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# Biopsychosocial Review of Eating Disorders

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## Abstract

Eating disorders are among the deadliest mental illnesses known to occur. Eating disorders directly cause 10,200 deaths each year, which is one death every 52 minutes. About 9% of the global population is affected by eating disorders at some point during their lifetime. This paper aims to provide a better understanding of the factors that contribute to the onset of eating disorders. Specifically, we examine biological factors, such as genetics, family history and the neuroscience behind eating disorders; furthermore, we explore psychological factors including other mental health conditions and their correlation, personality traits and behavioral risk factors; lastly, we consider social factors related to the onset of eating disorders, such as childhood and social environment, the media, and demographic factors.

## Keywords

Eating Disorders, Risk Factors, Etiology

## 1. Introduction

Eating disorders are behavioral illnesses characterized by severe disturbance in eating behavior accompanied by distressing thoughts and emotions [1]. Although there are many different types of eating disorders, this paper will focus on the three most prevalent: Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Binge Eating Disorder (BED). According to the *Diagnostic and Statistical Manual of Mental Disorders* (5th edition, DSM-5; American Psychiatric Association, 2013) [2], Anorexia Nervosa is characterized by severely restricting intake of food and can also include vomiting, use of diet pills, and overexercising. It is also paired with an intense fear of gaining weight, disturbance of body image, and a failure to recognize the severity of the disorder. There are two subtypes of AN: restriction type and purging type. Symptoms of AN typically include a substantial drop in weight, fatigue, absence of menstruation, low blood pressure, and more. The

DSM-5 classifies Bulimia Nervosa as eating an abnormally large amount of food in a short period of time and feeling uncontrolled, called binging, and then trying to get rid of the calories, called purging. The severity is measured based on how many compensatory behaviors occur per week. Purging can take multiple forms including vomiting, use of diet pills or laxatives. Binge Eating Disorder is characterized by the DSM-5 as constantly binging and feeling out of control during eating without using compensatory strategies. Its severity is measured in binging episodes per week [2].

Among all mental illnesses, eating disorders are among the most deadly, directly causing one death every 52 minutes. In the past several years, the rate of eating disorders has increased significantly. Studies have found that the prevalence rate from 2000 to 2006 was 3.5%, but it has increased to 7.8% from 2013 to 2018 [3]. Little is known about the causes and development of eating disorders. However, it is known that eating disorders feed off of an interaction of factors, including biological, psychological and social. Furthermore, there is stigma around both eating disorders and mental illnesses in general. This paper aims to help people understand the factors around eating disorders, and therefore help decrease negative stigma. Specifically, it focuses on the biological, psychological and social factors of eating disorders.

Genetics and biological factors play a significant role in the onset of mental health disorders, including eating disorders. In addressing biological factors of eating disorders, we examine the effects of family history and genetics in the development of eating disorders. This discussion is continued by exploring the hormones and brain regions associated with eating disorders.

Mental health disorders often coexist due to similar psychological patterns. Additionally, certain personality traits and behaviors increase risk for eating disorders. In addressing psychological factors, we examine the effect and correlation of co-occurring mental health disorders, personality traits, and behavioral risk factors.

Eating disorders are viewed as largely socially influenced mental health disorders due to the prevalence of dieting and body standards in society. However, there are more social factors influencing the onset of eating disorders than just cultural attitudes towards thinness. Finally, in addressing social factors, we explore factors related to the onset of eating disorders, such as childhood and social environment, the media and demographic factors.

## 2. Biological Factors

### 2.1. Genetics and Family History

Studies have shown that relatives of people with eating disorders have ten times a greater risk to develop the trait than those without due to familial aggregation, or the clustering of traits or disorders [4]. However, it is difficult to tell whether this is due to genetics or environment, which limits current genetic research. Typically, researchers use two methods to study the role of genetics in eating dis-

orders: twin studies and genome-wide studies. Twin studies utilize identical twins to distinguish between genetic and environmental factors, solving the limitation of the difficulty of separating these factors. Identical twins have the same DNA but different environmental experiences, so by observing identical twins, scientists can see if the trait is genetic or environmental. On the other hand, genome-wide studies study genomes, which are a complete set of genetic material, to look for particular genetic variations in people with the trait.

Twin studies have found that genetics account for a significant percentage of the inheritance of eating disorders. The genetic heritability, which is the percent chance a child has of inheriting the trait if a parent has it, of AN is estimated to be 33% to 84%, while it is estimated to be 28% to 83% for BN. The remaining variance can be accounted for by other factors, such as social context, mental health, or other psychological factors [4]. Overall, eating disorders are highly heritable, and family history can greatly increase the risk of development.

### 2.2. Hormones Associated

Scientists use case control association, which is genotyping and analyzing those who have the trait versus those who do not, to ascertain which genes are associated with eating disorders. They analyzed serontonic genes due to the relationship between weight, appetite regulation and serotonin, which is a neurotransmitter affecting cognitive brain functions and physiological processes [5]. Certain transporter and receptor genes have been studied, and certain alleles of these genes have been shown to be at higher risk. However, it is unlikely that serotonin is related uniquely to eating disorders, as they are also associated with other mental health conditions, such as depression. Therefore, serotonin is likely not a specific vulnerability factor for eating disorders, but one for mental health conditions in general [6]. This is consistent with the fact that depression and eating disorders are highly correlated, as explained in the co-occuring mental health disorders section.

Leptin, which is a hormone that helps prevent hunger when caloric intake is not needed, is affected in patients with eating disorders. Due to chronic starvation and therefore decreased fat mass in patients with AN, leptin regulation functions by inhibiting serotonin synthesis in the brain [7]. Therefore, increased leptin decreases serotonin, which increases hunger. Leptin levels in patients with AN are significantly lower than in those without [4]. In patients with AN-restriction type, chronic starvation leads to decreased fat mass [8]; therefore, leptin is not needed for negative reinforcement. This could explain the lower levels of leptin because it is not being used or needed, so therefore it is not being produced. Furthermore, low levels of leptin are associated with depression and other mood disorders, as leptin regulates levels of neurotransmitters [9]. This could explain the high correlation of AN and MDD (Major Depressive Disorder), as explained in the psychological section.

Epinephrine, norepinephrine, and dopaminergic genes have also been analyzed in association with eating disorders [4]. Epinephrine, also known as adre-

naline, triggers a "fight or flight" response. The "fight or flight" response is a reaction to high stress situations. Norepinephrine is the hormone that responds to stress. Dopamine is a reward hormone, which is released during situations that make people feel pleasure. Together, epinephrine, norepinephrine and dopamine make up a hormone family called catecholamines. Catecholamines are typically released during physical or emotional stress. Dopamine is implicated in patients with AN. Levels of dopamine are elevated in the brain as AN behaviors such as diet and exercise are rewarded by mesolimbic dopamine neurons. Overtime, this leads to unhealthy reliance on these behaviors, much like substance misuse and dependence, which is significant as substance misuse is correlated with eating disorders, as explained later. However, it is important to note that there are no existing comparisons between dopamine levels before and after AN, so it is possible that patients with AN had previously had elevated dopamine levels before developing the disorder [10]. Research has found that norepinephrine activity in the nervous symptoms of patients with eating disorders is decreased due to both starvation and intermittent dieting [11]. Due to high dopamine levels and reduced norepinephrine levels, it is likely that the dopamine beta hydroxylase, which converts dopamine to norepinephrine, is either at low levels or dysfunctions in patients with eating disorders.

Taken together, many hormones, especially those having to do with stress, mood, and hunger, are implicated in relation to eating disorders. Due to the physical effects of eating disorders and starvation, these hormones are either found in higher or lower levels than in controls.

### 2.3. Brain Regions Associated

Genes associated were found to be enriched in the prefrontal cortex of the brain [12]. The function of the prefrontal cortex is to set and achieve goals, to predict the consequences of one's action, to plan behaviors, and to make decisions. The prefrontal cortex receives impulses and input from different regions of the brain, and then processes the information and reacts. Furthermore, associations with eating disorders were found in striatal neurons, which react with enzymes to procedural learning and memory [13], and hippocampal neurons, which are responsible for learning, processing emotions and encoding memories [14]. Hyperactivity in the Cortico-Striatal-Thalamo-Cortical (CSTC) pathway, which connects the striatum, cortex and thalamus and controls habit formation and reward, is believed to underlie both OCD and AN [15]. Relatedly, pathway has also been thought to be involved in behaviors requiring sustained attention [16]. Those with BN are also thought to have structural and functional alterations in the CSTC pathway, causing persistence of the disorder [14]. In sum, key brain regions associated with eating disorder pathology are the prefrontal cortex, the striatum, hippocampus, and the CSTC pathway. These regions are responsible for a variety of functions, including planning, decision making, learning and memory, habit formation and reward, and sustained attention.

## 3. Psychological Factors

### 3.1. Co-Occuring Mental Health Conditions

Eating disorders are commonly comorbid with many psychiatric disorders. It has been estimated that between 55% to 95% of people with a diagnosis of an eating disorder will also meet diagnostic criteria for another mental health condition during their lifetime [17] [18]. Eating disorders are genetically correlated through Single-Nucleotide Polymorphism (SNP) to multiple mental health disorders including Obsessive Compulsive Disorder (OCD), Major Depressive Disorder (MDD), anxiety disorder, and substance use disorders [14]. Bulimic behaviors and alcohol use disorders are especially genetically correlated, with a genetic correlation of 0.33 to 0.61. Eating disorders are most strongly correlated with OCD, as those with eating disorders exhibit obsessions related to food. However, it is likely that they may also experience obsessions and compulsions unrelated to food, which warrants an OCD diagnosis [2]. Those with AN-restricting type may develop depressive behaviors, such as isolation, self harm, and suicidal ideations, due to the consequences of starvation [2]. Since eating disorders are an isolative disease, isolation is a shared trait with depression. Compared with controls, those with AN are eighteen more times likely to die by suicide, and those with BN are seven times more likely to [19].

### 3.2. Associated Personality Traits

Perfectionism, which is defined as setting exorbitant goals or standards of self-performance followed by critical self evaluations [20], has been correlated with eating disorders, especially those with AN. People with AN apply their high standards of self-performance to their restrictive eating habits in order to control their weight [21], which offers them social approval from others and the complete control of their bodies. Studies have found that females with AN had significantly higher scores on the Frost Multidimensional Perfection Scale than those without AN [22]. Furthermore, typically only those with AN experience self-oriented perfectionism, which is setting extremely high expectations for oneself and then evaluating one's own behavior, while socially prescribed perfectionism, believing that others have unrealistic expectations for them and not meeting these expectations leads to disappointment from others, is a symptom for many different eating disorders [23]. Perfectionism also increases eating disorder outcomes by interacting with other factors. For example, perfectionism can interact with body dissatisfaction to produce excessively thin body expectations [24].

Impulsivity, rash behavior without planning or consideration of the consequences, has been linked with both BN and BED. Both drive for reward and rash impulsivity are heightened in those with BED and BN. Individuals with BN are likely to behave rashly in response to negative effect. Eating releases dopamine, which gives a feeling of reward, therefore explaining heightened drive for reward in those with BED and BN [25]. This contrasts with individuals with AN re-

stricting type, as they are more prone to perfectionism and obsessive behaviors, which includes much planning [25]. Impulsivity is a shared trait with substance use disorders, explaining the high comorbidity rate between substance use disorders and BN/BED. However, comorbid pediatric patients tend to display a different type of impulsivity than those with just BN or BED, having been found to be more likely to have committed rash, unplanned acts such as behaviors consistent with diagnoses of conduct disorders and oppositional defiant disorders [25].

Neuroticism, one of the big five personality traits, is the vulnerability to experience negative emotions such as anger and anxiety. High neuroticism is associated with many mental illnesses, including eating disorders. Neuroticism can predict the development of eating disorders, and even diagnoses of eating disorders, including both AN and BN [26]. It has been suggested that genetics play a role in the link between neuroticism and disordered eating, with approximately 37% to 49% of the variance in factors leading to disordered eating being explained by genetics [27] [28].

Taken together, certain personality traits, such as perfectionism for AN, impulsivity for BN and BED, and neuroticism for all disorders, can increase vulnerability to eating disorders, as they are likely to develop behaviors and coping mechanisms which can evolve in an eating disorder.

### 3.3. Neurocognitive Processes

Cognitive flexibility is the ability to adjust attention from one task, operation or mental set to another [29]. Studies have shown that in individuals with AN, cognitive flexibility has been impaired compared to those without AN [26]. It is likely that this cognitive flexibility is a result of starvation and duration of the illness, as children and those who had AN for a shorter duration exhibited non-significant impairment compared to controls as opposed to adults with AN. Impaired cognitive flexibility results in hyperfixations due to the lack of ability to adjust content. This might explain the hyperfixation of food and body weight in patients with AN.

Inhibitory control is the behavior of suppressing an automatic motor or cognitive response to perform a less automatic one. Patients with BED and BN exhibit more inhibitory control impairment than those with AN-restricting type and control [30]. Neuroimaging shows eating disorder patients have reduced activation in frontostriatal circuits, which moderate motor and cognitive functions, explaining the inhibitory control impairment. In BED and BN, it is likely that reduced inhibitory control leads to interference with the automatic desire to not gain weight and not to eat hedonically [31].

In conclusion, those with eating disorders have impaired neurocognitive processes, such as lower cognitive flexibility and diminished inhibitory control, due to the physical and mental effects of the disease. These neurocognitive impairments, both falling under the broader category of executive function, or the cog-

nitive processes needed for goal driven behavior, can be reversed through cognitive training, such as Cognitive Behavioral Therapy (CBT) and pharmaceutically, through selective serotonin reuptake inhibitors [32] [33].

## 4. Social Factors

### 4.1. Adolescence and Social Environment

During adolescence, it is possible for caregivers to transfer eating disorder behaviors onto their children. For instance, parents often enforce rules at mealtimes, which could lead to eating disorder symptoms in the future [34]. Parents who are concerned with their own weight or the weight of their child are more likely to enforce food rules than those who do not [35]. Parents often either enforce restriction rules in hopes of keeping their child from becoming overweight or encourage them to finish their plate of food, ignoring their children's satiety cues. Both rules can often lead to weight gain, which in turn often leads to eating disorders.

Attachment styles, which are often developed during adolescence, are strongly related to eating disorders. According to Bartholemew and Horowitz's model of attachment styles, one's attachment style is based on thoughts about oneself and thoughts about others. Secure attachment, positive thoughts on both self and others, have healthy boundaries, are satisfied with intimacy and can healthily support others. Fearful, or anxious, attachment is negative thoughts about oneself but positive thoughts on others, which leads to the need for excessive attachment due to fear of abandonment. Avoidant, or dismissive, attachment is positive thoughts on oneself but negative thoughts on others, and leads to a lack of trust for others, closed off personality, and lack of intimacy. Finally, fearful-avoidant is negative thoughts on oneself and others, and fear of rejection leads to commitment issues [36]. Parenting styles often lead to the development of certain attachment styles. For example, parents who exhibit high control over their child's behavior leads to stress for the child, and can lead to fearful attachment [37]. People with insecure attachment styles (fearful, avoidant and fearful-avoidant) are more likely to have eating disorders [36]. Those with insecure attachment issues are more likely to have low self esteem and an increased feeling that others are rejecting them. This often leads them to go to extreme lengths for acceptance from others, such as adhering to society's strict body standards [37].

In conclusion, there is a clear relationship between parenting, attachment styles and the development of eating disorders. For example, modeling behaviors around meal times, such as enforcing restriction and encouragement rules, can later lead to disordered eating in the child. Certain parenting styles can also lead to more insecure attachment styles in children, therefore leading to an increased risk of eating disorder pathology.

### 4.2. Media

Higher social media use has been linked to higher body dissatisfaction, therefore

increasing eating disorders [38]. A study found that one in three teenage girls struggle with body image that is correlated with Instagram use [39] (Harringer *et al.*, 2022). Pressure to maintain a public image leads many people on social media to take photos showing off their bodies and manipulating those photos through filters, often distorting the photos to make themselves appear thinner. This leads to increased body image concerns, especially as photo-based social media apps such as Instagram and Snapchat are more linked to eating disorders than non-photo-based apps [40]. Furthermore, social media celebrities known as influencers tend to show off unrealistic body standards, which are usually edited to appear thinner. Exposure and internalization of these unrealistic body standards further perpetuates unhealthy coping mechanisms such as comparison, body dissatisfaction, and eating disorders.

Moreover, social media algorithms can lead users to be exposed to inappropriate content. Social media, such as the app TikTok, often uses personalized algorithms to generate content specific to the user's interests, which can include less monitored content such as pro-eating disorder content [39]. Social media, especially emotionally triggering content, is often addictive and algorithms are specifically built to keep users engaged with the media, leading to even greater exposure to these triggering videos over long periods of time. Although social media companies are beginning to monitor their content, emotionally triggering videos still bypass their monitoring and are seen by users, leading to eating disorders and other mental health issues [39] (Harringer *et al.*, 2022).

In summary, high use of media significantly increases risk of eating disorders by developing disordered habits due to exposure to unhealthy and unrealistic standards. Given that teenagers and young adults, who are at heightened risk of developing an eating disorder, spend a considerable amount of time on social media. Taken together, this represents a high risk environment for teenagers and young adults.

### 4.3. Cultural Differences

Eating disorders were assumed to be bound to "Western culture", which includes Europe, the United States, Australia and other countries settled by Europeans; however, research has shown eating disorders are found in the Middle East, Africa, Asia, and South America at similar or higher rates than Western countries [41]. Eating disorder rates are particularly high in East Asia, with Japan having the highest percentage of abnormal eating attitudes in the world at around 35% [41]. This is likely due to the slim, thin body standard perpetuated in East Asia. Political changes in Eastern Europe resulted in an increase in eating disorders. As society shifted from Communist to free market economies, women were given more liberties, but on the other hand, the removal of strict collective social structures removed previously held protection in terms of education, employment and childcare rights, all of which led to increased eating disorders [41]. One explanation for the spread of eating disorders is the "westernization" of

non-Western countries due to globalization [42]. As the two regions become more linked, it is theorized that societies will become more similar to Western societies and their values, increasing ED specific behaviors in these non-Western countries [43]. Another explanation for an increase in eating disorders globally is absence of data to confirm their existence in non-Western countries [41]). Overall, eating disorders are a global issue and are not currently confined to "Western culture".

### 4.4. Demographic Factors

Historically, eating disorders, especially AN, have been stereotyped as occurring mostly in white, upper-class, adolescent females; however, eating disorders occur in males, other ethnicities, middle age and older people, and those of lower socioeconomic status [42]. Although AN and BN are more prevalent in women, they are often left undiagnosed and untreated in men [43]. Due to the stigma of eating disorders being a "female disorder", it may cause men with eating disorders to overlook their symptoms and be hesitant to get treatment. BED is more common in men [42]. Furthermore, an increase in prevalence rates was reported in minority groups in the United States, such as African Americans, Hispanic, and Native American populations [41]. Immigrants are a vulnerable group for eating disorders, as they face pressure to fit into their host society, have urges to correct the negative stereotypes, and have high achievement orientation [41]. Acculturative stress, the stress when adapting to a new culture, has been linked to eating disorders [42]. Conversely to what researchers previously believed, eating disorders exist in all socioeconomic levels, as opposed to primarily upper class. In fact, studies have shown that BN and purging behaviors are more prevalent in those of lower socioeconomic status [44]. Overall, eating disorders occur in all genders, ethnicities, and socioeconomic levels.

## 5. Discussion

Overall, a multitude of factors contribute to the development of an eating disorder. Biological factors, such as family history and genetics, abnormal levels of hormones and abnormal activity in certain brain regions, can increase vulnerability to eating disorders. Psychological factors, such as certain personality traits, abnormality in neurocognitive processes and the presence of other mental health conditions, can also increase risk of eating disorders. Finally, exposure to social media, culture and demographics, such as ethnicity, socioeconomic status, gender and age, may affect risk of development. These factors often interact with one another to lead to eating disorders. Genetic predisposition to an eating disorder and environmental factors build on one another to cause the disorder. For example, someone with a genetic predisposition to an eating disorder has a higher chance of developing one than someone who does not have a genetic predisposition that is in the same environment. Furthermore, if two people who have a genetic predisposition are in different environments, the one in the environment

with more access to behaviors and media is more likely to develop the disorder. In conclusion, eating disorders are complex diseases caused by a variety of factors, and are not a choice, as many believe.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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