains a major challenge (e.g. Heymsfield, 2009).

The hope remains that future research will result in the development of an effective pharmacological treatment for obesity. In the meantime, the focus on a putative drug treatment for the 'brain disease' of obesity should not result in the relative de-emphasis of environmental influences.

### Behavioural model

The evidence suggests that what Brownell (2004) has described as the 'toxic food environment' is responsible for the relatively recent, rapid increase in rates of obesity. A defining feature of this environment is easy access to palatable high fat food. Modelling such a diet in rats leads to 'addiction-like reward deficits' (Johnson & Kenny, 2010). Other elements of the toxic food environment include the wide variety of calorically dense relatively inexpensive foods; deterioration of eating habits in the United States with excessive portion sizes; and aggressive marketing of high fat, unhealthy foods by the food industry (Brownell et al., 2009). Consensus also exists that a sedentary lifestyle contributes to the obesity epidemic. It is important to note that this behavioural component has nothing to do with the ingestion of a substance (food) that is the basis for the obesity-as-addiction model.

The distinctive pattern of relapse that characterizes obesity treatment programs makes good behavioural sense (Wilson, 1994). Vulnerable individuals, perhaps due to a genetically driven responsiveness to food cues (O'Rahilly & Farooqi, 2008), are exposed to a potent primary reinforcer (food) multiple times per day every day. Behaviour is powerfully controlled by positive reinforcing consequences. Regulating behaviour around consumption of this potent reinforcer within the greater toxic food environment requires unending, daily vigilance and self-control. For the majority of people who lose weight, their self-regulatory capacity is gradually eroded by the pressures of the toxic food environment. These pressures interact with metabolic changes that support a higher body weight (Goldsmith et al., 2010). The result is typically relapse that is associated with decreased dietary restraint and insufficient physical activity (Wing et al., 2008). Maintenance strategies can slow (Knowler et al., 2009) but seldom prevent relapse. Moreover, the expenditure of time and money required to implement resource-intensive maintenance strategies renders them impractical for

any large-scale, community-based intervention (Brownell, 2010).

Confronted with the twin failures of pharmacological and behavioural treatment in producing long-term weight control, several investigators have argued that the only realistic approach to managing obesity is to focus efforts on prevention and changes in public health policy, the details of which are beyond the scope of this paper (e.g. Brownell, 2004; Jeffery, 2002; Nestle & Jacobson, 2000; Seligman & Schilling, 2010).

### Concluding comments

The scientific case against the addiction model of eating disorders is compelling when evidence from laboratory eating studies, epidemiology, genetic and familial research and core psychopathology research is evaluated. Of particular importance is that adoption of an addiction model has treatment implications that explicitly contradict the currently most effective treatment for patients with an eating disorder (NICE, 2004; Wilson et al., 2007).

Neurobiological research has revealed commonalities in brain reward processes and developmental vulnerabilities between substance abuse disorders and obesity. Despite similarities between obesity and substance abuse disorders, important differences exist including the temporal course of relapse across these disorders and respective treatment outcomes. It is important that an emphasis on obesity and substance abuse as brain diseases not detract from the public health and prevention initiatives that derive directly from a behavioural model predicated on the influence of 'toxic food' environmental forces.

### Acknowledgements

The author is indebted to Rebecca Greif, Tanya Schlam, Tom Hildebrandt and Jessica Breland for their helpful feedback on this paper.

### References

- Alger, S. A., Schwalberg, M. J., Bigaouette, J. M., Howard, L. J., & Reid, L. D. (1991). Using drugs to manage binge-eating among obese and normal weight patients. In L. D. Reid (Ed.), *Opioids, bulimia, and alcohol abuse and alcoholism* (pp. 131–142). New York: Springer-Verlag.

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* 4th ed., Washington, DC: Author.
- Avena, N. M., Rada, P., & Hoebel, B. G. (2008). Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience and Biobehavioral Reviews*, *32*, 20–39.
- Bello, N. T., Guarda, A. S., Terrillion, C. E., Redgrave, G. W., Coughlin, J. W., & Moran, T. H. (2009). Repeated binge access to a palatable food alters feeding behavior, hormone profile, and hindbrain c-Fos responses to a test meal in adult male rats. *American Journal of Physiology—Regulatory, Integrative and Comparative Physiology*, *297*, 622–631.
- Bemis, K. M. (1985). Abstinence and nonabstinence models for the treatment of bulimia. *International Journal of Eating Disorders*, *4*, 407–437.
- Benton, D. (2010). The plausibility of sugar addiction and its role in obesity and eating disorders. *Clinical Nutrition*, *29*, 288–303.
- Bodell, L., & Devlin, M. (2010). Pharmacotherapy for binge-eating disorder. In C. M. Grilo, & J. E. Mitchell (Eds.), *The treatment of eating disorders* (pp. 402–414). New York: Guilford Press.
- Brownell, K. D. (2004). *Food fight*. New York: McGraw-Hill.
- Brownell, K. D. (2010). The humbling experience of treating obesity: Should we persist or desist? *Behaviour Research and Therapy*, *48*, 717–719.
- Brownell, K. D., Farley, T., Willett, W. C., Popkin, B. M., Chaloupka, F. J., Thompson, J. W., et al. (2009). The public health and economic benefits of taxing sugar-sweetened beverages. *New England Journal of Medicine*, *361*, 1599–1605.
- Bushnell, J. A., Wells, J. E., McKenzie, J. M., Hornblow, A. R., Oakley-Browne, M. A., & Joyce, P. R. (1994). Bulimia comorbidity in the general population and in the clinic. *Psychological Medicine*, *24*, 605–611.
- Davis, C., & Carter, J. C. (2009). Compulsive overeating as an addiction disorder: A review of theory and evidence. *Appetite*, *53*, 1–8.
- Fairburn, C. G. (1995). *Overcoming binge eating*. New York: Guilford Press.
- Fairburn, C. G. (2008). *Cognitive behavior therapy and eating disorders*. New York: Guilford Press.
- Fairburn, C. G., Marcus, M., & Wilson, G. T. (1993). Cognitive-behavioral therapy for binge eating and bulimia nervosa: A comprehensive treatment manual. In C. G. Fairburn, & G. T. Wilson (Eds.), *Binge eating* (pp. 361–404). New York: Guilford Press.
- Franco, D. L., Dorer, D. J., Keel, P. K., Jackson, S., Manzo, M. P., & Herzog, D. B. (2005). How do eating disorders and alcohol use disorder influence each other? *International Journal of Eating Disorders*, *38*, 200–207.
- Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., et al. (1995). Bulimia nervosa in a Canadian community sample: Prevalence and comparison of subgroups. *American Journal of Psychiatry*, *152*, 1052–1058.
- Goldschmidt, A. B., Hilbert, A., Manwaring, J. L., Wilfley, D. E., Pike, K. M., Fairburn, C. G., et al. (2010). The significance of overvaluation of shape and weight in binge eating disorder. *Behaviour Research and Therapy*, *48*, 187–193.
- Goldsmith, R., Joannise, D. R., Gallagher, D., Pavlovich, D., Shamoon, E., Leibel, R. L., et al. (2010). Effects of experimental weight perturbation on skeletal muscle work efficiency, fuel utilization, and biochemistry in human subjects. *American Journal of Physiology—Regulatory, Integrative and Comparative Physiology*, *298*, R79–88.
- Grilo, C. M., Crosby, R., Masheb, R., White, M., Peterson, C., Wonderlich, S., et al. (2009). Overvaluation of shape and weight in binge eating disorder, bulimia nervosa, and sub-threshold bulimia nervosa. *Behaviour Research and Therapy*, *47*, 692–696.
- Grilo, C. M., Hrabosky, J. I., White, M. A., Allison, K., Stunkard, A. J., & Masheb, R. M. (2008). Overvaluation of shape and weight in binge eating disorder and overweight controls: Refinement of BED as a diagnostic construct. *Journal of Abnormal Psychology*, *117*, 414–419.
- Gueorguieva, R., Wu, R., Donovan, D., Rounsaville, B. J., Cooper, D., Krystal, J. H., et al. (2010). Naltrexone and combined behavioral intervention effects on trajectories of drinking in the COMBINE study. *Drug and Alcohol Dependence*, *107*, 221–229.
- Haddock, C. K., & Dill, P. L. (2000). The effects of food on mood and behavior: Implications for the addictions model of obesity and eating disorders. *Drugs and Society*, *15*, 17–47.
- Heymsfield, S. (2009). Taranabant: A cautionary tale of weight suppression. *Talk Delivered at the Annual Meeting of the Eating Disorders Research Society*, New York, September.
- Hoebel, B. G., Avena, N. M., Bocarsly, M. E., & Rada, P. (2009). Natural addiction: a behavioral and circuit model based on sugar addiction in rats. *Journal of Addiction Medicine*, *3*, 33–41.
- Hunt, W. A., Barnett, L. W., & Branch, L. G. (1971). Relapse rates in addiction programs. *Journal of Clinical Psychiatry*, *27*, 455–456.
- Jansen, A., Van den Hout, M. A., & Griez, E. (1989). Does bingeing restore bulimics' alleged 5-HT-deficiency? *Behaviour Research and Therapy*, *27*, 555–560.
- Jeffery, R. W. (2002). Public health approaches to the management of obesity. In C. G. Fairburn, & K. D.

- Brownell (Eds.), *Eating disorders and obesity* (2nd ed., pp. 613–618). New York: Guilford Press.
- Jeffery, R. W., Drewnowski, A., Epstein, L. H., Stunkard, A., Wilson, G. T., Wing, R., et al. (2000). Long-term maintenance of a weight loss: Current status. *Health Psychology, 19*, 5–16.
- Johnson, J. G., Cohen, P., Kotler, L., Kasen, S., & Brook, J. S. (2002). Psychiatric disorders associated with risk for development of eating disorders during adolescence and early adulthood. *Journal of Consulting and Clinical Psychology, 70*, 1119–1128.
- Johnson, P. M., & Kenny, P. J. (2010). Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. *Nature Neuroscience, 13*, 635–641.
- Kalarchian, M., & Marcus, M. (2007). Drs. Kalarchian and Marcus reply. *American Journal of Psychiatry, 164*, 1268–1269.
- Keel, P. K., Klump, K. L., Miller, K. B., McGue, M., & Iacono, W. G. (2005). Shared transmission of eating disorders and anxiety disorders. *International Journal of Eating Disorders, 38*, 99–105.
- Kendler, K., Walters, E., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1995). The structure of the genetic and environmental risk factors for six major psychiatric disorders in women: Phobia, generalized anxiety disorder, panic disorder, bulimia, major depression and alcoholism. *Archives of General Psychiatry, 52*, 374–383.
- Kirshenbaum, A. P., Olsen, D. M., & Bickel, W. K. (2009). A quantitative review of the ubiquitous relapse curve. *Journal of Substance Abuse Treatment, 36*, 9–17.
- Knowler, W. C., Fowler, S. E., Hamman, R. F., Christophi, C. A., Hoffman, H. J., Brenneman, A. T., et al. (2009). 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet, 374*, 1677–1686.
- Kushner, M., Sher, K. J., & Beitman, B. (1990). The relationship between alcohol problems and the anxiety disorders. *American Journal of Psychiatry, 147*, 685–695.
- Leshner, A. (1997). Addiction is a brain disease, and it matters. *Science, 278*, 45–46.
- Lilenfeld, L. R., Kaye, W. H., Greeno, C. G., Merikangas, K. R., Plotnicov, K., Pollice, C., et al. (1997). Psychiatric disorders in women and bulimia nervosa and their first-degree relatives: Effects of comorbid substance dependence. *International Journal of Eating Disorders, 22*, 253–264.
- Lilenfeld, L. R., Kaye, W. H., Greeno, C. G., Merikangas, K. R., Plotnicov, K., Pollice, C., et al. (1998). A controlled family study of anorexia nervosa and bulimia nervosa: Psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Archives of General Psychiatry, 55*, 603–610.
- Lilenfeld, L. R., Ringham, R., Kalarchian, M. A., & Marcus, M. D. (2008). A family history study of binge-eating disorder. *Comprehensive Psychiatry, 49*, 247–254.
- Longabaugh, R., Wirtz, P. W., Gulliver, S. B., & Davidson, D. (2009). Extended naltrexone and broad spectrum treatment or motivational enhancement therapy. *Psychopharmacology, 206*, 367–376.
- Malik, V. S., Schulze, M. B., & Hu, F. B. (2006). Intake of sugar-sweetened beverages and weight gain: a systematic review. *American Journal of Clinical Nutrition, 84*, 274–288.
- Marcus, M., & Wildes, J. (2009). Obesity: Is it a mental disorder? *International Journal of Eating Disorders, 42*, 739–753.
- Measelle, J. R., Stice, E., & Hogansen, J. M. (2006). Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in female adolescents. *Journal of Abnormal Psychology, 115*, 524–538.
- Mitchell, J., Christenson, G., Jennings, J., Huber, M., Thomas, B., Pomeroy, C., et al. (1989). A placebo-controlled, double-blind crossover study of naltrexone in outpatients with normal weight bulimia. *Journal of Clinical Psychopharmacology, 9*, 94–97.
- National Institute for Clinical Excellence. (2004). Eating disorders – Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa, and related eating disorders (Clinical Guideline No. 9). London: Author. (Available at [www.nice.org.uk/guidance/CG9](http://www.nice.org.uk/guidance/CG9)).
- Nestle, M., & Jacobson, M. F. (2000). Halting the obesity epidemic: A public health policy approach. *Public Health Reports, 115*, 12–24.
- O’Rahilly, S., & Farooqi, I. S. (2008). Human obesity: A heritable neurobehavioral disorder that is highly sensitive to environmental conditions. *Diabetes, 57*, 2905–2910.
- Petry, N. M., Barry, D., Pietrzak, R. H., & Wagner, J. A. (2008). Overweight and obesity are associated with psychiatric disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychosomatic Medicine, 70*, 288–297.
- Reas, D. L., & Grilo, C. M. (2008). Review and meta-analysis of pharmacotherapy for binge eating disorder. *Obesity, 16*, 2024–2038.
- Redgrave, G., Coughlin, J., Heinberg, L., & Guarda, A. A. (2007). First-degree relative history of alcoholism in eating disorder inpatients: relationship to eating and substance use psychopathology. *Eating Behaviors, 8*, 5–22.
- Root, T., Pisetsky, E., Thornton, L., Lichtenstein, P., Pedersen, N., & Bulik, C. (2010). Patterns of co-morbidity of eating disorders and substance abuse in Swedish females. *Psychological Medicine, 40*, 105–115.
- Schuckit, M. A., Tipp, J. E., Anthenelli, R. M., Bucholz, K. K., Hesselbrock, V. M., & Nurnberger, J. I. (1996). Anorexia

- nervosa and bulimia nervosa in alcohol-dependent men and women and their relatives. *American Journal of Psychiatry*, 153, 74–82.
- Seligman, H. K., & Schilling, D. (2010). Hunger and socioeconomic disparities in chronic disease. *New England Journal of Medicine*, 363, 6–9.
- Shah, N., Passi, V., Bryson, S., & Agras, W. S. (2005). Patterns of eating and abstinence in women treated for bulimia nervosa. *International Journal of Eating Disorders*, 38, 330–334.
- Simon, G. E., Von Korff, M., Saunders, K., Miglioretti, D. L., Crane, P. K., van Belle, G., et al. (2006). Association between obesity and psychiatric disorders in the US adult population. *Archives of General Psychiatry*, 63, 824–830.
- Stice, E., Spoor, S., Bohon, C., Veldhuizen, M. G., & Small, D. M. (2008). Relation of reward from food intake and anticipated food intake to obesity: A functional magnetic resonance imaging study. *Journal of Abnormal Psychology*, 117, 924–935.
- Toornvliet, A. C., Tuinenburg, J. C., Elte-de Wever, B. M., Pieters, M. S., Frolich, M., Onkenhout, W., et al. (1997). Psychological and metabolic responses of carbohydrate craving obese patients to carbohydrate, fat, and protein-rich meals. *International Journal of Obesity*, 21, 860–864.
- Turner, M., Foggo, M., Bennie, J., Carroll, S., Dick, H., & Goodwin, G. M. (1991). Psychological, hormonal and biochemical changes following carbohydrate bingeing: A placebo controlled study in bulimia nervosa and matched controls. *Psychological Medicine*, 21, 123–133.
- Vandereycken, W. (1990). The addiction model in eating disorders: Some critical remarks a selected bibliography. *International Journal of Eating Disorders*, 9, 95–101.
- Volkow, N. D., & O'Brien, C. P. (2007). Issues for DSM-V: Should obesity be included as a brain disorder? *American Journal of Psychiatry*, 164, 708–710.
- Volkow, N. D., & Wise, R. A. (2005). How can drug addiction help us understand obesity? *Nature Neuroscience*, 8, 555–560.
- von Ranson, K. M., & Cassin, S. E. (2007). Eating disorders and addiction: Theory and evidence. In J. S. Rubin (Ed.), *Eating disorders and weight loss research* (pp 1–37). Huppauge, NY: Nova Science Publishers, Inc.
- Wadden, T. A., Berkowitz, R. I., Womble, L. G., Sarwer, D. W., Phelan, S., Cato, R. K., et al. (2005). Randomized trial of lifestyle modification and pharmacotherapy for obesity. *The New England Journal of Medicine*, 353, 2111–2120.
- Wadden, T. A., Butryn, M. L., & Byrne, K. J. (2004). Efficacy of lifestyle modification for long-term weight control. *Obesity Research*, 12, 1515–1625.
- Walsh, B. T. (1993). Binge eating in bulimia nervosa. In C. G. Fairburn, & G. Wilson (Eds.), *Binge eating: Nature, assessment, and treatment* (pp 37–49) New York, NY: Guilford Press.
- Wang, G. J., Volkow, N. D., Logan, J., Pappas, N. R., Wong, W. Z., Netusil, N., et al. (2001). Brain dopamine and obesity. *The Lancet*, 357, 354–357.
- Wardle, J. (1987). Compulsive eating and dietary restraints. *British Journal of Clinical Psychology*, 26, 47–55.
- Wilfley, D. E., Bishop, M., Wilson, G. T., & Agras, W. S. (2007). Classification of eating disorders; Toward DSM-V. *International Journal of Eating Disorders*, 40, S123–S129.
- Wilfley, D. E., Friedman, M. A., Douchis, J. Z., Stein, R. I., Welch, R. R., & Ball, S. A. (2000). Comorbid psychopathology in binge eating disorder: Relation to eating disorder severity at baseline and following treatment. *Journal of Consulting and Clinical Psychology*, 68, 641–649.
- Wilfley, D. E., Welch, R. R., Stein, R. I., Spurrell, E. B., Cohen, L. R., Saelens, B. E., et al. (2002). A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge eating disorder. *Archives of General Psychiatry*, 59, 713–721.
- Wilson, G. T. (1991). The addiction model of eating disorders: A critical analysis. *Advances in Behaviour Research and Therapy*, 13, 27–72.
- Wilson, G. T. (1994). Behavioral treatment of obesity: Thirty years and counting. *Advances in Behaviour Research and Therapy*, 16, 31–75.
- Wilson, G. T. (2010). Cognitive behavior therapy for eating disorders. In W. S. Agras (Ed.), *Handbook of eating disorders*. (pp. 331–347). New York: Oxford University Press.
- Wilson, G. T., Fairburn, C. G., Agras, W. S., Walsh, B. T., & Kraemer, H. D. (2002). Cognitive behavior therapy for bulimia nervosa: Time course and mechanisms of change. *Journal of Consulting and Clinical Psychology*, 70, 267–274.
- Wilson, G. T., Grilo, C., & Vitousek, K. (2007). Psychological treatment of eating disorders. *American Psychologist*, 62, 199–216.
- Wilson, G. T., & Latner, J. (2001). Eating disorders and addiction. In M. M. Hetherington (Ed.), *Food cravings and addiction* (pp. 585–605). England: Leatherhead Publishers.
- Wilson, G. T., Perrin, N., Rosselli, F., Striegel-Moore, R., DeBar, L., & Kraemer, H. (2009). Beliefs about eating and eating disorders. *Eating Behaviors*, 10, 157–160.
- Wilson, G. T., Wilfley, D. E., Agras, W. S., & Bryson, S. W. (2010). Psychological treatments for binge eating disorder. *Archives of General Psychiatry*, 67, 94–101.
- Wing, R. R., Papandonatos, G., Fava, J. L., Gorin, A. A., Phelan, S., McCaffery, J., et al. (2008). Maintaining large weight losses: The role of behavioral and psychological

- factors. *Journal of Consulting and Clinical Psychology*, *76*, 1015–1021.
- Yanovski, S. Z. (2005). Pharmacotherapy for obesity—promise and uncertainty. *The New England Journal of Medicine*, *353*, 2187–2189.
- Yanovski, S. Z., Leet, M., Yanovski, J. A., Flood, M., Gold, P. W., Kissileff, H. J., et al. (1992). Food selection and intake of obese women with binge eating disorder. *American Journal of Clinical Nutrition*, *56*, 975–980.
- Yanovski, S. Z., Nelson, J. E., Dubbert, B. K., & Spitzer, R. L. (1993). Association of binge eating disorder and psychiatric comorbidity in obese subjects. *American Journal of Psychiatry*, *150*, 1472–1479.