Eating disorders and obesity: two sides of the same coin?

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Abstract. The eating disorders anorexia and bulimia nervosa have traditionally been regarded as entirely separate from obesity. Eating disorders have been regarded as Western culture-bound syndromes, arising in societies with excessive emphasis on weight, shape and appearance, and best treated by psychological therapies, in particular cognitive behavioural therapy or family-based interventions. In contrast, obesity has been considered a medical illness with metabolic and genetic origins, and thought to be best treated by mainstream medicine, involving dietary, drug or surgical treatment. We believe that this polarisation is fundamentally flawed, and research and treatment of both types of disorder would be better served by greater appreciation of the psychosocial components of obesity and the biological and genetic components of eating disorders. There are similarities in phenotype (such as excessive attempts at weight control, binge eating behaviours) and in risk factors (such as low self-esteem, external locus of control, childhood abuse and neglect, dieting, media exposure, body image dissatisfaction, weight-related teasing and shared susceptibility genes). One example of shared genetic risk is the brain-derived neurotrophic factor (BNDF) gene, in which the valine allele of the Val66Met amino acid polymorphism predisposes to obesity, whereas the methionine allele predisposes to eating disorders. Thus the evidence suggests that these disorders will have both shared and distinct susceptibility factors; some will predispose to both types of disorder, some will push in opposite directions, and some will separate them.

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Over the last 20 years obesity prevalence has reached epidemic proportions in many countries of the developed world (World Health Organization, 2000). Less frequently reported but also worrying has been the parallel rise in incidence at least of eating disorders such an Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Binge Eating Disorder (BED). Eating disorders have a much lower prevalence than obesity – the National Comorbidity Survey replication data (Hudson *et al*., 2007) found lifetime prevalence of AN, BN and BED to be 0.9%, 1.5% and 3.5% respectively in females and 0.3%, 0.5% and 2% in males – but a recent study suggests the prevalence of eating disorders has increased over the past decade (Hay *et al*., 2008). Thus it seems that the same sociocultural changes in society may be increasing the incidence of both eating disorders and obesity.

However, sociocultural factors are only part of the risk for these disorders; both obesity and eating disorders are significantly and similarly heritable. Twins studies have shown that obesity, either defined by body mass index or adiposity is around 60-70% genetic in origin (Stunkard *et al*., 1986). AN and BN have also been shown to be heritable, with twin studies also attributing around 60% of the variance to genetic factors (Bulik *et al*., 2000). History of parental obesity and also childhood obesity may differentiate AN from BN as both are risk factors for BN (Fairburn *et al*., 1999; Jacobi *et al*., 2004); this is consistent with the notion of partially shared genetic overlap between the two disorders. It is quite possible that a propensity to obesity interacts with dieting behaviour, given that obesity and BN are associated with dissatisfaction with weight, a desire to become slimmer (Shin *et al*., 2008), psychological distress (Gibson *et al*., 2008) and indeed, dieting itself (Terres *et al*., 2006).

Binge eating may represent the link between these disorders; 20-50% of people with obesity meet the criteria for binge eating disorder, whereas it is one of the diagnostic criteria for BN; most women with AN also engage in binge-eating behaviour. Binge eating is a controversial measure as the diagnostic criteria may not be ideal (Bulik *et al*., 2008); also there is no simple definition, as it can involve either an objective or subjective loss of control over food consumption. However, it is heritable (Bulik *et al*., 1998) and thus very likely to represent shared genetic risk between these disorders.

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Despite reasonable efforts to identify genes involved in the aetiology of these disorders, there are a limited number of robust findings, especially for eating disorders, where molecular genetic research is a more recent activity. For obesity, the FTO gene has been identified as a genetic risk factor (Frayling *et al*., 2007), with the 16% of adults who are homozygous for the risk allele weighing about 3 kilograms more and having a 1.67-fold increased risk of obesity when compared with those not inheriting a risk allele. More recent studies have uncovered a further series of obesity genes, including MC4R, TMEM18, KCTD15, GNPDA2, SH2B1, MTCH2, NEGR1, BDNF and ETV5 (Loos *et al*., 2008; Willer *et al*., 2009; Thorleifsson *et al*., 2009).

Thus, one could argue that people homozygous for the obesity protective allele, who have less fat mass than the population on average, might be at increased risk of AN. On the other hand, since binge eating and family history of obesity are features of BN, one could also argue that obesity genes will be shared with this diagnosis. Certainly for neuropsychiatric disorders such as bipolar disorder, schizophrenia, epilepsy and intellectual impairment, genetics has tended to clump disorders together rather than separate them (St Clair, 2009). However there are no large, well powered genetic studies of eating disorder which have addressed this issue, and the extent and nature of any shared risk genes genetic factors is largely unknown. However one intriguing finding is the association of Brain Derived Neurotrophic Factor (BDNF) with both obesity (Gunstad *et al*., 2006a; Thorleifsson *et al*., 2009) and eating disorders (Ribases *et al*., 2004), in consistent directions, i.e. the met66 allele of the valine66methionine polymorphism is associated with eating disorders and lower BMI and the val66 allele with higher BMI.

Socioeconomic status (SES) is one environmental factor which might differentiate between eating disorders and obesity (Taylor *et al*., 2005). SES shows a linear relationship with BMI, with higher rates of obesity in low SES groups and higher rates of eating disorders in high SES groups (O'Dea *et al*., 2008; Power *et al*., 2003; Nevonen *et al*., 2004). The higher rates of obesity in low SES groups can be attributed to the relatively low cost of high fat energy dense foods and lack of access or motivation to physical activity. Conversely, amongst high SES groups there will be an expanded range of diet choice, including more expensive foods such as food and vegetables, and propensity to exercise, greater pressure on weight and shape and higher parental expectations to succeed and conform.

Gender is also a factor. Women are disproportionately over-represented in both eating disorder and obesity (Hoek *et al*., 2003). The role of sex in modulating to weight and body fat distribution is undoubtedly metabolic but may also be due to differential sociocultural pressures on body shape, especially in relation to body dissatisfaction in women. Thus there is good evidence for shared risk factors between eating disorders and obesity involving dieting, media exposure, body image dissatisfaction and weight-related teasing (Haines & Neumark-Sztainer, 2006). Body dissatisfaction is found in obesity and across the eating disorder diagnoses (Troisi *et al*., 2006) and even part of the diagnostic criteria for AN, BN and BED (American Psychiatric Association, 1994). Likewise dieting is often a prominent risk factor in eating disorders, except for restricting anorexia, which has a constant low intake. Dieting with binging can also occur in weight gain and obesity (Roehrig *et al*., 2009), and is frequently found in obese individuals and those with eating disorders (Jacobi *et al*., 2004), particularly BN and BED.

Adolescence is the typical period of onset for EDs, The age of menarche has a common relationship with weight status being earlier in obese children and later in those who are underweight (Bau *et al*., 2009). Early menarche, controlled by hormone levels and affected by body fat content, is associated with obesity and early menarche and physical maturation and have been identified as risk factors for BN (Mangweth-Matzek *et al*., 2007).

Another risk factor which contributes towards both eating disorders and obesity is sexual abuse during childhood and adolescence (Rohede *et al*., 2008; Pinhas-Hamiel *et al*., 2009). Sexual abuse has been shown to be a significant and even specific risk factor for eating disorders, again particularly those involving binge eating (Preti *et al*., 2006; Pawlowska *et al*., 2007). Sexual abuse is a commonly reported antecedent event in those affected by eating disorders, particularly BN (Sanci *et al*., 2008). A longitudinal study found those who were sexually abused as a child were at elevated risk for subsequent eating pathology in early adulthood (Johnson *et al*., 2002). Characteristically however, sexual and physical abuse are acknowledged as non-specific risk factors for BN, associated with general development of psychiatric illness, rather than representing risk factors specific only to the development of eating disorders (Jacobi *et al*., 2004). Although there is a paucity of research in the area, there is evidence that abuse and neglect are also substantial risk factors for the development of obesity (Gilbert *et al*., 2009; Gunstad *et al*., 2006b; Lissau & Sørensen, 1994).

Stress and other psychological risk factors or trait disorders are seen in both eating disorders and obesity. Low self-esteem is common amongst sufferers of eating disorders (Brytek, 2006) which typically manifests as negative moods and depression; body dissatisfaction; social and

general anxiety and other internalising disorders. Likewise anxiety and depressive disorders are common comorbid diagnoses in individuals with eating disorders (Jordan *et al*., 2008) as is alcohol/drug abuse in those with eating disorders involving bingeing behaviours but not restrictive AN. This might suggest that traits commonly found in those with BN, BED, binge-purging AN and those reporting substance abuse, (namely impulsivity, high novelty-seeking, risk taking and reward response) mediate the link.

Whilst eating disorders are typically seen as based in psychological malfunction, psychological disorders are commonly thought of as stemming from obesity rather than being causative of obesity development. However there is some evidence that psychological ill health precedes obesity development (Pine *et al*., 2001; Hallstrom & Noppa, 1981). Certainly obesity and overweight and eating disorders show common co-morbidities with poor psychological health. Obesity is associated with depression, anxiety (at least in women) low self-esteem, an external locus of control, behavioural problems, and even parental depression (Davis *et al*., 2008). Whilst some of these associations are no doubt a result of obesity, it is likely that the direction of causality runs both ways. Psychological ill health itself can cause disordered eating which in turn leads to obesity (Lee, 2007). A more external locus of control has also been linked with BN (Shisslak *et al*., 1990; Grace *et al*.,1985), AN (Harding & Lachenmeyer, 1986), as well as disordered eating behaviours (Fouts *et al*., 2002) such as binge eating (Moyer *et al*., 1997). Some dimension of impulsivity is involved in obesity development (Goudie *et al*., 2008; Nederkoorn *et al*., 2006); for example disinhibited eating as measured by the Three factor eating questionnaire is associated with obesity (Yeomans *et al*., 2008). Impulsivity is considered a common trait in those with eating disorders and even considered a risk factor for the development of BN (Wonderlich *et al*., 2004; Fernandez-Aranda *et al*., 2008).

In summary, it is clear that disorder eating, which manifests itself as eating disorders with low or normal weight (e.g. AN and BN) and obesity, have both commonalities and differences in aetiology. This aetiology is highly complex, consisting of many genetic and environmental risk factors which have a small effect and interact with each other. There will be risk factors which are distinct between eating disorders and obesity, individual shared risk factors such as the BDNF gene Val66Met polymorphism which separate the phenotype by driving body fat composition in opposite directions, and fully shared risk factors such as childhood abuse and neglect.

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