



keynote article

Is obesity an eating disorder?

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The relationship between obesity and eating disorders has always attracted interest. In both conditions, weight, eating and body image are central features, and in both conditions societal and cultural influences play a major role. The two disorders have been linked with one another in many different aetiological models. Historically, obesity was considered to be a psychiatric disorder characterised by emotionally-driven abnormalities of eating behaviour (Kaplan & Kaplan, 1957). More recently, there have been suggestions that binge eating disorder plays a significant aetiological role in obesity (Hasler et al., 2004). Coming from an analysis of socio-cultural influences, societal idealisation of thinness has been blamed for body dissatisfaction which in turn has been hypothesised to drive efforts at weight control that are either too effective (resulting in anorexia nervosa and bulimia nervosa) or counterproductive (resulting in weight gain and obesity) (Ruderman & Wilson, 1979). From a similar perspective, obesity prevention programmes have been challenged as potentially increasing the risk of development of eating disorders (Carter & Bulik, 2008).

This paper presents a brief review of the phenomenology and aetiology of obesity to evaluate whether it is appropriate to identify it as a psychiatric disorder in which problems of eating behaviour and body image are primary features.

Eating disorders

DSM-IV recognises three eating disorder diagnoses: anorexia nervosa (AN), bulimia nervosa (BN), and eating disorder not otherwise specified (EDNOS) which currently includes binge eating disorder (BED) although it may ultimately become a separate diagnostic category. As indicated by the name, abnormalities of eating behaviour are common to all three (restriction, binge eating, purging and other compensatory behaviours). Disturbances of body image including over-valuation of weight and denial of seriousness of low body weight (in AN), are also critical diagnostic features of AN and BN. Severe underweight is only diagnostic for AN; body weight may be normal in BN and even high in BED. Amenorrhea is also only diagnostic for AN, where it is most likely part of a generalised endocrine disturbance caused by low body weight.



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The age of onset of eating disorders is usually in the teens and early twenties, with apparent cases of late-onset probably representing delayed diagnosis (Scholtz, Hill, & Lacey, 2009). Women have strikingly higher rates of AN and BN than men; although BED shows little evidence for sex differences (Decaluwe, Braet, & Fairburn, 2003). AN used to be thought to be more common in higher socioeconomic status (SES) groups, but current evidence indicates relatively little association with SES (Gard & Freeman, 1996); also true for BN and EDNOS. The prevalence of AN increased rapidly prior to the 1980s, but has been fairly stable since then, while BN, which was only identified in the late 1970s, showed increases over a longer period, though rates have stabilised (Currin, Schmidt, Treasure, & Jick, 2005).

In terms of aetiology, societal idealisation of thin body sizes for women, which began with the popularity of the English model Twiggy in the 1960s and has been perpetuated in the vogue for 'heroin-chic' and the 'size zero' model, is still accepted as playing a major role. However, the growing body of evidence that both genetic and early life factors are important has led to a more nuanced model in which individual susceptibility interacts with social pressures and life stress to determine risk (Mazzeo & Bulik, 2009).

Obesity

Obesity is defined entirely by weight (or more accurately, body fat); usually indexed by weight/height ratio (BMI) or waist circumference. High levels of ►

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body fat are necessary and sufficient for a diagnosis of obesity; which makes it inconsistent with a diagnosis of AN or BN, although not ruled out in EDNOS. There are established physiological correlates of obesity, including the elements of metabolic syndrome (e.g. insulin resistance, abdominal adiposity). Amenorrhea and infertility are also common in severely obese women. None of these are essential to the diagnosis of obesity, although they may be influential in determining priority for treatment.

Obesity rates were fairly stable in most developed countries until the early 1980s, but then started to rise; going from around 7% in 1984 to a current prevalence of 24% in the UK (Health Survey for England, 2007). Rates of obesity are similar in men and women, but overweight is more common in men. There is a weak negative association between obesity prevalence and SES in men, but a much stronger association in women, and high SES women are the only group in the UK to have shown no increase in obesity rates in the past 10 years. Age of onset is becoming earlier, with obesity emerging in the pre-school years and Type 2 diabetes (a comorbidity of obesity) appearing in paediatric populations for the first time (Jackson-Leach & Lobstein, 2006; Lobstein & Jackson-Leach, 2006). However, adiposity also increases throughout adult life, falling only in old age (Health Survey for England, 2007; <http://www.ic.nhs.uk/webfiles/publications/HSE07/HSE07%20Summary.pdf>).

In terms of aetiology, there are a small number of single gene disorders with obesity as a primary feature (e.g. congenital leptin deficiency); but these are rare (Farooqi & O'Rahilly, 2007). Most cases are so-called 'common obesity'; representing the upper part of the normal distribution of weight within the population. But even in common obesity there is strong evidence for genetic factors (Wardle, Carnell, Haworth, & Plomin, 2008). Changes in the food supply and in food consumption norms, together with reduction in the need for physical activity in daily life, have almost certainly provided the engine for increases in obesity over the past 25 years (Hill, Wyatt, Reed, & Peters, 2003), with genetic characteristics determining susceptibility to these environmental exposures (Friedman, 2009). A gene-environment interaction model gains support from evidence that the greatest gains in weight in recent years have been at the higher end of the weight distribution: thinner people are almost as thin as they ever were, but weights at the upper end of the weight distribution have increased greatly, with worryingly high increases in severe obesity (Wardle & Boniface, 2007).

Body image in obesity

Body image disturbance is not a diagnostic feature

of obesity, however, there has been speculation that unrealistically thin body shape ideals could play an important initiating role. The theory is that body dissatisfaction motivates attempts at weight control which are ultimately counterproductive because they disturb the appetite control system and increase the reward value of food. Essentially, this is the 'restraint-as-cause' model (body dissatisfaction → restraint → overeating → weight gain). The role of restraint in the aetiology of overeating has been an area of intensive psychological inquiry, although most of the literature has focused on women because they are the primary targets of the thin-ideal and eating disorders are a predominantly female problem. Restraint theory fits less comfortably in a context where both obesity and BED show little sign of sex differences.

The existing evidence does not support the idea that obese people either favour especially low body sizes or over-estimate their own body size. Most obese adults do not believe that they are obese and few obese adults either favour or seek extremely low body weights (Johnson, Cooke, Croker, & Wardle, 2008). Among parents, fewer than 20% of those with overweight or obese children identify their child's weight as too high (Carnell, Edwards, Croker, Boniface, & Wardle, 2005). Adolescents have been shown to be more likely to under-estimate than over-estimate their body size, with under-estimation increasing with higher body weight and despite the dramatic increases in obesity over the past decade, there has been no increase in the numbers of adolescents who feel overweight (Standley, Sullivan, & Wardle, 2009). The proportion of adults who regard themselves as overweight has actually fallen (Johnson et al., 2008).

Studies examining perceptual and cognitive aspects of body image in obesity have not demonstrated the kinds of systematic distortions that characterise eating disordered populations (Williamson, Muller, Reas, & Thaw, 1999). Nonetheless, most obese people are dissatisfied with their body size and shape, with a linear association between BMI and body dissatisfaction in adolescents (unpublished data from the HABITS study). Body dissatisfaction can be a significant source of psychological distress in some obese people; but in most cases, body dissatisfaction does not have pervasive effects on self-esteem or mood (Wadden, Foster, Stunkard, & Linowitz, 1989; Wardle, Williamson, Johnson, & Edwards, 2006). Self-esteem is not notably lower in obese than normal-weight groups (Wardle, 2005), nor are depression rates increased except among severely obese adults (Cooke & Wardle, 2005). However, emotional distress may undermine efforts at weight control, either by ►



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depleting self-regulatory ability or because strategies for coping with distress include over-eating. We have shown in adolescents that body dissatisfaction is associated with a range of eating problems (including binge eating), but the effect is not mediated by restraint, which if anything is associated with fewer signs of eating pathology (Johnson & Wardle, 2005).

Eating behaviour in obesity

Binge eating

Binge eating is the eating behaviour that has most often been implicated in obesity, although there is also evidence for a pattern of eating called 'night-eating syndrome' (Allison et al., 2006). Community studies show that binge eating rates are higher in obese than normal-weight adults, although still comparatively low (below 5%) (Smith, Marcus, Lewis, Fitzgibbon, & Schreiner, 1998). However, it appears that there are important differences between obese patients with and without BED. BED is associated with a range of psychiatric comorbidities including depression, anxiety disorders and personality disorders, and a stronger family history of substance abuse (Yanovski, Nelson, Dubbert, & Spitzer, 1993). BED is also associated with poorer treatment outcomes independently of psychiatric co-morbidities (Pagoto et al., 2007).

Emotional eating

Over-eating in response to negative emotional states has long been identified in the clinical literature, although the scientific evidence for its role in the aetiology of obesity is less clear (Allison & Heshka, 1993). An early review suggested that emotional eating (EE) was highly prevalent among obese adults in treatment (Ganley, 1989), although community studies have not always found such strong effects. In clinical populations, EE may be specifically linked with other signs of emotional distress: in a sample of severely obese adults, a quarter reported no EE at all while half had maximum scores on the EE scale of the Three Factor Eating Questionnaire (Karlsson, Persson, Sjostrom, & Sullivan, 2000). The high EE group were also more depressed and anxious, making it difficult to be certain whether they were always prone to emotional eating, or their current emotional state accentuated the problem.

External Eating

Two eating behaviour traits were strongly identified with obesity in a series of elegant laboratory studies carried out by Schachter and colleagues: responsiveness to external food cues and responsiveness to internal satiety cues (Schachter, 1968). These two aspects of eating behaviour are echoed in the dual-process model of food intake regulation (Berridge, 2009) which distinguishes homeostatic processes (related to biological need and

signalled by the state of the internal milieu), and hedonic processes (related to the perceived reward value of food).

Recent work using psychometric measures of external responsiveness in adults has produced mixed results with some studies finding higher external eating in the obese but others finding few differences (Cappelleri et al., 2009). However, the picture appears clearer in children. Jansen et al (2003) used an experimental protocol to show that obese children ate more than normal-weight controls following food cue exposure. We have also found that heavier children are more likely to overeat in response to exposure to food cues (Hill et al., 2008), and that parents of heavier children identify them as more responsive to food (Carnell & Wardle, 2008; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009). Adult-child differences in these results may be due to adults exerting more deliberate control.

A closely-related construct is the reinforcing value of food. Epstein has showed that obese children and adults will do relatively more 'work' for food than non-food rewards compared with normal-weight adults (Epstein, Leddy, Temple, & Faith, 2007), and we recently showed that the reinforcing value of food at age 7 predicted weight gain prospectively over a year (Hill, Saxton, Webber, Blundell, & Wardle, 2009). We also found that speed of eating – potentially an indicator of the reward value of food – was higher in heavier children (Llewellyn, van Jaarsveld, Boniface, Carnell, & Wardle, 2008).

Satiety responsiveness

Schachter's other signature obesogenic behaviour was reduced responsiveness to internal food cues. Since then, a number of studies have shown that obese children are less responsive to preloads (i.e. down-regulate meal consumption less than normal weight children if they had been given a pre-meal) (Birch & Fisher, 1998); and we have replicated this in the school setting, showing that heavier children are less responsive to variation in the caloric content of a mid-morning snack. Our recent work has taken this approach further by using a psychometric measure (completed by parents) (Wardle, Guthrie, Sanderson, & Rapoport, 2001) which makes it possible to assess very large samples. These data show that satiety responsiveness is strongly associated with weight in children, and is also heritable (Carnell, Haworth, Plomin, & Wardle, 2008) and associated with the weight-related gene, *FTO* (Wardle et al., 2008).

Is it useful to call obesity an eating disorder?

In terms of meeting any of the criteria for currently recognised eating disorders, this is not the case for ►

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the majority of the obese population. Most obese people experience some body dissatisfaction, but this is not more than might be expected given their deviations from ideals for body weight, and they do not consistently show any sign of disturbances of body image. Their eating behaviour is not highly restrictive. Binge eating is relatively uncommon except in the small sub-set that meet the diagnostic criteria for BED, and as such, could be defined as having an eating disorder. Comparing the epidemiology of obesity and eating disorders also gives little evidence for common processes: sex distributions, SES distributions, age of onset, and trends over time, are all different.

So do obese people eat 'normally'? Excluding the subset that meet diagnostic criteria for BED, most obese people show no sign of dramatic abnormalities in eating behaviour. However the studies described above link obesity with more subtle variations in appetite. Lower responsiveness to internal satiety cues and higher responsiveness to external food cues are heritable characteristics that are likely to confer vulnerability to over-eating in environmental conditions where the food supply is palatable and accessible (Carnell & Wardle, 2008; O'Rahilly & Farooqi, 2008). But these are not characteristics that are specific to obesity; rather they are traits expressed to a greater or lesser extent across the weight distribution.

We see no case for defining the majority of obese people as eating-disordered and little benefit in 'pathologising' an increasingly large proportion of the population. Nonetheless, sharing knowledge and expertise between the fields of obesity and eating disorders is likely to benefit both (Neumark-Sztainer, 2009), and a better understanding of the role of appetitive processes in obesity risk should offer new strategies for prevention and treatment. ■

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