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Obesity

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Definition

Obesity is defined by the World Health Organization (2014) as the accumulation of excess body fat, which may negatively impact health. As highlighted by this physiological definition, obesity is not a psychiatric condition, nor is it defined by any behavioral, psychological, or cognitive profile.

Measurement

Obesity is most validly assessed using body fat composition techniques, such as dual-energy X-ray absorptiometry or densitometry techniques. However, these techniques can be costly and are not appropriate or accessible in all settings. As such, the most commonly used indicator of obesity is body mass index (BMI). BMI is a weight-to-height ratio, calculated as weight in kilograms divided by height in meters squared. Obesity in adults is indicated by a BMI $\geq$30 kg/m^2. In children and adolescents, obesity is classified according to age and sex-specific BMI percentiles with reference to established growth curves. The use of BMI to assess obesity is particularly suited to population-based surveillance systems and screening tools. However, as BMI is not a direct indicator of body fat accumulation and cannot differentiate fat mass from fat-free mass, the accuracy with which BMI can detect excess adiposity has been questioned. A recent meta-analysis suggests that BMI has a high specificity but low sensitivity to detect excess adiposity, indicating that a BMI cutoff of 30 kg/m^2 reliably detects the absence of excess adiposity but can often fail to detect the presence of excess adiposity (Okorodudu et al. 2010).

Current Knowledge

Epidemiology

Obesity is associated with poorer physical health (e.g., type 2 diabetes, cardiovascular disease, hypertension, asthma, some cancers, and sleep apnea), quality of life, and mental health (e.g., depression). A comprehensive review and analysis conducted by Ng and colleagues (2014) estimated global trends in obesity prevalence from 1980 to 2013. In this time period, obesity prevalence increased in both developed and developing countries, although rates of obesity have stabilized in developed countries in recent years. Globally, it is estimated that 671 million people had obesity in 2013; 62% of these individuals lived in developing countries. Obesity prevalence is

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higher among developed than developing countries overall, but substantial variability is observed within these categories. Obesity prevalence is higher among women than men in both developed and developing countries. The prevalence estimates of obesity among western countries/regions in 2013 were as follows: Western Europe (21.0% women, 20.5% men, 6.4% girls, 7.2% boys), North America (32.5% women, 30.6% men, 13.0% girls, 12.1% boys), and Australia (29.8% women, 27.5% men, 7.3% girls, 7.0% boys).

Etiology

The concept of energy balance, informed by the thermodynamic law of energy conservation, is universally invoked to explain the physiological underpinnings of obesity. Put simply, it is argued that body fat accumulation occurs when energy in (i.e., calories consumed in food) exceeds energy out (i.e., energy burned in bodily processes or expelled from the body). Therefore, it is commonly thought that obesity is caused by eating too much and exercising too little. While diet composition and physical activity are undoubtedly involved, research indicates that the etiology of obesity is decidedly more complex than this simple account.

There is increasing evidence that metabolic and hormonal processes must be considered as part of the energy balance equation. Diet macronutrient content, stress, sedentary behavior, and sleep can influence metabolic processes, which in turn influence the regulation of body weight (see review by Wells 2013). Further investigation of the complex physiological etiology of obesity is required.

In addition, a range of social and environmental factors related to the food and physical activity environment has also been implicated in the etiology of obesity. The impact of social and environmental factors may be inequitably distributed across the population, such that those with lower socioeconomic status receive the greatest disadvantage. For example, reduced access to physical activity facilities and safe, walkable neighborhoods in lower socioeconomic status areas may contribute to social disparities in physical activity. While individuals are often held personally responsible for their obesity, this neglects the complex social and environmental factors that may influence and restrict personal choices and contribute to obesity at the population level. Further work is needed to delineate the role of social and environmental factors in the etiology of obesity (for a review see Kirk et al. 2010).

Comorbidity with Eating Disorders

In adult community samples, obesity is more prevalent among those with an eating disorder than those without an eating disorder. In the 2007 British National Psychiatric Morbidity Survey, a population-based survey of adults aged 50 years and above, the prevalence of obesity was significantly greater among those who screened positively for a current eating disorder (38.7%) than among those who did not screen positively for a current eating disorder (19.5%) (Ng et al. 2013). After controlling for age, gender, race, marital status, education, and psychiatric disorder, positive screening for an eating disorder was associated with a 2.31 increased likelihood of current obesity (Ng et al. 2013). There is also evidence from an Australian sample that the cooccurrence of obesity and eating-disordered behaviors (i.e., binge eating, purging, and fasting) may be increasing in the general population, from 1% in 1995 to 3.5% in 2005 (Darby et al. 2009).

The comorbidity between obesity and eating disorders varies by eating disorder diagnosis. Anorexia nervosa (AN) is characterized by a low BMI (e.g., <18.5 kg/m^2), and therefore current AN cannot be comorbid with current obesity. Bulimia nervosa (BN) and binge eating disorder (BED) are both characterized by repeated episodes of objective binge eating, which involves episodes of eating a large amount of food with a sense of having lost control over eating. In addition, those with BN engage in behavior designed to compensate for the calories consumed during binge eating, such as self-induced vomiting, laxative abuse, strict dieting, or excessive exercise, while those with BED do not. The population-based US National Comorbidity Survey of adults investigated the relationship between current

obesity and a lifetime history of each eating disorder diagnostic category (Hudson et al. 2007). The prevalence of current obesity was lower among those with a lifetime history of AN and did not differ between those with a lifetime history of BN and those without a lifetime history of an eating disorder. However, those with a lifetime history of BED had almost a fivefold increased likelihood of current "severe" obesity (BMI >40 kg/m^2) than those without a lifetime history of BED. Similarly to community samples, obesity is more commonly comorbid with BED than BN in adult treatment seeking samples.

As with adults, obesity is also comorbid with eating disorders in adolescents. In an adolescent community sample in Canada, adolescents with obesity were more likely to have a binge-type eating disorder than adolescents without obesity (Flament et al. 2015). Specifically, girls with obesity were 3.27 times more likely to have a binge-type eating disorder, and boys with obesity were 7.86 times more likely to have a binge-type eating disorder than those adolescents without obesity.

Current Treatments

Behavioral and Cognitive-Behavioral Therapy

Behavioral weight loss treatments typically aim to decrease calorie consumption and increase physical activity in order to induce a negative energy balance. Cognitive-behavioral weight loss treatments employ similar behavioral techniques but also focus on reducing mood-related eating, improving body image, and implementing strategies for weight loss maintenance. In the short term, both behavioral and cognitive-behavioral weight loss interventions are typically successful in inducing moderate weight loss. However, weight is frequently regained in the long term following the cessation of treatment. An illustrative example is a randomized controlled trial for obesity treatments conducted at Oxford University (Cooper et al. 2010). Patients lost weight following 44 weeks of either behavioral therapy (12.7% of body weight lost) or cognitive-behavioral therapy (8.9% of body weight lost). However, most of this weight was regained 3 years following treatment; 89.9% of lost body weight was regained following behavior therapy, and 88.6% of lost body weight was regained following cognitive-behavioral therapy. If such treatments are to be offered, ethical practice requires that all patients undergoing behavioral or cognitive-behavioral treatment for obesity are informed of the poor long-term maintenance of weight loss following treatment. Otherwise, participants may experience psychological harm from perceived failure to maintain weight loss.

Bariatric Surgery

Bariatric surgery treatments for obesity include gastric bypass, adjustable gastric banding, and sleeve gastrectomy procedures. On average, bariatric surgery results in large reductions in weight (e.g., 60% loss in excess body weight 1 year post-surgery) that are largely maintained over the follow-up period of treatment trials. Surgical complications (e.g., bleeding, vomiting, or leaking) occur following 10–17% of bariatric surgery procedures, and 7% of procedures require reoperation. A mortality rate of 0.31% is observed postoperatively (for a meta-analysis, see Chang et al. 2014).

Current Issues

Weight Stigma and Obesity

People with obesity can experience significant weight stigma across multiple settings, including healthcare, education, family, and workplace environments. Negative attitudes and stereotypes about people with obesity are frequently endorsed. While it is popularly thought that weight stigma increases motivation for health behavior change, research indicates that weight stigma has a negative impact on psychological health (e.g., low self-esteem, depressive symptoms, and body image) and healthcare utilization. The public health implications of weight stigma and weight bias, particularly in healthcare settings, are receiving growing attention (for a review, see Puhl and Heuer 2009).

Understanding the Comorbidity Between Obesity and Eating Disorders

The causes of the observed comorbidity between obesity and eating disorders require further investigation. It is likely that the comorbidity between obesity and eating disorders is partly accounted for by shared risk factors (i.e., factors that prospectively predict the development of eating disorders and obesity). Sánchez-Carracedo et al. (2012) argue for a multilevel socio-ecological framework for understanding shared risk factors across eating and weight-related problems, including individual level factors (e.g., dieting and body dissatisfaction), family- and peer-level factors (e.g., weight-related teasing and modeling of diet behavior), and societal level factors (e.g., weight- and shape-related sociocultural norms and weight stigma or discrimination). Evidence for shared risk factors supports the use of prevention strategies that concurrently target both obesity and eating disorder risk factors. School-based prevention programs have been developed that target shared risk factors, including body dissatisfaction and dieting, and work is likely to build in this area.

In addition to shared risk factors, obesity and eating disorders may be linked by indirect causal pathways. First, research suggests that obesity may sometimes precipitate onset of both restrictive-type eating disorders and binge-type eating disorders. In this manner, obesity may lead to body dissatisfaction, weight-related teasing, and dieting or unhealthy weight control behavior, which in turn increase risk for eating disorders. In the reverse direction, binge eating among those with BED may increase risk for obesity, although this requires further investigation.

Future Directions

Complex problems such as obesity require complex, systemic, sensitive, multifactor solutions. Those leading the way in this area highlight the importance of promoting individual and community health by creating inclusive environments that support mental and physical well-being. In addition to advocating a multilevel, population-based, government-wide strategy, this approach adopts a focus on well-being rather than weight and may be well suited to the prevention of both obesity and eating disorders (e.g., see Provincial Health Services Authority 2013). This approach may represent the future direction of prevention in this area but substantial work is needed.

Cross-References

- ▶ Body Mass Index: Self and Parents
- ▶ Obesity and Eating Disorders

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Obesity and Eating Disorders

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Synonyms

Co-morbidities; Eating disorders; Obesity; Risk factors

Definitions

Obesity

Approximately one-third of children and two-thirds of adults in the United States are overweight or obese, utilizing the definitions of a body mass index (BMI, weight in kg/height in m^2) $\geq$25 for overweight and $\geq$30 for obesity in adults and a BMI percentile (based on age and sex) $\geq$85th percentile for overweight and $\geq$95th percentile for obesity in children. Although the rates of overweight and obesity have plateaued, the rates of extreme obesity among children and adults have continued to rise. These trends are extremely problematic given that overweight and obesity in both childhood and adulthood are associated with myriad health consequences, including increased insulin resistance, type II diabetes, fatty liver disease, hypertension, dyslipidemia, orthopedic complications, and cardiovascular complications. Additionally, the psychosocial consequences of obesity are staggering, with significantly lower quality of life and self-esteem among individuals with obesity across the lifespan. Increased rates of mental health conditions are also present in an overweight/obese population, including eating disturbances. These include diagnosable eating disorders based on DSM-5 criteria as well as subthreshold pathological eating behaviors that contribute to significant distress and impairment above and beyond the impairment directly related to obesity. The development and maintenance of eating disorders and obesity can be intertwined, with high rates of comorbidity due in part to a host of shared risk factors and the fact that each disorder can uniquely precipitate the development of the other.

Current Knowledge

Prevalence

Due in large part to increasing rates of severe obesity, rates of comorbidity between obesity and eating disordered behaviors are on the rise as well. Regardless of current weight, individuals with eating disorders, particularly binge-type pathology, frequently have a history of childhood

obesity or lifetime obesity (i.e., those with a history of childhood obesity and/or a BMI $\geq$30 during adulthood). More specifically, up to one-third of individuals with binge eating disorder (BED) and up to 40% of those with bulimia nervosa (BN) report a history of childhood obesity. Lifetime obesity rates are even higher among those with BED; upwards of 80% of individuals with BED have a history of obesity at some point during the lifespan. Those with subthreshold pathology consistent with other specified feeding and eating disorders (OSFED) have lifetime obesity rates lower than what is seen in BED or BN, but rates still approach 20%. Comorbidities between obesity and eating disorders are similarly high when examining prevalence rates of concurrent obesity and eating pathology. Approximately half of those with BED are currently overweight, according to community studies, and up to 30% of individuals presenting for weight loss treatment meet criteria for BED. Furthermore, community studies also demonstrate that up to two-thirds of those with BN are currently overweight or obese. While anorexia nervosa (AN), by definition, cannot co-occur with obesity, up to 5% of individuals with diagnoses of AN have a lifetime history of obesity, and among adolescents seeking treatment for restrictive eating disorders (i.e., AN and restrictive-type OSFED), approximately one-third have a history of overweight or obesity.

Correlates and Consequences

Independently, eating disorders and obesity are associated with myriad negative physical and psychosocial consequences, including mortality for the most severe forms of both conditions (e.g., AN, morbid obesity). When obesity and some types of eating pathology are experienced concurrently, particularly binge-type pathology, symptomatology and impairment are often exacerbated compared to when either problem is experienced on its own. Higher BMI and lifetime obesity are positively correlated with severity of eating pathology as well as rates of general psychopathology; thus, among those with binge-type eating pathology, individuals with the highest levels of obesity are likely to experience the most serious forms of disordered eating paired with other psychological comorbidities. Furthermore, individuals with obesity and eating disordered attitudes and behaviors, particularly those evidencing an overvaluation of weight and shape, experience more severe psychosocial consequences characteristic of eating disorders, including depressive symptoms, anxiety, low self-esteem, social problems, and substance use.

Additionally, obesity and eating disorders can perpetuate each other. Disordered eating behaviors, particularly unhealthy weight control practices and binge eating behaviors, put individuals at risk for weight gain. A hallmark behavior of both BED and BN is the consumption of objectively large amounts of food paired with a loss of control over those eating episodes. Thus, individuals with BED or BN are regularly consuming excess calories, and this calorie surplus can contribute to excess weight gain. It should be noted that compensatory behaviors utilized by those with BN do not remove all excess calories from the body; some of the calories consumed during binge episodes are absorbed regardless of subsequent behavioral attempts to prevent this process. In addition to binge eating, both cross-sectional and prospective studies have identified loss of control eating and unhealthy weight control strategies to be risk factors for obesity via an increase in weight and/or body fat. Thus, eating disordered behaviors contribute to the maintenance of overweight and obesity and may exacerbate the medical comorbidities associated with excess weight. Additionally, the converse is also true – individuals with overweight or obesity are at risk for the development of eating pathology, particularly unhealthy weight control strategies, via the initiation of restrictive and maladaptive dieting behaviors, thus, perpetuating the cycle between weight gain and disordered eating.

Comorbid eating pathology and obesity may also make both conditions more challenging to treat utilizing traditional treatment methodologies. Given the link between behaviors like binge eating and weight gain, achieving weight loss in behavioral weight loss treatments may be more challenging until the eating disorder is addressed, particularly in individuals with binge-purge pathology. Early weight loss success (e.g.,

in the first 2 months) of a behavioral weight loss treatment predicts long-term weight loss success; therefore, individuals with eating pathology, who may struggle with initial weight loss in a behavioral weight loss program due to binge eating behaviors, may experience more difficulty with weight loss and maintenance over time. In such cases, it would be best to treat the eating disorder before behavioral weight loss efforts are initiated so as not to exacerbate weight and shape concerns and maladaptive weight control behaviors via an emphasis on weight loss and to increase the likelihood of sustained weight loss success. Additionally, high rates of weight and shape concerns as well as experiences of weight-related stigma and teasing in individuals with obesity may precipitate avoidance of physical activity, further decreasing the potential to create a calorie deficit among those in behavioral weight loss treatments and further complicating the goal of reducing weight and shape concerns among those in treatment for eating pathology where overvaluation of weight and shape is a primary feature. Thus, addressing body image and the social skills needed to address teasing and stigmatization may be particularly important in order for interventions for obesity and eating pathology to be as successful as possible.

Risk Factors and Etiology

Childhood obesity. A primary explanation for the link between eating disorders and obesity is the fact that the two conditions have shared risk and maintenance factors. One of the most robust predictors of obesity throughout the lifespan is childhood obesity, given that it tracks into adulthood; approximately 82% of children with obesity go on to become adults with obesity, and concordance rates increase with severity of childhood obesity. The commonly held view that children will grow out of their obesity as they age has no empirical support. Childhood obesity is also associated with greater risk for the development of not only eating disorders but a variety of eating disordered behaviors, including dieting and other maladaptive weight control methods, overvaluation of weight and shape, and loss of control and binge eating in both children and adults.

Dieting behavior. One of the mechanisms by which childhood obesity contributes to adult obesity and eating disorders may be the initiation of dieting. Dieting behavior has shown to be a robust predictor of the presence of eating disorders and obesity, especially when dieting is initiated at a younger age. Although dietary modifications are the foundation of weight loss, research has consistently demonstrated that self-directed dieting and other restrictive weight control methods actually predict the development and maintenance of obesity. According to restraint theory, binge eating behaviors occur when individuals who are dieting interpret an episode of eating as being in violation of their strict dietary rules, and subsequently abandon their dietary restriction temporarily, giving rise to loss of control eating. Over time, such eating habits can contribute to an increase in adiposity, the maintenance of obesity, and the development of eating disorders. Dieting within the household may precipitate dieting attempts among youth, in particular. Parents who frequently focus on body weight and image in a maladaptive manner significantly increase the likelihood of their children developing disordered eating. One mechanism by which parental dieting may precipitate eating pathology in the child is modeling. Adolescents often imitate or internalize the eating attitudes and behaviors of their parents, and when their parental model is demonstrating eating disordered thoughts, the youth may experience elevated shape and weight concerns and adopt similarly maladaptive dieting behaviors, putting them at greater risk for the development of overweight and obesity.

Body dissatisfaction. Weight and shape concerns are common among individuals with overweight and obesity and also frequently occur among individuals of normal weight, to an extent that body image disturbances are considered to be normative in western cultures, particularly among young women. In turn, body image disturbances are a well-known risk factor for the development of negative affect and disordered eating. Over time, these eating disordered behaviors can contribute to excess weight gain and obesity. Media exposure is one of the avenues by which body image disturbance develops. The emphasis of

body size and shape in the media, paired with an internalization of this unattainable thin ideal, or in other words, accepting the notion that one must be thin to be valued, contributes to body dissatisfaction. Weight- and appearance-related teasing is another mechanism for the development of body dissatisfaction. Children with overweight and obesity are often victims of bullying and teasing by their peers, and these experiences can contribute to the development of negative self-esteem and body image, particularly among those youth who are more interpersonally sensitive or have internalized the thin ideal.

Current Controversies

Risk of inducing eating disorders via *obesity management*. Due to the multiple connections between obesity and disordered eating, there is a fear among some in the psychological community that treating obesity can lead to the development of an eating disorder via the focus on calorie restriction. However, behavioral weight loss programs in youth have been shown to actually reduce aspects of disordered eating, including drive for thinness, unhealthy dieting, and weight and shape concerns. Similarly, eating disorder prevention programs have been shown to reduce eating symptomatology and obesity risk. Supervised behavioral lifestyle programs for weight loss are far superior to personal dieting attempts. Diets that are promulgated by the mass media, commercial programs, and peers often are highly restrictive and unsustainable methods that result in rapid weight loss, feelings of deprivation, and, frequently, a weight rebound via the initiation of loss of control eating behaviors. On the other hand, behavioral weight loss programs stress meal regulation, sensible calorie reductions, and slow, steady weight loss, components that predict sustainable weight loss in the long term.

Due to issues concerning cost and reimbursement for care, individuals may not have access to these evidence-based behavioral lifestyle programs that have been shown to produce clinically significant reductions in body weight in healthy, sustainable ways. Thus, a multipronged approach is needed; the psychological community should continue to work toward increasing access to evidence-based weight management programs, educating the public about the consequences of engaging in self-directed restrictive dieting, and screening and continuously monitoring appropriately for the presence of eating disordered attitudes and behaviors in individuals who present for behavioral weight loss treatment. Referrals for eating disorder treatment with a specialist should be made and weight loss interventions halted if it becomes clear that significant disordered eating behaviors are primary in the individual's presentation and do not cease after meal regulation strategies are initiated in a behavioral weight loss program. However, these complications are rare and typically occur as a result of pre-existing eating pathology that was not appropriately screened for or addressed prior to treatment, as opposed to being induced by the behavioral weight loss intervention.

Future Directions

Given the strong association between overweight and obesity throughout the lifespan and the development of psychopathology, including eating disorders, an important area of investigation is obesity prevention and early intervention. While behavioral lifestyle programs yield clinically meaningful reductions in weight, long-term maintenance of weight loss is challenging and access to treatment is limited. Obesity experts should continue to work toward the goals of implementing robust programs that are cost-effective, are wide in reach and scale, and target individuals with obesity before their obesity becomes severe and chronic. Additionally, given the comorbidity between obesity and eating pathology, more research examining comprehensive treatments that involve both aspects of treatments for obesity (e.g., self-monitoring, calorie modification, physical activity promotion) and eating disorders (e.g., cognitive restructuring, reducing shape and weight concerns, reducing binge eating) is warranted, for instance, cognitive behavioral therapy. Such treatment could prove to be cost-effective in comparison to treating comorbid obesity and eating disorders separately. Overall, given

the prevalence of overweight and obesity, the link between obesity and eating disorders, and the negative comorbidities and outcomes associated with both disorders, establishing procedures for addressing both disorders concurrently is of paramount importance.

Cross-References

- Anorexia Nervosa
- Binge-Eating Disorder
- Body Mass Index: Self and Parents
- Body Image
- Bulimia Nervosa
- Comorbidities: Anxiety Disorders
- Coping: Escape Avoidance
- Depressive Disorders
- Diabetes
- Loss of Control (LOC) Eating in Children
- Obesity
- Other Specified Feeding or Eating Disorder (OSFED)
- Overevaluation of Shape and Weight and Its Assessment
- Self-Criticism and Low Self-Esteem
- Spiral Model of Dieting and Disordered Eating
- Structured Clinical Interview for DSM-IV (SCID)

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Obesity Prevention and Its Impact on Eating Disorders

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Introduction

Over the past two decades, the prevalence of children and adults with overweight or obesity has risen dramatically throughout the United States and in many parts of the world, making obesity prevention a top public health priority. In the United States, overweight or obesity affects about one-third of youth and two-thirds of adults. Further, overweight and obesity are unequally distributed by gender, family income, and race; the prevalence in certain subgroups of children and adults approaches 50% and 80%, respectively. Significant health consequences have been documented for individuals whose weight-to-height ratio identifies them as "obese"

including metabolic syndrome, type 2 diabetes, hypertension, hyperlipidemia, sleep disorders, and, among females, polycystic ovary syndrome. Individuals who are overweight also report psychosocial difficulties, such as depression, lower self-esteem, lower quality of life, and less perceived social acceptance. Both the physical and emotional consequences of overweight have the potential to place a significant burden on the individual, healthcare system, and society in general when the prevalence of overweight is so high.

Public health and societal focus on obesity has increased together with the growing epidemic; yet, despite this recent intense national focus on obesity and obesity prevention, obesity prevention efforts have been largely unsuccessful. As public health professionals continue to explore ways to successfully prevent and reduce obesity across the lifespan, it is crucial for them to also consider what impact, if any, their obesity prevention efforts have on eating disorders and eating disorder risk factors. This question is particularly significant given that the evidence is mounting that eating disorders and obesity are linked in a myriad of important ways.

Comorbidity of Obesity and Eating Disorders

Research suggests that obesity, binge eating, disordered eating, and eating disorders represent a spectrum of unhealthy weight-related problems that often occur simultaneously, increase in severity over time, and often lead to the onset of different weight-related problems. One research study in particular sought to describe in full the co-occurrence of disordered eating behaviors, binge eating, and overweight within a population-based sample of adolescents; the level of co-occurrence was high.

Among young women, 44% had at least one of the three weight-related problems, and 13.4% had more than one problematic outcome. Among overweight girls, 10% reported both binge eating and disordered eating behaviors, 6.4% reported binge eating only, and 23.5% reported disordered eating behaviors only. Among young men, the overall prevalence of these behaviors, and thus levels of co-occurrence, was lower; 29.2% had at least one of the three problematic weight-related outcomes, and 4.7% had more than one weight-related problem. Among overweight boys, 1.9% reported both binge eating and disordered eating behaviors, 2.7% reported binge eating only, and 12.3% reported disordered eating behaviors only. These self-reported data serve to confirm that the prevalence of weight-related problems is high and provide evidence to suggest that for many individuals, these problems occur simultaneously. Public health researchers who seek to develop effective interventions aimed at decreasing obesity must consider this high level of co-occurrence during the intervention design process.

One way to view weight-related disorders, including obesity, disordered eating, and eating disorders, that has been discussed some within the literature is on a spectrum. This proposed "spectrum of weight-related disorders" has obesity on one end, anorexia and bulimia nervosa on another end, and a range of other weight-related disorders in the middle, including anorexia or bulimic behaviors, unhealthy dieting, and overeating. This spectrum approach does have its limitations; for example, we know that anorexia nervosa is not the opposite of obesity, and, as discussed above, we know that obesity and disordered eating can, and often do, co-occur. However, thinking of this spectrum of weight-related disorders can be very useful for considering and understanding how we might work toward simultaneous prevention of a variety of weight-related disorders.

Obesity and Eating Disorders: Shared Risk Factors

In response to the high prevalence and co-occurrence of obesity, eating disorders, and disordered eating behaviors, researchers in both the obesity and eating disorder fields have sought to identify shared risk factors for the development of the spectrum of weight-related problems; to date, research has identified several risk factors shared between these weight-related disorders.

- *Dieting*: Dieting has been commonplace within American culture for decades. The assumptions that dieting will yield the long-term benefits of improved health and sustained weight

loss have been rarely questioned, until recently. Evidence from cross-sectional and prospective research studies has established dieting as a known risk factor for the development of eating disorders among youth and young adults. A number of studies involving clinical samples have found that the majority of individuals with eating disorders report that they started to diet before they initiated their disordered eating behaviors. Further evidence of the association is provided by prospective studies within community samples of adolescents. Within observational studies, self-reported dieting has been shown to predict increased risk of disordered eating behaviors and subthreshold eating disorders. These results suggest that self-reported dieting among adolescents may lead to more severe eating pathology. Dieting has also been shown to be ineffective at achieving sustained weight loss in the majority of individuals. Cross-sectional data have consistently shown BMI to be positively correlated with dieting behaviors among children, adolescents, and adults. While these cross-sectional data do not provide evidence regarding the direction of the association, prospective data from several large observational studies have shown that dieting predicts weight gain over time.

- *Media use*: Media are ubiquitous in Western society. Media use and the internalization of the messages promoted by the media have been explored as putative risk factors for both obesity and eating disorders. In general, cross-sectional and prospective studies have shown a positive association between media use and BMI in children and adolescents. Stronger evidence of this association has also been provided by two school-based obesity prevention intervention trials, which found that reducing television use predicted decreases in obesity prevalence and BMI in youth. Additionally, due to the ubiquitous nature of media in our culture and its relentless promotion of the thin beauty ideal, media use has been considered a potential risk factor for the development of eating disorders for a long time. Several cross-sectional surveys and prospective studies have found a positive association between media use and disordered eating behaviors in both children and adolescents; numerous laboratory-based experiments have also demonstrated short-term effects of exposure to media images.
- *Body dissatisfaction*: Body dissatisfaction is common, particularly among children and adolescents of both genders, as well as young adult and adult women. Body dissatisfaction has been associated with binge eating, with worse nutritional and physical activity behaviors, and with increased weight gain over time. In short, youth who like their bodies are more motivated to take better care of them by making healthy, sustainable, lifestyle choices. Body dissatisfaction is also one of the most consistent and robust risk factors for the development of eating disorders.
- *Weight-based teasing*: Weight-based teasing is prevalent among adolescents and children with overweight youth reporting higher levels of weight-related teasing compared to their normal-weight peers. Weight-based teasing has been shown to be prospectively associated with binge eating among both overweight and normal-weight individuals. It is thought that being teased about weight might lead to depression or body dissatisfaction, which may lead to binge eating behaviors. Binge eating, in turn, can lead to increased risk for weight gain and obesity. Teasing has also been associated with disordered eating behaviors, such as purging and restricting behaviors.

An Integrated Approach for the Prevention of Obesity and Eating Disorders

As a result of the high prevalence of obesity and eating disorders, the evidence suggesting that these weight-related problems often coexist and the knowledge that these weight-related problems share a common set of risk factors, there has been increasing interest among obesity and eating disorder researchers to develop interventions that are successful at the prevention of this broad spectrum of weight-related problems. There are both conceptual and practical reasons for pursuing an integrated approach to the prevention of obesity

and eating disorders. At a conceptual level, research has demonstrated that weight-related disorders may not be conceptually distinct from one another. Research has shown that multiple weight-related disorders can coexist within an individual and that there might be crossover from one disorder to another within an individual. Practical considerations provide further justification for the integration of eating disorder and obesity prevention. For example, time constraints are often noted as an obstacle for prevention programs within school or clinical settings; however, by addressing shared risk factors, less time is needed to address the prevention of the broad spectrum of weight-related disorders, rather than addressing each separately. Further, this type of approach helps to avoid conflicting messages that may confuse or frustrate those on the receiving end of these messages.

Although there are both conceptual and practical reasons for pursuing an integrated approach to the prevention of obesity and eating disorders, there are also several challenges associated with this integrated approach. One of these challenges stems from the widely varied perspectives of those professionals who work primarily in the obesity field, as compared to those professionals working primarily in the eating disorder prevention field. These two fields, while interconnected, each boast their own set of research priorities, a language common to their specific field and a set of messages that they hope to use to reach out to public with the goal of prevention in mind. Finding a way to build a bridge between these two unique fields is certainly its own challenge, but discussions across disciplines and resulting controversies are likely to yield to the development of better prevention-focused interventions in the long term. It is through this type of challenging discussion across disciplines that we are able to best challenge our own point of view and to find new partners for collaborations.

Another inherent challenge to the development of integrated prevention programs is the challenge of obtaining funding. Often, institutions are interested in providing funds for projects that closely align with the mission of the institution; it can be easiest to make this connect in an application when the target health issue is very clearly defined. Reviewers critiquing a program aimed at preventing the broad spectrum of weight-related problems might raise concerns about whether there are enough shared risk factors between obesity and eating disorders to warrant an integrated approach; it is the job of the individual writing the grant to shoulder the burden of making this connection clear to the potential funder.

Finally, an additional challenge to the development of integrated prevention approaches lies in the struggle to develop suitable messages. It is considerably, and understandably, more complex to develop messages aimed at preventing the broad spectrum of weight-related disorders than it is to develop a message for a specific disorder (e.g., obesity). The development of messages suitable for the prevention of a broad spectrum of weight-related disorders remains an area in which additional research is needed.

Integrated Programming and Next Steps

Research to date on the prevalence and consequences of obesity, eating disorders, and disordered eating behaviors clearly supports the importance of interventions aimed at preventing the broad spectrum of weight-related disorders. Along these lines, it is important to note highlight that there have been a handful of intervention studies that have successfully pursued an integrative approach to the prevention of a spectrum of weight-related problems. An early example of an intervention that aimed to prevent both obesity and eating disorders was *The Weigh to Eat*, a 10-week school-based program created for Israeli school girls. *The Weigh to Eat* included components addressing nutrition, physical activity, healthy weight maintenance, media literacy, and assertiveness training on social pressure in the context of food, eating, and weight. *Planet Health* is another school-based program that, while originally designed as an obesity prevention program, also demonstrated positive effects on eating disorder pathology. *Planet Health* is a 2-year-long intervention which is delivered to students by

their teachers within the classroom setting; this intervention has been evaluated in three separate research trials over the past decade. Researchers at Stanford created *Student Bodies*, a program for college women with high weight and shape concerns. This program ran online for 8 weeks and focused on topics including body dissatisfaction and body image, healthy weight management and nutrition, and increasing knowledge of eating disorder risk factors. *The Body Project* is a dissonance-based body-acceptance program designed to help high school girls and college-aged women to resist the thin-ideal standard of beauty within our culture with the goal of reducing their engagement in disordered eating behaviors. Adaptations of *The Body Project* have been made since the original design, and this program has been disseminated widely to classrooms, colleges, and sororities across the United States. The *Healthy Weight* program, which was originally designed as a comparison intervention in the *Body Project* research trials, is another program that has demonstrated success in promoting healthy weight control techniques as well as reducing eating disorder pathology among participants. *New Moves* is a school-based program geared toward high school girls who are overweight or at risk for becoming overweight. This program includes elements of both eating disorder and obesity prevention and utilizes principals of social cognitive theory to promote behavior change. Finally, *Media Smart* is a program that has had demonstrated success in reducing both disordered eating and obesity risk factors within adolescent males and females in Australia. *Media Smart* was presented over a series of eight lessons and focused on reducing media internalization, perceived pressure to be thin/muscular, and weight concern.

These programs are examples of intervention studies that have demonstrated success in reaching young people with educational programming aimed at reducing risk factors common across the spectrum of weight-related disorders. That said, a crucial step moving forward will be to find a way to *continue* to bridge the gap between professionals in the fields of obesity and eating disorders, allowing for the easy sharing of ideas for prevention across disciplines. It is through the sharing of ideas and resources that these two fields will be able to pursue the cultivation of programs focused on the integrated prevention of obesity and eating disorders. Further, a continued focus on conducting research that explores the effectiveness of different types of integrated prevention approaches is needed and should be considered a top priority across both fields.

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O

Objectification

▶ Objectification Theory Model of Eating Disorders

Objectification Theory Model of Eating Disorders

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Synonyms

Male gaze; Objectification; Self-objectification; Sexual objectification; Sexualization

Definition

Objectification theory (Fredrickson and Roberts 1997) provides a framework for understanding the experience of being female in a culture that sexually objectifies the female body. As such, the theory serves as the foundation for an empirically testable model for understanding mental health risks such as eating disorders, which are disproportionately experienced by girls and women. The model derived from objectification theory may be classified as a *feminist* psychological model of eating disorders in that it assumes that experiences of anorexia, bulimia, binge eating, and obesity are best understood not as reflections of individual mental illness and deviance but rather are consistent with widely dispersed cultural ideals of body appearance.

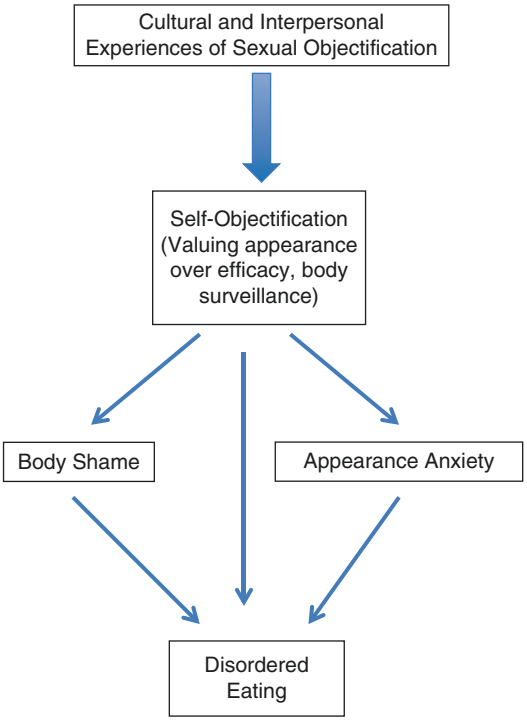
An objectification theory-based model of eating disorders begins with the premise that girls and women, more so than boys and men, are sexually objectified. That is, their bodies are valued primarily for their outward appearance, and their body parts and sexual functions may be separated from their personhood, reduced to the status of mere instruments, or regarded as capable of representing them entirely. Within this cultural (e.g., mass media, marketing, advertising) and interpersonal (e.g., ogling, catcalling) milieu, the theory argues girls and women are socialized to internalize an observer's perspective as the primary perspective on their physical selves, a phenomenon known as *self*-objectification. Self-objectification is characterized by a chronic monitoring of the body's outward appearance. This embodied form of self-consciousness takes up time and resources (attentional, physical, and economic). Self-objectification, in turn, leads to a number of cognitive, emotional, and motivational consequences, including increased feelings of shame, anxiety, and even disgust toward the bodily self, which more often than not fails to meet idealized cultural standards of beauty.

Disordered eating may be predicted to occur within this model via two routes. First, like dieting, using cosmetics, or other body-altering activities, eating disorders may be seen as a direct consequence of self-objectification itself. That is, eating disorders are time- and resource-intensive practices that reinforce the rewards and punishments of the (typically thin) cultural ideal for the female body. Secondly, disordered eating may be a consequence of the accumulation of such negative experiences as body shame and appearance anxiety resulting from chronic self-objectification. As Tiggemann (2013) argued, "self-objectification is the very crux of Objectification Theory" (p. 37), and indeed this particular form of embodied self-consciousness is what sets the model derived from objectification theory apart from other, more general feminist framings on eating disorders and what enables empirical testing of its tenets (Tiggemann 2013).

Self-Objectification and Its Consequences

Objectification theory (Fredrickson and Roberts 1997) argues that self-objectification can be thought of as both a trait and a state. Evidence for trait-level self-objectification comes from research demonstrating that individuals vary in the extent to which they prioritize their body's outward appearance over its efficacy and functioning, as measured by the Self-Objectification Questionnaire (Noll and Fredrickson 1998), as well as in their tendency to engage in habitual body surveillance, as measured by the Objectified Body Consciousness Scale (McKinley and Hyde 1996). State self-objectification has been induced via several methods, from having participants try on and evaluate swimwear in a dressing room (e.g., Fredrickson et al. 1998), to being subjected to actual male "gazing" or anticipating such gazing (e.g., Calogero 2004), to exposure to words or images of idealized female bodies commonly found in media (e.g., Roberts and Gettman 2004).

Correlational evidence has been found directly linking trait self-objectification to disordered eating (the abovementioned first route) and, in other cases, supporting a meditational model wherein body shame and/or appearance anxiety mediate the relationship (the second route) between self-objectification and eating disorders, as predicted by the original theory (e.g., Noll and Fredrickson 1998). Experimental



Objectification Theory Model of Eating Disorders, Fig. 1 Pathways predicted by Objectification Theory for development of eating disorders

inductions of state self-objectification have yielded both body shame and appearance anxiety in participants, and these, in turn, have predicted eating-related outcomes such as restraint and problematic eating attitudes and behaviors on self-report scales (e.g., Fredrickson et al. 1998; Moradi et al. 2005). Figure 1 shows the basic model for eating disorders for which objectification theory has provided evidence (modified from Moradi et al. 2005).

Developmental Trajectory

In addition to providing a model for understanding the antecedents and meditational pathways through which self-objectification may lead to disordered eating, objectification theory also proposes a developmental trajectory to girls' and women's troubled and disordered eating, which has been supported indirectly by cross-sectional research. Fredrickson and Roberts (1997) argued that the experiential and mental health consequences of self-objectification ought to change over the life course, intensifying in early adolescence and lessening in late middle age, corresponding with readily observable changes in the shape and reproductive functioning of the female body. Indeed, eating disorders such as anorexia, bulimia, and binge eating disorder are most commonly diagnosed in girls and women between the ages of 12 and 35 (American Psychiatric Association). Some studies suggest that aging is associated with lower body dissatisfaction and lower rates of eating disorders, at least for those women who "opt out" of self-objectifying practices (e.g., Grippo and Hill 2008). Furthermore, the increasing cultural sexualization of very young children – especially girls – appears to have occurred in step with increasing concerns with dieting, restrained eating, and even eating disorder symptomatology among children as young as 8–10 years old (Zurbriggen and Roberts 2013).

Future Directions

The ample existing research literature on eating disorders has yet to embrace the decidedly feminist social constructivist objectification theory to quite the extent it has other, more individual differences oriented or genetic frameworks. However, the model derived from the theory appears to provide a parsimonious contribution to our understanding of disordered eating. Further tests of the model, specifically targeted at examining the direct or mediated links among self-objectification's cognitive, emotional, and motivational consequences, are merited for predicting, treating, and preventing eating disorders.

Cross-References

- ▶ Body Image
- ▶ Media Literacy Approaches to Prevention
- ▶ Sociocultural Environment and Internalization of the Thin Ideal as Eating Disorder Risk Factors
- ▶ Weight and Shape Concern and Body Image as Risk Factors for Eating Disorders

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Objective Binge Eating

▶ Binge Eating Scale (BES)

Obsessions

▶ Obsessive-Compulsive Disorder and Eating Disorder Comorbidity

Obsessive Compulsive Disorder

▶ Obsessive-Compulsive Disorder and Eating Disorder Comorbidity

Obsessive-Compulsive Disorder and Eating Disorder Comorbidity

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Synonyms

Anxiety disorder; Compulsions; Obsessions; Obsessive compulsive disorder; Rituals

Definition

The definition of obsessive-compulsive disorder (OCD) has remained mostly unchanged from the DSM-IV to the DSM-5, although now is categorized in a new section entitled, "Obsessive-Compulsive and Related Disorders" along with the following disorders: hoarding, body dysmorphic, trichotillomania (hairpulling), and excoriation (skin picking). This new categorization is designed to reflect increasing evidence of these disorders' relatedness to each other and distinction from other anxiety disorders. These disorders share obsessive preoccupation and repetitive behaviors. There also have been changes to diagnostic specifiers, such that for OCD they have expanded from the DSM-IV which included just "with poor insight," now to include "good or fair insight," "poor insight," "absent or delusional

obsessive-compulsive disorder beliefs (i.e., complete conviction that obsessive-compulsive disorder beliefs are true)," and "tic related." The DSM-5 definition of OCD is as follows:

Either obsessions or compulsions are required. Obsessions are defined by both of the following:

1. Recurrent and persistent thoughts, urges, or images that are experienced, at some time during the disturbance, as intrusive and unwanted, and that in most individuals cause marked anxiety or distress.
2. The individual attempts to ignore or suppress such thoughts, urges, or images or to neutralize them with some other thought or action (i.e., by performing a compulsion).

Compulsions are defined by both of the following:

1. Repetitive behaviors (e.g., hand-washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly.
2. The behaviors or mental acts are aimed at preventing or reducing anxiety or distress or preventing some dreaded event or situation; however, these behaviors or mental acts are not connected in a realistic way with what they are designed to neutralize or prevent or are clearly excessive. (Note: Young children may not be able to articulate the aims of these behaviors or mental acts.)

Additional DSM-5 requirements are that the obsessions or compulsions are time-consuming (e.g., take more than 1 h per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. Importantly, the content of the obsessions or compulsions must not be restricted to another disorder (e.g., ritualized eating behavior in the presence of an eating disorder, compulsive hairpulling in the presence of trichotillomania, preoccupation with substances in the presence of a substance use disorder). Lastly, the OCD symptoms must not be due to the physiological effects of a substance or another medical condition.

The definition of OCD in the ICD-10 is similar, but not identical, to the DSM-5 definition. According to the ICD-10, obsessional symptoms or compulsive acts, or both, must be present on most days for at least two successive weeks and be a source of distress or interference with activities. By contrast, the DSM-5 does not have a symptom duration requirement. ICD-10 requires that the obsessional symptoms have the following characteristics:

1. They must be recognized as the individuals' own thoughts or impulses.
2. There must be at least one thought or act that is still resisted unsuccessfully, even though others may be present which the sufferer no longer resists.
3. The thought of carrying out the act must not in itself be pleasurable (simple relief of anxiety is not regarded as pleasure in this sense).
4. The thoughts, images, or impulses must be unpleasantly repetitive.

ICD-10 has its own set of potential specifiers:

1. Predominantly obsessional thoughts or ruminations
2. Predominantly compulsive acts [obsessional rituals]
3. Mixed obsessional thoughts and acts
4. Other obsessive-compulsive disorders
5. Obsessive-compulsive disorder, unspecified

Historical Background

Obsessive-compulsive disorder was first described by Robert Burton in 1621 in *The Anatomy of Melancholy*. Having unwanted thoughts and repetitive behavior was considered a type of melancholia in the seventeenth century and largely attributed to not being sufficiently religiously devout. More modern concepts of OCD further evolved in Europe during the nineteenth century. The start of the twentieth century led to

the most significant advancement, with Sigmund Freud and Pierre Janet probably most responsible for advancing our understanding of OCD as we know it today.

OCD appeared as a diagnosable mental disorder in the first edition of the *Diagnostic and Statistical Manual of Mental Disorders*. It was labeled "obsessive-compulsive reaction" and was classified in the section entitled "Psychoneurotic Disorders." The DSM-I definition was as follows.

In this reaction the anxiety is associated with the persistence of unwanted ideas and of repetitive impulses to perform acts which may be considered morbid by the patient. The patient himself may regard his ideas and behavior as unreasonable, but nevertheless is compelled to carry out his rituals. The diagnosis will specify the symptomatic expression of such reactions, as touching, counting, ceremonials, hand-washing, or recurring thoughts (accompanied often by a compulsion to repetitive action). This category includes many cases formerly classified as "psychasthenia."

Current Knowledge

The lifetime prevalence rate of OCD is estimated to be 2.3% (Kessler et al. 2012). The lifetime rates of OCD among individuals with eating disorders are typically much higher than that found in the general population, approximately 40% among those with anorexia nervosa or bulimia nervosa (e.g., Kaye et al., 2004). OCD is typically found to be one of the most common, if not the most common, comorbid anxiety disorder among individuals with anorexia nervosa or bulimia nervosa. Most individuals with eating disorders develop OCD before the onset of the eating disorder (as is true for most other comorbid anxiety disorders). The fact that OCD typically precedes the onset of the eating disorder supports the notion that OCD and eating disorders may share a common vulnerability factor or, alternatively, could suggest that OCD itself may be a risk factor for the development of anorexia nervosa or bulimia nervosa. The limited existing prospective data have found that OCD during childhood is indeed a risk factor for eating disorders later in life.

Likewise, eating disorders occur at elevated rates (estimates range from 11% to 42%) in probands with OCD (e.g., Sallet et al., 2010), compared to much lower rates of eating disorders in the general population (lifetime prevalence of anorexia nervosa is approximately 0.5%; bulimia nervosa is 1.5%; binge eating disorder is 2.8%). It appears that individuals who have both OCD and an eating disorder have comparably severe OCD symptoms compared to those individuals who have OCD but no eating disorder; however the comorbid group does appear to have more severe general psychiatric symptomatology and additional comorbidity.

Family studies allow one to examine patterns of comorbidity in identified probands and their relatives. Family studies utilizing probands with eating disorders (e.g., Bellodi et al. 2001; Lilenfeld et al., 1998) and family studies utilizing probands with OCD (e.g., Bienvenu et al., 2000) have yielded mixed findings. All such studies have found that eating disorder probands have elevated rates of OCD, as do the relatives of these probands. Likewise, OCD probands have elevated rates of eating disorders, as do the relatives of those probands. However, the patterns of psychopathology found in relatives after stratification by proband comorbidity status are inconsistent. Some data suggest that the reason for the elevations of the other disorder in the relatives of the proband is because the proband herself has elevated rates of that disorder. For instance, particularly elevated rates of OCD have been found in the relatives of those eating disorder probands who also have OCD themselves. This makes sense since OCD is indeed familial. The same pattern has been found in family studies of OCD probands, where especially elevated rates of eating disorders have been found in the relatives of those OCD probands who have eating disorders themselves. Yet there are interesting opposing data that suggest that the familial link between these conditions is not due to proband comorbidity status, but rather due to a likely shared etiology between the two conditions. Specifically, some family study data demonstrate that the risk for OCD in the relatives of eating disorder probands is similarly elevated, regardless of whether the

proband herself has OCD. The reason this would occur is due to shared familial liability between OCD and the eating disorder.

While most twin studies have not specifically examined the comorbidity of eating disorders and OCD, several have examined the comorbidity of eating disorders and anxiety disorders. These study findings suggest partially shared genetic transmission of eating disorders and anxiety disorders more generally. In fact, recent data from an extremely large study of OCD and anorexia nervosa, specifically, found that females with OCD have a 16-fold increased risk of having a comorbid diagnosis of anorexia nervosa, compared to the general population, while males with OCD have a 37-fold increased risk. Longitudinal risk for later anorexia nervosa among those first diagnosed with OCD, as well as risk for later OCD among those first diagnosed with AN, is likewise approximately twice as high among males than females. The particularly strong link between these disorders among males could have a number of possible explanations. One is that males may require a relatively higher genetic/familial loading, compared to females, in order to develop anorexia nervosa, possibly because environmental risk factors for eating disorders are more salient for the average female. Overall, individuals first diagnosed with OCD have a fourfold increased risk of developing anorexia nervosa later in life; those first diagnosed with anorexia nervosa have a tenfold increased risk of developing a later diagnosis of OCD. The strong link between OCD and anorexia nervosa is partially explained by the moderate, but significant, degree of genetic overlap between the two conditions. It appears that both shared genetic factors and disorder-specific genetic factors contribute to the comorbidity of OCD and anorexia nervosa. Yet, the exact nature of the shared etiology is still not clear.

However, several putative shared etiological factors have been highlighted in the literature. One hypothesis is a shared underlying personality trait, such as perfectionism or impulsivity (e.g., Lilenfeld et al. 2006). Perfectionism is a core personality trait underlying all types of eating disorders, as well as OCD. In fact, there is evidence to suggest that it is a mediator in the relationship between eating disorder and OCD symptoms (e.g., Bernert et al., 2013). Impulsivity is an additional personality trait elevated in both those with OCD and those individuals who have eating disorders with binging or purging symptomatology. This trait has also been suggested as part of an underlying shared etiology that may explain the co-occurrence of these two disorders.

Current Controversies

The primary controversy is whether the frequent comorbidity observed between eating disorders and OCD is indeed explained by a shared core disease process and/or shared maintaining mechanisms. This theory is compelling, particularly given the overlapping phenomenology of obsessive thinking and ritualistic behavior in both conditions. For instance, both involve repetitive preoccupations about a feared stimulus (e.g., food/weight gain with eating disorders, contamination or harm with OCD). Both also typically involve compensatory behaviors (e.g., food restriction or purging with eating disorders, washing or checking with OCD) to alleviate anxiety.

Indeed, there are data that support the "common-cause model." However, there are numerous other reasons disorders may be comorbid other than shared liability, such as one causing the other, the conjunction of independent risk factors, a third variable leading to both, or simply chance co-occurrence (Klein & Riso, 1993). The latter possibility is particularly likely when clinical samples are utilized in such studies because individuals who have more than one disorder are more likely to seek treatment than those with one disorder ("Berkson's bias").

Future Directions

Family studies and multivariate twin studies are among the best methodological designs to utilize to evaluate whether a shared underlying factor is responsible for the frequent co-occurrence of two disorders. Relatively few such studies have been

conducted to evaluate the reasons for the comorbidity of eating disorders and OCD. Early identification of identified shared risk factors for both disorders may lead to advances in early intervention and, theoretically, prevention of both disorders.

Cross-References

► Comorbidities: Anxiety Disorders

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Obsessive-Compulsive Personality Disorder as a Risk Factor for Eating Disorders

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Synonyms

Obsessive-compulsive personality traits as a risk factor for anorexia nervosa; Obsessive-compulsive personality traits as a vulnerability for eating disorders

Definition

The definition of obsessive-compulsive personality disorder (as well as all other personality disorders) has remained unchanged from the DSM-IV (American Psychiatric Association 1994) to the current version, the DSM-5 (American Psychiatric Association 2013).

Obsessive-Compulsive Personality Disorder

A pervasive pattern of preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency, beginning by early adulthood and presented in a variety of contexts, is indicated by four (or more) of the following:

1. Is preoccupied with details, rules, lists, order, organization, or schedules to the extent that the major point of the activity is lost.
2. Shows perfectionism that interferes with task completion (e.g., is unable to complete a project because his or her own overly strict standards are not met).
3. Is excessively devoted to work and productivity to the exclusion of leisure activities and friendships (not accounted for by obvious economic necessity).
4. Is overconscientious, scrupulous, and inflexible about matters of morality, ethics, or values (not accounted for by cultural or religious identification).
5. Is unable to discard worn-out or worthless objects even when they have no sentimental value.
6. Is reluctant to delegate tasks or to work with others unless they submit to exactly his or her way of doing things.
7. Adopts a miserly spending style toward both self and others; money is viewed as something to be hoarded for future catastrophes.
8. Shows rigidity and stubbornness (American Psychiatric Association 2013).

In the ICD-10 (World Health Organization 1992), this personality style is given the label of *anankastic personality disorder* with the following definition provided:

Personality disorder characterized by feelings of doubt, perfectionism, excessive conscientiousness, checking and preoccupation with details, stubbornness, caution, and rigidity. There may be insistent and unwelcome thoughts or impulses that do not attain the severity of an obsessive-compulsive disorder.

Obsessive-Compulsive Personality Disorder Versus Obsessive-Compulsive Disorder

While some individuals have both obsessive-compulsive disorder and obsessive-compulsive personality disorder, it is more common that they occur without the comorbid condition. However, obsessive-compulsive personality disorder is the most commonly co-occurring personality disorder alongside obsessive-compulsive disorder.

Historical Background

Obsessive-compulsive personality was first described by Freud over 100 years ago. It appeared as a diagnosable mental disorder in the first edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association 1952). Since then, along with other disorders in the manual, a shift occurred from a brief description of the condition to an operationalized definition in which a specific number of criteria need to be met in order to obtain the diagnosis. For instance, the definition of obsessive-compulsive personality in the personality disorders section of the DSM-II (American Psychiatric Association 1968) was as follows:

This behavior pattern is characterized by excessive concern with conformity and adherence to standards of conscience. Consequently, individuals in this group may be rigid, over-inhibited, overconscientious, over-dutiful, and unable to relax easily. This disorder may lead to an *obsessive-compulsive neurosis*, from which it must be distinguished.

The distinction between obsessive-compulsive personality disorder and obsessive-compulsive disorder is long-standing. The transition to operationalized polythetic criteria sets occurred with the publication of the DSM-III (American Psychiatric Association 1980), and the definition of compulsive personality disorder was as follows:

At least four of the following are characteristics of the individual's current and long-term functioning, are not limited to episodes of illness, and cause either significant impairment in social or occupational functioning or subjective distress:

1. Restricted ability to express warm and tender emotions, e.g., the individual is unduly conventional, serious and formal, and stingy.
2. Perfectionism that interferes with the ability to grasp "the big picture," e.g., preoccupation with trivial details, rules, order, organization, schedules, and lists.
3. Insistence that others submit to his or her way of doing things and lack of awareness of the feelings elicited by this behavior, e.g., a

husband stubbornly insists his wife complete errands for him regardless of her plans.
4. Excessive devotion to work and productivity to the exclusion of pleasure and the value of interpersonal relationships.
5. Indecisiveness: decision-making is either avoided, postponed, or protracted, perhaps because of an inordinate fear of making a mistake, e.g., the individual cannot get assignments done on time because of ruminating about priorities.

This is not identical, but similar, to the polythetic criteria system in which any four (or more) symptoms (now of eight possible symptoms rather than five, as described in the prior section) may be present in order to meet the criteria for the disorder.

Current Knowledge

Obsessive-compulsive personality disorder is estimated to occur in approximately 3–8% of the general population but at higher rates among those with eating disorders, with widely ranging estimates of 3–60%, depending upon the sampling and assessment strategies utilized.

Indeed, obsessionality and perfectionism are known among both clinicians and researchers to be core features in many individuals with eating disorders and restricting-type anorexia nervosa in particular. However, the question is whether such traits, often captured within the diagnosis of obsessive-compulsive personality disorder, are truly risk factors for eating disorders. This question can be most definitively answered using a prospective research design by assessing the personality of individuals who do not have any signs of an eating disorder and following them over time to track who later develops an eating disorder. Due to the very challenging nature of such research, it is limited; however, the existing data do suggest that obsessive-compulsive personality is a likely risk factor for the development of eating disorders. That is, those individuals who have such traits (in the form of maladaptive levels of perfectionism, rigidity, or an obsessive- compulsive personality disorder diagnosis) are more likely to later develop an eating disorder than those who have low levels of these traits or the absence of obsessive- compulsive personality disorder.

A different methodological strategy is the family study, in which one examines patterns of disorders in relatives of probands with an identified disorder, such as the presence of obsessive-compulsive personality disorder in the relatives of individuals with an eating disorder. These few such studies that have been conducted have found elevated rates of obsessive-compulsive personality disorder in the relatives of probands with restricting-type anorexia nervosa, even when the individual with anorexia nervosa did not have obsessive-compulsive personality disorder. This suggests that this form of eating disorder shares some familial liability with obsessive-compulsive personality traits. Thus, family study evidence points toward shared etiology, and other prospective study, as well as retrospective study, evidences point toward obsessive-compulsive personality disorder as a risk factor for eating disorders.

Importantly, rigid perfectionism, considered a hallmark symptom of obsessive-compulsive personality disorder, has been found to function as both a risk and maintaining factor for eating disorders. That is, those high in maladaptive perfectionism develop eating disorders at greater rates than those lower in perfectionism. In addition, those with high levels of perfectionism are more likely to persist in their disorder than those who are less perfectionistic. While these findings characterize the state of the field's understanding of this personality trait and its relationship to eating disorders, it should be noted that it is not a relationship that is exclusive to eating disorders, as perfectionism likewise has been identified as both a risk and maintaining factor for depression and anxiety disorders.

Most of this research in the field of eating disorders has focused upon the diagnosis of anorexia nervosa. In fact, numerous authors have noted the remarkable homogeneity in personality presentation among those individuals who persist with restricting-type anorexia nervosa. There are some data to suggest that obsessive-compulsive personality traits are associated with a poor

outcome in anorexia nervosa. Thus, some recent efforts have been made to utilize personality traits in order to classify individuals with eating disorders in a more clinically useful way using empirically derived personality subtypes, as well as tailor treatment approaches based upon personality and temperament. Cognitive-behavioral interventions for perfectionism and cognitive rigidity have been developed as novel adjunctive treatment approaches for eating disorders in response to the above findings.

Current Controversies

There are two separate areas of controversy worthy of note. The first concerns methodological challenges specific to identifying risk factors for eating disorders. The second concerns the categorical approach to defining obsessive-compulsive personality, a critique which applies to all personality disorders.

The first issue is a challenge for other areas of psychopathology (e.g., depression) but for none more so than eating disorders. In order to definitively identify a risk factor for a disorder, that risk factor must be established before the onset of the disorder and be predictive of the development of that disorder. The necessary requirements for such a study include assessment at least at two different points in time in which the individual changes status on the outcome of interest (Jacobi et al. 2004). A prospective design is the ideal research design through which to identify risk factors because for obsessive-compulsive personality disorder (or any other condition) to be a true risk factor, it must temporally precede the outcome variable of interest, in this case, the eating disorder (Lilenfeld et al. 2006). However, there are significant challenges in conducting such research. Due to the relatively low base rates of eating disorders, prospective studies are extremely difficult to execute with this population. As a result of this challenge, other methodological designs have been utilized, but they pose problems in definitively identifying a risk factor. A recovered study design utilizes individuals who have recovered from an eating disorder in order to identify disorders or traits that remain elevated after recovery, with the assumption that this may be reflective of the individual's premorbid state. A major problem with this approach is that eating disorders (as well as other disorders like depression) are thought to potentially leave a "scar" upon personality or other characteristics such that although there is recovery from the disorder, the individual may have changed as a result of having experienced the disorder and, thus, a recovered presentation may not reflect the premorbid presentation. This is particularly important to note with eating disorders that may involve profound physiological impact as a result of starvation and other disruptive eating and weight-control behaviors. A second alternative research design that has been utilized to obviate the need for the more difficult prospective study is a retrospective recall study. The individual and sometimes family informants are asked to report on the individual's traits before the onset of the disorder in which risk factors are being sought. The obvious limitation of this design is recall bias. These limitations being noted, both recovered study and retrospective study designs have identified obsessive-compulsive personality disorder (or hallmark traits such as perfectionism) as likely eating disorder risk factors. A final related note is that because personality disorder diagnoses ought not to be made until early adulthood, or late adolescence at the earliest, requiring the categorical definition of obsessive-compulsive personality disorder is not sensible, since the most typical age of onset for eating disorders is mid-adolescence through young adulthood. Therefore, identifying this pathology on a continuum, as with maladaptive perfectionism, utilizing a well-established psychometrically sound measure such as one of two Multidimensional Perfectionism Scales (Frost et al. 1990; Hewitt and Flett 1991), may be preferable.

Regarding this second issue, the debate over whether a categorical or a dimensional approach to classifying psychopathology has been ongoing for decades. There is no area of psychopathology in which this has been more hotly debated than personality disorders. While most clinicians, as well as personality and psychopathology researchers, agree that personality pathology falls on a continuum, discrete disorder categories

have advantages, such as ease of communication among professionals and facilitating efforts to find causes of discrete syndromes. These, among other reasons, have led to the retention of a predominantly categorical system in the DSM. While there was some move toward increased dimensionality in the DSM-5, the categorical structure was retained. Personality disorders is the one area which was poised to move toward a much more hybrid categorical-dimensional model, but very shortly before publication of the manual, it was decided that the original personality disorder categories from the DSM-IV would be retained with no changes "in order to preserve continuity with current clinical practice," despite ample research supporting a more dimensional classification of personality pathology. This hybrid model, which is likely to be used by many researchers, can be found in Section III "Emerging Measures and Models" under the heading "Alternative DSM-5 Model for Personality Disorders." Personality disorders are defined by impairments in both personality functioning and pathological personality traits. Obsessive-compulsive personality disorder was one of six of the original ten personality disorders retained in this alternative model. The proposed diagnostic criteria for this alternative model of obsessive-compulsive personality disorder are as follows:

(a) Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:
   1. *Identity*: Sense of self derived predominantly from work or productivity; constricted experience and expression of strong emotions.
   2. *Self-direction*: Difficulty completing tasks and realizing goals, associated with rigid and unreasonably high and inflexible internal standards of behavior; overly conscientious and moralistic attitudes.
   3. *Empathy*: Difficulty understanding and appreciating the ideas, feelings, or behaviors of others.
   4. *Intimacy*: Relationships seen as secondary to work and productivity; rigidity and stubbornness negatively affect relationships with others.

(b) Three or more of the following four pathological personality traits, one of which must be (1) rigid perfectionism:
   1. *Rigid perfectionism* (an aspect of extreme conscientiousness (the opposite pole of detachment)): Rigid insistence on everything being flawless, perfect, and without errors or faults, including one's own and others' performance, sacrificing of timeliness to ensure correctness in every detail, believing that there is only one right way to do things, difficulty changing ideas and/or viewpoints, and preoccupation with details, organization, and order.
   2. *Perseveration* (an aspect of negative affectivity): Persistence at tasks long after the behavior has ceased to be functional or effective; continuance of the same behavior despite repeated failures.
   3. *Intimacy avoidance* (an aspect of detachment): Avoidance of close or romantic relationships, interpersonal attachments, and intimate sexual relationships.
   4. *Restricted affectivity* (an aspect of detachment): Little reaction to emotionally arousing situations, constricted emotional experience and expression, and indifference or coldness.

Any clinician who treats individuals with eating disorders can attest that the above definition aptly captures the personality style of nearly everyone with the restricting subtype of anorexia nervosa who does not transition to another type of eating disorder. This proposed move to require rigid perfectionism as the hallmark trait in obsessive-compulsive personality disorder is indeed consistent with clinical and research findings of most individuals with eating disorders, particularly those with restricting-type anorexia nervosa.

Future Directions

The hybrid dimensional-categorical personality disorders model presented in the DSM-5 may

hold the greatest promise for future research on personality risk factors for eating disorders. In addition, in order to most definitively identify a true risk factor, that factor must be present before the onset of the disorder. This poses methodological challenges given the relatively low base rates of eating disorders (though notably higher when disordered eating is conceptualized on a continuum), because large samples are needed to be studied through the period of risk (for which there is some variation) in order to adequately power a prospective study of personality risk factors for eating disorders. Very few such studies have been conducted. Therefore, more sophisticated, longitudinal, prospective research is needed. Most prospective research in the field of eating disorders has been limited by inconsistent outcome and predictor measures, small sample sizes, modest follow-up periods, and failure to control for baseline eating pathology. Prospective research is time-consuming and costly, but necessary to identify true personality risk factors for an illness. Collaborative efforts are advisable in order to accomplish this task.

Cross-References

- ▶ Anorexia Nervosa
- ▶ Perfectionism
- ▶ Personality Disorders as Comorbidities in Eating Disorders
- ▶ Personality-Based Approaches to Classification

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Obstetric – Complications

- ▶ Perinatal Complications

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Olanzapine as an Adjunctive Treatment for Adolescent Anorexia Nervosa

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Background

Anorexia nervosa (AN) is a severe, potentially life-threatening illness that often begins in adolescence, and threatens the physical and cognitive

development of children and youth. AN affects 0.9–2.2% of women over a lifetime, with a prevalence of at least 1% in children and adolescents. Medical complications can be multisystemic and include cardiovascular, endocrine, gastrointestinal, reproductive, and hematological systems (Watson and Bulik 2013). It is estimated that 50% of children with new onset AN have life-threatening medical complications (Watson and Bulik 2013). In addition to medical morbidity, AN has the highest mortality rate of all mental illnesses, with substantial proportions of patients experiencing a chronic course (Watson and Bulik 2013).

Management of AN is often multidisciplinary in nature, with a focus on nutritional, medical, and psychological rehabilitation. The success of employed treatment modalities are influenced by a host of factors, including individual patient characteristics (both medical and psychological), degree of malnutrition, life interference, and at times, depending on patient age and prior treatment trajectory, patient (and caregiver) motivation, and resource availability.

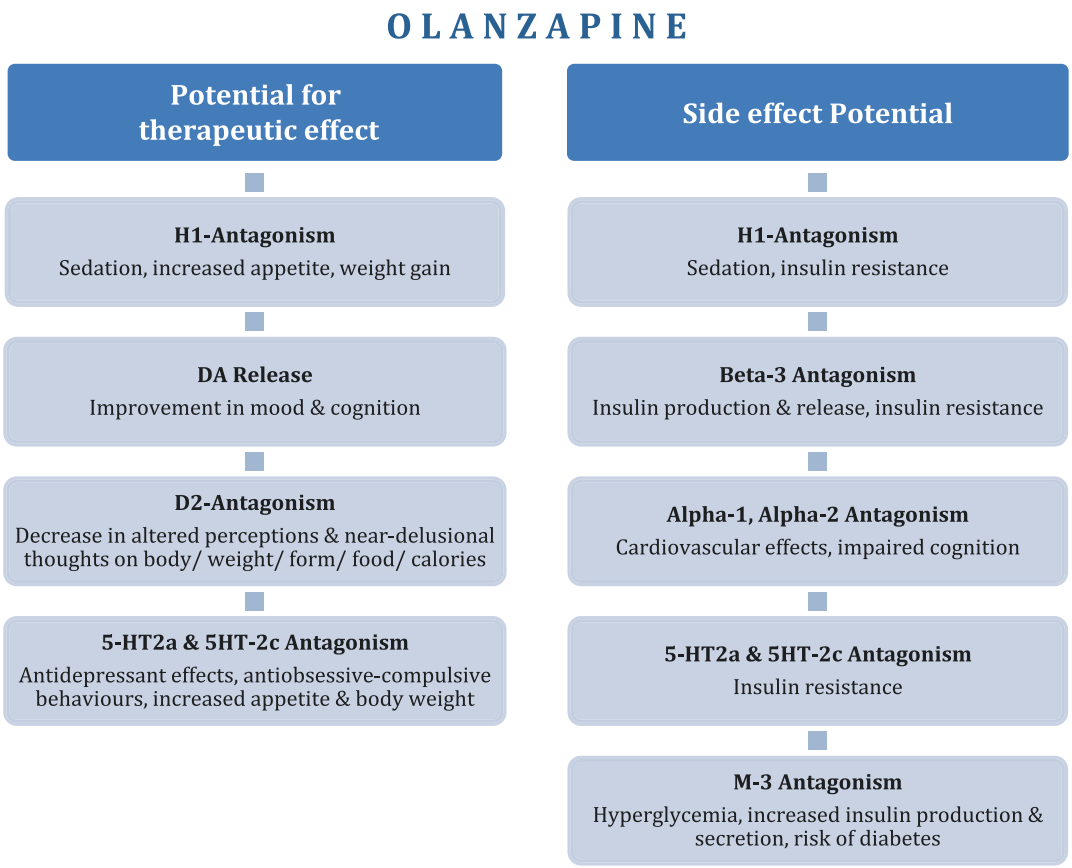
In children and adolescents, current evidence suggests that treatment results are optimized through the use of family-based therapy (FBT). In a proportion of cases, outpatient treatment may not be suitable or advisable. Factors including degree and rate of weight loss, risk of refeeding syndrome, medical instability, as well as the presence of profound psychiatric symptoms or distress can all influence decision-making to admit patients to hospital. Pharmacological interventions are not recommended as the primary or sole treatment for AN, and guidelines caution about pharmacotherapy use, given that weight restoration by way of nutritional rehabilitation on its own improves depressive symptoms, anxiety, obsessive-compulsive tendencies, and restlessness (Watson and Bulik 2013). Studies that have investigated the use of augmented pharmacotherapy with treatment for AN have not been shown to consistently impact treatment or outcomes of patients. Despite this, a recent study has suggested that medications, including olanzapine, are utilized frequently by care providers (Watson and Bulik 2013). Olanzapine is the most commonly studied medication of its class in patients with AN and is the only AP classified as grade B evidence by the World Federation of Societies of Biological Psychiatry (WFSBP) for efficacy in this patient population (van den Heuvel and Jordaan 2014).

Pharmacology of Olanzapine

Olanzapine's primary mechanism of action is assumed to lie in its ability to block dopaminergic (D1-4) and serotonergic (5HT 2A & 2C) receptors (Norris et al. 2011). As the neurobiology of AN is thought to involve altered dopamine and serotonin pathways, SGAs like olanzapine have been hypothesized as theoretically reasonable pharmaco-therapeutic interventions (Dold et al. 2015). As seen in Fig. 1, D2 receptor antagonism has the propensity to reduce inner tension, obsessions, delusional beliefs, and increased motor activity, and improve (auto)-aggressive behaviors (Fabrazzo et al. 2013). Additionally, 5-HT2/D2 receptor affinity ratio may help reduce affective symptoms, irritability, and anxiety. SGAs also act to enhance the properties of adipocytokines, such as leptin (peripheral satiety hormone), tumor necrosis factor alpha (TNFα), and interleukin-6, which in turn, can result in weight gain (Fabrazzo et al. 2013). It is also postulated that the weight gain effect of SGAs through increased food intake is mediated through their ability to elevate serum ghrelin (hunger hormone) (Fabrazzo et al. 2013).

Olanzapine Use in Adolescent AN: Reflections from the Last Decade

Although our knowledge of the efficacy of olanzapine in the treatment of AN has improved since the first case reports appeared at the turn of the century, the number of high quality, sufficiently powered studies involving children and adolescents remains very limited. This reality likely reflects challenges associated with the execution and completion of adequately powered trials in this cohort. Problems associated with recruitment, controlling inherent biases (such as ambivalence and motivation for treatment), high



Olanzapine as an Adjunctive Treatment for Adolescent Anorexia Nervosa, Fig. 1 Postulated receptor mechanisms of olanzapine action in patients with AN showing potential for increased therapeutic effect on left and side effect potential on right (Adapted from Fabrazzo et al. 2013)

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rates of attrition and drop out, as well as choosing "the right" treatment outcomes all contribute to these difficulties (Watson and Bulik 2013; Norris et al. 2010). If we reflect on our own experience over the last 15 years of study in this area (utilizing retrospective, open label, and randomized placebo controlled trial designs), we faced each of these challenges first hand. In the case of our retrospective case control study, we found that despite our best attempt to match patients on age, diagnosis, and treatment modality undertaken, the cohort who were treated with olanzapine exhibited greater rates of psychopathology and complexity as evidenced by higher rates of comorbid diagnoses and more intensive and longer treatment courses (Norris et al. 2011). Simultaneously, we embarked on an ambitious randomized placebo-controlled trial that had been designed with help from international experts in research design and methodology (Spettigue et al. 2008). Despite moving ahead with a "gold standard" study, it was closed prematurely after 3 years on account of the fact that recruitment efforts failed miserably. Of the 92 patients who were assessed and treated over the study timeframe, only 27 patients (29%) met full criteria for inclusion, of which just 7 enrolled (26% of those eligible). The most common reasons for study refusal related to fears associated with medication effects and refusal to consider medication as a treatment option (70%) (Norris et al. 2010). Our experience highlights that even the best-designed well-funded studies are fraught with challenges. Not wanting to give up, we

sought consultation from other experts and landed on an open label design that attempted to mimic "real world conditions" as best as possible, giving patients the option to begin (or stop once starting) olanzapine at any point 8 weeks after starting treatment with our program. We monitored all enrolled participants closely, regardless of their choice to use olanzapine or not. All patients were offered the medication at the time of study enrollment, and at various points thereafter. With this design, we completed the largest study to date of olanzapine as adjunctive treatment for adolescent anorexia nervosa; recruiting a total of 32 patients, 22 of whom took olanzapine at some point in their treatment course. Our results showed that patients who augmented treatment with olanzapine gained weight at a significantly faster rate than those who did not take olanzapine (Spettigue et al. 2015).

Keeping all of these challenges in mind, it is no wonder that the evidence base in pediatrics (with the exception of one very small randomized control trial [RCT]) remains limited to case series, retrospective chart reviews, and open label trials. A recent systematic review by Balestieri et al. (2013) that evaluated the study of antipsychotic use in adolescent and adult populations with AN highlighted this reality. Although olanzapine demonstrated favorable effects on BMI, ED symptoms, and functional impairment in a proportion of trials studied, pooled data (completed before the release of our 2015 data) failed to support the use of olanzapine in the treatment of adolescent AN.

The question remains: Has the evidence base been sufficiently developed to allow for a definitive answer as to whether olanzapine can offer benefit to children or youth with AN? If we consider potential areas of bias and limitations within these studies and reviews, can we say with certainty that the results of this review are valid and that further study is not warranted? With close examination, it becomes clear that we likely cannot, for the following reasons:

1. The total number of controlled studies that has been completed in youth is very limited, including just one small, RCT. Kafantaris et al.'s (2011) RCT included a total of 20 adolescents AND young adult patients (age 12–21 years, mean age 17.1 years). Although results showed no significant differences in measures of BMI, psychopathological features, or ED attitudes/behavior between groups, it is noteworthy that only 20 of 94 potentially study-eligible patients participated in the trial, and only 15 completed the trial (7 in the medication group) (Kafantaris et al. 2011). Only two open label trials (both with variable study designs) of olanzapine for adolescent AN have been completed. One conducted by Leggero et al. (2010) followed 13 girls with AN (mean age 13.7 years) treated with low-dose olanzapine and showed some modest improvements in 7/13 in weight and eating attitudes, with a significant improvement in the symptom of hyperactivity for these deemed responders. Our own open label study (Spettigue et al. 2015) of 33 adolescents found that those treated with olanzapine gained weight at a significantly greater rate than those that who did not take olanzapine.
2. The sample size in each of the outlined studies is quite small. Given the low incidence of AN, the reluctance of patients to seek and accept treatment (and enrol in studies that set weight gain as a primary outcome), as well as high rates of attrition and drop-out, recruitment and retention of patients is a tremendous challenge.
3. As one of the key features of AN is that patients are terrified of weight gain, researchers are faced with the added challenge of asking patients to willingly enrol into a study that is using a medication hypothesized to cause weight gain. How do we capture the extent to which selection bias potentially impacts the results obtained? To what extent does caregiver pressure or motivation impact the decision of patients to enrol? And how do we control for this tremendous confounder?
4. How do we best control all of the potential confounders that impact treatment? Factors such as nutrition prescription, mode of nutritional delivery, presence of meal support, concomitant individual psychotherapy, group

therapy and family-based treatment, family resources, and therapist skills vary across most treatment settings and yet are rarely described in detail in the study methodology.

5. To what extent does drug dosing impact treatment response? In each of the studies outlined above, olanzapine dosing ranged considerably. Some studies use fixed dosing and others use variable dosing. If a patient agrees to enrol in a study but cannot tolerate an olanzapine dose greater than 2.5 mg daily, can we say that the medication was not useful or rather should we say that the medication was not tolerated to an extent typically required for treatment response? The study by Leggero reported a mean olanzapine dose of 4.1 mg/day, and in our own study the average dose was 5 mg/day. If trials in youth report negative findings, is this because olanzapine is not helpful, or because higher doses are required to show a significant impact from the medication?
6. Should the role of epigenetics be addressed? No study to date has controlled for factors related to the pharmacogenetic profiles of patients.
7. How should treatment response be defined and should this terminology be standardized across studies? Should weight be tracked over the duration of treatment, over the first 8–12 weeks only, or over some other as of yet unspecified timeframe? Should response be limited to the timeframe necessary for weight restoration? Should 3-, 6-, or 12-month time points after the discontinuation of medication matter? For example, our open-label trail planned to follow patients on olanzapine for 12 weeks, and to administer measures at the end of that time. However, in reality, almost 100% of the participants in the medication group had reached their healthy weight before 12 weeks, leading their treating physicians to discontinue the medication before that time. Even by 8 weeks there were few patients left in the medication group (Spettigue et al. 2015).

In summary, despite the fact that specialists have been using olanzapine in various capacities for the augmented treatment of AN for over 15 years, there remains a paucity of evidence to demonstrate its effectiveness in this population; this lack of evidence may reflect limitations of the medication itself to combat the illness, but it may also reflect the challenges associated with studying olanzapine in this cohort. These challenges include the huge resources necessary to conduct randomized controlled psychopharmacological trials in youth, recruitment and retention challenges associated with this population (including fear of taking medication), dosing issues, and difficulties controlling for other factors during treatment, including treatment settings, types of therapy provided, and family support. In addition to these challenges, the unproven efficacy of the medication must be weighed against increasing evidence of risks associated with the use of SGAs in this population.

Medical Monitoring

If and when considered, the use of olanzapine should be accompanied by clinical, metabolic, as well and cardiac as monitoring, including of monitoring the corrected QT interval on electrocardiogram (Pringsheim et al. 2011). Most adolescent studies report that the medication is tolerated without serious issues; however, our own experience suggests that over half of proportion of patients experience clinically significant though mild side effects (Norris et al. 2011; Spettigue et al. 2015). While few studies speak specifically about the degree to which side effects are monitored during trials, serious side effects including neuromuscular malignant syndrome (NMS) (Ayyıldız et al. 2016) and diabetes (Yasuhara et al. 2007) have been described. Although we lacked a controlled comparator in our retrospective study, our early experience suggested that a proportion of malnourished patients developed dyslipidemia and transaminitis (Norris et al. 2011). More recently, results from our open label study (which contained a control group) affirmed the need to monitor the metabolic profile and liver function of patients treated with olanzapine (Spettigue et al. 2015).

Conclusions and Future Considerations

Based on current published literature, there is insufficient evidence to support the use of olanzapine for the pharmacological treatment of pediatric AN; readers should understand that there have been only a handful of small trials and case reports on this topic published over the last 15 years, and our overall state of knowledge remains limited. It is clear that more study is required. The need for larger randomized controlled trials of olanzapine as adjunctive treatment for pediatric anorexia nervosa remains just as pressing as it did 15 years ago when we set out to study this question. Although it seems logical to consider multicenter randomized controlled trials as a means of providing greater clarity to the question of the role of olanzapine in the treatment of adolescent AN, the number and degree of confounding variables as well as demonstrated barriers involved with such trials cannot simply be dismissed and should not be ignored. We live in an age where research funding has become more competitive, accountability has increased, and resources are more limited. Given this, it is time that specialists in the field come together to better delineate methods (e.g. agree upon and standardize protocols, instruments, and outcome-measures) that best answer the question of whether olanzapine is effective for the adjunctive treatment of pediatric anorexia nervosa.

Cross-References

- Family Meal in Family Therapy for Anorexia Nervosa: Is It Important?
- Family-Based Treatment
- Treating Adolescent Anorexia Nervosa when Family-Based Treatment Is Insufficient or Contraindicated

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ω-3 Fatty Acids

► Omega 3 Fatty Acids as Adjunctive Treatment for Eating Disorders

Omega 3 Fatty Acids as Adjunctive Treatment for Eating Disorders

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Synonyms

ω-3 fatty acids; *n*-3 fatty acids; polyunsaturated fatty acids (PUFAs)

Definition

Omega 3 fatty acids are fatty acids that contain more than one double bond in their backbone.

Historical Background

Due to the side effect profiles and low acceptability of many psychoactive drugs, the research community is now turning to more natural products for the treatment of many mental health conditions. Poly unsaturated fatty acids (PUFAs) are essential fatty acids that cannot be synthesized by the human body; thus, there is complete reliance on dietary intake. Linoleic acid (LA) and α-linoleic acid (LNA) are two forms of these essential fatty acids that are the main dietary precursors of omega-6 and omega-3 fatty acids, respectively. The omega-3 and omega-6 fatty acids are both essential components of the phospholipid membrane that is predominant in the brain. Their balance is important for normal functioning and proper membrane fluidity. The phospholipid hypothesis of mental illness proposes that neurotransmitter receptor functioning is affected by the fatty acid composition of the cell membrane, particularly the ratio of omega-6 to omega-3 fatty acids. A high ratio of omega-6 to omega-3 fatty acids can alter cell membrane properties and increase production of inflammatory mediators, as arachidonic acid (an omega-6 fatty acid) is a precursor of inflammatory eicosanoids, such as prostaglandins and thromboxanes (Simopoulos 2002). In contrast, omega-3 fatty acids are anti-inflammatory (Simopoulos 2002). It has also been found that diets deficient in fatty acids severely affect the composition of neuronal and glial membranes, linked to alterations in brain function (Ayton 2004). By this mechanism, increased omega-3 fatty acid concentrations in cell membranes have been shown to regulate serotonin and dopamine neurotransmission, particularly within the frontal cortex. With reduced omega-3 fatty acids, the composition of the cell membrane would be altered, leading to improper neurotransmitter binding and psychopathology.

The Role of Fatty Acids in Mental Health

Omega-3 fatty acid treatment has been shown to be effective for several psychiatric conditions such as depression, bipolar disorder, dementia, psychosis, and attention deficit hyperactivity

disorder. Multiple reviews have identified omega-3 fatty acid supplementation for mood disorders as a beneficial intervention (Liperoti et al. 2009). The most common omega-3 fatty acids studied have been eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). For young adults with depression, a recent RCT showed a significant reduction in the Beck Depression Inventory over a 21-day period compared to placebo with a dose of 1000 mg of EPA combined with 400 mg of DHA (Ginty and Conklin 2015). In addition, a recent meta-analysis showed a beneficial effect of the combination of EPA and DHA compared to placebo on depressed mood for women with depression (Yang et al. 2015). More specifically for adolescents, Wozniak and colleagues conducted an 8-week open-label treatment with omega-3 fatty acids in teens with bipolar disorder (Wozniak et al. 2007). The study reported a modest improvement in manic symptoms with a dosage range of 1290–4300 mg of combined EPA and DHA (Wozniak et al. 2007). A more recent meta-analysis of 10 trials for attention deficit children found a small but significant benefit of omega-3 fatty acid monotherapy or supplementation, with EPA dose ranging from 80 to 750 mg daily (Bloch and Qawasmi 2011). The study found a significant association between higher EPA dose and improvement in ADHD symptoms (Bloch and Qawasmi 2011).

Fatty Acids and Eating Disorders

More recent attention has been paid to the function of fat deprivation in the onset and duration of eating disorders and the potential role that fatty acid supplementation can play as an adjunctive treatment. In the Western diet, omega-6 fatty acids or their precursors (LA) are much more abundant than omega-3 fatty acids or their precursors (LNA). Hence, it has been suggested that our diets lack sufficient omega-3 fatty acids, and under fasting conditions, these levels are further lowered (Ayton 2004).

The rationale for possible fatty acid effectiveness for treating eating disorders is present in two types of studies. The first are those studies which demonstrate abnormalities in levels of fatty acids and membrane fluidity in patients with anorexia nervosa (AN). For example, Swenne and Rosling (2012) conducted a study of 24 teenage girls being treated for eating disorder not otherwise specified (EDNOS) or AN. These patients originally presented with negative energy balance and low fat intake. As expected, these patients had lower levels of some fatty acids within their erythrocyte cell membranes. Swenne et al. (2011) have reported similar findings among 217 teenage patients diagnosed with AN, Bulimia Nervosa (BN) and EDNOS. They noted that the end products of omega-3 fatty acids were lower in erythrocyte cell membranes, particularly in depressed patients with eating disorders. A third study conducted with 22 adult patients with restrictive and purging types of eating disorders also found that fatty acid composition of erythrocytes was very different between patients and controls, with very low levels of omega-3 fatty acids among the clinical sample (Caspar-Bauguil et al. 2012).

Previously published case reports, case series, and trials have examined the effectiveness of omega-3 fatty acid supplementation in the treatment of eating disorders and suggest continued investigations in this area. For example, a RCT conducted by Barbarich et al. (2004), compared fluoxetine supplemented with either fatty acids or placebo in 26 adult patients with AN. The 6-month trial suggested that nutritional supplements containing essential fatty acids did not increase the efficacy of fluoxetine in terms of weight gain, perfectionism, or anxiety. However, these authors did not examine mood symptoms and did not include adolescents. Mauler et al. (2009) compared a diet rich in omega-3 fatty acids to a control diet in their study involving 25 female subjects (12–45 years of age) with AN. They reported that the body weight of patients in the omega-3 fatty acid group showed greater improvement than the body weight of subjects in the control group. Similarly, Ayton et al. (2004b) reported gradual improvement in diet, weight, and mood in a case report of a 15-year-old patient with AN who was treated with 1000 mg of EPA daily. And finally, Ayton et al. (2004a) reported a case series of seven adolescents with AN treated with 1000 mg of EPA daily in addition to standard treatment. These

authors note that the omega-3 supplementation caused little side effects, with three of the seven patients having recovered from their illness and the remaining four subjects showing significant improvement in all symptoms domains. The conclusion of these authors was that RCTs are needed to empirically confirm these findings.

Current Knowledge

We have recently completed a pilot open trial of omega-3 fatty acids in 21 children and adolescents with eating disorders (Woo et al. 2016). In contrast to psychopharmacological studies, our acceptance rates were high. Only four eligible patients who were approached declined to participate in the study, one due to an inability to swallow pills, two due to driving distance to the clinic, and one who did not wish to consume extra calories. We had no dropouts from our study. All participants received standard treatment coupled with omega-3 supplementation for 8 weeks. Depending on the type and severity of symptoms, standard treatment consisted of family-based model of psychotherapy, cognitive behavioral therapy, or dialectical behavioral therapy, along with psychotropic medications.

Participants took one capsule of Swiss Natural Product's "Swiss: Omega-3 (NPN: 80004899)" per day as a source of 300 mg of EPA and 200 mg of DHA. The dosage was decided according to the American Dietetic Association and Dietitians of Canada's daily recommended intake of EPA and DHA (Kris-Etherton et al. 2007). We ensured that the dosage consisted of 60% EPA, based on previous findings that combined EPA-DHA supplementation is most effective if the proportion of EPA is at least 60%. The study included 21 participants aged 9–17 years, with a mean age of 15.29 $\pm$ 2.03. The sample was 90% female. Diagnoses consisted of AN (15/21), Other Specified Feeding or Eating Disorders (4/21), and Bulimia Nervosa (2/21). When asked about the general acceptability of omega-3, three participants reported that the fishy aftertaste of the pill was bothersome at first; however, the taste ceased to be noticeable after the first week. In terms of preliminary assessment of outcomes, percent IBW ranged between 79% and 110% at baseline, and between 82% and 106% at week eight. Mean percent IBW was significantly greater at week eight compared to baseline ($p < 0.01$). There were no significant differences in mean scores or clinically elevated levels of EDI-3, CDI-2, and MASC between baseline and week eight.

Current Controversies

Studies to date, including our own, have been limited by small sample sizes and comprised largely of individuals with AN. Our sample was largely in partial remission at the time of enrollment (57%), making it difficult to generalize our finding to patients of lower body weight. The treatment periods are generally short and thus may not capture all potential side effects or benefits of omega-3. There are few well-designed RCTs on omega-3s in this patient population making it difficult to draw any conclusions about efficacy. In addition, the dosage of omega-3 used in the studies with patients with eating disorders has been relatively low, compared to studies using omega-3 in other patient populations. While there is little consistency in doses used in the literature, the American Psychiatric Association recommends 1 g/day of combined EPA and DHA for adults with psychiatric disorders (Freeman et al. 2006).

Future Directions

Despite these limitations, omega-3 fatty acids are generally acceptable and well tolerated by children and adolescents with eating disorders. In the future, a RCT of longer duration using a higher dosage of EPA and DHA is needed to help draw stronger conclusions about the effectiveness of omega-3 fatty acids in this population. Omega-3 fatty acid levels could also be compared pre- and posttreatment in order to further elucidate the relationship between fatty acids and eating disorder symptomology. In addition, it may be helpful

to monitor inflammatory markers to search for biological indicators of treatment effect. Lastly, the impact of clinical and demographic characteristics such as gender, age, comorbidities, medications, psychotherapeutic treatment, and type and severity of eating disorder diagnosis on omega-3 treatment outcomes should be examined.

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Origin

► Ethnicity and Eating Disorders: More Similarities than Differences

Osteoporosis

► Bone Health

Other Specified Feeding or Eating Disorder (OSFED)

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Definition

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association 2013) introduced a new diagnostic category to replace the previous residual category of *eating disorder not otherwise specified* (EDNOS). This new category, *other specified feeding or eating disorder* (OSFED), describes individuals who have a clinically significant feeding or eating disorder that does not meet criteria for an officially recognized diagnosis (i.e., anorexia nervosa, bulimia nervosa, binge-eating disorder, or avoidant/restrictive food intake disorder). *DSM-5* identifies five OSFED examples by name: *atypical anorexia nervosa*, *bulimia nervosa (of low frequency and/or limited duration)*, *binge-eating disorder (of low frequency and/or limited duration)*, *purging disorder*, and *night eating syndrome*.

A diagnosis of *atypical anorexia nervosa* is given if a person meets all criteria for anorexia nervosa except for low weight (i.e., the individual's weight falls within or above the normal body mass index range despite significant weight loss). *Bulimia nervosa (of low frequency and/or limited duration)* resembles *DSM-5* bulimia nervosa except that the duration and/or frequency criteria are not met. For example, an individual who meets all diagnostic criteria for bulimia nervosa but engages in binge eating and inappropriate compensatory behaviors less than once per week and/or for a duration of less than 3 months would be diagnosed with this subthreshold presentation. Similarly, *binge-eating disorder (of low frequency and/or limited duration)* features all criteria for *DSM-5* binge-eating disorder except that the individual with this subthreshold presentation engages in binge eating less than once per week and/or for less than 3 months. In contrast, individuals with *purging disorder* resemble those with bulimia nervosa except that they do not engage in objective binge episodes. By definition, individuals with purging disorder engage in some form of purging (e.g., self-induced vomiting, laxative misuse, diuretic misuse) to influence body shape or weight. These individuals may or may not also exhibit subjective binge eating, which is characterized by a sense of loss of control while eating a small or normal amount of food. Finally, *night eating syndrome* is characterized by either nocturnal ingestions (i.e., waking up in the middle of the night to eat) or evening hyperphagia (i.e., consuming at least 25% of daily calorie intake after the evening meal). Individuals with night eating syndrome are fully awake and alert for nocturnal ingestions (in contrast to sleep-related eating disorder, in which individuals may not recall episodes of night eating), which cause marked distress or impairment in functioning. Night eating syndrome must not be better accounted for by substance use, a medical condition, or an unrelated disturbance in a person's sleep/wake cycle.

If an individual's presentation is clinically significant, does not meet criteria for any of the officially recognized feeding or eating disorders, and does not resemble one of the five named OSFED examples, the diagnostic label *OSFED-other* is given. An example of OSFED-other would be an individual who engages in subjective binge eating as well as either fasting or excessive exercise to counteract the potential effects of loss-of-control eating on shape and weight. Some have referred to this pattern as "compensatory eating disorder" (Davis et al. 2014), though the term is not listed in *DSM-5* and would technically be classified as OSFED-other.

Another conceptually related diagnosis that is separate from OSFED is *DSM-5 unspecified feeding or eating disorder* (UFED). This label is given if a clinically significant eating disorder is present but the clinician does not have sufficient information to provide a more specific feeding or eating disorder diagnosis (e.g., in an emergency room setting). Of note, a UFED diagnosis would rarely be conferred in research settings where respondents respond fully to structured diagnostic interviews, because it is the lack of information – rather than the nature of the eating disorder presentation itself – that characterizes the UFED diagnosis.

Historical Background

The first residual eating disorder category, labeled "atypical eating disorder" in *DSM-III*, debuted in 1980 to capture individuals who did not meet criteria for anorexia nervosa or bulimia. *DSM-III-R* renamed the category EDNOS in 1987 and provided three specific example presentations. These examples included (1) recurrent purging to influence body weight or shape in the absence of binge eating, (2) all symptoms of anorexia nervosa except amenorrhea, and (3) all symptoms of bulimia nervosa except that binge eating and inappropriate compensatory behaviors occurred less than twice per week. These presentations were described with numbered examples but not explicitly named. In 1994, *DSM-IV* included these three original presentations and added three new examples including (1) binge-eating disorder, (2) a syndrome characterized by repeatedly chewing and spitting out food, and (3) a presentation featuring all symptoms of anorexia nervosa except low weight. Binge-eating disorder was the only EDNOS clinical description to feature specific diagnostic criteria listed in the appendix as a disorder warranting further research. The description and examples of EDNOS did not change when *DSM-IV-TR* was later published in 2000.

The current feeding and eating disorder classification is based on *DSM-5* (2013). *DSM-5* introduced important revisions that broadened the diagnostic criteria for anorexia nervosa and bulimia nervosa, recognized binge-eating disorder as an "official" eating disorder, and classified feeding disorders together with eating disorders. Additionally, the residual diagnostic category of EDNOS was divided into OSFED (with the five named examples described under *Definition*) and UFED.

Current Knowledge

In studies conducted prior to *DSM-5*, *DSM-IV* EDNOS was often the most prevalent eating disorder diagnosis, comprising 40–60% of patients at specialty eating disorder clinics, up to 90% of eating disorder cases in general psychiatric settings, and approximately 75% of eating disorder diagnoses in community-based studies (Thomas et al. 2009). Thus, a primary aim of *DSM-5* revisions was to reduce the proportion of residual diagnoses by broadening the diagnostic criteria for anorexia nervosa and bulimia nervosa and including binge-eating disorder as an officially recognized diagnosis. Current evidence largely supports that *DSM-5* revisions reduced reliance on residual eating disorder categories by at least 50%. When diagnoses conferred under *DSM-5* are compared to those conferred under *DSM-IV*, rates of residual diagnoses (i.e., EDNOS/OSFED) are lower, rates of anorexia nervosa are higher, and rates of bulimia nervosa are either higher or stable in treatment seeking samples (e.g., Fisher et al. 2015; Mancuso et al. 2015; Thomas et al. 2015). However, OSFED remained the most prevalent diagnosis in a study of individuals with overweight or obesity seeking weight-loss treatment (Thomas et al. 2014), suggesting that sample characteristics and study setting likely influence the diagnostic distribution of eating disorders.

Regarding the prevalence of individual OSFED examples, atypical anorexia nervosa is typically the most prevalent OSFED presentation in clinical samples (Fisher et al. 2015; Thomas et al. 2015). For example, in a clinical sample of 309 adolescents seeking an eating disorder evaluation in an adolescent medicine practice, 30.10% had atypical anorexia nervosa, 5.83% had purging disorder, 0.65% had subthreshold bulimia

nervosa, and 0.65% had subthreshold binge-eating disorder (the remaining 62.77% had an unspecified or full-threshold eating disorder; Fisher et al. 2015). In contrast, subthreshold bulimia nervosa and purging disorder have typically emerged as the most prevalent OSFED diagnoses in epidemiological studies. For example, in a large community sample of adolescent females in which 13.1% met criteria for a lifetime eating disorder diagnosis, 4.4% had subthreshold bulimia nervosa, 3.6% had subthreshold binge-eating disorder, 3.4% had purging disorder, and 2.8% had atypical anorexia nervosa (Stice et al. 2013). The estimated prevalence of night eating syndrome ranges from 1.5% (general population; Rand et al. 1997) to 6.0% (weight-loss treatment-seeking sample; Thomas et al. 2014).

Current Controversies

Assigning specific labels to OSFED examples is helpful in facilitating recognition of clinically significant disordered eating patterns, enhancing communication among treatment providers, and stimulating research to better understand these distinct presentations. However, despite these improvements, controversies remain. For example, because the current OSFED nomenclature is based on prototype matching rather than formalized diagnostic criteria, high inter-rater reliability may be difficult to achieve. For example, kappas for individual OSFED examples ranged from 0.05 (Thomas et al. 2015) to 0.15 (Thomas et al. 2014) in two recent studies comparing clinician versus researcher diagnoses. Relatedly, OSFED examples have no empirically derived diagnostic cut-offs (e.g., there is no weight-loss criterion to determine whether an individual warrants a diagnosis of atypical anorexia nervosa versus normal dieting) or trumping scheme (e.g., a diagnosis of subthreshold bulimia nervosa does not necessarily take precedence over a diagnosis of purging disorder if a patient presents with features of both syndromes). In addition, OSFED examples are meant to be mutually exclusive, yet demonstrate considerable symptom overlap (e.g., alternating evening hyperphagia and binge eating occurring twice per month for 2 months could be potentially classified as either subthreshold binge-eating disorder or night eating syndrome). In summary, lack of clear diagnostic criteria and boundaries for each OSFED example has the potential to generate more questions than answers regarding prevalence, correlates, and prognosis of individual presentations.

Finally, unlike full-threshold eating disorders, OSFED and its individual example presentations do not have specific empirically supported treatments. In contemporary practice, clinicians often adapt existing treatment manuals meant for officially recognized disorders to fit the presenting problem (e.g., atypical anorexia nervosa in an adolescent might be treated with family-based treatment, whereas subthreshold bulimia nervosa in an adult might be approached with cognitive-behavioral therapy). In contrast, enhanced cognitive-behavioral therapy assumes a transdiagnostic perspective that employs similar strategies for addressing distinct eating disorder presentations.

Future Directions

As our understanding of eating disorder psychopathology becomes more nuanced, our ability to make finer distinctions across clinical presentations will continue to increase. There may come a point, however, when incorporating these increasingly fine distinctions into our diagnostic nomenclature will reach the asymptote of clinical utility. Recent initiatives including the Research Domain Criteria set forth by the National Institute of Mental Health suggest an alternative dimensional approach to the classification of psychiatric disorders. Further examination of the validity and utility of highlighting differences (categorical approach) versus similarities (dimensional approach) among eating disorder presentations is warranted. Relatedly, there is a need for identification of efficacious treatments for individuals with OSFED, as current research has not yet guided us toward specific (i.e., one for each OSFED example) versus general (i.e., a transdiagnostic approach) management. Lastly, specific guidelines considering both the sensitivity

and specificity of the distinction between OSFED and normal dieting across the "almost anorexic" spectrum (Thomas and Schaefer 2013) must be developed so that individuals with clinically significant presentations can be appropriately identified and offered the care they deserve.

Cross-References

- ▶ Binge-Eating Disorder
- ▶ Night Eating Syndrome History Inventory (NESHI)/Night Eating Questionnaire (NEQ)
- ▶ Starvation in Children, Adolescents, and Young Adults: Relevance to Eating Disorders

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Outcome Evaluation

- ▶ Current Status of Eating Disorder Prevention Research
- ▶ Evidence-Based Prevention Program Delivery

Outreach Programs: An Alternative Model of Care for Individuals with Enduring Eating Disorders

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Definition

Outreach programs are a relatively new and innovative form of care recommended for individuals who have not responded to traditional treatment (e.g., inpatient hospitalization, intensive day programs). The primary barrier to the success of traditional care for outreach program candidates is a mismatch between the action-oriented nature of treatment and the patient's readiness for change. As such, patients who benefit most from outreach programs are those with an enduring course of illness, a history of repeat hospitalizations, and/or comorbid medical or psychiatric conditions. Typically, the mandate of outreach

programs is to address and maintain medical and psychiatric stability while working on goals that enhance the patient's quality of life.

Rationale

An underlying premise of outreach programs is that the repetition of previously unsuccessful treatment is counterproductive for the patient, care team, and health-care system. For instance, lengthy inpatient hospitalizations are expensive and evidence supporting their efficacy in this population is poor. Instead, outreach programs offer treatment focused on improving aspects of patients' lives that are of value to *them*. This involves matching treatment goals to patient readiness and working to reduce harms associated with the eating disorder such as social isolation, medical complications, and crisis hospitalizations.

Description

Outreach programs typically make use of motivational interviewing, psychosocial rehabilitation, and harm reduction models. Thus, although patients are often not focused on achieving full recovery, they are actively working on reducing the costs associated with the eating disorder. These may include medical issues (i.e., stress fractures secondary to malnutrition), financial stress due to daily binge/purge episodes, or life disruption resulting from unplanned crisis medical stabilization hospitalizations.

Outreach programs are offered by multidisciplinary teams and typically include a medical provider, dietician, social worker, psychologist, and outreach mental health worker. A unique feature of outreach programs is that a portion of the care that patients receive occurs outside of traditional medical or clinic settings. That is, health workers may meet with patients in their community, such as in coffee shops, community centers, parks, or in the patient's own home.

Examples of outreach goals are to decrease isolation, anxiety, or depression, increase autonomy, improve quality of life, and increase hope for the future. Thus, an outreach worker may help an anxious or depressed patient register for and participate in a course, attend a medical appointment, or work on budgeting or time management. Examples of goals that are eating disorder related but not oriented toward full recovery include decreasing time spent bingeing and purging, reducing amount of time spent exercising, and maintaining connection with medical professionals.

Outreach programs are flexible and tailored to the needs of the individual. For patients who are involved in multiple harmful activities (e.g., bingeing and purging, substance use, cutting), the program may first assist the patient to identify and reduce the activity that is most damaging in order to lessen overall risk or harm. Subsequent goals often emerge as the disruption caused by primary harmful behaviors is lessened.

One of the ways in which outreach programs balance patient safety while maximizing patient autonomy is by having clear nonnegotiable guidelines, or therapeutic boundaries, which patients and outreach program staff commit to at the onset of treatment. Nonnegotiables provide clarity to patients and outreach team members about the therapeutic frame and are especially helpful in providing guidelines about what will happen if the patient's medical or psychiatric condition worsens. A treatment philosophy for outreach programs ensures that nonnegotiables have a sound rationale, are not experienced by patients as a surprise, are implemented consistently, and patient autonomy is maximized. An example of how a nonnegotiable philosophy is helpful in medical management is described in the following section.

Medical Management

Many patients in outreach programs have a history of poor health requiring hospitalizations for renourishment and/or to decrease harms associated with the eating disorder. One of the primary medical goals of outreach programs is to increase the efficiency of and reduce or eliminate

involuntary medical admissions. As hospital admissions are often disruptive, patients are usually pleased to be involved in this planning process.

Built into outreach programs is a regular means for monitoring the patient's medical status. These appointments provide feedback to the care team on critical health indicators. In order to *reduce surprises*, the conditions under which patients would require hospitalization are made clear as early as possible. This decreases anxiety and provides patients with the opportunity, if desired, to use program support to stabilize their health to avoid a hospital admission. This could occur by meeting with an outreach worker for goal setting around electrolyte replacement, reducing use of compensatory behaviors, or increasing hydration.

Explaining the *rationale* for medical monitoring appointments (including regular weighing and blood work in order to address issues early on and reduce the need for an admission) is also important for all team members. Having regular contact with patients promotes planned admissions, when necessary, that are linked to the patient's therapeutic goals. For instance, a patient who wishes to complete a course (e.g., job training) may be motivated to use program support to improve her nutritional intake and not have her life sidetracked by a hospitalization. Patients can thus be provided with timely and appropriate feedback and choose to work with their outreach program team to avoid inpatient care. Over time, reliance on hospitalization may be decreased.

Patients who have a history of malnourishment and low weight requiring medical intervention will have a nonnegotiable involving body mass index and other medical indicators. At the onset of treatment, the team and the patient meet to determine a threshold below which the patient would need to be hospitalized for renourishment. It is important for nonnegotiable thresholds to be *enforced consistently* by all team members at all times and across patients. This creates an environment of safety for both the patient and for outreach team members and reduces feelings of favoritism or persecution and testing of nonnegotiable limits.

Finally, patients benefit from having choices throughout treatment in order to *maximize autonomy*. This handing over of responsibility as much as possible is a key component of outreach programs and helps to reduce dependence on care teams. There may be times when none of the patient's choices are desirable. For instance, a patient who requires renourishment while hospitalized for medical stabilization may choose from eating a prescribed meal plan, drinking meal replacement, or receiving a nasogastric tube. While the patient may dislike each of these options, having support from her outreach worker to choose the renourishment method with which she is most comfortable increases her sense of autonomy and participation in her health goals.

A nonnegotiable philosophy of no surprises, sound rationale, consistent implementation, and maximizing autonomy is thus integral to outreach program treatment planning and helps ensure a respectful and safe delivery of care.

Current Knowledge

Given that outreach programs are a relatively new form of care for eating disorder patients, little is known about outcomes. However, a small number of studies have shown improvements in motivation, psychiatric symptoms, eating disorder symptoms, body mass index, and quality of life. There is some evidence that outreach programs better meet the needs of a proportion of patients who would otherwise be admitted for lengthy inpatient hospitalizations and/or who relapse quickly once discharged. Efficient use of outreach programs can therefore reduce the frequency and reliance on hospitalization and cut costs. Moreover, there are reports of patients who after experiencing the benefits of decreased social isolation, increased autonomy, and improved quality of life resulting from outreach programs eventually engage with and benefit from action-oriented treatment.

Controversies

Individuals who benefit from outreach programs may at times present with ethical and clinical

challenges. Some issues that are important to consider for such programs include:

Involuntary admissions. Ideally, a meeting is held early in treatment in which the outreach program team meets with the patient and any other individuals identified by the patient to discuss treatment options in the event of serious medical risk. The use of an ethical decision-making framework that considers principles of beneficence and autonomy is helpful in developing nonnegotiables around involuntary admissions. This process allows the patient to contribute to a plan that has as its mandate to use involuntary admissions only as a last resort and that prioritizes patient autonomy.

Weight gain. As noted earlier, outreach programs do not focus on full recovery and may not require patients to gain weight. This may be controversial, given that early weight gain is emphasized in traditional eating disorder treatment. It should be noted that candidates for outreach programs have a history of either leaving treatment or being dismissed from treatment for not gaining weight. Despite weight not being an initial focus, maintaining medical stability is nevertheless desirable for this population. Furthermore, as noted earlier, some patients have gone on with the support of their outreach program to benefit from action-oriented treatment and make significant weight gains. Thus, for some patients, flexibility regarding the timing of weight gain may be beneficial.

Cost-effectiveness: Outreach programs may appear to require a significant investment of time and money. For example, there may be significant expenses incurred from having a full staff attend meetings to discuss ethical issues and determine nonnegotiables. In addition, transport to and from patient meetings may also be considerable. These and similar time commitments may be perceived as costly given that many patients do not show overt signs of improvement in some of their eating disorder symptoms (e.g., weight gain). Economic analysis of outreach programs suggests that total treatment costs are in actual fact small compared to those incurred in the absence of such programs where greater reliance on lengthy hospitalizations is necessary. Caring for patients in the community via outreach programs is likely the most cost-effective option.

Future Directions

Outreach programs for individuals with eating disorders are a promising, flexible, yet relatively novel form of care for individuals who have not benefitted from traditional, action-oriented treatment. Although a small number of review papers have described outreach programs (see references), more information is needed about outcomes. Given that a range of disciplines can be involved in providing outreach support, further research is required to determine what level of training is optimal, what level of supervision is needed, and what intensity and duration of treatment is most favorable. Outreach programs offer an additional component of care that may help us to determine the best treatment for which patient at what time.

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Overall Low Self-Regard

► Self-Criticism and Low Self-Esteem

Overconcern with Shape and Weight

► Overevaluation of Shape and Weight and Its Assessment

Overevaluation of Shape and Weight and Its Assessment

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Synonyms

Body Image; Overconcern with shape and Weight; Undue influence of shape and weight on self-evaluation

Definition

It has been proposed that individuals with eating disorders share a distinctive "core psychopathology" (Cooper and Fairburn 1993; Fairburn et al. 2003). This is the overevaluation of shape and weight and their control. Most individuals with eating disorders judge their self-worth largely, or even exclusively, in terms of their shape and weight and their ability to control them. This is in contrast to the majority of people who evaluate themselves on the basis of their perceived performance in a wide range of areas, for example, their work performance and the quality of their relationships.

It is important to distinguish overevaluation of shape and weight from body dissatisfaction. Dissatisfaction or dislike of one's appearance (sometimes referred to as "normative discontent") is not peculiar to people with an eating disorder: indeed, it is common among women in the general population.

Overevaluation of Shape and Weight in the Classification of Eating Disorders

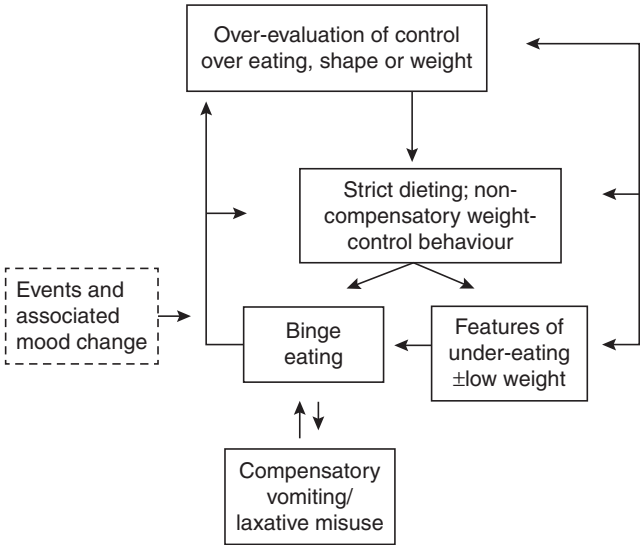
In terms of the classification of eating disorders, a broader but related construct, "persistent overconcern with shape and weight," first appeared in the diagnostic criteria of bulimia nervosa in DSM-III-R. DSM-IV later refined this criterion to take the form of the more stringent criterion of overevaluation of shape and weight. The overevaluation of shape and weight has been retained in DSM-5 for bulimia nervosa and is also included as part of the body weight and shape criterion for anorexia nervosa.

Shape and weight overevaluation is not included in the criteria for binge eating disorder. However, overevaluation has been shown to be present in a subcategory of this group. It has been proposed that it may be a useful diagnostic specifier to indicate individuals with BED who have greater levels of general psychopathology and social dysfunction and who may require modified interventions as a result (Grilo 2013).

Why Is Overevaluation of Shape and Weight Important?

According to the transdiagnostic theory of eating disorders, most features of eating disorders are secondary to the overevaluation of shape and weight and to its consequences (Fairburn et al. 2003; Fairburn 2008). In other words, overevaluation of shape appears to be the "engine" driving the eating disorder.

This core psychopathology is expressed in several ways and there are differences between people, so that the specific features seen in any individual vary (see Fig. 1 for the "transdiagnostic formulation" of the processes that cause eating disorder features to persist). In terms of eating habits, overevaluation of shape and weight leads to dietary restraint, where an individual attempts to limit his or her food intake. This behavior is



Overevaluation of Shape and Weight and Its Assessment, Fig. 1 The "transdiagnostic" cognitive behavioral theory (Reproduced with permission from *Cognitive Behavior Therapy and Eating Disorders* by Christopher G. Fairburn. Copyright 2008 from the Guilford Press)

seen across most of the eating disorders (other than some forms of binge eating disorder, see below). This "dieting" takes the form of multiple, extreme, and highly specific dietary rules. Attempting to follow such rules tends to be highly impairing as it requires a great deal of effort, often provokes anxiety, and can make socializing difficult if not impossible. The presence of strict dietary rules also leads to further problems. If the attempts to limit eating are successful, there may be severe weight loss or, more commonly, the development of binge eating.

The overevaluation of weight concern also often leads to frequent weight checking (weighing) and a consequent preoccupation with trivial changes in the number on the scale. Alternatively, it may result in a complete avoidance of weighing, with an associated "fearing the worst" and a high degree of concern about weight. In terms of body shape, it leads to similar behavior, either frequent body checking or body avoidance, both of which also serve to maintain the overconcern. Such overevaluation of shape and weight can have a harmful effect on relationships, due to a dislike of other people seeing or touching one's body.

It is worth noting that in a subgroup of eating disorder patients the core psychopathology takes a different form. This variant is the overevaluation of control over eating per se rather than the overevaluation of shape and weight (although it is worth noting that the two can coexist). In this case, individuals judge their self-worth in terms of their ability to control their eating for its own sake, rather than simply as a means of influencing shape and weight. This type of presentation is most common in young people and especially those with a short history and those who are underweight. It is also seen in non-Western cases. Such individuals tend to be very concerned about the details of their eating (e.g., exactly what food is eaten, when they eat, and how many calories are consumed) but without concerns about their body shape and weight.

Why Assess Overevaluation of Shape and Weight?

There are two main reasons why it is useful to assess the overevaluation of shape and weight. Firstly, it informs diagnosis and, more broadly speaking, an understanding of the nature and severity of the eating problem present. Secondly, assessing this overevaluation is an important part of deciding how best to proceed in management.

Overevaluation of shape and weight is a key feature of most eating problems. Therefore, if overevaluation of shape and weight is detected

during a clinical assessment, in combination with dysfunctional eating behavior and impairment, it is highly suggestive of an eating disorder. Furthermore, given that overevaluation of shape and weight is proposed to be a core maintaining mechanism underlying most of the features of eating disorders, it suggests that treatment needs to target this in order to produce long-lasting benefits. Indeed, clinical experience and research evidence suggest that unless the overevaluation is effectively addressed, patients are at considerable risk of relapse (Fairburn et al. 1993).

If eating difficulties are present but overevaluation of shape and weight is absent, then it is important to explore other features which may be driving the problem with eating. This may be a feature which is not particular to the eating disorders. For example, difficulties with eating may be secondary to a mood disturbance (e.g., extreme weight loss as a result of a clinical depression) or to anxiety (e.g., difficulty eating with others due to social anxiety), or as a result of straightforward overeating in someone with obesity. Alternatively, as noted earlier, some patients do have an eating disorder but without overevaluating shape and weight.

How to Assess Overevaluation of Shape and Weight?

Clinical Assessment of Overevaluation of Shape and Weight

Overevaluation of shape and weight should be assessed during the initial clinical evaluation interview, which is usually the first step for any psychiatric problem. This would be part of a wider assessment by a clinician designed to establish the nature of the problem and to engage the client or patient.

The assessment of overevaluation includes direct questions about the importance placed on shape and weight in terms of self-worth as well as queries about those experiences which tend to be associated with it. The client should be asked about his or her view of their body shape and weight (e.g., "How do you feel about your body? Do you have any concerns about your weight?"), the importance of shape and weight in their self-evaluation (e.g., "Does your body or your weight have an effect on how good or bad you feel about yourself as a person?"), whether or not they engage in body checking (e.g., "Do you scrutinize or examine your body, for example by looking in the mirror?"; "Do you compare your body with other people's bodies?") or avoidance (e.g., "Do you ever try to avoid seeing your own body or showing it to others, for example by wearing baggy clothes or getting dressed in the dark?"), and whether or not they have experiences of feeling fat (e.g., "Do you ever have times where you suddenly 'feel fat'?").

In addition, standardized information on overevaluation and shape and weight can be obtained through administering the Eating Disorder Examination Questionnaire (EDE-Q 6: Fairburn and Beglin 2008) (see "▶ Eating Disorder Examination (EDE)/(EDE-Q)").

Research Assessment of Overevaluation of Shape and Weight Using the EDE

The Eating Disorder Examination (EDE) interview (Fairburn et al. 2008a) provides a thorough assessment of current eating disorder features including overevaluation of shape and weight (see "▶ Eating Disorder Examination (EDE)/(EDE-Q)"). This interview is too in depth and time-consuming for routine clinical use. However, it is the measure of choice for assessing overevaluation for research purposes as it provides a well-established, reliable, and valid assessment of features of eating disorders, with community norms available.

The EDE includes Weight Concern and Shape Concern subscales. These measure the degree of concern about weight and shape, respectively. Each subscale is made up of several items assessing different aspects of weight and shape concern. Within these there are two specific items assessing "importance of weight" and "importance of shape": "*Over the past four weeks has your weight (the number on the scale) been important in influencing how you feel about (judge, think, evaluate) yourself as a person?*" and "*What about your shape? How has it compared in importance with your weight in*

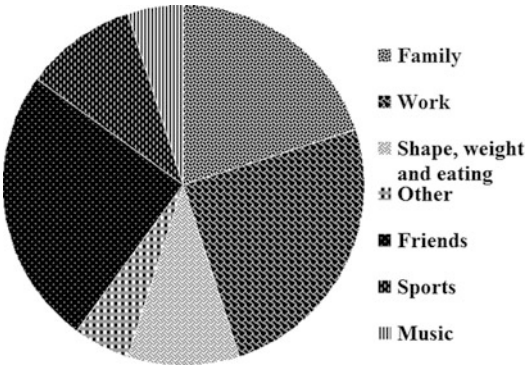
influencing how you feel about yourself?". Further instructions for helping participants to understand these more complex items are provided by the EDE.

Guidance is also provided in the EDE for the threshold levels of overevaluation needed to generate DSM-5 eating disorder diagnoses. It is specified that shape and weight must be at least of moderate importance in terms of self-evaluation (equivalent to "definitely one of the main aspects of self-evaluation") in order to meet the criterion that "self-evaluation is unduly influenced by shape and weight."

Assessing the overevaluation of shape and weight in children and adolescents should be conducted using the version of the EDE specifically designed for use with this group (Bryant-Waugh et al. 1996).

Assessing Overevaluation of Shape and Weight in the Context of "Enhanced Cognitive Behavior Therapy" (CBT-E)

Enhanced cognitive behavior therapy (CBT-E) is the latest version of the leading evidence-based treatment for eating disorders (see "► Enhanced Cognitive Behavior Treatment: Transdiagnostic Theory and Treatment" entry). It has its origins in a treatment for adults with bulimia nervosa (CBT-BN) which has since been "enhanced" to improve its potency and to address "transdiagnostic" processes across the eating disorders. Within CBT-E, the overevaluation of shape and weight occupies a central position in the case formulation (see Fig. 1). It is assessed within treatment as part of the intervention designed to address the overevaluation of shape and weight (Fairburn et al. 2008b). This intervention begins with a general discussion of the concept of self-evaluation. It is explained by the therapist that if we are meeting our personal standards in the areas of life we value we tend to feel good about ourselves, but if we are not then we tend to feel bad. Therapists give examples of possible domains that people might judge themselves in terms of, such as "performance at work" or "quality of friendships." It is suggested that a good clue as to the importance of an area is how we feel when that area is going badly. If we feel really bad about ourselves as a person as a result of feeling that an area of life is not going well, then it indicates that this aspect is important to one's self-evaluation.



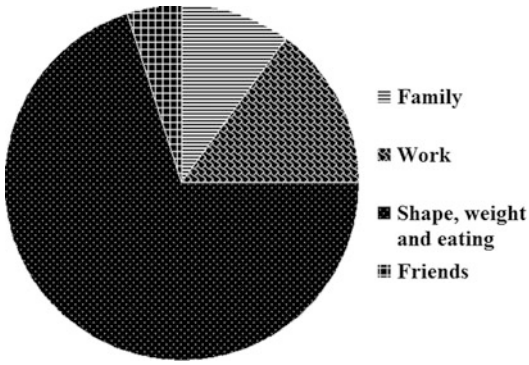
Overevaluation of Shape and Weight and Its Assessment, Fig. 2 A pie chart of a young woman without an eating problem (Reproduced with permission from *Cognitive Behavior Therapy and Eating Disorders* by Christopher G. Fairburn. Copyright 2008 from the Guilford Press)

The therapist then helps the patient to create a list of those areas which are important to his or her own system of self-evaluation. During this the therapist may need to help the patient distinguish between those areas which he or she regards as "important" in general (for example, because they are regarded by society as such; e.g., work or relationships with family members) but which in practice don't actually influence the way they view themselves, and things that form part of their current evaluation. Occasionally, shape and weight may not be mentioned and in such instances the therapist should raise this by asking whether appearance, and body shape and weight, influence their self-worth.

It is then suggested that a pie chart would be a helpful way to represent the patient's system of self-evaluation. The various slices within the pie chart, and their sizes, can be used to illustrate the importance of areas of life in his or her scheme of self-worth. The therapist and patient then draw out a tentative pie chart to visually represent the patient's self-evaluative scheme. Two pie charts are shown in Figs. 2 and 3, with one typical of a person without an eating disorder and the other typical of someone with overevaluation of shape and weight. It can be seen that the patient's pie chart is dominated by a large slice representing the

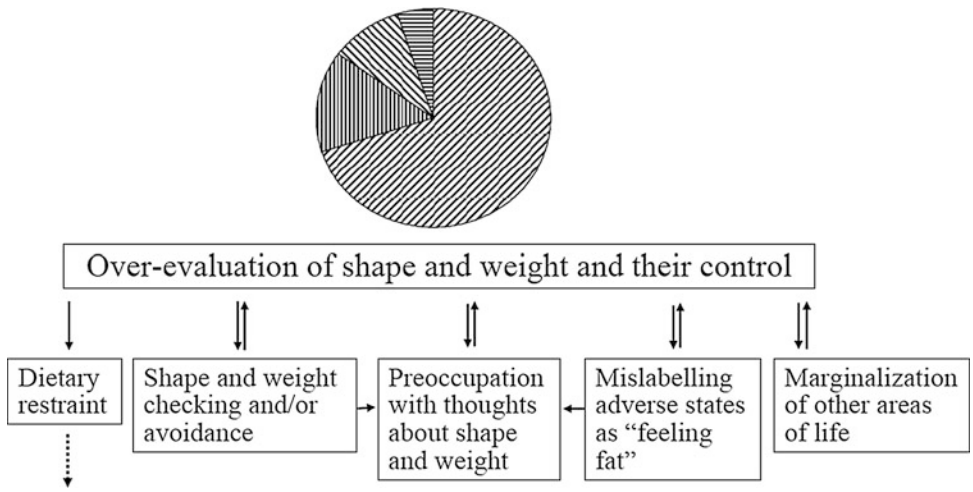
overevaluation of shape and weight and their control.

This in-treatment assessment of overconcern about shape and weight would also include the creation of an "extended formulation" to show the secondary effects. The therapist asks directly about shape and weight checking/avoidance, experiences of feeling fat, and whether other aspects of life have become "squeezed out" as a result of the dominance of weight and shape concerns. The therapist shows how these features are consequences of the overevaluation. The extended formulation also depicts the various vicious circles that exist which serve to maintain the concerns about shape and weight (see Fig. 4). It is explained that in treatment each of these features will be addressed, alongside increasing the importance of other domains for self-evaluation. In this way there should be a gradual reduction in the overevaluation of shape and weight, with an associated improvement in the eating problem.



Overevaluation of Shape and Weight and Its Assessment, Fig. 3 The pie chart of a patient (Reproduced with permission from *Cognitive Behavior Therapy and Eating Disorders* by Christopher G. Fairburn. Copyright 2008 from the Guilford Press)

Towards the end of treatment, the pie chart can be redrawn and, hopefully, patients will be pleased to see that the original shape and weight slice is significantly smaller and that new slices have appeared. At the end of CBT-E a clinical interview, and the EDE-Q, would be used to reassess overevaluation of shape and weight. If the overevaluation of shape and weight has persisted largely unchanged then it is suggestive of the fact that treatment has not been sufficient to address the eating disorder. However, it should be noted that of all the features of an eating disorder the overevaluation of shape and weight is likely to take the longest to address. There may still be some residual shape and weight concerns at the end of treatment, but these should improve over the period of follow-up.



Overevaluation of Shape and Weight and Its Assessment, Fig. 4 The overevaluation of control over shape and weight: an "extended formulation" (Reproduced with permission from *Cognitive Behavior Therapy and Eating Disorders* by Christopher G. Fairburn. Copyright 2008 from the Guilford Press)

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Cross-References

- ▶ Eating Disorder Examination (EDE)/(EDE-Q)
- ▶ Enhanced Cognitive Behavior Treatment: Transdiagnostic Theory and Treatment

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Oxytocin in Feeding and Eating Disorders, Role of

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Synonyms

Hyperphagia: Overeating; Pituitary gland: Hypophysis; Posterior pituitary lobe: Neurohypophysis

Definition

Gene: A section of DNA that codes for the production of a specific protein.
Hormone: A biochemical messenger that is carried in the blood until it binds to receptors in or on its target cell.
Hypothalamus: A region of the lower brain that integrates the neural and endocrine systems through its effect on the pituitary gland.
Oxytocin: A hormone/neurotransmitter that is released by neurons in the hypothalamus and serves many functions related to lactation, partition, and the regulation of feeding and anxiety.
Neurotransmitter: A molecule released by a neuron that acts to influence the activity of other neurons.
Nucleus: A tightly packed grouping of neuronal cell bodies in the central nervous system.
Pituitary Gland: An endocrine gland that receives chemical and neural signals directly from the hypothalamus. Through the release of a variety of hormones, the pituitary gland exerts control over a wide range of bodily functions.
Prader-Willi Syndrome: A genetic disorder resulting from a deletion of genes on the copy of chromosome 15 inherited from the father, which results in physical and behavioural abnormalities including compulsive overeating.

Background

Feeding and eating disorders arise and are maintained through the interaction of many different factors, including personality traits, social interactions, food availability, and aspects of the cultural environment. Additionally, there is evidence to suggest that certain biological factors make some individuals more prone to developing a feeding or eating disorder. Secondary to the onset of the disorder, abnormal eating and weight-loss behaviors can cause further disruptions in the neural and hormonal systems that control appetitive and emotional regulation. Some of these physical effects then have the flow-on consequence of altering physiology or impacting mood in such a way that further reinforces behaviors such as binging or restricting food intake.

The physiological systems governing appetitive and emotional state are highly complex, involving the interaction of many different hormones and patterns of neural activity. One hormone that has recently received increasing attention by eating disorders researchers is oxytocin. Oxytocin is primarily produced by neurons in the paraventricular nucleus and supraoptic nucleus of the hypothalamus. (Note: A nucleus is a tightly packed group of neuron cell bodies within the brain.) Oxytocin doubles as a neurotransmitter that acts directly on oxytocin receptors in the hypothalamus and in other central brain structures. Oxytocin is also secreted to the periphery via the posterior pituitary lobe. In humans, oxytocin has long been known for its effects on social functioning and for its ability to induce childbirth and milk letdown during lactation.

Findings from Animal Studies

In addition to the social and reproductive functions that oxytocin serves in humans, there is a wide base of evidence from animal studies to demonstrate that oxytocin also has an impact on eating behavior. In general, greater activity of oxytocin on oxytocin receptors tends to be associated with reduced feeding. This effect, however, is conditional on a number of social and environmental factors, which will be discussed further below.

Genes

The *SIM1* gene has been found to play an important role in coding for a protein that supports the normal development of the paraventricular nucleus of the hypothalamus. As discussed above, these neurons contribute greatly to the brain's production of oxytocin. Mice who lack one copy of this gene have been found to exhibit oxytocin levels that are reduced by as much as 80%. These same mice have also been found to exhibit pronounced hyperphagia (overeating) and obesity.

Similarly, mice lacking the specific gene for oxytocin production also exhibit greater consumption of a sucrose solution versus water when compared to wild-type mice (with normal copies of the oxytocin gene). This effect is observed in conditions of stress and non-stress.

Both of these findings support the hypothesis that oxytocin activity tends to inhibit eating behavior, and insufficiencies in this system tend to lead to disinhibited eating behavior.

Effect of Oxytocin Administration on Animal Behavior

It has been repeatedly found that injecting oxytocin directly into certain areas of the brain results in a decrease in subsequent food consumption in rats, with a particularly strong effect on the consumption of carbohydrates. By contrast, pretreatment with chemicals that block oxytocin receptors (oxytocin antagonists) has been found to evoke greater feeding when animals are given access to food in isolated cage conditions.

Physiological Pathways

It should be noted that oxytocin does not act alone to influence eating, but is rather one component in a large and interconnected appetitive system. For example, oxytocin acts as an intermediary step in some pathways that result from rising nutrition levels in the bloodstream (following a meal).

Oxytocin is also secreted in response to conditions where continued eating would lead to harm (e.g., in the presence of certain toxins and when the stomach is greatly distended).

Conditional Effects

The effect that oxytocin has on suppressing feeding depends in part on social and environmental factors. For example, animals tend to exhibit reduced feeding in response to the anxiety provoked by novel foods and environments. However, administering chemicals that act on oxytocin receptors in the same fashion as oxytocin (oxytocin agonists) tends to eliminate the undereating that is typically observed in conditions of novelty-induced stress.

Therefore, on the whole, the breadth of empirical literature examining oxytocin suggests that the effect of oxytocin on feeding varies, generally acting to suppress feeding behavior, while enhancing feeding in conditions of anxiety, with the ultimate consequence of promoting a healthy state of homeostasis.

Effects on Eating Behavior in Humans

To summarize thus far, the pattern of evidence from animal studies implicates oxytocin as an important agent acting within complex appetitive systems, which generally acts to suppress eating behaviors.

Genes

Similarly, in humans, it has been found that some genetic mutations disrupting the normal development of oxytocin systems result in extreme patterns of overeating and obesity. One such example is the genetic condition, Prader-Willi syndrome. This syndrome is caused by a deletion of several genes on the copy of chromosome 15 inherited from the father, which results in a reduction in the number of oxytocin-producing neurons in the paraventricular nucleus of the hypothalamus. Similar to the overeating behavior seen in oxytocin knockout mice, Prader-Willi syndrome is typically characterized by extreme, compulsive overeating.

Effect of Oxytocin Administration on Human Behavior

Several pilot studies have found that the administration of an intranasal oxytocin spray has also been found to reduce eating and weight gain in humans. This effect, however, is dependent upon the extent of participants' hunger, the types of food provided, and the presence of a preexisting eating disorder. Of particular note, Ott and colleagues (2013) found that oxytocin administration acted to reduce the amount of a cookie snack consumed by healthy males whilst already full, without affecting the consumption of a full meal in a hunger condition. This may indicate a specific role for oxytocin in reducing eating for pleasure, as opposed to affecting eating for the purpose of sating hunger.

Oxytocin Abnormalities in Eating Disorders

In line with evidence indicating a role for oxytocin in influencing eating behavior, abnormalities in oxytocin systems have been found in both anorexia nervosa and bulimia nervosa. Individuals with anorexia nervosa-restricting subtype have been found to have lower levels of oxytocin in cerebrospinal fluid than healthy controls, altered expression of the oxytocin receptor gene, heightened levels of oxytocin after feeding, and a reduction in the level of reactive oxytocin secretion that is generally seen in conditions of hypoglycemia (low blood sugar). However, it is unclear whether these findings reflect a causal role for oxytocin abnormalities in anorectic behavior or psychopathology or are rather a result of inadequate nutrition in the acute stage of the disorder.

Women with bulimia nervosa have been found to have similar levels of peripheral oxytocin when compared to healthy control participants, but there is evidence to suggest that some genetic variants of oxytocin receptor genes are associated with greater bulimic behaviors among women. Due to the fact that the effects from genotype to behavior can only move in one direction, this evidence supports the hypothesis that alterations in oxytocin systems have a causal role in predisposing some individuals to develop bulimia nervosa.

Oxytocin as a Novel Therapeutic Target

Given preliminary evidence that artificial administration of oxytocin can deter both reward-based eating and anxiety-induced undereating, oxytocin systems have more recently been explored as a potential target in the treatment of feeding and eating disorders. For example, Kim et al. (2015b) have tested the effect that the artificial administration of oxytocin exerts on eating behaviors among healthy control women, women with anorexia nervosa, and women with bulimia nervosa. They did not find that oxytocin administration had an effect on how much apple juice drink was consumed by individuals in any diagnostic group following a 3.5-h fast. However, they did record a reduction in the total quantity of food consumed by participants with bulimia nervosa over the following 24-h period after oxytocin administration (when compared to a placebo condition). The same team also found that intranasal oxytocin reduced the attentional bias toward images of feared foods and body shape-related images in anorexia nervosa.

An Australian team has also tested whether daily administration of an intranasal oxytocin spray over a 4-week period had significant therapeutic benefit for individuals with anorexia nervosa. Preliminary findings from this trial indicate reductions in the participants' levels of eating concern, although this was not associated with greater weight restoration.

Effects on Stress: An Explanatory Mechanism?

On the whole, the evidence from animal studies and studies in healthy humans points to a role for oxytocin in affecting appetite and eating behaviors. An alternative explanation for results observed in clinical populations, however, is that the effects of oxytocin on eating behavior might occur due to an indirect consequence of the hormone's effects on stress. It is well established that restricting and binging behaviors are often used by individuals in an attempt to regulate emotions. Therefore, it could be that reductions in binging behaviors in bulimia nervosa and reductions in eating concerns among individuals with anorexia nervosa result arise, at least in part, due to oxytocin's anxiety-reducing (anxiolytic) effects. This hypothesis receives supports from studies which have found that the intranasal administration of oxytocin can subdue the anxiety response to feared stimuli.

Summary

In summary, administering oxytocin has been found to suppress feeding behavior in healthy humans and some diagnostic subgroups under some conditions. However, it is still unclear as to why the effects of oxytocin on eating behavior are so varied and what factors explain the differences seen in different studies. It may be the case that oxytocin consistently reduces snacking for pleasure after individuals are already full, and this effect may account for the specific reductions in food consumption observed among healthy participants after the consumption of a meal and over the following 24-h period among participants with bulimia nervosa (a diagnostic group characterized by regular binge-eating episodes).

Research is currently underway to further clarify the mechanism of these effects and continues to explore the potential for oxytocin administration to enhance recovery from symptoms experienced across the feeding and eating disorder spectrum.

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