Exploring the Cultural and Biopsychological Limitations of the Tripartite Influence Model of Body Dissatisfaction and Eating Disturbance

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Abstract
Anorexia nervosa (AN) is a psychological disorder predominantly manifesting in post-pubertal women. It is characterized by a distorted body image and a fear of gaining weight, which results in the restriction of food intake. Consequently, these food intake restrictions lead to severe and unhealthy weight loss. The Tripartite Influence Model of body dissatisfaction and eating disturbance focuses on the interactions among peers, parents, and the media with thin-ideal internalizations and social comparisons. These complex interactions can lead to body dissatisfaction and food restriction behaviours, contributing to the emergence of AN. Although these psychosocial factors play an important role in the development of AN, the Tripartite Influence Model is limited in its ability to conceptualize AN in non-Westernized cultures and it neglects the significance of biological factors specifically present after the onset of puberty. Levels of ovarian hormones, serotonin, hypothalamic-pituitary-adrenal axis activation, and genetics have all been proposed to influence the risk of AN onset. As such, there is a need in AN research for a biopsychosocial model, which would expand our current understanding of the complex etiologies of the disorder, allow for the development of more effective treatments and add to the general understanding of AN prevention.

According to the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5; American Psychiatric Association, 2013), anorexia nervosa (AN) is an eating disorder characterized by body image issues, a fear of gaining weight, and restricted food intake, all of which result in a low body weight. Unlike other psychological disorders, AN is mainly diagnosed after puberty (Klump, 2013) among women between the ages of 15-19 years old (Espie & Eisler, 2015). With the new DSM–5 criteria, it is estimated that by the age of 20, the prevalence of AN among women is 0.8% (Stice, Marti, & Rohde, 2013). In 2010, it was estimated that eating disorders contribute to 2.16 million years lost globally due to disability and premature death, a 65.7% increase from 1990 (Murray et al., 2012). Therefore, compared to a healthy individual’s life, patients suffering from an eating disorder have a drastically reduced lifespan and quality of life—a problem that appears to be increasing over the years. Compared to a population of the same sex and age, individuals suffering from AN are three times more likely to die within a year following hospital discharge (Hoang, Goldacre, & James, 2011). Considering the detrimental effects AN has on an individual’s quality of life, it is crucial to understand the disorder’s etiology in order to better engage in prevention strategies.

Extensive research in the area of AN has focused on the possible psychosocial etiologies of the disorder. The Tripartite Influence Model of body dissatisfaction and eating disturbance highlights the importance of social comparisons and the internalization of thin-ideals (i.e., the belief that being thin is the ideal body type), which are influenced by peers, parents, and the media. Research has shown that both of these components contribute towards body dissatisfaction and an increased risk for developing AN (Keery, van den Berg, & Thompson, 2004). Due to the importance Westernized cultures place on being thin (Swami et al., 2010),
the fear of gaining weight as one of the AN diagnostic criteria may limit the ability to diagnose AN in non-Westernized cultures (Lee, 1995). This also limits the Tripartite Influence Model’s ability to conceptualize AN risk cross-culturally. Furthermore, because AN diagnoses are often made in post-pubertal women (i.e., after the onset of puberty), it has been proposed that changes in hormones and gene expression, not just social factors outlined by the Tripartite Influence Model, play a role in the development of AN. Ovarian hormones, particularly estradiol and progesterone, have been implicated in the development of AN due to their effects on thin-ideal internalization and body dissatisfaction (Racine et al., 2012), assist in the development of secondary sex characteristics, and restrict food intake (Asarian & Geary, 2006). Serotonin contributes to an increase in AN risk by altering food intake (Rivera, Santollo, Nikonova, & Eckel, 2010) and mood (Gauthier et al., 2014), while the activation of the hypothalamic-pituitary-adrenal (HPA) axis maintains self-starvation behaviours (Bergh & Sodersten, 1996). Finally, genes interact with ovarian hormones, as well as heighten the sensitivity and vulnerability to thin-ideal internalization and body weight concerns to increase the risk of developing AN (Suisman et al., 2012). The focus of the Tripartite Influence Model on psychosocial determinants of AN neglects the importance of these biological factors that also contribute to AN risk. The aforementioned limitations of the Tripartite Influence Model will be critically analyzed with the goal of outlining the importance of cultural and biopsychological risk factors for AN in order to develop a new integrative model.

**Tripartite Influence Model of Body Dissatisfaction and Eating Disturbance**

According to the Tripartite Influence Model, peer influences have been shown to play a large role in body dissatisfaction and the desire to lose weight. One study (Clark & Tiggeman, 2006) showed that with greater thin-ideal internalization, girls are more likely to engage in appearance-related conversations with their friends. These conversations establish peer appearance norms that can ultimately lead to body dissatisfaction if there is a discrepancy between the perceived norm and a girl’s current body size. Body dissatisfaction is also affected by appearance-based comparisons between a girl and individuals in her social circle. One study (Lev-Ari, Baumgarten-Katz, & Zohar, 2014) examined how appearance-based comparisons with a girl’s mother, sister, and female friend affected body dissatisfaction, desire to be thin, and ideal body image. Comparisons between friends, in particular, showed the highest correlation with body dissatisfaction and a drive to be thin, as well as as the greatest discrepancy between one’s current body size and their ideal body size. Overall, having a female friend that is thinner than one’s self leads to greater body dissatisfaction and desire to lose weight.

Peers have the capacity to greatly affect body dissatisfaction; however, their influence also extends to food intake behaviours. Girls in grades 6-8, who are exposed to peers engaged in dieting behaviours, are more likely to restrict their own eating as well (Levine, Smolak, Moodey, Shuman, & Hessen, 1994). Appearance-based comparisons among peers also have implications for dieting behaviours. Girls who believe popularity and attractiveness are based on thinness are more likely to engage in restricted eating (Lieberman, Gauvin, Bukowski, & White, 2001). Therefore, social comparisons between peers have important implications for body dissatisfaction and dieting behaviours, ultimately contributing to the development of AN. However, peers alone do not account for the emergence of body dissatisfaction and thin-ideal internalization. It is important to also examine the effects parents have on their children’s susceptibility to AN risk factors.

Girls who experience pressure to be thin from their parents have a greater risk for developing body dissatisfaction. On average, girls not only desire to be thinner, but also believe their parents and best female friends want them to be skinnier (Shneider et al., 2013). The perceived pressure to be thin, whether or not it is explicitly expressed by parents, has implications for body dissatisfaction and restricted food intake. Research has shown that girls who are teased by family members about their weight are more likely to engage in restricted eating behaviours and have greater body dissatisfaction (Neumark-Sztainer et al., 2010). There have also been studies showing that girls are more likely to diet when
shown that social comparison amongst peers mediates the relationship between parents, body dissatisfaction, and food intake (van den Berg, Thompson, Obremski-Brandon, & Coovert, 2002). Therefore, peers play an important role in propagating media-induced thin-ideals, thereby increasing the risk of developing body dissatisfaction and AN. They also play an essential role in mediating the relationship parents have on body dissatisfaction. Altogether, examining psychosocial risk factors is imperative to understanding the etiology of AN. However, psychosocial factors are often shaped by culture, providing a unique set of influences that affect the risk of AN development.

Cultural Influences

Since the fear of gaining weight is one of the diagnostic criteria of AN, some researchers argue that the disorder is culture-bound (Lee, 1995). Western cultures view a thinner body as more beautiful and attractive, thereby motivating the internalization of thin-ideals and, ultimately, generating a fear of gaining weight among many women. Not all cultures, however, share the same body weight ideals. A study (Swami et al., 2010) assessing the beauty standards in 26 countries around the world explored the interactions between physical attractiveness ideals and body dissatisfaction. Some variations in body weight ideals were found between different cultures. However, the greatest variation was found in cultures with both low and high socioeconomic status (SES) areas. In low SES areas of Malaysia and South Africa, a heavier body was considered to be more attractive due to its association with increased resources. In contrast, high SES areas showed a preference for low body weights. Furthermore, low SES areas in Malaysia and South Africa showed lower levels of body dissatisfaction than higher SES areas. In addition, increased exposure to Western media, was associated with greater desire to be thinner, regardless of the world region. These thin-ideals also led to an increase in body dissatisfaction among women. As a result of the variation in body weight ideals between and within cultures, it could be argued that AN may also manifest differently depending on the cultural context.

A study (Lee, Ho, & Hsu, 1993) examining Chinese patients with AN discovered that, with the exception of
the fear of gaining weight, Chinese patients with AN displayed similar symptoms to Western anorexic patients. However, Chinese patients with AN were more likely to restrict their food intake due to a lack of appetite or stomach bloating. Therefore, it can be argued that the fear of gaining weight is a manifestation of AN, which occurs from Westernization, instead of being an integral aspect of the disorder itself. A recent systematic review and meta-analysis examined the cross-cultural literature of eating disorders and found that AN is not culturally bound as there are similar expressions of the disorder historically and across different cultures. One exception, however, is the fear of gaining weight diagnostic criteria, which is influenced by Western culture (Keel & Klump, 2003). The Tripartite Influence Model focuses on thin-ideal internalization as an integral part of AN development, thereby limiting its conceptualization of AN cross-culturally. In order to better understand the etiology of AN, integrating both psychosocial and cultural factors is required. However, psychosocial and cultural influences alone do not explain why the incidence of AN is greatest following puberty. Therefore, it is essential to examine how physiological factors, such as hormones, may also affect the development of AN.

### Hormonal Factors

Hormone levels, particularly after puberty, have been implicated in the development of AN. Estradiol and progesterone are steroid hormones released by the ovaries in females. These hormones function by binding to proteins called nuclear receptors within cell nuclei to influence the transcription of genes throughout the body. By altering transcription, these hormones have various widespread effects on the body. These include, but are not limited to, effects on adipose tissue (Kim, Cho, & Kim, 2014), bone tissue growth, reproduction, and brain functioning (Edwards, 2005). In post-pubertal women, ovarian hormone levels fluctuate over the course of their menstrual cycle. These changes in hormone levels affect cognition and emotional processing in female brains (Toffoletto, Lanzenberger, Gingnell, Sundstrom-Poromaa, & Comasco, 2014). To examine estradiol and progesterone’s effect on psychosocial factors that contribute to disordered eating, one study (Racine et al., 2012) followed women across their menstrual cycle, and measured body dissatisfaction and thin-ideal internalization daily. Both body dissatisfaction, and a desire to be thin peaked at the midluteal phase of the menstrual cycle, when estradiol and progesterone levels are the highest. The relationship between body dissatisfaction and progesterone was not influenced by emotional eating or negative affect; however, the relationship between body dissatisfaction and estradiol was mediated through negative affect. This suggests that progesterone has more direct effects on body dissatisfaction than estradiol. The desire to be thin, however, was directly affected by both ovarian hormones. Not only do estradiol and progesterone affect body dissatisfaction and the desire to be thin, but they are also implicated in altered cognitive processing among individuals with AN. Women with AN who experience irregular menstrual cycles and, therefore, lower levels of estradiol and progesterone, have been shown to have deficits in language, math, and reading scores compared to AN patients with regular menstrual cycles (Chui et al., 2008). Therefore, it could be argued that ovarian hormones play an important role in cognition, which may affect the risk of AN among post-pubertal women. According to the Tripartite Influence Model, thin-ideals result from media exposure, which contributes to the development of body dissatisfaction. However, because estradiol and progesterone are important for determining body dissatisfaction and the desire to be thin, it is important to acknowledge these ovarian hormones in the development of AN instead of focusing entirely on psychosocial determinants. As demonstrated, ovarian hormones are important in altering cognition, but both estradiol and progesterone are also crucial for physical changes that occur in women around puberty.

Secondary sex characteristics, such as increased body mass and breast development, occur during puberty in young women. Research has shown that puberty is associated with increased levels of body dissatisfaction, a desire to be thin, and eating concerns among girls. One study (McNicholas, Dooley, McNamara, & Lennon, 2012) showed that girls who mature earlier than their peers tend to be the least happy with their bodies, have the greatest desire to be thin, and show the highest overall eating concerns. The increased weight gain
and physical maturity that is associated with puberty is thought to drive these effects. Secondary sex characteristics are largely affected by increased levels of ovarian hormones throughout the body. It has been suggested that estradiol regulates adipose tissue storage through estrogen receptor signaling, which increases body mass in women (Kim et al., 2014). This increased body mass conflicts with the thin-ideals valued in Westernized cultures. Studies have shown that body dissatisfaction is influenced by both an increase in body mass and thin-ideal internalization (Stice & Whitenton, 2002). This is intuitive as ovarian hormones at the time of puberty increase body weight, thereby increasing the desire to be thin in order to attain a Western ideal of beauty. These thin-ideals then drive the production of body dissatisfaction. Therefore, it is important to acknowledge the role of ovarian hormones through secondary sex characteristics in AN risk. The increased body dissatisfaction induced by secondary sex characteristics may lead to a restriction of food intake to attain thin-ideals. However, ovarian hormones, estradiol in particular, have also been studied in relation to directly affecting food intake.

To study the effects of ovarian hormones on food intake, ovaries are removed (i.e., ovariectomy) in rat models to drastically reduce the levels of ovarian hormones in the body. As a result, the animals will increase their food intake and gain weight. With estrogen supplementation, the rats will decrease their food intake and lose weight (Asarian & Geary, 2002). When examining human subjects, women reduce their food intake during their pre-ovulatory phase, when estrogen levels are high, compared to the other phases of the menstrual cycle (Asarian & Geary, 2006). This research suggests estradiol acts as a mediator between restricted food intake and AN, rather than just body dissatisfaction as a motivator for food restriction behaviours. Therefore, higher levels of estradiol may contribute to dieting behaviours that lead to the development of AN. One of the proposed mechanisms of estradiol’s relationship with food intake restriction is through the neurotransmitter serotonin.

Serotonin regulates both food intake and mood. In ovariectomized rats, it has been shown that estradiol binds to the serotonin receptors in the brainstem, thereby decreasing the rats’ food intake (Rivera et al., 2010). Estradiol has also been shown to increase serotonin production throughout many other brain regions such as the amygdala, hypothalamus (McQueen, Wilson, & Fink, 1997), and frontal cortex (Matsuda, Hirano, & Watanabe, 2002). The increase in estradiol-induced serotonin may result in changes in mood, cognition, and behaviour (McQueen et al., 1997). Serotonin has also been well studied in its relation to symptoms of depression and anxiety, which are often co-morbidly expressed with AN. One study (Gauthier et al., 2014) demonstrated that an increased level of serotonin was associated with greater depression and anxiety symptoms when AN patients were discharged from treatment centers following re-feeding. However, compared to individuals without AN, patients with AN had lower overall serotonin levels likely due to severe malnutrition. Another study (Rodgers, Paxton, & Chabrol, 2010) found that depression increased the perceived pressure that girls experienced from both their peers and the media. This resulted in increased body dissatisfaction and a desire to be thin. Considering estradiol activates serotonin receptors, rises in estradiol levels that accompany puberty may also increase serotonin levels in the body. Theoretically, this would increase depressive symptoms, leading to an increase in body dissatisfaction, thin-idealization, and ultimately increase the risk of AN. This proposed mechanism is neglected by the Tripartite Influence Model, which states that body dissatisfaction is influenced by thin-ideal internalization and social comparison. Therefore, this model fails to properly conceptualize body dissatisfaction. It neglects the interactions between serotonin and estradiol levels and, consequently, the influence of serotonin on mood and cognition. Although the interaction with serotonin and estradiol may explain the emergence of dieting behaviours, it neglects the importance of stress for maintaining food intake restriction.

One of the mechanisms behind self-starvation is the activation of a stress pathway called the HPA axis. Starvation imposes a stress on the body, causing the hypothalamus to release corticotropin-releasing hormone (CRH). CRH indirectly increases glucocorticoid levels in the body. One of the actions of glucocorticoids, a class of hormones released by the adrenal glands, is to
activate dopaminergic neurons. Research has implicated glucocorticoids in feelings of reward (Keating, 2011). One study demonstrated that rats will self-administer glucocorticoids to maintain optimal levels of the hormones in the blood (Piazza et al., 1993). This results in an increase in dopamine levels within half an hour of the glucocorticoid injections. In the context of AN, it has been proposed that initial feelings of starvation activate the HPA axis, thereby coupling the starvation with a reward mechanism through dopamine release. Therefore, for individuals with AN, it will feel rewarding to experience starvation (Bergh & Sodersten, 1996). A second model used to understand the relationship between dopamine and AN is the lack of reward associated with food consumption. Generally speaking, food can be considered rewarding; however, alterations to the dopamine system among individuals with AN may limit the rewards associated with food intake (Kontis & Theochari, 2012). Although these two proposed models are simplistic and require future research, it highlights the importance of examining the HPA axis and dopamine in the maintenance of AN symptoms rather than focusing on body dissatisfaction and thin-ideal internalization as the only explanations for food intake restriction. Examining how hormones act in the body is crucial for understanding the emergence of AN in post-pubertal women. However, there is a strong connection between hormones and genetics that contribute to AN risk, particularly following the onset of puberty.

### Genetic Factors

Research on AN etiologies has recently focused on the role of genetics that predispose an individual to developing AN. This includes increasing the susceptibility to internalizing thin-ideals, effecting body weight concerns, and interacting with ovarian hormones. Thin-ideal internalization is a major risk factor for the development of AN. However, not all individuals adopt thin-ideals to the same extent. Research has shown that genetic factors influence thin-ideal internalization among young women. Using a twin study design, one study (Suisman et al., 2012) concluded that there were significant differences in thin-ideals among non-shared environments, with little effect on thin-ideals among shared environments. In other words, genetic factors are likely influencing a large proportion of the variation in thin-ideal internalization among twins, whereas environmental factors, such as media exposure, have a smaller effect. It was proposed that personality traits might mediate the relationship between genetics and thin-ideal internalizations by altering the vulnerability to adopt thin-ideals. Genetics not only play a role in the susceptibility to value thin-ideals, but they also alter the concerns one has about their body weight. Recent research has determined that weight and shape concerns have heritability estimates of 64-66% (Spanos, Burt, & Klump, 2010). Due to the heritability of weight and shape concerns, body dissatisfaction is not just a product of thin-ideal internalization and social comparison. As such, the Tripartite Influence Model misses important factors to explain the development of body dissatisfaction and, ultimately, the emergence of AN. Although the heritability of genes influence many risk factors of AN, it is also important to understand how hormones interact with genes to influence AN susceptibility.

Estradiol and progesterone’s mechanism of action is to bind to nuclear receptors to alter the transcription of genes. These hormones, therefore, interact with the genes responsible for disordered eating behaviours. Studies have shown that 0% of the variance in disordered eating could be accounted for by genetic influences before puberty (Klump, Perkins, Burt, McGue, & Iacono, 2007). However, following puberty, 44% of the variance could be explained by genetics. The difference in genetic influence before and after puberty can be attributed to the increase in ovarian hormones at the time of puberty as these hormones alter the transcription of genes (Klump et al., 2007). Furthermore, there is little heritability in disordered eating among twins with low estradiol levels, but high heritability when estradiol levels are high (Klump, Keel, Sisk, & Burt, 2010). Therefore, the genetic influences driving AN are likely to be mediated by ovarian hormones, particularly estradiol. Overall, it is important to acknowledge the role of genetics in the development of AN. However, the Tripartite Influence Model fails to consider how an individual’s genetic makeup can lead to disordered eating. This limits the model’s ability to predict disordered eating, especially at the time of puberty when increases in ovarian hormones interact with underlying genetic
differences.

**Conclusion**
The Tripartite Influence Model of body dissatisfaction and eating disturbance provides a robust overview of psychosocial factors that increase the risk of developing AN. Many studies have supported its claims that influences from peers, parents, and the media affect thin-ideal internalization and social comparison, leading to body dissatisfaction and restricted food intake. Although it focuses on psychosocial etiologies of AN, the Tripartite Influence Model limits its conceptualization of AN to Westernized cultures. The model also neglects a range of possible biological influences that contribute to AN risk.

Considering the Tripartite Influence Model lacks in its comprehension of underlying physiology, a biopsychosocial model is required to better understand the potential etiologies of AN, particularly after the onset of puberty. In order to develop such a model, research connecting the psychosocial factors to underlying physiological mechanisms is needed. Examining correlates, for instance, between social comparison of appearance to other girls, and ovarian hormones across the menstrual cycle would allow for a better understanding of estradiol’s role in AN risk. Understanding the link between social comparison, and ovarian hormone levels could further help in identifying girls at risk for developing AN by allowing for the analysis of estradiol and progesterone levels in the body. Investigating estradiol levels cross-culturally, and examining whether they are related to the prevalence of AN within that culture, is another viable approach to the problem.

Integrating psychosocial, cultural, and physiological factors that contribute to AN risk is also important for treatment of the disorder. Current treatment methods include family counseling (Espie & Eisler, 2015), cognitive-behavioural therapy, and re-nourishment to regain healthy weight (Watson & Bulik, 2013). Some studies have assessed the effectiveness of selective serotonin reuptake inhibitors (SSRIs) to improve weight gain and to treat the anxiety and depressive symptoms associated with AN. The effectiveness of this approach depends on the SSRI used, which results in inconsistent outcomes (Watson & Bulik, 2013). Treatment plans that address psychosocial determinants of AN, such as family counseling, with physiological-based treatments, such as medications, may provide the most effective form of treatment for AN by targeting multiple facets of the disorder. Further research is required to assess the efficacy of such treatment plans.

Overall, the Tripartite Influence Model of body dissatisfaction and eating disturbance encompasses many of the psychosocial factors that may lead to the development of AN. However, integrating psychosocial, cultural, and biological factors into a biopsychosocial model will provide the most robust understanding of AN etiology. Ultimately, this will aid in developing more effective treatment plans as well as provide insight into prevention of the disorder.

**References**


