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Citation

LI, Norman P., SMITH, April R., YONG, Jose C., & BROWN, Tiffany A.. (2014). Intrasexual competition and other theories of eating restriction. In *Evolutionary Perspectives on Human Sexual Psychology and Behavior* (pp. 323-346). Berlin: Springer. Available at: https://ink.library.smu.edu.sg/soss_research/1472

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Intrasexual Competition and Other Theories of Eating Restriction

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Published in Evolutionary Perspectives on Human Sexual Psychology and Behavior, 2014, Springer, pp. 323-346.

https://doi.org/10.1007/978-1-4939-0314-6_17

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Abstract

Various forms of disordered eating and unhealthy eating practices, including excessive dieting, vomiting, binging and purging, and diet-motivated drug use, negatively affect and are potentially fatal to millions of individuals. We describe the etiology of disordered eating as well as various hypotheses on this phenomenon, both from traditional, non-evolutionary perspectives and from evolutionary perspectives. In particular, we explore in detail the intrasexual competition hypothesis, which draws on a broad evolutionary theory: intrasexual selection. From this perspective, women are thought to have evolved to compete intrasexually on thinness, which would have indicated youth and nubility in the ancestral past (Abed, 1998). In modern societies, however, an oversaturation of nubile-looking females, both real and virtual, may overstimulate this competitive mechanism, leading to unresolved body image dissatisfaction and eating restriction to the point of ill health. We discuss the theory, research, and implications of intrasexual competition and then provide a consideration of future directions for research on disordered eating.

Over 24 million people in the United States and as many as 70 million worldwide are afflicted with an eating disorder, and many more are undiagnosed (Renfrew Center Foundation for Eating Disorders, 2003). Indeed, 80 % of women report being dissatisfied with their body shape (Smolak, 1996), and half of teenage girls skip meals, vomit, or engage in other extreme weight control practices (Neumark-Sztainer, 2005). In this chapter, we describe the etiology and epidemiology of disordered eating and review both traditional, non-evolutionary perspectives and evolutionary perspectives, with particular attention given to the intrasexual competition hypothesis. We then close the chapter by considering unanswered questions and future directions for research on disordered eating.

Disordered Eating

Three major categories of eating disorders exist in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association, 2000): anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED). Individuals with a clinically significant disorder of eating that does not meet the criteria for AN, BN, or BED, are diagnosed with an other specified feeding or eating disorder (OSFED). Subsumed within OSFED are atypical AN, subthreshold BN, subthreshold BED, purging disorder (PD), and night eating syndrome (NES).

DSM-5 AN is primarily characterized by the restriction of food intake leading to significantly low body weight (i.e., less than minimally normal for age, sex, developmental trajectory, and physical health). Additional criteria for AN include an intense fear of gaining weight or becoming fat or persistent behavior that interferes with weight gain, and body image disturbance. AN also includes two subtypes, to denote the presence or absence of binge eating/purging during the current episode. The restricting subtype specifies presentations with weight loss occurring as a result of dieting, fasting, and/or excessive exercise. The binge eating/purging subtype includes individuals who have regularly engaged in binge eating or purging (self-induced vomiting, laxatives, diuretics) or both. AN affects disproportionately more women than men, with lifetime prevalence rates approximating 0.9 % in women and 0.3 % in men (Hudson, Hiripi, Pope, & Kessler, 2007).

BN is primarily characterized by recurrent episodes of binge eating and inappropriate compensatory behaviors to prevent weight gain. Binge eating episodes are typified by both eating an amount of food that is definitely larger than what most people would consume within a 2-h period and experiencing a sense of loss of control over the eating episode. Inappropriate compensatory behaviors can either include purging (self-induced vomiting, laxative use, or diuretics) or non-purging behaviors (fasting or excessive exercise), to influence weight or shape. To meet full criteria for BN, these binge eating and compensatory behaviors must occur, on average, once per week for 3 months. Additionally, BN is characterized by an undue influence of weight and shape on self-evaluation and cannot be diagnosed concurrently with AN. As with AN, BN affects a greater proportion of females, with lifetime prevalence estimates approximating 1.5 % of women and 0.5 % of men (Hudson et al., 2007).

BED is characterized by recurrent binge eating episodes in the absence of any recurrent compensatory behaviors. To meet criteria for BED, three of several cognitive and behavioral features must also be associated with the binge episodes, including: eating more rapidly, eating until uncomfortably full, eating when depressed, eating in the absence of hunger, eating alone due to embarrassment over food consumption, or feeling disgusted or guilty after eating. To meet full criteria for BED, binge eating episodes must occur on average at least once per week for three months. BED affects more females than males, with lifetime prevalence estimates at 3.5 % and 2.0 %, respectively; however, the gender ratio is far less skewed than in AN and BN (Hudson et al., 2007). As a residual category, OSFED includes any clinically significant disorder of eating that does not meet criteria for AN, BN, or BED, including subthreshold and atypical forms of AN, BN, and BED, along with alternative symptom configurations. These alternative symptom configurations include PD and NES. PD is characterized

by recurrent episodes of purging (vomiting, laxative, or diuretic use) to control weight or shape in the absence of binge eating episodes among normal weight individuals. NES is primarily characterized by recurrent episodes of night eating (either eating after awakening from sleep or by excessive food consumption after the evening meal). NES must be associated with clinical distress/impairment and cannot be better explained by another eating, mental, or medical disorder. Given that the DSM-5 has just recently been published, there is not enough information to determine the prevalence of OSFED. The lifetime prevalence estimate of DSM-IV eating disorder not otherwise specified (EDNOS) was approximately 4.62 %, which was greater than those observed for both AN and BN (Le Grange, Swanson, Crow, & Merikangas, 2012); however, given that the changes made to DSM-5 AN, BN, and BED were meant to reduce the preponderance of the residual EDNOS category, the estimates of OSFED are likely to be somewhat lower than those for EDNOS.

Regarding course and outcome, AN has typically been associated with a more chronic course and poorer prognosis compared to BN and EDNOS (Keel, Brown, Holland, & Bodell, 2012; Steinhausen, 2002). Indeed, AN is associated with lower remission rates compared to BN over the course of 10 or more years of follow-up (approximately 50 % for AN and 75 % for BN; Keel & Brown, 2010). Relapse affects a substantial minority of individuals who achieve remission, with one study finding that relapse occurred in 26.0 % of patients with AN compared to 17.7 % of patients with BN (Castellini et al., 2011). These remission rates support the relatively more chronic course of AN. Consistent with this, the longitudinal stability of AN is more common than diagnostic crossover; however, approximately 18 % of individuals initially diagnosed with AN crossover to a diagnosis of BN at some point (Keel et al., 2012). Further supporting the severity of the disorder, AN has been consistently associated with increased mortality (Herzog et al., 2000; Sullivan, 1995) and suicidality (Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). Indeed, risk for death by suicide among individuals with AN was found to be approximately 28-fold that of the general population (Preti et al., 2011).

Research has supported a slightly more favorable prognosis for BN as compared to AN. BN appears to have lower remission rates compared to bulimic-type EDNOS and BED at shorter-term follow-up (Agras, Crow, Mitchell, Halmi, & Bryson, 2009; Milos, Spindler, Schnyder, & Fairburn, 2005); however, rates between diagnoses appear more comparable at longer-term follow-up (Fichter & Quadflieg, 2007; Grilo et al., 2007; Keel, Gravener, Joiner, & Haedt, 2010). Similar to AN, BN is more likely to remain stable over time than to crossover to another eating disorder. Among those who do change diagnoses, the most common crossover patterns are from BN to AN (7 %) and from BN to BED (2 %; Keel et al., 2012). Notably, the crossover rate from BN to AN is lower than that of AN to BN, supporting the greater severity of AN. Evidence supports elevated mortality rates among individuals with BN (Franko & Keel, 2006; Nielsen, 2003); however, these rates have typically been lower than those observed in AN. Individuals with BN are also at increased risk for suicide (Crow et al., 2009; Preti et al., 2011; Smith et al., 2013), with an approximately 14-fold higher risk of suicide compared to that of the general population (Preti et al., 2011).

BED appears to have a more favorable course and outcome compared to both AN and BN. Remission rates are generally higher for individuals with BED (up to 82 %) and individuals with BED appear to achieve remission in a shorter amount of time than either those with AN or BN (Agras et al., 2009). Remission among 11.4 % of patients with BED (Castellini et al., 2011). Unlike the diagnostic stability observed across AN and BN, BED is actually more likely to crossover to BN than to remain stable (Fichter & Quadflieg, 2007; Keel et al., 2012). Also unlike AN and BN, there does not appear to be an increased risk of death by suicide in BED (Keel et al., 2012). Given the new designation of named syndromes (i.e. atypical AN, PD) and heterogeneity within the category of OSFED, relatively few studies have examined course and outcome in this group, which limits definitive conclusions regarding prognosis for specific types of OSFED. Studies examining DSM-IV EDNOS have supported more encouraging outcomes as compared to AN and BN in the short term (Keel & Brown, 2010). While higher remission rates for EDNOS compared to AN persist over longer-term follow-up,

differences between EDNOS and BN tend to diminish over time (Keel & Brown, 2010). Among those who do achieve remission, relapse rates are comparatively lower to those observed in AN, supporting a more favorable outcome across EDNOS diagnoses. Indeed, Castellini and colleagues (2011) found that among EDNOS diagnoses, relapse occurred in 4.4 % of those with subthreshold AN, 15.6 % of those with subthreshold BN, and 12.1 % of those with subthreshold BED. Although few studies have examined the stability of PD, one short-term study provides evidence for greater stability of diagnosis than crossover, with crossover to BN being relatively low (4 %; Keel, Haedt, & Edler, 2005). Studies have also provided evidence for an elevated rate of death among EDNOS, somewhat comparable to BN, but lower than that for AN (Button, Chadalavada, & Palmer, 2010; Crow et al., 2009). EDNOS also appears to have increased risk for suicide, similar to rates observed for BN (Crow et al., 2009).

Traditional (Non-evolutionary) Perspectives on Disordered Eating

Since the description of AN in 1873 by Sir William Gull, thousands of articles and books have been written on the possible causes of eating disorders (bulimia was not described until 1979; thus, earlier perspectives on eating disorder etiology focused on AN). Early models tended to highlight specific factors, like puberty or family, to be at the root of the development of eating disorders. However, most current researchers agree that the etiology of eating disorders is complex and multiply determined, and modern researchers discuss the development of disordered eating in the context of an integrated biopsychosocial model. Despite this, various etiological perspectives differ by the relative importance they accord for the role of factors such as family, peers, culture, emotion regulation, interpersonal strategies, cognitive processes, and biology. Below, we briefly review each of these perspectives.

The Onset of Puberty

One of the early influential writers on AN was Hilde Bruch, who focused on puberty as a pivotal trigger for the development of AN. Puberty is a time when young women's bodies go through a variety of changes, including increased fat accumulation. Further, puberty is also associated with greater challenges in terms of role expectations and peer relationships. A large proportion of eating disorders onset around puberty; thus, Bruch suggested that AN results in those adolescent girls who experience puberty as overwhelming. She speculated that these adolescent girls desired to revert to a prepubertal stage, one before the overwhelming demands of puberty were placed on them (1978). Similarly, writing some 20 years later, Crisp (1997) speculated that young women were motivated to engage in self-starvation as a way to revert to an earlier pubertal stage. In fact, Crisp hypothesized that women with AN had such a pronounced phobic avoidance of their adult body that they engaged in severe dietary restriction in order to avoid developing an adult physique.

Although these theories hold some intuitive appeal, they are largely untested. Further, they fail to account for various aspects of disordered eating. For instance, they do not address the development of disordered eating in boys and men, despite the fact that men account for approximately 10–25 % of individuals diagnosed with an eating disorder (Carlat, Camargo, & Herzog, 1997; Weltzin et al., 2005). Additionally, these explanations do not take into account the development of eating disorders in prepubertal or postpubertal women, though these two groups are noted to make up a proportion of eating disorder cases (e.g., Keel et al., 2010; WCEDCA, 2007). Moreover, these accounts fail to explain the development of eating disorders in individuals who do not express maturity fears.

Psychosomatic Families

Around the same time as Bruch, Minuchin and colleagues (1975, 1978) developed a theory of AN that laid the etiological blame on the family, coining the term "psychosomatic family" to describe families of girls with AN. According to Minuchin et al. (1978), these families were characterized by high conflict avoidance, high enmeshment, and great emphasis on bodily functions. Minuchin stated that

the child with AN used her illness as a way to gain control in the context of an overcontrolled family. According to Minuchin, "for the sick child, the experience of being able to protect the family by using the symptoms may be a major reinforcement for the illness" (p. 31). However, although family therapy is a successful form of treatment for adolescents with AN (e.g., Lock, 2011), the role of the psychosomatic family in the development of AN has not been empirically supported (e.g., Eisler, 2005). Thus, although families are important in the treatment process, current theories do not suggest that controlling families "cause" eating disorders.

Western Culture, Media, and the Thin Ideal

More recent researchers have speculated that eating disorders are a product of modern Western culture. Support for this comes from the fact that eating disorders are most common in Western societies, and there appears to be some evidence that the incidence of bulimia is increasing along with Westernization (Keel & Klump, 2003). More specifically, some researchers have suggested that the internalization of the Western beauty ideal, which has become thinner over the past 60 years (Seifert, 2005), is a major contributor to the development of eating disorders (e.g., Striegel-Moore, Silberstein, & Rodin, 1986; Sypeck, Gray, & Ahrens, 2004).

Thin-ideal internalization results when individuals internalize attitudes that are revered by sources such as peers, media, and family. It is hypothesized that failure to live up to the thin ideal creates body dissatisfaction (Thompson & Stice, 2001). Body dissatisfaction is a well-established risk factor for disordered eating (Stice & Shaw, 2002); thus, it is believed that thin-ideal internalization leads to body dissatisfaction, which in turn leads to disordered eating. Other researchers have emphasized the role of social comparison processes (Festinger, 1954) in the development of body dissatisfaction. According to the appearance comparison perspective, exposure to a host of idealized, thin images forces individuals to make upward comparisons between themselves and the idealized images; the result of these upward comparisons is believed to be the dissatisfaction in one's appearance (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999). These upward comparisons are thought to be particularly pernicious for individuals who internalize the thin ideal (Thompson, van den Berg, Roehrig, Guarda, & Heinberg, 2004).

There is a large body of research that supports positive relationships between media consumption, body dissatisfaction, and disordered eating (cf. Grabe, Ward, & Hyde, 2008). Additionally, studies have found cross-cultural support for the association between Western media and problematic eating behaviors. For example, Becker, Burwell, Herzog, Hamburg, and Gilman (2002) examined eating behaviors among adolescent Fijian girls before and after the arrival of Western TV. After 3 years of Western TV exposure, the authors reported self-induced vomiting went from being nonexistent in the population to being endorsed by 11.3 % of the population. Further, there was a reported 16.5 % increase in clinical levels of disordered eating attitudes (as measured by a score of 20 or above on the Eating Attitudes Tests; Becker et al., 2002).

Experimental studies have also supported this relationship. In a typical design, participants are randomly assigned to view either thin images or normal weight images; researchers have continuously found that participants report greater body dissatisfaction after being exposed to images exhibiting the thin ideal as compared to the participants who do not view this ideal (e.g., Birkeland, Thompson, & Herbozo, 2005; Dittmar & Howard, 2004). These findings are supported by a recent meta-analysis of experimental and correlational studies, which found small to moderate effects for the impact of thin-ideal media exposure on body image concerns among women (Grabe et al., 2008).

Although current evidence suggests that culture may play a role in the development of eating disorders, culture is clearly not the whole story, as not everyone who is exposed to and internalizes the thin ideal develops an eating disorder. In addition, although there is evidence that the incidence of bulimia has been increasing since its introduction into the DSM in 1983, the incidence of AN does not appear to be increasing (Keel & Klump, 2003). Further, there are numerous accounts of AN that predate the rise of thin ideal in Western culture, including saints like Catherine of Sienna, who engaged in severe dietary restriction and sometimes fasted to the point of death (Keel, 2005).

Peer Influence

Some researchers place more emphasis on the role of peer influence over societal influence in the development of eating disorders. Peer influence models build off of learning theory and suggest that peers inculcate certain behaviors (e.g., laxative use) and beliefs (e.g., the importance of a slim body) in other peers (Levine, Smolak, & Hayden, 1994). In a study involving friendship groups, Paxton, Schutz, Wertheim, and Muir (1999) found that cliques were similar with respect to their body image concerns, use of compensatory behaviors, and dietary restraint. Further, these authors found that a clique's use of compensatory behaviors accounted for unique variance in the prediction of an individual clique member's engagement in compensatory behaviors, over and above a host of well-known contributors to disordered eating, like BMI, depression, and self-esteem. As with sociocultural models, limitations of peer influence models include a lack of specificity. In other words, peer influence is so broad that if it was a necessary contributor to eating disorders, then we would expect much higher rates of eating disorders.

Interpersonal Formulation

Building off of a peer influence model, the interpersonal formulation model of eating disorders (Rieger et al., 2010) proposes that in response to negatively valenced social interactions, individuals may engage in disordered eating in an attempt to repair self-esteem and regain their sense of self. Supporting evidence for this model comes from multiple studies which have found that difficult interpersonal situations trigger binge-like behavior (e.g., Baumeister, DeWall, Ciarocco, & Twenge, 2005; Tanofsky-Kraff, Wilfley, & Spurrell, 2000). For instance, study participants who were told that they had been rejected by their peers ate a significantly larger amount of cookies as compared to non-rejected participants (Baumeister et al., 2005).

The interpersonal formulation model (Rieger et al., 2010) further stipulates maintenance factors. Engagement in disordered eating behaviors is believed to increase interpersonal problems; in turn, these interpersonal problems exacerbate eating disorder symptoms. Support for the escalation of disordered eating in response to interpersonal difficulties comes from a longitudinal study which examined the effect of negative feedback seeking on eating disorder-related variables (Joiner, 1999). Over the course of 5 weeks, this study found that among college-aged women, interest in negative feedback led to body dissatisfaction and, in turn, increased bulimic symptoms. Additionally, Rieger and colleagues (2010) hypothesize that indirect sources of evaluative information, such as social comparisons, lead to increased body dissatisfaction and disordered eating. In support of this claim, an experimental study found that female participants exposed to a thin confederate reported worse body dissatisfaction as compared to female participants exposed to a normal weight confederate (Krones, Stice, Batres, & Orjada, 2005).

Cognitive Biases

Leading cognitive theories of AN and BN hold that extreme overvaluation of shape and weight is central to the disorders (e.g., Fairburn, Shafran, & Cooper, 1998). Disordered cognitions and cognitive biases are believed to play a major role in the development and maintenance of eating

disorders (e.g., Cooper, 1997, 2005; Shafran, Lee, Cooper, Palmer, & Fairburn, 2007). Specifically, cognitive theories posit that people with eating disorders hold dysfunctional beliefs about their eating habits, shape, and weight. These core beliefs perpetuate negative automatic thoughts and attentional biases in the processing of information (e.g., attending only to information regarding one's body size). Thus, behaviors that reduce these negative thoughts, such as restricting food intake, are highly reinforcing and contribute to the chronicity of the disorder.

Recent work by Guardia and his colleagues (2012) suggests that individuals with eating disorders not only think they are bigger than they actually are, but they perceive themselves that way as well. In their study, individuals with AN exhibited marked distortions regarding the size of their bodies as compared to controls. Specifically, when asked to indicate whether or not they would be able to pass through a door opening that was definitely large enough for them to pass through, the participants with AN were more likely to indicate that they could not as compared to the controls. Further, this perceptual disturbance was found to be specific to their own bodies; individuals with AN could correctly judge whether someone else could or could not fit through a door opening.

Experiments that have used implicit tasks have found support for attentional biases to shape- and weight-related cues among individuals with disordered eating. For example, Ferraro, Andres, Stromberg, and Kristjanson (2003) found that individuals who were at risk for developing an eating disorder were faster at responding to fat-related words (e.g., heavy, plump, cellulite) than words unrelated to fat, whereas control subjects were faster at recognizing "nonfat" words than "fat" words. Additionally, Ahern, Bennett, and Hetherington (2008) found that participants who had positive implicit attitudes toward images of underweight women had higher drive-for-thinness scores on the Eating Disorder Inventory-2 and chose lower ideal body sizes than did participants who had more positive implicit attitudes toward normal weight models.

Emotion Regulation

Although cognitive processes figure prominently in many models of eating disorder development, emotion regulation is considered by some to be particularly important in the development of eating disorders that involve binge eating (i.e., BN, BED). The affect regulation model makes two primary predictions about the relationship between emotion and binge eating: (1) increases in negative affect trigger binge eating and (2) binge eating has a palliative effect and thus reduces negative affect (Hawkins & Clement, 1984). In their meta-analysis of 36 studies using ecological momentary assessment (EMA) methodologies, Haedt-Matt and Keel (2011) examined the validity of both of the hypotheses of the affect regulation model. Their findings indicated that negative affect preceded binge eating; however, the meta-analysis also found that negative affect increased following a binge, in opposition to the second hypothesis of the affect regulation model.

Escape

The escape model of binge eating (Heatherton & Baumeister, 1991) is related to the affect regulation model in that it gives affect a primary role; however, it holds that individuals engage in binge eating as a way to escape from negative emotional states (as opposed to an attempt at decreasing negative emotional states). This theory suggests that in the face of negative affect, individuals turn to binge eating as a way to narrow cognitive processes and reduce aversive self-awareness. Due to methodological considerations, it has been difficult to design studies that can measure affective states during a binge episode, thus making it difficult to garner concrete support for the escape model of binge eating. However, with the development of psychophysiological ambulatory monitoring, which allows for the measurement of psychophysiological correlates of emotion, such as heart rate variability, respiratory sinus arrhythmia, and skin conductance (Blascovich, Mendes, Vanman, &

Dickerson, 2011), it may be possible for future studies to use these types of methods to more accurately assess affective responses during a binge episode.

Biology

With the advent of other new technologies like the functional magnetic resonance imaging (fMRI) and methodologies like genome-wide association studies (GWAS), biological perspectives on the etiology of eating disorders have come to prominence. Beginning in the 1980s, family and twin studies have repeatedly shown evidence of familial aggregation of eating disorders (Bulik et al., 2006; Strober, Freeman, Lampert, Diamond, & Kaye, 2000). Using findings from twin studies, researchers have estimated the heritability of AN to be 33–84 % and bulimia to be 28–83 % (Zerwas & Bulik, 2011). Thus far there has been only one twin adoption study that has been published (Klump, Suisman, Burt, McGue, & Iacono, 2009). Participants for this study were 123 adopted and 56 biological female siblings. This study found that the majority of variance (59–82 %) in eating disorder symptoms was accounted for by genetic factors, while the remainder was accounted for by non-shared environmental factors. Interestingly, shared environmental factors, which include the family environment, did not account for a significant proportion of the variance.

Two primary approaches have been used in order to uncover potential candidate genes that may play a role in the development of eating disorders: association studies and GWAS. Thus far, findings from various association studies suggest that likely gene candidates are those that are involved in the serotonergic and dopaminergic systems and in weight regulation (Hebebrand & Remschmidt, 1995). Currently, only one GWAS study has been published; this study included over a thousand individuals with AN and close to 4000 control subjects (Wang et al., 2010). The authors reported several suggestive single nucleotide polymorphisms (SNPs), which are involved in the transmission and regulation of neurotransmitters; however, none were significant at the genome-wide threshold. The lack of significant findings is likely due to the small sample size for this type of study. Generally speaking, studies are only able to detect SNPs with at least five times as many ill participants as in the Wang et al. (2010) study (Kim, Zerwas, Trace, & Sullivan, 2011). Thus, the results of these types of studies are promising, but very preliminary, and ultimately, the field needs more studies with greater power in order to detect effects. Fortunately, a GWAS with a target sample size of 3000 subjects with AN is underway (Bulik, Collier, & Sullivan, 2011).

Recent work also suggests that hormones likely play an important role in the development of eating disorders (e.g., Klump et al., 2012; Quinton, Smith, & Joiner, 2011; Smith, Hawkeswood, & Joiner, 2010). Specifically, several studies have found that prenatal testosterone levels were higher among controls as compared to women with bulimic symptoms, and thus, prenatal testosterone may protect against bulimic disorders through its organizational effects on the brain (Culbert, Breedlove, Burt, & Klump, 2008; Klump et al., 2006; Smith et al., 2010). Further, Klump and colleagues (2012) have posited that ovarian hormones released at puberty play a role in the development of disordered eating and help explain both the sex difference in eating disorders and the timing of onset, which is often during puberty. Specifically, they speculate that puberty and the attendant effects of ovarian hormones may activate genetic risk in girls (Klump et al., 2012). These findings mesh with the observations of earlier writers, such as Bruch and Crisp, who suggested that puberty is a trigger for eating disorders; however, these findings suggest a more central role for biological factors that onset at puberty as opposed to environmental factors, though likely both are important.

Further, there appear to be important neurobiological weaknesses in individuals with eating disorders. For instance, individuals with AN have been noted to have altered reward processing, poor setshifting, and loose central coherence (e.g., Lopez, Tchanturia, Stahl, & Treasure, 2009; Roberts, Tchanturia, & Treasure, 2010). In a recent study by Danner et al. (2012), three groups of participants (women with current AN, recovered women, and healthy control women) completed a battery of neuropsychological instruments (e.g., Berg's Card Sorting Task, Rey-Osterrieth Complex Figure Test, Iowa Gambling Task). This study found that both ill and recovered women with AN demonstrated set-shifting problems; further, individuals with impaired set-shifting also displayed central coherence weaknesses. These findings suggest that a rigid and inflexible thinking style may be associated with the development and maintenance of AN. However, currently there is not strong evidence that these weaknesses play a role in the development of eating disorders due to a lack of prospective studies examining potential neuropsychological impairments in eating disorders. Thus, it is unclear if these impairments predate the onset of the disorder or are a consequence of the disorder.

The above summary of leading etiological perspectives is far from exhaustive; due to space limitations, we did not discuss more general factors, such as depression and low self-esteem, or highly intrapersonal factors, like personality or the experience of traumatic events, like sexual abuse, though all of these other factors have been found to be associated with disordered eating.

All in all, there is a multitude of psychological perspectives through which eating disorders have been examined. Together, the extensive research suggests that many proximate causes may be involved in the onset and maintenance of disordered eating. In addition, biological research, including the results of various behavioral genetics studies, indicates a significant genetic component. Looking for broader, more ultimate explanations, some theorists have proposed how eating restriction and disordered eating might reflect underlying psychological mechanisms that have evolved to provide adaptive benefits. We now examine each of these evolutionary perspectives.

Evolutionary Perspectives on Disordered Eating

Evolutionary psychology provides ultimate explanations for various human thoughts, feelings, and behaviors (Buss, 1995). Although starving oneself can be quite detrimental to one's health or even fatal, there are reasons to believe that adaptive mechanisms may underlie eating restriction and the prevalence of disordered eating. In this part, we consider various evolutionary hypotheses that propose how negative eating attitudes and practices may represent adaptations.

Biological Functions of Fat

In order to understand how an evolutionary perspective can account for eating disorders in women, consideration should be given to the biological functions of adipose tissue (i.e., fat) in mammals and, more specifically, mammalian females. Adipose tissue has been viewed as having two main survival functions for mammals. First, it primarily serves as storage for calories through a reserve of lipids, which are metabolized to meet the energy needs of the body (Cahill, 1982; Norgan, 1997; Pond, 1978). Second, adipose tissue may have evolved as an adaptation for thermal insulation, accumulating in subcutaneous tissue and providing protection from heat and cold (Gesta, Tseng, & Kahn, 2007), although this latter point has been more controversial (cf. Pond, 1998). (For a more thorough review of the biological functions of human adipose tissue, see Wells, 2012.)

In addition to these functions, fat has also been implicated for female mammals in the onset and maintenance of ovulation (Frisch, 1990), working through the organism's endocrine function (Fishman et al., 1975; Frisch et al., 1981; Nimrod & Ryan, 1975). Furthermore, fat is important as a source of calories for the success of pregnancy and lactation (Brown & Konner, 1987). Sex differences in the distribution and abundance of adipose tissue in humans (e.g., Enzi et al., 1986) additionally indicate that natural selection has played a critical role in shaping the anatomy and development of fat, lending further credence to the view that fat serves important survival and reproductive functions. Thus, if attitudes toward fatness in women, perceived either in others or

oneself, have any evolutionary importance, that importance ultimately depends on some biological function of adipose tissue. Evolutionary hypotheses about standards of physical appearance and beauty are hence, at some level, attempts to explain how observed patterns in attitudes toward female fatness and in eating behavior could have, at least in ancestral conditions, improved the fitness of individuals relative to other possible patterns.

Reproductive Suppression Hypothesis

One of the most prominent evolutionary theories for eating disorders is the reproductive suppression hypothesis (Condit, 1990; Salmon, Crawford, Dane, & Zuberbier, 2008; Surbey, 1987; Voland & Voland, 1989). This hypothesis suggests that natural selection may have shaped a mechanism in women that alters their proportion of body fat in order to adjust their reproduction in accord with socioecological conditions. This hypothesis was borne out of two well-known biological concepts that were derived from the observation that fat affects the onset and maintenance of ovulation.

The first concept, adaptive reproductive suppression, argues that as reproduction is a highly risky and energetically demanding endeavor for female mammals (Williams, 1966), a female may increase her lifetime reproductive success by timing her reproductive attempts to occur during desirable conditions and, correspondingly, curtailing her reproductive activity at other less favorable times. Natural selection may therefore select for individuals who undergo reproductive suppression under suboptimal reproductive conditions (Wasser & Barash, 1983).

The second concept, known as the critical fat hypothesis (Frisch, 1985, 1990), points out that there is a positive relationship between body fat and the likelihood of ovulation and menstruation. On average, adipose tissue must make up at least 22 % of a woman's body weight to maintain ovulation. Because a female's body fat contains considerable quantities of estrogen and converts androgens to estrogen, changes in the rate of weight gain among adolescent girls or in the weight of lean adult women can influence whether ovulation occurs (Rippon, Nash, Myburgh, & Noakes, 1988). Indeed, for female athletes who are bordering on this threshold, menstruation can be activated or deactivated by the gain or loss of only a few pounds (Frisch et al., 1981). As such, in response to cues relating to reproductive conditions, weight control could have been an effective mechanism for ancestral females to adjust reproductive effort (Becker, Breedlove, & Crews, 1993; Frisch, 1990). Such socioecological cues might include stressful sexual attention from undesirable males and elevated levels of social competition between females.

In modern urban cultures, socioecological cues, which would have signaled the need for temporary postponement of reproduction in ancestral environments, may now be experienced at unprecedented levels of intensity and duration. For some women, the heightened and prolonged body image fears and anti-fat attitudes that abound in today's society may result in reproductive suppression mechanisms being engaged continuously from preadolescence to adulthood and, thus, in the onset and maintenance of disordered eating attitudes and behaviors during that time (Salmon et al., 2008).

Although quite plausible, the reproductive suppression hypothesis nevertheless has some limitations. For instance, it does not directly explain the function of distorted body image; why some more direct and less costly means to stop menstruation did not evolve; why women in modern urban cultures, who are economically well off and have easy access to food, would face poor reproductive prospects; and why men are afflicted. Importantly, as noted earlier, amenorrhea is no longer considered to be a useful indictor of AN and has been deleted as a criterion for AN in DSM-5 (American Psychiatric Association, 2000).

Model of Parental Manipulation

Voland and Voland's (1989) model of parental manipulation provides an interesting account for why eating disorders tend to occur more among wealthier, higher class individuals. This model draws on kin selection theory (Hamilton, 1964) and asserts that AN may be adaptive insofar as it increases the helping behavior of an individual with AN toward her own kin and aids their survival and reproduction while suppressing her own reproductive activity. Such a kin selection-based "helper at the nest" mechanism would have been particularly beneficial in large family units, which were prevalent in human history until recently.

The parental manipulation model suggests that anorexia is instigated by parental dominance influential parents who are highly involved in the control of resources, offspring livelihood, and family outcomes. Particularly for wealthy large families, males may have relatively better reproductive potential than females, as an abundance of resources contributes to male mate value more than female mate value (Trivers, 1972; Trivers & Willard, 1973). Thus, if affluent parents favor and bestow their resources onto sons and spur the restriction of reproduction of one or a few daughters (via induced anorexia), better inclusive fitness outcomes may be achieved, as those daughters can divert resources that would otherwise have gone to their own offspring toward a male kin.

Consistent with this model, correlational studies have shown that anorexic individuals tend to worry constantly about the well-being of their families, and members of anorexic families possess mutually overprotective attitudes (Minuchin et al., 1975). Specifically, the likelihood of AN development is significantly correlated with having dominant and overprotective mothers (Steiger, Bruce, & Israël, 2003) as well as overly controlling parents (Bruch, 1988). When a daughter is overprotected and dominated by her mother, her ability to find a mate may be reduced, further decreasing her likelihood of producing offspring while increasing the relative benefits of helping her male kin. The parental manipulation model, however, does not account for desires for thinness or anorexic behaviors that derive from sources external to the family.

Adapted-to-Flee-Famine Hypothesis

The curious case of anorexic symptoms leading to a decline in individual fitness by decreasing the carrier's fertility and increasing the risk of death by starvation, while at the same time increasing the carrier's hyperactivity levels, led Guisinger (2003) to propose that anorexia nervosa could exist in its modern form because humans are adapted to flee famine. Like other primates and most mammals, humans cannot store many extra calories as fat; yet, they consistently faced periodic waves of famine. As such, in nomadic tribes, anorexia nervosa might have been a way to help humans overcome food shortages during periods of famine or while traversing vast distances, by facilitating the migration from depleted environments to greener pastures.

Findings from nonhuman populations provide preliminary evidence supporting this view. Foodrestricted rats with access to a running wheel and lean-bred pigs with wasting pig syndrome have been found to reject food (Epling & Pierce, 1988; Treasure & Owen, 1997). More generally, Mrosovsky and Sherry (1980) documented the cessation of eating and weight loss of a number of nonhuman species during their seasonal migrations. Animals also increase activity in times of food shortage. When starved in the laboratory, a number of mammal species ignore their food and exercise excessively (Epling & Pierce, 1992). Lastly, when individuals starve, neurochemical signals of hunger normally are raised, and signals for satiety and activity are typically lowered. In anorexics, however, neuromodulators and hormones regulating appetite and activity have been found to go against this usual trend and appear instead to facilitate movement and activity (e.g., Leibowitz, 1992; Prentice et al., 1992). Taken together, these findings are consistent with the possibility that there are adaptations to deactivate desires for eating and activate traveling and suggest that AN as an adaptive mechanism might have benefited our ancestors who were faced with food shortages to overcome the pain of hunger and energize them to migrate to more food-abundant locations.

Although the evidence is encouraging, the adapted-to-flee-famine hypothesis does have its own set of limitations. It does not address why individuals with AN resist eating food when food is readily available and why AN is more prevalent in women than men. Furthermore, some studies have shown that not all individuals with AN are hyperactive throughout the entire phase of anorexia.

Restricted Eating as Response to Threat

Gatward (2007) proposed that response to threat may be a reason why individuals get trapped in an anorexic cycle. Because humans are social animals whose survival depends on group inclusion, there will inevitably be competition for status within the group (Baumeister & Leary, 1995). Within-group competition leads to the threat of being expelled from the safety of one's social group, which, in the ancestral past fraught with myriad dangers, would have likely meant certain death. Demonstrating status is thus of paramount importance, as status is an indicator of one's worth to remain in the group. Until recently, fatness was a sign of good resources, as only the wealthy could afford to be overweight. In many cultures today food is relatively abundant and cheap, and resistance to food has become a modern sign of status and self-control (Stevens & Price, 2000). Eating restriction could thus have emerged in order to signal high status as a response to the threat of social exclusion.

Gatward borrows from the adapted-to-flee-famine hypothesis and the adaptive suppression hypothesis to explain the onset and maintenance of AN. Perceived threat of exclusion may activate dietary restriction, which in turn may trigger the adaptive response of decreased appetite and increased hyperactivity to a newly perceived threat of famine (caused by the dietary restriction), and thus cause the individual to experience the symptoms of AN. As these multiple threats of social exclusion and famine, real or imagined, suggest undesirable socioecological conditions, females are also likely to undergo suppressed reproductive behavior. Because severe weight loss removes people from competition for status, subsequent weight gain could also be felt as threatening because it could signify reentering within-group competition and risking attack by others and further exclusion. These multiple perceived sources of threat thus maintain restricted eating behaviors in individuals, particularly females. A limitation of this model is that it is difficult to evaluate; furthermore, it does not effectively rule out alternative hypotheses.

Perceived Vulnerability to Disease and Anti-fat Attitudes

Another perspective argues that eating disorders arise out of the association between fat and character undesirability. Specifically, when being fat implies negative traits such as laziness, irresponsibility, lack of self-control, and other qualities pertaining to character and lifestyle flaws (e.g., Björvell, Edman, Rössner, & Schalling, 1985; Fassino et al., 2002), anti-fat attitudes emerge which in turn may result in restricted eating in order to avoid gaining weight and, as a consequence, being associated with those aversive, negative traits.

While such a perspective has tended to reside within the grounds of proximate, social, and nonevolutionary factors, Park, Schaller, and Crandall (2007) explored the possibility that humans have evolved to view obesity as a heuristic cue connoting pathogen transmission. As signal detection of pathogens is often imperfect, humans may have evolved to associate a wide range of superficial cues, such as facial birthmarks and physical disabilities, with pathogens. Humans' behavioral immune system, in the form of aversion, can be triggered by the perception of substantial morphological deviations; thus, perceived obesity may trigger a behavioral immune system of aversion because gross obesity represents one such deviation from species-typical morphological norms. Relatedly, overweight people are commonly stereotyped as unattractive, unclean, and unhealthy, and images of overweight people arouse visceral emotions such as disgust (Harvey, Troop, Treasure, & Murphy, 2002). Antipathy toward overweight people could thus be more fundamentally the result of a pathogen-avoidance mechanism.

Pathogen-avoidance mechanisms typically involve hypervigilance and risk aversion, and avoidant responses to individuals marked by disease-connoting cues are particularly strong when perceivers feel especially vulnerable to disease transmission (Schaller, Park, & Faulkner, 2003). These features allow for an adaptive rejection of individuals afflicted with actual contagious diseases.

Consistent with their hypotheses, the authors found that perceived vulnerability to disease significantly predicted antipathy toward overweight people independently from other variables, such as self-determination ideologies (Schaller et al., 2003). In addition, participants who were primed with images depicting contagious diseases and disease-causing agents were more likely to associate overweight people (as opposed to thin people) with disease using the implicit association test compared to when they were exposed to either accident primes (which eliminate the possibility of negatively valenced primes as a factor), or primes pertaining to work ethics (which eliminate the likelihood that self-determination attitudes accounted for the pattern of results).

The perceived vulnerability to disease and anti-fat attitudes model focuses on antipathy toward overweight individuals other than oneself. Nevertheless, evolved disease- and pathogen-avoidance mechanisms may play a role in shaping attitudes toward one's own weight status and, thus, in the development of restricted eating. This may be especially true for individuals who perceive a lack of social standing in their groups.

In the next part, we consider an additional evolutionary explanation that is perhaps most consistent with all the other theories and is also guided by a fundamental evolutionary theory: intrasexual selection (Darwin, 1871).

Intrasexual Selection Hypothesis

Intrasexual selection involves members of one sex competing among themselves, usually for access to mates or resources. Heritable behaviors or features that provide an advantage in this competition tend to be selected and passed down over the generations. Intrasexual selection is traditionally associated with male–male competition, as in massive male elephant seals battling viciously for a large territory on a beach before female seals arrive for the mating season (e.g., Gould & Gould, 1989). However, recent evidence suggests that females of many species also engage in various forms of intense intrasexual competition (e.g., Clutton-Brock, 2007; Rosvall, 2011).

In humans, intrasexual competition tactics employed by one sex tend to reflect the mate preferences of the other sex (Buss, 1988; Walters & Crawford, 1994). For instance, women's fertility tends to peak at a relatively early age and decreases rapidly after 30. Thus, when considering potential mates, men may have evolved to especially value appearance-related cues that indicate sexual maturity and youth. Such preferences allowed ancestral men to choose mates with greater fertility and reproductive value and, thus, to outreproduce men who did not have such preferences. In contrast to women's fertility, men's fertility declines significantly slower over the lifespan; thus, there may have been less selective pressure for women to strongly prefer similar cues in their mates. However, because ancestral men varied in their ability to provide key resources essential for offspring survival and eventual reproduction (e.g., Geary, 2009), women may have evolved to prefer men with status and resources (Symons, 1979).

Indeed, numerous studies have found that men value physical attractiveness in their mates more than women do, and women value status and resources more than men do (e.g., Buss, 1989; Li, Bailey, Kenrick, & Linsenmeier, 2002; Li, Valentine, & Patel, 2011; Shackelford, Schmitt, & Buss, 2005). These sex-specific preferences appear to be ingrained in people's self-concepts: when considering themselves as potential long-term mates, men prioritize having status and resources, whereas women prioritize having physical attractiveness (Li, 2007). In line with these differences, women, more than men, express greater usage of intrasexual competition tactics related to physical appearance, including dieting to improve one's figure. Such tactics are also judged to be more effective for female versus male intrasexual competition (Buss, 1988). Furthermore, men are more distressed when a rival surpasses them on financial prospects, job prospects, and physical strength, whereas women are more distressed when a rival surpasses them on facial and bodily attractiveness (Buss, Shackelford, Choe, Buunk, & Dijkstra, 2000).

The intrasexual competition model. Drawing on principles of sexual selection, Abed (1998) proposed an intrasexual competition hypothesis for disordered eating, laying out the following logic. In the ancestral past, the hourglass shape [i.e., waist-to-hip ratio (WHR), Singh, 1993] was a reliable indicator of a female reproductive condition and capacity. As such, men evolved to strongly prefer females with low WHRs as mates because such females had high reproductive capacity and were also not currently pregnant or lactating. As women age, they not only lose the hourglass shape but also tend to gain body mass. This is especially the case when women's bodies are subjected to cycles of pregnancy, childbirth, and nursing, as they would have been from a relatively early age in the ancestral past. Thus, in addition to the hourglass shape, bodily thinness may also have been reliably associated with nubility—a state of fertility with no reproductive history. Whereas WHRs are largely influenced by estrogen levels (Cashdan, 2008; Singh, 1993), thinness may not only be influenced by hormones but may also be controllable through dieting. As such, women may have evolved to be sensitive to perceptions of premature obesity and to strive for being as thin as or thinner than other young nulipara. Such an adaptation may have given ancestral women a competitive edge in attracting long-term mates.

In modern, industrial environments, however, several factors may lead to a destabilization of longterm relationships and, consequently, to a "runaway" intrasexual competition process in which an eating restriction strategy is triggered and maintained to the point of ill health or fatality. With the formation of modern, industrial societies, the size and unity of extended families—which tended to be patrilocally organized (centered around men's genetic relatives) in the ancestral past (e.g., Wrangham, 1999)—greatly decreased. Accordingly, the role of kin as well as the power of men in influencing mating markets and women's sexual behavior greatly diminished, leaving women to promote themselves in the mating markets and with greater autonomy in selecting mates. According to Abed (1998), such forces likely decreased men's paternity confidence and, thus, their paternal investment, which in turn led to a destabilization of marriage and, at the same time, an increase among women in pursuing short-term relationships. Thus, in modern societies, there are more individuals available for a longer time on the mating market and more women competing with each other for mates for many more years than in the ancestral past.

Additionally, children in modern societies are no longer needed to tend to family farms or trades and instead, represent net economic losses. Accordingly, reproduction in modern societies—in particular, urban environments—is curtailed and delayed, and the birth spacing interval is greater, thereby allowing women to retain a nubile shape for much longer than would be the case in an ancestral or traditional society. Moreover, the population density in modern environments can reach several millions per city—an extremely large-scale increase over the lightly populated ancestral village where 150 might be a maximum total population (Dunbar, 1992). Thus, in modern environments, women are

exposed to an unnaturally high number of potential competitors who may appear nubile and trigger intrasexual competition on thinness (Salmon et al., 2008).

Furthermore, in modern environments, the person's perceived intrasexual competitors are not limited to actual competitors. Various lines of research have demonstrated that people (their evolved mechanisms) cannot distinguish between real individuals encountered in the flesh and those seen on television, magazines, Internet, and other forms of media (e.g., Kanazawa, 2002). For example, many people evaluate their social lives more positively after having watched television (Kanazawa, 2002). After being exposed to pictures of physically attractive women, women evaluate themselves more negatively as potential mates and men express reduced commitment to their long-term romantic partners (Kenrick, Neuberg, Zierk, & Krones, 1994). The multibillion dollar pornography industry attests to the ability of psychological mechanisms to be triggered by two-dimensional images (Kenrick, Gutierres, & Goldberg, 1989). These days, people consume electronic media and can expose themselves to more individuals in 1 day than our ancestors encountered in a lifetime. The effects of such exposure are now coming into light. For instance, a recent study indicated that many healthy men in their 20s who regularly consume Internet pornography cannot maintain erections with their actual partners, who likely compare unfavorably to images of naked women on the Internet (Italian men suffer 'sexual anorexia' after Internet porn use, 2011).

All in all, individuals, including women, face an inordinate number of real and virtual same-sex competitors in the modern world. One result of all this competition is that for some women, intrasexual competition mechanisms via dietary control may be excessively triggered, thereby leading to unhealthy dieting practices and, in some instances, disordered eating. We note here that although in this part we refer to disordered eating in general, Abed (1998) makes a distinction between the two main forms. That is, AN is viewed as a manifestation of intrasexual competition with a relatively early onset in which standards for thinness are set extremely low, whereas BN involves a reactivation of intrasexual competition mechanisms for thinness with a somewhat later onset.

Compatibility with other perspectives and research. The intrasexual competition model is compatible with many of the findings on disordered eating reviewed earlier. For instance, various researchers have noted of the high incidence of eating disorders in adolescent females (e.g., Bruch, 1978; Crisp, 1997). Indeed, although both young girls and older women can evidence disordered eating, the average age of onset for anorexia nervosa is 17, and rates tend to drop off after 25 (Substance Abuse and Mental Health Services Administration, 2003). As mentioned earlier, Bruch (1978) suggested that AN might be especially likely to occur in female adolescents who experience puberty as overwhelming. From an intrasexual competition perspective, puberty may be particularly overwhelming and a time when eating disorders are likely to occur because this is precisely when competition for mates has not only set in but is especially intense. Similarly, attempts to repair self-esteem as proposed by the interpersonal formulation model (Rieger et al., 2010) may center around efforts to regain mate value or social status, which, for women, tends to revolve around physical attractiveness and youth. Consistent with this line of reasoning, one study found that women with anorexia were less likely to be married than aged-matched controls (45 % vs. 16 %; Sullivan, Bulik, Fear, & Pickering, 1998).

From the intrasexual competition perspective, the prevalent thin ideal (e.g., Thompson & Stice, 2001) that is thought to be a product of Western culture may represent women's mental composites (Symons, 1979) of the youngest and most fertile-looking potential competitors for mates. Although adaptive for setting comparison standards in a small village setting, this composite in the modern world includes an unnaturally high number of thin, nubile-looking competitors, both real and virtual, thereby leading to perpetual shortcomings between one's self-evaluation and one's ideal, desired

state. As described above, such gaps are associated with body dissatisfaction and attempts to eliminate the gaps via various forms of unhealthy caloric restriction and weight reduction practices.

Similarly, the intrasexual competition perspective also offers insights into the numerous studies showing links between women's exposure to thin images and body dissatisfaction, unhealthy eating attitudes, and various eating disorders. That is, the ultimate reason why people—in particular, young women—are susceptible to comparisons of thinness in the first place is because of psychological mechanisms that evolved to promote successful intrasexual competition for mates. Such mechanisms may also be responsible for various cognitive distortions and biases relating to one's own size and shape (e.g., Cooper, 1997), which, alongside body dissatisfaction, serve to motivate eating restriction and weight loss.

From an intrasexual competition perspective, women's interest in consuming fashion-based media and the featured thin models may be stemming from innate, adaptive mechanisms to socially compare and learn from same-sex individuals of higher mate value. Along these lines, one study examined the eye movements of women presented with various target faces. Women with relatively high bulimotypic symptomatology tended to fixate on physically attractive female faces versus average female faces or male faces (Maner et al., 2006).

Empirical investigations of the intrasexual competition model. Various studies have provided direct support for the intrasexual competition hypothesis. For instance, a correlational study found, through structural equation modeling, that female intrasexual competitiveness for mates was the underlying factor behind competition for status, perfectionism, body dissatisfaction, drive for thinness, and both BN and AN (Faer, Hendriks, Abed, & Figueredo, 2005).

Another study examined the relationships between life history strategy, intrasexual competition, and eating disorders (Abed et al., 2012). Life history strategy was developed by evolutionary biologists to explain how organisms (including humans) adaptively allocate energy, time, and resources across their lifetime toward different activities (e.g., Charnov, 1993; Daan & Tinbergen, 1997). Whereas a slow life history strategy is associated with greater somatic effort (development of body, mind, skills, etc.) and parental investment, a fast life history strategy is associated with greater mating and reproductive effort. A structural equation model indicated that intrasexual competitiveness was related to disordered eating behaviors; moreover, a slow life history strategy had negative effects on disordered eating behavior both directly and through its negative effect on intrasexual competitiveness. A subsequent study found evidence that the protective effects of a slow life history strategy may be due to its association with greater behavioral regulation, which is negatively associated with intrasexual competitiveness and disordered eating behaviors (Salmon, Figueredo, & Woodburn, 2009).

Strong evidence for the intrasexual competition model also comes from two recent investigations using an experimental paradigm. First, Li, Smith, Griskevicius, Cason, and Bryan (2010) conducted two studies in which participants were exposed to a series of ten personal profiles allegedly written by same-sex target individuals, describing their interests, school and community activities, and job. The profiles conveyed either a competitive and status-seeking orientation (e.g., playing to win, taking leadership positions, aiming for success) or a noncompetitive and non-status-seeking orientation (e.g., playing for fun, joining but not leading organizations, and being content to get by). Each profile was accompanied by a facial photograph of an individual of average physical attractiveness and normal weight.

In the first study, people saw these profiles and then completed the Eating Attitudes Test (Garner & Garfinkel, 1979), which measures thoughts and feelings related to disordered eating, including restriction (e.g., "I avoid eating when hungry"), purging (e.g., "I have the impulse to vomit after meals"), and a strong desire for thinness (e.g., "I am preoccupied with a desire to be thinner"). Women who were exposed to the competitive target profiles indicated having significantly more negative eating attitudes (some were at clinical levels) than women exposed to the noncompetitive profiles. No effects were found for men, who tended to have low negative eating attitudes in both conditions. Thus, this study indicated that even in the absence of attractiveness and thinness-related cues, instrasexual status competition motives are capable of triggering negative/restrictive eating attitudes.

A unique strength of the intrasexual competition hypothesis is that it addresses eating disorders in men. Although eating disorders predominantly occur in women, they do affect some men, and they are disproportionately represented in gay men (e.g., Herzog, Norman, Gordon, & Pepose, 1984) but not lesbian women (Striegel-Moore, Tucker, & Hsu, 1990). To examine this phenomenon, in their second study, Li et al. (2010) also investigated the effects of sexual orientation in their second study. Similar to heterosexual men, gay men also place great value on youth and physical attractiveness in their mates (Bailey, Gaulin, Agyei, & Gladue, 1994; Kenrick, Keefe, Bryan, Barr, & Brown, 1995). Thus, like heterosexual women, gay men compete intrasexually on appearance and may develop similar issues of body image and eating restriction due to such competition. Indeed, heterosexual women, but not heterosexual men, responded to the competitive target profiles by reporting more negative eating attitudes and worse body image. On the other hand, gay men, but not lesbian women, reported more negative attitudes and worse body image after viewing the competitive profiles. This specific pattern of results reflected the differing values that individuals' mates place on physical attractiveness according to their sex and sexual orientation and lent further support to the intrasexual competition model.

If intrasexual competition underlies the prevalence of eating disorders in modern societies, then competitive individuals who are especially oriented toward the attainment of social status would be expected to be particularly at risk. To investigate this possibility, Smith, Li, and Joiner (2011) measured women's status aspiration with the status aspiration subscale of the Achievement Motivation Scale (Cassidy & Lynn, 1989; e.g., "I would like an important job where people look up to me"). Participants were then exposed to a series of ten same-sex individuals who were either thin or heavy in their photographs and came across as either successful or unsuccessful in their alleged self-descriptions. After being exposed to thin, successful targets, women who were high on status aspiration reported significantly worse body satisfaction and greater ineffectiveness (i.e., a lack of control over their lives) than women who were low on status aspiration. Together with perfectionism, body dissatisfaction and ineffectiveness have been implicated in the development of bulimic symptoms (Bardone-Cone, Abramson, Vohs, Heatherton, & Joiner, 2006) and the maintenance and exacerbation of bulimic symptoms (Joiner, Heatherton, Rudd, & Schmidt, 1997). Thus, together with the other findings, these results suggest that intrasexual competitiveness is a significant factor that underlies the development of modern-day eating disorders.

In summary, an intrasexual competition model provides a promising evolutionary account for the development of disordered eating behaviors. It addresses several of the shortcomings of the other evolutionary hypotheses and is largely consistent with the demographic profile of affected individuals and much of the other research on eating disorders, including the well-established links between media consumption and women's body image and eating attitudes. The model has also been supported by various investigations that have both correlationally and experimentally demonstrated the link between intrasexual competition and body dissatisfaction and disordered eating attitudes. Furthermore, because intrasexual competition is a key evolutionary process for both sexes, the intrasexual competition model addresses why eating disorders occur in some men. Finally, the model

is consistent with separate lines of research indicating that various modern-day ills may be due to a mismatch between current living conditions and the ancestral environment in which human psychological mechanisms evolved to function (e.g., Buss, 2000; Kennair, 2002; Nesse & Williams, 1995).

Future Directions and Treatment Implications

As noted in this chapter, numerous theories, including those that embrace an evolutionary perspective, have been offered as explanations for the prevalence of eating disorders. Each of the available theories has various limitations and/or is unable to explain certain aspects of eating disorders. The intrasexual competition model appears to offer a comprehensive account of eating disorder etiology and maintenance; however, more empirical investigations of the intrasexual competition model are needed to substantiate this theoretical framework and to further demonstrate its precision and predictive power compared to existing frameworks.

It will also be important for future work on the intrasexual competition model to incorporate biological factors. Given recent findings, which have found associations with disordered eating and hormonal factors both prenatally (e.g., Quinton, Smith, & Joiner, 2011; Smith et al., 2010) and at puberty (Klump et al., 2012), it will be informative for future work to investigate the ovulatory cycle. Because women are more likely to accentuate their physical appearance during ovulation (Durante, Li, & Haselton, 2008; Haselton, Mortezaie, Pillsworth, Bleske-Recheck, & Frederick, 2007), it may be that women are especially likely to indicate restrictive eating attitudes in response to cues of status competition around the time of ovulation.

It will also be important for future work to continue to investigate potential moderators, like achievement motivation, and situational factors, like power, that may activate intrasexual competition motives. For instance, as women continue to enter the global economy in record numbers (Aguirre, Sabbagh, Rupp, & Hoteit, 2012), power may be a particularly salient factor to examine with respect to disordered eating, as power has been found to activate goal pursuits (e.g., Galinsky, Gruenfeld, & Magee, 2003; Kunstman & Maner, 2011). Thus, success in the workforce could have potentially negative downstream consequences among women whose goals include dieting and weight loss.

The current review also provides suggestions for eating disorder treatment and prevention efforts. Given that the abundance of idealized media images is believed to over-activate intrasexual competition motives, and given that individuals are likely to continue to be increasingly bombarded by such images, interventions, such as media literacy programs, may be particularly indicated for the prevention of eating disorders. These programs are designed to teach participants to be informed consumers of media and typically include psychoeducational components and the viewing of presentations on the treatment of media images (e.g., utilizing techniques such as air brushing and Photoshopping to make images look more "perfect"). Media literacy programs are based on inoculation theory; specifically, they operate under the assumption that by providing participants with facts about advertising and media images, participants will be less susceptible to thin ideal internalization and the pressure to be thin (Wilksch, Durbridge, & Wade, 2008). Further, it is hypothesized that reducing susceptibility to internalization of the thin ideal will improve body image and decrease behaviors that are associated with the development of eating disorders, such as dieting (Yager & O'Dea, 2008).

Recent studies provide support for the effectiveness of media literacy programs in reducing eating disorder-related cognitions. For instance, Watson and Vaughn (2006) found that the female college students who took part in a 4-week intervention group, which consisted of watching a movie about the

realities of female images in the media and participating in exercises and discussions to increase media literacy, evidenced less awareness of the thin ideal and greater body satisfaction as compared to the control group. Further, the effects of media literacy programs have been found to persist long term. Specifically, Wilksch and Wade (2009) found that after participating in an eight-session media literacy program, adolescent girls had significantly lower shape and weight concerns, dieting concerns, body dissatisfaction, feelings of ineffectiveness, and depression at post-intervention and at a 30-month follow-up.

Dissonance-based approaches, which are based on Festinger's cognitive dissonance theory (1957), ask participants to take a counter-attitudinal stance against the thin ideal and have also been found to be highly effective at reducing eating disorder-related cognitions and behaviors (Becker, Smith, & Ciao, 2006; Stice, Chase, Stormer, & Appel, 2001; Yager & O'Dea, 2008). For example, Stice, Shaw, Burton, and Wade (2006) found that individuals in the dissonance-based program had lower thin-ideal internalization, dieting, and eating pathology symptoms at a 1-year follow-up compared to assessment only controls. Thus, interventions that combine media literacy and dissonance approaches may be particularly effective in the prevention of disordered eating.

Conclusion

In conclusion, eating restriction is a highly nuanced phenomenon that has been viewed from many perspectives and linked to many different factors. The intrasexual competition model has been especially promising, and further examination is needed on the intrasexual competition processes among people afflicted with disordered eating. Such information would prove useful for the development of prevention and intervention programs that utilize an understanding of both the ancient, adaptive mechanisms that underlie eating restriction and the proximate factors that are likely to trigger these mechanisms in modern environments.

References

Abed, R. T. (1998). The sexual competition hypothesis for eating disorders. British Journal of Medical Psychology, 71, 525–547.

Abed, R., Mehta, S., Figueredo, A. J., Aldridge, S., Balson, H., Meyer, C., et al. (2012). Eating disorders and intrasexual competition: Testing an evolutionary hypothesis among young women. Scientific World Journal, 2012(290813), 1–8.

Agras, W. S., Crow, S., Mitchell, J. E., Halmi, K. A., & Bryson, S. (2009). A 4-year prospective study of eating disorder NOS compared with full eating disorder syndromes. International Journal of Eating Disorders, 42, 565–570.

Aguirre, D., Sabbagh, K., Rupp, C., & Hoteit, L. (2012). Empowering the third billion: Women and the world of work in 2012. Retrieved from http://www.booz.com/global/home/what_we_think/third_billion

Ahern, A. L., Bennett, K. M., & Hetherington, M. M. (2008). Internalization of the ultra-thin ideal: Positive implicit associations with underweight fashion models are associated with drive for thinness in young women. Eating Disorders, 16, 294–307.

American Psychiatric Association. (2000). Diagnostic and statistical manual of mental disorders (5th ed.). Washington, DC: Author.

Bailey, J. M., Gaulin, S., Agyei, Y., & Gladue, B. A. (1994). Effects of gender and sexual orientation on evolutionary relevant aspects of human mating psychology. Journal of Personality and Social Psychology, 66, 1081–1093.

Bardone-Cone, A. M., Abramson, L. Y., Vohs, K. D., Heatherton, T. F., & Joiner, T. E., Jr. (2006). Predicting bulimic symptoms: An interactive model of self-efficacy, perfectionism, and perceived weight status. Behaviour Research and Therapy, 44, 27–42.

Baumeister, R. F., DeWall, C. N., Ciarocco, N. J., & Twenge, J. M. (2005). Social exclusion impairs self-regulation. Journal of Personality and Social Psychology, 88(4), 589.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. Psychological Bulletin, 117, 497–529.

Becker, J. B., Breedlove, S. M., & Crews, D. (1993). Behavioral endocrinology. Cambridge, MA: MIT Press.

Becker, A. E., Burwell, R. A., Herzog, D. B., Hamburg, P., & Gilman, S. E. (2002). Eating behaviours and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. The British Journal of Psychiatry, 180, 509–514.

Becker, C. B., Smith, L. M., & Ciao, A. C. (2006). Peer-facilitated eating disorder prevention: A randomized effectiveness trial of cognitive dissonance and media advocacy. Journal of Counseling Psychology, 53, 550–555.

Birkeland, R., Thompson, J., & Herbozo, S. (2005). Media exposure, mood, and body image dissatisfaction: An experimental test of person versus product priming. Body Image, 2, 53–61.

Björvell, H., Edman, G., Rössner, S., & Schalling, D. (1985). Personality traits in a group of severely obese patients: A study of patients in two self-chosen weight reducing programs. International Journal of Obesity, 9, 257–266.

Blascovich, J., Mendes, W. B., Vanman, E., & Dickerson, S. (2011). Social psychophysiology for social and personality psychology. London: Sage.

Brown, P. J., & Konner, M. (1987). An anthropological perspective on obesity. Annals of the New York Academy of Sciences, 499, 29–46.

Bruch, H. (1978). The golden cage: The enigma of anorexia nervosa. Cambridge, MA: Harvard University Press.

Bruch, H. (1988). Conversations with anorexics. Northvale, NJ: Aronson.

Bulik, C. M., Collier, D., & Sullivan, P. (2011). WTCCC3 and GCAN: A genomewide scan for anorexia nervosa. International Conference on Eating Disorders, Miami, FL.

Bulik, C. M., Sullivan, P. F., Tozzi, F., Furberg, H., Lichtenstein, P., & Pedersen, N. L. (2006). Prevalence, heritability, and prospective risk factors for anorexia nervosa. Archives of General Psychiatry, 63, 305–312.

Buss, D. M. (1988). The evolution of human intrasexual competition: Tactics of mate attraction. Journal of Personality and Social Psychology, 54, 616–628.

Buss, D. M. (1989). Sex differences in human mate preferences: Evolutionary hypotheses tested in 37 cultures. Behavioral and Brain Sciences, 12, 1–49.

Buss, D. M. (1995). Evolutionary psychology: A new paradigm for psychological science. Psychological Inquiry, 6, 1–30.

Buss, D. M. (2000). The evolution of happiness. American Psychologist, 55, 15–23.

Buss, D. M., Shackelford, T. K., Choe, J., Buunk, B. P., & Dijkstra, P. (2000). Distress about mating rivals. Personal Relationships, 7, 235–243.

Button, E. J., Chadalavada, B., & Palmer, R. L. (2010). Mortality and predictors of death in a cohort of patients presenting to an eating disorders service. International Journal of Eating Disorders, 43, 387–392.

Cahill, G. C., Jr. (1982). Starvation. Transactions of the American Clinical and Climatological Association, 94, 1–21.

Carlat, D. J., Camargo, C. A., & Herzog, D. B. (1997). Eating disorders in males: A report on 135 patients. American Journal of Psychiatry, 154, 1127–1132.

Cashdan, E. (2008). Waist-to-hip ratio across cultures: Trade-offs between androgen- and estrogendependent traits. Current Anthropology, 49, 1099–1107.

Cassidy, T., & Lynn, R. (1989). A multidimensional approach to achievement motivation: The development of a comprehensive measure. Journal of Occupational Psychology, 62, 301–312.

Castellini, G., Lo Sauro, C., Mannucci, E., Ravaldi, C., Rotella, C. M., Faravelli, C., et al. (2011). Diagnostic crossover and outcome predictors in eating disorders according to DSM-IV and DSM-V proposed criteria: A 6-year follow-up study. Psychosomatic Medicine, 73, 270–279.

Charnov, E. L. (1993). Life history invariants. Oxford: Oxford University Press.

Clutton-Brock, T. (2007). Sexual selection in males and females. Science, 318, 1882–1885.

Condit, V. K. (1990). Anorexia nervosa: Levels of causation. Human Nature, 1, 391-413.

Cooper, M. J. (1997). Cognitive theory of anorexia nervosa and bulimia nervosa: A review. Behavioural and Cognitive Psychotherapy, 25, 113–145.

Cooper, M. J. (2005). Cognitive theory in anorexia nervosa and bulimia nervosa: Progress, development and future directions. Clinical Psychology Review, 25, 511–531.

Crisp, A. H. (1997). Anorexia nervosa as a flight from growth: Assessment and treatment based on the model. In D. M. Garner & P. E. Garfinkel (Eds.), Handbook of treatment for eating disorders (2nd ed., pp. 248–277). New York: Guilford Press.

Crow, S. J., Peterson, C. B., Swanson, S. A., Raymond, N. C., Specker, S., Eckert, E. D., et al. (2009). Increased mortality in bulimia nervosa and other eating disorders. American Journal of Psychiatry, 166, 1342–1346.

Culbert, K. M., Breedlove, S. M., Burt, S. A., & Klump, K. L. (2008). Prenatal hormone exposure and risk for eating disorders: A comparison of opposite-sex and same-sex twins. Archives of General Psychiatry, 65, 329.

Daan, S., & Tinbergen, J. M. (1997). Adaptation of life histories. In J. R. Krebs & N. B. Davies (Eds.), Behavioural ecology: An evolutionary approach (pp. 311–333). Oxford: Blackwell Science.

Danner, U. N., Sanders, N., Smeets, P. A., van Meer, F., Adan, R. A., Hoek, H. W., et al. (2012). Neuropsychological weaknesses in anorexia nervosa: Set-shifting, central coherence, and decision making in currently ill and recovered women. International Journal of Eating Disorders, 45, 685–694.

Darwin, C. (1871). The descent of man and selection in relation to sex. London: Murray.

Dittmar, H., & Howard, S. (2004). Thin-ideal internalization and social comparison tendency as moderators of media models' impact on women's body-focused anxiety. Journal of Social and Clinical Psychology, 23, 768–791.

Dunbar, R. I. M. (1992). Neocortex size as a constraint on group size in primates. Journal of Human Evolution, 22, 469–493.

Durante, K. M., Li, N. P., & Haselton, M. G. (2008). Changes in women's choice of dress across the ovulatory cycle: Naturalistic and experimental evidence. Personality and Social Psychology Bulletin, 34, 1451–1460.

Eisler, I. (2005). The empirical and theoretical base of family therapy and multiple family day therapy for adolescent anorexia nervosa. Journal of Family Therapy, 27, 104–131.

Enzi, G., Gasparo, M., Biondetti, P. R., Fiore, D., Semisa, M., & Zurlo, F. (1986). Subcutaneous and visceral fat distribution according to sex, age and overweight, evaluated by computed tomography. Journal of Clinical Nutrition, 44, 739–746.

Epling, W. F., & Pierce, W. D. (1988). Activity-based anorexia: A biobehavioral perspective. International Journal of Eating Disorders, 5, 475–485.

Epling, W. F., & Pierce, W. D. (1992). Solving the anorexia puzzle. Toronto, Ontario, Canada: Hogrefe & Huber.

Faer, L. M., Hendriks, A., Abed, R. T., & Figueredo, A. J. (2005). The evolutionary psychology of eating disorders: Female competition for mates or for status? Psychology and Psychotherapy: Theory, Research and Practice, 78, 397–417.

Fairburn, C. G., Shafran, R., & Cooper, Z. (1998). A cognitive behavioral theory of anorexia nervosa. Behavior Research and Therapy, 37, 1–13.

Fassino, S., Leombruni, P., Pierò, A., Daga, G. A., Amianto, F., Rovera, G., et al. (2002). Temperament and character in obese women with and without binge eating disorder. Comprehensive Psychiatry, 43, 431–437.

Ferraro, F. R., Andres, M., Stromberg, L., & Kristjanson, J. (2003). Processing fat-related information in individuals at risk for developing an eating disorder. The Journal of Psychology, 137, 467–475.

Festinger, L. (1954). A theory of social comparison processes. Human Relations, 7, 117-140.

Festinger, L. (1957). A theory of cognitive dissonance. Stanford, CA: Stanford University Press.

Fichter, M. M., & Quadflieg, N. (2007). Long-term stability of eating disorder diagnoses. International Journal of Eating Disorders, 40, S61–S66.

Fishman, J., Fishman, J. H., Nisselbaum, J. S., Menendez-Botet, C., Schwartz, M. K., Martucci, C., et al. (1975). Measurement of the estradiol receptor in human breast tissue by the immobilized antibody method. Journal of Clinical Endocrinology and Metabolism, 40, 724–727.

Franko, D. L., & Keel, P. K. (2006). Suicidality in eating disorders: Occurrence, correlates, and clinical implications. Clinical Psychology Review, 26, 769–782.

Frisch, R. E. (1985). Fatness, menarche, and female fertility. Perspectives in Biology and Medicine, 28, 611–633.

Frisch, R. E. (1990). Body fat, menarche, fitness, and fertility. In R. E. Frisch (Ed.), Adipose tissue and reproduction (pp. 1–26). Basel, Switzerland: Karger.

Frisch, R. E., von Gotz-Welbergen, A., McArthur, S., Albright, T., Witschi, J., Bullen, B., et al. (1981). Delayed menarche and amenorrhea of college athletes in relation to age of onset of training. Journal of the American Medical Association, 246, 1559–1563.

Galinsky, A. D., Gruenfeld, D. H., & Magee, J. C. (2003). From power to action. Journal of Personality and Social Psychology, 85, 453–466.

Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. Psychological Medicine, 9, 273–279.

Gatward, N. (2007). Anorexia nervosa: An evolutionary puzzle. European Eating Disorders Review, 15, 1–12.

Geary, D. C. (2009). Male, female: The evolution of human sex differences. Washington, DC: APA.

Gesta, S., Tseng, Y.-H., & Kahn, C. R. (2007). Developmental origin of fat: Tracking obesity to its source. Cell, 131, 242–256.

Gould, J. L., & Gould, G. C. (1989). Sexual selection (2nd ed.). New York: Scientific American Library.

Grabe, S., Ward, L. M., & Hyde, J. S. (2008). The role of the media in body image concerns among women: A meta-analysis of experimental and correlational studies. Psychological Bulletin, 134(3), 460–476.

Grilo, C. M., Pagano, M. E., Skodol, A. E., Sanislow, C. A., McGlashan, T. H., Gunderson, J. G., et al. (2007). Natural course of bulimia nervosa and of eating disorder not otherwise specified: 5-Year prospective study of remissions, relapses, and the effects of personality disorder psychopathology. Journal of Clinical Psychiatry, 68, 738–746.

Guardia, D., Conversy, L., Jardir, R., Lafargue, G., Thomas, P., Dodin, V., et al. (2012). Imagining one's own and someone else's body actions: Dissociation in anorexia nervosa. PLoS One, 7, e43241.

Guisinger, S. (2003). Adapted to flee famine: Adding an evolutionary perspective on anorexia nervosa. Psychological Review, 110, 745–761.

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. Psychological Bulletin, 137, 660–681.

Hamilton, W. D. (1964). The genetical evolution of social behaviour. I and II. Journal of Theoretical Biology, 7, 1–52.

Harvey, T., Troop, N. A., Treasure, J. L., & Murphy, T. (2002). Fear, disgust, and abnormal eating attitudes: A preliminary study. International Journal of Eating Disorders, 32, 213–218.

Haselton, M. G., Mortezaie, M., Pillsworth, E. G., Bleske-Recheck, A. E., & Frederick, D. A. (2007). Ovulation and human female ornamentation: Near ovulation, women dress to impress. Hormones and Behavior, 51, 40–45.

Hawkins, R. C., & Clement, P. F. (1984). Binge eating: Measurement problems and a conceptual model. In R. C. Hawkins, W. J. Fremouw, & P. F. Clement (Eds.), The binge purge syndrome: Diagnosis, treatment, and research (pp. 229–251). New York: Springer.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. Psychological Bulletin, 110, 86–108.

Hebebrand, J., & Remschmidt, H. (1995). Anorexia nervosa viewed as an extreme weight condition: Genetic implications. Human Genetics, 95, 1–11.

Herzog, D. B., Greenwood, D. N., Dorer, D. J., Flores, A. T., Ekeblad, E. R., Richards, A., et al. (2000). Mortality in eating disorders: A descriptive study. International Journal of Eating Disorders, 28(1), 20–26.

Herzog, D. B., Norman, D. K., Gordon, C., & Pepose, M. (1984). Sexual conflict and eating disorders in 27 males. American Journal of Psychiatry, 141, 989–990.

Hudson, J. I., Hiripi, E., Pope, H. G., Jr., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biological Psychiatry, 61, 348–358.

Italian men suffer 'sexual anorexia' after internet porn use. (2011). Retrieved December 29, 2012, from

http://www.ansa.it/web/notizie/rubriche/english/2011/02/24/visualizza_new.html_1583160579.html

Joiner, T. E., Jr. (1999). Self-verification and bulimic symptoms: Do bulimic women play a role in perpetuating their own dissatisfaction and symptoms? International Journal of Eating Disorders, 26, 145–151.

Joiner, T. E., Jr., Heatherton, T. F., Rudd, M. D., & Schmidt, N. B. (1997). Perfectionism, perceived weight status, and bulimic symptoms: Two studies testing a diathesis-stress model. Journal of Abnormal Psychology, 106, 145.

Kanazawa, S. (2002). Bowling with our imaginary friends. Evolution and Human Behavior, 23, 167–171.

Keel, P. K. (2005). Eating disorders. Upper Saddle River, NJ: Pearson Education.

Keel, P. K., & Brown, T. A. (2010). Update on course and outcome in eating disorders. International Journal of Eating Disorders, 43, 195–204.

Keel, P. K., Brown, T. A., Holland, L. A., & Bodell, L. P. (2012). Empirical classification of eating disorders. Annual Review of Clinical Psychology, 8, 381–404.

Keel, P. K., Gravener, J. A., Joiner, T. E., Jr., & Haedt, A. A. (2010). Twenty-year follow-up of bulimia nervosa and related eating disorders not otherwise specified. International Journal of Eating Disorders, 43, 492–497.

Keel, P. K., Haedt, A., & Edler, C. (2005). Purging disorder: An ominous variant of bulimia nervosa? International Journal of Eating Disorders, 38, 191–199.

Keel, P. K., & Klump, K. L. (2003). Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. Psychological Bulletin, 129, 747–769.

Kennair, L. E. O. (2002). Evolutionary psychology: An emerging integrative perspective within the science and practice of psychology. Human Nature Review, 2, 17–61.

Kenrick, D. T., Gutierres, S. E., & Goldberg, L. L. (1989). Influence of popular erotica on judgments of strangers and mates. Journal of Experimental Social Psychology, 25, 159–167.

Kenrick, D. T., Keefe, R. C., Bryan, A., Barr, A., & Brown, S. (1995). Age preferences and mate choice among homosexuals and heterosexuals: A case for modular psychological mechanisms. Journal of Personality and Social Psychology, 69, 1166–1172.

Kenrick, D. T., Neuberg, S. L., Zierk, K. L., & Krones, J. M. (1994). Evolution and social cognition: Contrast effects as a function of sex, dominance, and physical attractiveness. Personality and Social Psychology Bulletin, 20, 210–217.

Kim, Y., Zerwas, S., Trace, S. E., & Sullivan, P. F. (2011). Schizophrenia genetics: Where next? Schizophrenia Bulletin, 37(3), 456–463.

Klump, K. L., Culbert, K. M., Slane, J. D., Burt, S. A., Sisk, C. L., & Nigg, J. T. (2012). The effects of puberty on genetic risk for disordered eating: Evidence for a sex difference. Psychological Medicine, 42, 627–637.

Klump, K. L., Gobrogge, K. L., Perkins, P. S., Thorne, D., Sisk, C. L., & Breedlove, S. (2006). Preliminary evidence that gonadal hormones organize and activate disordered eating. Psychological Medicine, 36, 539–546.

Klump, K. L., Suisman, J. L., Burt, S. A., McGue, M., & Iacono, W. G. (2009). Genetic and environmental influences on disordered eating: An adoption study. Journal of Abnormal Psychology, 118, 797.

Krones, P. G., Stice, E., Batres, C., & Orjada, K. (2005). In vivo social comparison to a thin-ideal peer promotes body dissatisfaction: A randomized experiment. International Journal of Eating Disorders, 38, 134–142.

Kunstman, J. W., & Maner, J. K. (2011). Sexual overperception: Power, mating motives, and biases in social judgment. Journal of Personality and Social Psychology, 100, 282–294.

Le Grange, D., Swanson, S. A., Crow, S. J., & Merikangas, K. R. (2012). Eating disorder not otherwise specified presentation in the US population. International Journal of Eating Disorders, 45, 711–718.

Leibowitz, S. (1992). Neurochemical-neuroendocrine systems in the brain control macronutrient intake and metabolism. Trends in Neuroscience, 15, 491–497.

Levine, M. P., Smolak, L., & Hayden, H. (1994). The relation of sociocultural factors to eating attitudes and behaviors among middle school girls. Journal of Early Adolescence, 14, 471–490.

Li, N. P. (2007). Mate preference necessities in long- and short-term mating: People prioritize in themselves what their mates prioritize in them. Acta Psychologica Sinica, 39, 528–535.

Li, N. P., Bailey, J. M., Kenrick, D. T., & Linsenmeier, J. A. W. (2002). The necessities and luxuries of mate preferences: Testing the tradeoffs. Journal of Personality and Social Psychology, 82, 947–955.

Li, N. P., Smith, A. R., Griskevicius, V., Cason, M. J., & Bryan, A. (2010). Intrasexual competition and eating restriction in heterosexual and homosexual individuals. Evolution and Human Behavior, 31, 365–372.

Li, N. P., Valentine, K. A., & Patel, L. (2011). Mate preferences in the U.S. and Singapore: A crosscultural test of the mate preference priority model. Personality and Individual Differences, 50, 291– 294.

Lock, J. (2011). Evaluation of family treatment models for eating disorders. Current Opinion in Psychiatry, 24, 274–279.

Lopez, C., Tchanturia, K., Stahl, D., & Treasure, J. (2009). Weak central coherence in eating disorders: A step towards looking for an endophenotype of eating disorders. Journal of Clinical and Experimental Neuropsychology, 31, 117–125.

Maner, J. K., Denoma, J. M., Van Orden, K. A., Gailliot, M. T., Gordon, K. H., & Joiner, T. E. (2006). Evidence of attentional bias in women exhibiting bulimotypic symptoms. International Journal of Eating Disorders, 39, 55–61.

Milos, G., Spindler, A., Schnyder, U., & Fairburn, C. G. (2005). Instability of eating disorder diagnoses: Prospective study. British Journal of Psychiatry, 187, 573–578.

Minuchin, S., Baker, L., Rosman, B. L., Liebman, R., Milman, L., & Todd, T. C. (1975). A conceptual model of psychosomatic illness in children. Archives of General Psychiatry, 32, 1031–1038.

Minuchin, S., Rosman, B. L., & Baker, L. (1978). Psychosomatic families: Anorexia nervosa in context. Cambridge, MA: Harvard University Press.

Mrosovsky, N., & Sherry, D. F. (1980). Animal anorexias. Science, 207, 837-842.

Nesse, R. M., & Williams, G. C. (1995). Evolution and healing: The new science of Darwinian medicine. London: Weidenfeld and Nicolson.

Neumark-Sztainer, D. (2005). I'm, like, SO fat! New York: Guilford Press.

Nielsen, S. (2003). Standardized mortality ratio in bulimia nervosa. Archives of General Psychiatry, 60, 851.

Nimrod, A., & Ryan, K. J. (1975). Aromatization of androgens by human abdominal and breast fat tissue. Journal of Clinical Endocrinology and Metabolism, 40, 367–372.

Norgan, N. (1997). The beneficial effects of body fat and adipose tissue in humans. International Journal of Obesity, 21, 738–754.

Park, J. H., Schaller, M., & Crandall, C. S. (2007). Pathogen-avoidance mechanisms and the stigmatization of obese people. Evolution and Human Behavior, 28, 410–414.

Paxton, S. J., Schutz, H. K., Wertheim, E. H., & Muir, S. L. (1999). Friendship clique and peer influences on body image concerns, dietary restraint, extreme weight-loss behaviors, and binge eating in adolescent girls. Journal of Abnormal Psychology, 108, 255–266.

Pond, C. M. (1978). Morphological aspects and the ecological and mechanical consequences of fat deposition in wild vertebrates. Annual Review of Ecology and Systematics, 9, 519–570.

Pond, C. M. (1998). The fats of life. Cambridge: Cambridge University Press.

Prentice, A. M., Diaz, E., Goldberg, G. R., Jebb, S. A., Coward, W. A., & Whitehead, R. G. (1992). Famine and refeeding: Adaptations in energy metabolism. In G. H. Anderson & S. H. Kennedy (Eds.), The biology of feast and famine: Relevance to eating disorders (pp. 22–46). San Diego, CA: Academic Press.

Preti, A., Rocchi, M. B., Sisti, D., Camboni, M. V., & Miotto, P. (2011). A comprehensive metaanalysis of the risk of suicide in eating disorders. Acta Psychiatrica Scandinavica, 124, 6–17.

Quinton, S. J., Smith, A. R., & Joiner, T. E. (2011). The 2nd to 4th digit ratio (2D:4D) and eating disorder diagnosis in women. Personality and Individual Differences, 51, 402–405.

Renfrew Center Foundation for Eating Disorders. (2003). Eating disorders 101 guide: A summary of issues, statistics and resources. Retrieved June 4, 2012, from http://www.renfrew.org

Rieger, E., Van Buren, D. J., Bishop, M., Tanofsky-Kraff, M., Welch, R., & Wilfley, D. E. (2010). An eating disorder-specific model of interpersonal psychotherapy (IPT-ED): Causal pathways and treatment implications. Clinical Psychology Review, 30, 400–410.

Rippon, C., Nash, J., Myburgh, K. H., & Noakes, T. D. (1988). Abnormal eating attitude test scores predict menstrual dysfunction in lean females. International Journal of Eating Disorders, 7, 617–624.

Roberto, C., Steinglass, J., Mayer, L., Attia, E., & Walsh, B. T. (2008). The clinical significance of amenorrhea as a diagnostic criterion for anorexia nervosa. International Journal of Eating Disorders, 41, 559–563.

Roberts, M., Tchanturia, K., & Treasure, J. (2010). Exploring the neurocognitive signature of poor set-shifting in anorexia and bulimia nervosa. Journal of Psychiatric Research, 44, 964–970.

Rosvall, K. A. (2011). Cost of female intrasexual aggression in terms of offspring quality: A cross-fostering study. Ethology, 117, 1–13.

Salmon, C., Crawford, C., Dane, L., & Zuberbier, O. (2008). Ancestral mechanisms in modern environments: Impact of competition and stressors on body image and dieting behavior. Human Nature, 19, 103–117.

Salmon, C., Figueredo, A. J., & Woodburn, L. (2009). Life history strategy and disordered eating behavior. Evolutionary Psychology, 7, 585–600.

Schaller, M., Park, J. H., & Faulkner, J. (2003). Prehistoric dangers and contemporary prejudices. European Review of Social Psychology, 14, 105–137.

Seifert, T. (2005). Anthropomorphic characteristics of centerfold models: Trends towards slender figures over time. International Journal of Eating Disorders, 37, 271–274.

Shackelford, T. K., Schmitt, D. P., & Buss, D. M. (2005). Universal dimensions of human mate preferences. Personality and Individual Differences, 39, 447–458.

Shafran, R., Lee, M., Cooper, Z., Palmer, R. L., & Fairburn, C. G. (2007). Attentional bias in eating disorders. International Journal of Eating Disorders, 40, 369–380.

Singh, D. (1993). Adaptive significance of female physical attractiveness: Role of waist-to-hip ratio. Journal of Personality and Social Psychology, 65, 293–307.

Smith, A. R., Fink, E. L., Anestis, M. D., Ribeiro, J., Gordon, K. H., Davis, H., et al. (2013). Exercise caution: Over-exercise is associated with suicidality in bulimia nervosa. Psychiatry Research, 206(2–3), 246–255.

Smith, A. R., Hawkeswood, S. E., & Joiner, T. E. (2010). The measure of a man: Associations between digit ratio and disordered eating in males. International Journal of Eating Disorders, 43, 543–548.

Smith, A. R., Li, N. P., & Joiner, T. (2011). The pursuit of success: Can status aspirations negatively affect body satisfaction? Journal of Social and Clinical Psychology, 30, 531–547.

Smolak, L. (1996). National Eating Disorders Association/next door neighbors puppet guide book. Seattle, WA: National Eating Disorders Association.

Steiger, H., Bruce, K., & Israël, M. (2003). Eating disorders. In G. Stricker, T. A. Widiger, & I. B. Weiner (Eds.), Handbook of psychology (Vol. 8, pp. 173–194). New York: Wiley.

Steinhausen, H. C. (2002). The outcome of anorexia nervosa in the 20th century. American Journal of Psychiatry, 159, 1284–1293.

Stevens, A., & Price, J. (2000). Evolutionary psychiatry: A new beginning (2nd ed.). London: Routledge.

Stice, E., Chase, A., Stormer, S., & Appel, A. (2001). A randomized trial of a dissonance-based eating disorder prevention program. International Journal of Eating Disorders, 29, 247–262.

Stice, E., & Shaw, H. E. (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology. Journal of Psychosomatic Research, 53, 985–993.

Stice, E., Shaw, H. E., Burton, E., & Wade, E. (2006). Dissonance and healthy weight eating disorder prevention programs: A randomized efficacy trial. Journal of Consulting and Clinical Psychology, 74, 263–275.

Striegel-Moore, R. H., Silberstein, L. R., & Rodin, J. (1986). Toward an understanding of risk factors for bulimia. American Psychologist, 41, 246–263.

Striegel-Moore, R. H., Tucker, N., & Hsu, J. (1990). Body image dissatisfaction and disordered eating in lesbian college students. International Journal of Eating Disorders, 9, 493–500.

Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: Evidence of shared liability and transmission of partial syndromes. American Journal of Psychiatry, 63, 305–312.

Substance Abuse and Mental Health Services Administration. (2003). Handout-eating disorder. Retrieved June 4, 2013, from http://mentalhealth.samhsa.gov/publications/allpubs/ken98-0047/default.asp

Sullivan, P. F. (1995). Mortality in anorexia nervosa. American Journal of Psychiatry, 152, 1073–1074.

Sullivan, P. F., Bulik, C. M., Fear, J. L., & Pickering, A. (1998). Outcome of anorexia nervosa: A case–control study. American Journal of Psychiatry, 155, 939–946.

Surbey, M. K. (1987). Anorexia nervosa, amenorrhea, and adaptation. Ethology and Sociobiology, 8, 47–61.

Symons, D. (1979). The evolution of human sexuality. New York: Oxford University Press.

Sypeck, M. F., Gray, J. J., & Ahrens, A. H. (2004). No longer just a pretty face: Fashion magazines' depictions of ideal female beauty from 1959 to 1999. International Journal of Eating Disorders, 36, 342–347.

Tanofsky-Kraff, M., Wilfley, D. E., & Spurrell, E. (2000). Impact of interpersonal and ego-related stress on restrained eaters. International Journal of Eating Disorders, 27, 411–418.

Thompson, J. K., Heinberg, L. J., Altabe, M., & Tantleff-Dunn, S. (1999). Exacting beauty: Theory, assessment, and treatment of body image disturbance. Washington, DC: American Psychological Association.

Thompson, J. K., & Stice, E. (2001). Thin-ideal internalization: Mounting evidence for a new risk factor for body-image disturbance and eating pathology. Current Directions in Psychological Science, 10, 181–183.

Thompson, J. K., van den Berg, P., Roehrig, M., Guarda, A. S., & Heinberg, L. J. (2004). The Sociocultural Attitudes Towards Appearance Scale-3 (SATAQ-3): Development and validation. International Journal of Eating Disorders, 35, 293–304.

Treasure, J. L., & Owen, J. B. (1997). Intriguing links between animal behavior and anorexia nervosa. International Journal of Eating Disorders, 21, 307–312.

Trivers, R. L. (1972). Parental investment and sexual selection. In B. Campbell (Ed.), Sexual selection and the descent of man (pp. 136–179). Chicago: Aldine.

Trivers, R. L., & Willard, D. E. (1973). Natural selection of parental ability to vary the sex ratio of offspring. Science, 179, 90–92.

Voland, E., & Voland, R. (1989). Evolutionary biology and psychiatry: The case of anorexia nervosa. Ethology and Sociobiology, 10, 223–240.

Walters, S., & Crawford, C. B. (1994). The importance of mate attraction for intrasexual competition in men and women. Ethology and Sociobiology, 15, 5–30.

Wang, K., Zhang, H., Bloss, C. S., Duvvuri, V., Kaye, W., Schork, N. J., et al. (2010). A genomewide association study on common SNPs and rare CNVs in anorexia nervosa. Molecular Psychiatry, 16, 949–959.

Wasser, S. K., & Barash, D. P. (1983). Reproductive suppression among female mammals: Implications for biomedicine and sexual selection theory. Quarterly Review of Biology, 58, 513–538.

Watson, R., & Vaughn, L. M. (2006). Limiting the effects of the media on body image: Does the length of the intervention make a difference? Eating Disorders: The Journal of Treatment and Prevention, 14, 385–400.

Wells, J. C. K. (2012). The evolution of human adiposity and obesity: Where did it all go wrong? Disease Models and Mechanisms, 5, 595–607.

Weltzin, T. E., Weisensel, N., Franczyk, D., Burnett, K., Klitz, C., & Bean, P. (2005). Eating disorders in men: Update. The Journal of Men's Health & Gender, 2, 186–193.

Wilksch, S. M., Durbridge, M. R., & Wade, T. D. (2008). A preliminary controlled comparison of programs designed to reduce risk of eating disorders targeting perfectionism and media literacy. Journal of the American Academy of Child and Adolescent Psychiatry, 47, 939–947.

Wilksch, S. M., & Wade, T. D. (2009). Reduction of shape and weight concern in young adolescents: A 30-month controlled evaluation of a media literacy program. Journal of the American Academy of Child and Adolescent Psychiatry, 48, 652–661.

Williams, C. G. (1966). Natural selection, the costs of reproduction, and a refinement of Lack's principle. American Naturalist, 100, 687–690.

Workgroup for Classification of Eating Disorders in Children and Adolescents (WCEDCA). (2007). Classification of child and adolescent eating disturbances. International Journal of Eating Disorders, 40, S117–S122.

Wrangham, R. W. (1999). Evolution of coalitionary killing. Yearbook of Physical Anthropology, 42, 1–30.

Yager, Z., & O'Dea, J. A. (2008). Prevention programs for body image and eating disorders on University campuses: A review of large, controlled interventions. Health Promotion International, 23, 173–189.

Zerwas, S., & Bulik, C. M. (2011). Genetics and epigenetics of eating disorders. Psychiatric Annals, 41, 532–538.