ABSTRACT

The Genetic, Environmental, and Sociocultural Factors in Eating Disorder Relapse, and Opportunities for Prevention

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Eating disorders rank among the top 10 causes of disability among women, thereby emphasizing the profound impacts of bulimia and anorexia nervosa on both the female population and society as a whole. Their etiology shows support for a combination of genetic, environmental, and sociocultural factors in the development and maintenance of the mental disorder(s). Fewer studies have explored, however, the circumstances under which one might be prone to relapse, as opposed to a full recovery post-treatment. In this literature review, the nature versus nurture debate highlights some of the hereditary and acquired risk factors for eating disorders (Mazzeo et al., 2009), as well as their impact on levels of severity (Polivy & Herman, 2002). Treatment techniques such as stepped-care models of intervention, cognitive-behavioral therapy (CBT), and family-based therapy (FBT) vary by type of eating disorder (Treasure et al., 2021); however, research pertaining to the extent of their promise and long-term effectiveness must be further investigated. Findings reveal that 20% to 50% of those with eating disorders will suffer from relapse (Keel et al., 2005), but predictors of this must continue to be explored. Risk factors leading to relapse seem to point toward 1) the extent of body image disturbance as well as 2) overall worse psychological function, but such findings require further support.

Keywords: eating disorders, anorexia nervosa, bulimia nervosa, relapse, recovery

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THE GENETIC, ENVIRONMENTAL, AND SOCIOCULTURAL FACTORS IN EATING DISORDER RELAPSE, AND OPPORTUNITIES FOR PREVENTION

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DEDICATION

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CHAPTER ONE

Introduction

Eating disorders have become increasingly more prevalent across societies over the course of the last several decades. Only in the 1960s did scientists in western societies characterize anorexia nervosa (AN), a presently well-known eating disorder subtype, as an obscure and rare disorder characterized by starvation and extremely low weight. Despite significant research progress and definitional clarity provided by the Diagnostic and Statistical Manual (DSM) of mental disorders over the years, there is still undoubtedly gray area concerning the diagnostic criteria surrounding these disorders, which poses questions as to whether or not an individual should fall into a certain category. This chapter will explore the current definitions and qualifiers for eating disorders, as well as challenges that rise from gaps in the DSM-5. In addition, it will examine the risk factors in the development of each of these eating disorders, as it is imperative to differentiate between subtypes. Due to the availability of prior research and for the purposes of this paper, the following findings will focus primarily on anorexia nervosa (AN) and bulimia nervosa (BN).

Overview of Eating Disorders

Anorexia nervosa and bulimia nervosa are two of the most prominent eating disorder subtypes which are both characterized by abnormal eating behaviors, as well as distorted attitudes and perceptions surrounding weight and body shape (Kaye et al., 2000). Each of these disorders are distinct in their symptomatology and vary greatly

across individuals. Victims of AN are divided into one of two diagnostic subgroups: 1) in the restricting subtype, abnormal (low) body weight and a persistent malnourished state are maintained due to food avoidance; 2) in the binge eating/purging subtype, this weight loss and malnutrition also occur, but is accompanied by episodes of binge eating and compensatory action (i.e., self-induced vomiting or laxative use) (Kaye et al., 2000). BN victims, on the other hand, maintain a normal body weight while engaging in repetitive episodes of binge-eating, compensatory self-induced vomiting, laxative abuse, and/or extreme exercise (Kaye et al., 2000). Bulimic individuals, too, are split into two distinct subtypes: purging type (e.g., self-induced vomiting, laxatives, and enemas) and non-purging type (e.g., fasting and exercise) (Kaye et al., 2000). The key difference between these two mental disorders is whether or not the individual is at a disturbingly low weight (15% below) based on their height and age (and thus would qualify for AN), or at a normal weight despite their unhealthy behaviors (and thus would qualify for BN). The aforementioned symptoms, however, are far too broad in theory and vary greatly by individual case in terms of magnitude and intensity. Although the disorders are differentiable, the problem lies in whether or not a person meets the required criteria to qualify for a medical diagnosis.

The DSM-5 has evidently made great progress in identifying and diagnosing these disorders in comparison to its prior editions. Substantial changes from the DSM-4 include the distinct clinical entity of binge eating disorder, criteria for AN and BN and the split of eating disorder not otherwise specified (EDNOS) into two distinct categories: 1) other specified feeding or eating disorder (OSFED) and 2) unspecified feeding or eating disorder (UFED) (Mancuso et al., 2015). This substantial change allowed for individuals

who met similar eating disorder criteria (that did not fall under one of the other 3 subtypes) to be grouped together, and segregated them from those who might exhibit more unique symptoms. Altered criteria for anorexia now include a drop in the prior amenorrhea criterion, as well as a marginal increase in the weight threshold; similarly, the frequency threshold has been lowered for binge eating and compensatory behaviors with regard to bulimia (Mancuso et al., 2015). These adjustments have allowed for individuals who may display "milder" forms of the illness to be recognized with a formal diagnosis. Such alterations from the previous text have unsurprisingly generated significant changes with regard to specific ED prevalence rates; most notably, OSFED/USFED diagnoses decreased (53% v. 25%) and anorexia nervosa diagnoses increased (8% v. 29%) (Mancuso et al., 2015); similar results are stated to be mirrored in other findings as well. Hence, those with previously "borderline" AN/BN may be given a more official subtype diagnosis, as opposed to being grouped into a larger population of OSFED/USFED.

Risk Factors in the Development of Eating Disorders

Risk factors surrounding the initial onset of anorexia and bulimia unsurprisingly surround the widely-known nature-nurture debate, with previous studies supporting a multitude of genetic and environmental factors that collectively may play key roles in eating disorder development. Family and twin studies provide the bulk of support for hereditary implications in both eating disorder subtypes. Although family and twin studies comprise the majority of studies that help to display this inherited potential, molecular genetic studies have been applied in the form of association studies and linkage studies (Striegel-Moore & Bulik, 2007). Association studies are conducted when

the pathology of a trait points to specific gene candidates, whereas linkage analysis uses genetic markers to identify chromosomal regions that may contain genes influencing the trait of interest (Striegel-Moore & Bulik, 2007). A variety of different studies display the numerous environmental conditions that might provoke or inhibit eating disorder tendencies. In-depth analyses of potential genetic and environmental contributions in the manifestation of AN and BN are further discussed below. It is important to note that the extent to which heredity and life experiences play a role can vary depending on the eating disorder subtype in question.

As previously stated, family and twin studies are the most effective way to assess the extent of genetic implications for eating disorders; however, such evidence is limited due to the inevitable entanglement of biological and environmental factors that occur within the family. High heritability coefficients have consistently been claimed across multiple studies for both AN and BN, with 50-83% of the variance being genetic (Polivy & Herman, 2002). More recent findings, however, suggest more exact estimates of heritability; BN ranges from 28-83%, whereas AN ranges from 56-76% (Peterson et al., 2016). Although the heritability range for anorexia is lower than that of bulimia, it suggests a more significant genetic component, with the low end of genetic factors hovering above 50%. When controlling for other factors, the role for the shared environment was found to be negligible, with the bulk of factors from the unique (unshared) environment coming in second place (Striegel-Moore & Bulik, 2007). Such unshared environmental factors may include (but are not limited to) peer influences, birth order, and parent-child relationships. One particular problematic finding in relation to its heredity surrounds the fact that the eating disorder, along with its associated attitudes

(concerns, fears, preoccupations), are both heritable (Polivy & Herman, 2002); therefore, it is difficult to distinguish whether the inherited attitudes caused the disorder, or vice versa.

Further genetic studies display the extent of neurological influences in the manifestation of anorexia nervosa. Genes in the serotonergic system (i.e., serotonin receptor 2A, 2C, serotonin transporter gene) and dopaminergic system (i.e., D3 receptor, D4 receptor, dopamine transporter), which both play a profound role in mood and appetite, are believed to be central to the etiology of eating disorders (Striegel-Moore & Bulik, 2007). The elevated activity in 5-Hydroxytryptamine (HT) (otherwise known as serotonin, a neurotransmitter which helps to stabilize one's mood) is consistent with AN patients' generally obsessive-compulsive life approach. Even after recovery from their ED; the disorder's response to fluoxetine (similar to OCD) leads some to speculate that anorexia may simply be a variant of the latter disorder (Polivy & Herman, 2002). Striegel-Moore & Bulik (2007) also state that previously conducted linkage studies for AN yield statistically significant results, particularly in examining the classic AN restricting subtype; restricting a heterogenous sample to relative pairs yielded evidence for the presence of a susceptibility locus on chromosome 1, with areas of interest also isolated on regions 2 and 13. These researchers further cite evidence that two genes in particular, serotonin 1D (HTR1D) and delta opioid (OPRD) receptor, exhibit significant associations with anorexia nervosa. The question simply lies in the extent to which these genetic susceptibilities play a role in triggering the onset of the eating disorder itself. Surely an individual can have a genetic predisposition for an eating disorder, but these

genes may be activated or never manifest themselves depending on the person's environment.

With regard to bulimia, there is evidence of a genetic component in self-induced vomiting (SIV). In the context of a BN diagnosis, SIV is the symptom most strongly influenced by genetics, and its inclusion in molecular genetic linkage studies have allowed researchers to detect significant genetic signals more easily (Peterson et al., 2016). In a twin study conducted by Peterson et al., the researchers used a causal-contingent-common pathway model to assess the extent of genetic and environmental factors in SIV behaviors. Results indicate that genetic factors are substantial in both SIV initiation and progression, with heritability estimated at 61% and 51%, respectively. That is, over half of those who are (recorded) victims of self-induced vomiting are more prone solely due to their genetic makeup. 100% of the genetic factors influencing the progression of self-induced vomiting were shared with its initiation; no new factors emerged between the two.

Though heritability rates vary more in bulimia nervosa as compared to anorexia nervosa, several biological factors are also implicated in this disorder (hence alluding to a strong genetic component). Association studies cited by Striegel-Moore & Bulik (2007) show that genes implicated in BN closely parallel those of AN, with researchers examining factors associated with deficits in the serotonin transporter (5-HTTPLR). This stands in stark contrast to the aforementioned AN findings by Polivy and Herman, which instead found heightened 5-HT levels in anorexic individuals. Recovered BNs continue to show persistent abnormalities in serotonin function, suggesting that such factors underly not only the progression, but the onset, of bulimia (Polivy & Herman, 2002). Striegel-

Moore & Bulik (2007) also cite only one linkage study, which displayed significant linkage on chromosome 10p13, along with suggestive evidence for linkage on 10p14 and 14q22-23. A second analysis refined the initial study, and narrowed the linkage peak on chromosome 10p13.

An inherent flaw in isolating genetic factors, as mentioned above, is the profound entanglement of genetics and environment; therefore, most studies focus on G-E correlations (i.e., genetic differences in exposure to particular environments). 3 particular types of G-E correlations have been previously described in literature: passive, evocative, and active. Passive gene-environment correlations occur when non-adoptive children receive genes from the same individuals who create their family environment (such as parents and siblings), and whose parents may also model eating disorder behaviors and attitudes in the household (Mazzeo & Bulik, 2009). Thus, these children may be receiving a "double-dose" or eating disorder risk that stems from both genetic and environmental exposures. Such examples of this include, but are not limited to, parental role modeling of eating disorders, problematic feeding behaviors in the household, and a parental over-emphasis on their child's weight and shape. Evocative G-E correlations occur in situations where an individual with a genetic predisposition may seek out appearance-related comments by parents and peers, or "evoke" the disorder. The positive or negative comments that the individual receives in turn reinforces their tendency to over-value appearance, therefore promoting both the initiation and maintenance of disordered behavior (Mazzeo & Bulik, 2009). Teasing and weight-related criticisms, therefore, may play a key role in this type of correlation. Lastly, active G-E correlations occur when those with a genetic vulnerability seek out an environment that reinforce a

strong emphasis on appearance, such as modeling (Mazzeo & Bulik, 2009). Media and peer group selection is therefore imperative when considering this gene-environment correlation. With these interplays in mind, purely genetic research for eating disorders truly lies in infancy.

A vast array of environmental factors, most notably the family environment, play a critical role in the development of eating disorders, regardless of their subtype. Differences in individual environmental factors may contribute to which subtype is manifested later on. With regard to AN individuals and normal controls, several studies show elevated family problems (Schmidt et al., 1997). Factors correlated with the restrictive subtype include the parents' tendency to give a double message of nurturant affection combined with neglect of their child's need to express themselves, more deaths in first-degree relatives, and high maternal overprotectiveness (Schmidt et al., 1997). Purging-subtype anorexics, on the other hand, tend to rate their families as more isolated, more contradictory in their communications, and less involved, supportive, and nurturing (Schmidt et al., 1997). This contrast is intriguing, for although both populations controlled for AN individuals, the difference in restricting and purging subtypes were astounding, and surprisingly opposite. The restrictive subtype reported an almost unhealthy level of nurturing and attention, whereas the purging subtype tended to experience neglect. These familial factors certainly are not necessary; there are just as many AN individuals whose symptoms are not manifested as a manipulative tool in response to their home environment (Polivy & Herman, 2002). In bulimia nervosa, family problems as a whole seem to be more profound than in individuals with AN. Bulimics may generally report poorer general family functioning, with higher conflict

and less cohesion, empathy, expressiveness, and independence (Schmidt et al., 1997). This environment heavily resembles that of the purging subtype of anorexia, in which families tend to neglect, rather than nurture, the child.

Hypothesis

The purpose of this paper is to examine the risk factors in eating disorder relapse and potentially chronic eating disorders, rather than simply their initial onset. To do so, etiological and risk factors for their initial onset must first be examined in order to better grasp what might contribute to poorer outcomes later on. In the following chapters, I will touch on the implications of relapse, eating disorder treatments, and comparisons between long-term full recovery and relapse patients. Such discussions will aim to pinpoint the multitude of factors that can result in overall poorer outcomes of both bulimics and anorexics. I will argue that the factors which play into AN and BN individuals displaying poorer outcomes (including relapse and chronic illness) rests not in their environment, but rather primarily in their genetics and personality; these patients likely have a predisposition to engage in unhealthy behavior, as opposed to simply being heavily influenced by their surroundings.

CHAPTER TWO

Outline of Current Potential Treatments

The role of adequate treatment for eating disorders is critical, if not absolutely essential, for starting and maintaining the grueling process of recovery. Forms of treatment vary by eating disorder subtype (in the context of this paper, AN and BN), and several studies stress that some types of treatment may prove to be more beneficial than others. However, it is important to note that both the eating disorder manifestations, as well as the process of recovery, vary highly by individual; thus, any studies that generalize such findings should be interpreted with caution. This chapter will aim to delve into depth on the most commonly utilized forms of eating disorder treatment across anorexia and bulimia nervosa. Previously explored methods include, but are not limited to, family-based therapy (FBT), cognitive behavioral therapy (CBT), and psychopharmacotherapy. Additionally, it will seek to examine the elements of eating disorder treatment that are believed to be "necessary ingredients" in the recovery process. The aforementioned elements will provide leeway into Chapter 3, as the role of treatment itself can play a potential role in the risk of relapse.

Although there is no universally accepted AN treatment due to the inevitable individuality of the disorder, one of the most popular approaches is the Maudsley method, otherwise termed the Maudsley Model of Treatment for Adults with Anorexia Nervosa (MANTRA). This technique aims to target maintaining factors related to thinking styles, including perfectionism, OCD personality traits, emotion avoidance, and pro-anorectic beliefs (Wade et al., 2011). Through focusing on the adjustments that need

to be made both physically and cognitively, it succeeds in creating a more individualized approach to recovery by targeting the patient's own bad habits and tendencies. In doing so, it breaks free of the stereotypes that have the potential to tarnish several alternative forms of treatment. Throughout AN treatment for adults, the Maudsley model consists of assessment sessions, treatment history, and discussion regarding the desire and willingness to change. In the context Wade et al.'s 2011 study, therapy is delivered in 25 one-hour sessions over a 10-month period, consisting of 20 weekly sessions followed up with 5 monthly sessions. The time frame and amount of therapy administered may vary by those giving the treatment, as well as individual circumstances.

The Maudsley method has been adapted and now serves as the primary treatment of adolescent anorexia nervosa, and serves as a more intensive, family-based treatment (FBT) in comparison to its adult-based counterpart. The approach consists of three phases; 1) mobilizing parents to take the lead on weight restoration, 2) transitioning food control back over to the individual at an age appropriate level, and 3) subsequently focusing on other issues pertaining to adolescent development and potentially triggering eating disorder behavior (Treasure et al., 2021). Feedback from both the caregivers and anorexic victims themselves support the theory that family involvement is beneficial in eating disorder recovery (Treasure et al., 2021). However, in taking these things to mind, it is important to note that the family environment varies highly by individual; thus, environmental factors should be heavily considered in deciding which treatment method would be most effective. That is, those who have unhealthy relationships with family members may perceive the Maudsley method as detrimental, rather than beneficial, to their eating disorder recovery.

Another form of treatment used across both AN and BN individuals is cognitive behavioral therapy (CBT-ED). This technique is primarily used in the context of bulimia nervosa, but can occasionally be applied across anorexic individuals. Traditionally, CBT was designed to be delivered in an outpatient setting, consisting of 20 sessions delivered over 5 months (Atwood & Friedman, 2020). Proponents of this method developed a transdiagnostic theory which acknowledges that there are maintaining processes across all eating disorders, such as overemphasizing weight, shape, and dietary restriction, as well as maintaining processes specific to one or more disorders (e.g., binging and purging in BN) (Atwood & Friedman, 2020). Additionally, this theory includes internal conflicts such as perfectionism, low self-esteem, interpersonal difficulties, and mood intolerance in combination with other external factors that can together create obstacles and impede recovery (Atwood & Friedman, 2020). CBT emphasizes the role of food as a source of intrapsychic conflict for eating disorder victims, and primarily seeks to heal the tense relationship through therapeutic means (Thoma et al., 2015). In recent years, two advanced versions of CBT-ED have been developed; the first is a "focused" version of CBT (CBT-Ef), which addresses focuses on the core eating disorder pathology and mood intolerance. The second is a more "broad" therapy (CBT-Eb), which addresses one or more of the eating disorder maintaining processes and ties them into the individual's conceptualization (Atwood & Friedman, 2020). Thus, interventions that employ cognitive-behavioral therapy for both AN and BN emphasize improving general functioning through managing food intake, reducing purges, and addressing distorted cognitions pertaining to weight and body shape (Thoma et al., 2015). However, while the therapy outcomes show promise for reducing acute eating disorder symptoms, the results

are less favorable in the long-term. Nevertheless, study findings show overall more hopeful outcomes for bulimic individuals (in comparison to those with anorexia), and suggest that CBT may serve as an equivalent alternative when FBT is not as promising (Craig et al., 2019).

Another attempted path to recovery that falls under both aforementioned subcategories of eating disorders is pharmacotherapy. With regard to anorexia, although many medications have been considered, the majority have been rendered either disappointing or outright useless. Unfortunately, pharmacotherapy does not traditionally serve as a stand-alone treatment for AN; rather, it is used in conjunction with other treatment plans when initial responsiveness rates fall through (Davis & Attia, 2017). Due to the disorder's substantial overlap with psychiatric disorders such as depression and anxiety, antidepressants were assumed to be a promising option; however, medications such as fluoxetine have neither enhanced nor maintained significant weight gain in anorexic individuals (Davis & Attia, 2017). However, second-generation antipsychotics (SGAs) have shown more promise in reducing intense anxiety; more specifically, olanzapine has been reported to modestly improve weight gain, as well as improvement in obsessionality scores (Davis & Attia, 2017). In contrast, pharmacotherapy options for bulimia nervosa show significantly more promise than its anorexia counterpart. The antidepressant fluoxetine, which demonstrated no improvement in anorexic individuals, has essentially the opposite on bulimics; this medication has shown to be effective in reducing binge-purge episodes by over 50% (Davis & Attia, 2017). Similarly, other medications such as topiramate (an anti-epileptic) show promise in reducing binge-purge episodes, as well as improved psychological measures (Davis & Attia, 2017). In

considering the above medications, it is important not to treat these as stand-alone treatments; while they may provide promising outcomes, particularly with regard to BN, they are best used in conjunction with additional recovery techniques.

Although these treatments seem particularly effective in delaying and/or hindering the portrayal of eating disorder behavior, such techniques primarily provide short-term benefit for acute symptoms. All three of the aforementioned approaches have relatively high relapse rates that seem to occur shortly after treatment has halted; this alludes to the theory that perhaps such behaviors are only inhibited while actively participating in the recovery treatment. The long-term implications for FBT, CBT, and pharmacotherapy hardly show substantial promise in the years following.

CHAPTER THREE

Contributors of Relapse

While some treatments undoubtedly prove to be successful in the long-term recovery of anorexics and bulimics, others are unfortunately susceptible to relapse. In adults, relapse rates are estimated to be between 22% and 63% (McFarlane et al., 2008); the high variability of these numbers is likely partly due to the subtype of eating disorder, methodological differences, and variability in definition with regard to "relapse" and "remission." Due to the fact that ED treatment is uniform across many individuals, it raises the question of why some fall back into their disordered patterns while others do not. This chapter give rise to the potential contributors of relapse, including genetic, environmental, and sociocultural implications, and will hopefully put forth findings that will help to solve this mystery.

Psychological and Genetic Implications

A study conducted by Olmsted et al. (1994) sought to provide a relapse estimate in bulimic patients and identify indexes of functioning before and after treatment that might predict a subsequent regression. Participants were assessed before and after the study; these individuals consisted of 48 female patients ranging from ages 18-57 who met DSM-3 criteria for bulimia nervosa, did not have concurrent diagnosed anorexia nervosa, and received their treatment two years prior at a full-time, day hospital program for eating disorders at The Toronto Hospital. This participation criteria allowed for wide age variability, elimination of those who might have comorbid eating disorders, and treatment

standardization. However, due to the older nature of the study and the utilization of DSM-3 criteria, its results should be interpreted with caution; most individuals during this time period who met eating disorder criteria had more "extreme" symptomatology, as the DSM-3 did not adopt the same criteria flexibility as the DSM-5. In the scope of this study, the term "remission" encompassed both full and partial remission, and was defined as a maximum of one binge eating/purging episode during the last 4 weeks of treatment; "relapse" was defined as a change from full or partial remission to full bulimia nervosa (binging and/or vomiting at least twice weekly for three months).

With these criteria in mind, 31.3% (15) of the participants relapsed within the two follow-up period, two-thirds of which did poorly over the entire study period and would have met the DSM-3 criteria for bulimia nervosa at least 50% of the time (Olmstead et al., 1994). With respect to demographic variables, age was the only significant predictor of relapse and accounted for 8% of relapse variance; concerning pretreatment variables, vomiting frequency and the score on the bulimia subscale of the 26-item Eating Attitudes Test were the only significant predictors. Regarding posttreatment variables, vomiting frequency and the score on the interpersonal distrust subscale of the Eating Disorder Inventory were the only two to approach statistical significance. Surprisingly, difference in measures related to self-esteem, depression, and social adjustment did not meet statistical significance. Additionally, the amount of treatment received during the two-year follow up was not significantly correlated with relapse status (Olmstead et al., 1994). Rather, the three notable factors with regard to relapse surrounded vomiting frequency, age, and pre and post-treatment scores on the Eating Attitudes Test and Eating Disorder Inventory, respectively.

While most research surrounding eating disorder course and recovery follows clinical populations after treatment discharge, a study carried out by Zerwas et al. (2013) examined the anorexia nervosa recovery in a sample of 680 female participants in a multi-site genetic study. Participants were to meet the DSM-4 criteria for anorexia nervosa, a weight less than the 5th percentile of BMI, onset before age 25, weight controlled by restricting and/or purging, age between 13-65, and a diagnosis at least 3 years prior to entering the study. The researchers aimed to examine the association between prognostic factors (i.e., eating disorder features, personality traits, and psychiatric comorbidity) and the likelihood of recovery; they hypothesized an earlier age of onset and higher novelty seeking would predict a higher likelihood of recovery. "Recovery" in this study was deemed the offset of AN symptoms (e.g., low weight, dieting, compensatory behaviors) for at least one year (Zerwas et al., 2013). The Structured Interview for Anorexia Nervosa and Bulimic Disorders (SIAB), Temperament and Character Inventory (TCI-9), and Structured Clinical Interview for DSM-4 Personality Disorders (SCID-II) were administered to participants in order to measure compensatory behaviors, personality, and comorbidity, respectively.

Results showed that only 18.1% of participants met the study criterion for recovery (Zerwas et al., 2013). Impulsivity was discovered to be a significant factor in recovery; at anorexia nervosa onset, participants had a higher probability of recovery when endorsing more impulsivity. Other significant predictors of recovery included vomiting and trait anxiety (both displayed negative associations; in other words, the higher the likelihood of these two factors, the lower the likelihood of recovery). Additionally, avoidant personality disorder was a negative prognostic factor, but was not

shown to be significant in the final model (Zerwas et al., 2013). Strangely, factors such as age, laxative abuse, fasting, exercise, reward dependence, persistence, comorbid OCD, MDD, alcohol or drug abuse/dependence, PTSD, and borderline personality disorder were not significant predictors. The reason for vomiting frequency is unclear, but may be due to heightened psychological disturbance (Zerwas et al., 2013).

With respect to anorexia nervosa, a study set forth by Carter et al. (2012) similarly suggests that perhaps a heightened innate disturbance may be a significant factor contributing to eating disorder relapse. In the study, 100 patients meeting the DSM-IV criteria for AN were admitted to a hybrid inpatient/day treatment eating disorder program which focused on restoring weight, reducing binging and purging symptoms, and group psychotherapy. The participants were comprised of 95% females, 94% Caucasians, had a mean age of 25.4 years old, an average BMI of 15.1, and a median illness duration of 6.3 years (Carter et al., 2012). Additionally, a majority of the patients (67%) had AN-R (restricting subtype), while the remaining 33% had AN-BP (binging-purging) subtype of anorexia. “Relapse” in this study was defined as a BMI of less than or equal to 17.5 for 3 consecutive months of at least one episode of binging-purging behavior per week for 3 consecutive months during the 1-year follow-up period (Carter et al., 2012). 3 groups of variables were examined to measure relapse: 1) intake variables (e.g., BMI at admission to the program, AN subtype, history of self-harm, motivation score), 2) treatment process variables (e.g., the rate at which one gained weight throughout the program, change in motivation score), and 3) discharge variables (e.g., BMI at program discharge, motivation score).

Using the Kaplan-Meier survival analysis, results showed that the highest risk of relapse fell between 4 to 9 months after treatment discharge (Carter et al., 2012). Additionally, only 59% of participants did not meet relapse criteria during the 1-year follow-up, whereas the other 41% did; 28% of those who met the criteria for relapse fell at or below a 17.5 BMI for 3 consecutive months, and 20% reported at least 1 binge-purge episode per week for 3 consecutive months. When conducting multivariate Cox regressions, the eating disorder subtype was found to be a significant predictor of relapse; only 30% of patients with the restrictive anorexia subtype met relapse criteria after 12 months compared to 70% of those within the binging-purging subtype. Decreases in level of motivation throughout treatment were also found to be a significant predictor of relapse in the one-year follow-up (Carter et al., 2012). A third predictor of relapse was the high pre-treatment severity on the Padua Inventory, which measured obsessive-compulsive disorder symptoms. These findings suggest that certain individuals are psychologically more vulnerable to relapsing, perhaps due to diminished emotion regulation skills.

Similarly, an analysis by Keel et al. (2005) implies that post-remission predictors of relapse in both anorexia and bulimia nervosa may be correlated with psychological, rather than environmental, factors. The researchers utilized a longitudinal design and conducted interviews every 6-12 months for approximately 9 years following the initiation of the study in 1987. Four predictors were examined: 1) body image disturbance, 2) other axis I disorders, 3) treatment, and 4) psychosocial function. The MacArthur guidelines were used to provide a standard definition of remission and relapse: remission was defined as having minimal or no symptoms of one's eating

disorder for 8 consecutive weeks, and relapse was described as a return to full disorder criteria after a period of remission (Keel et al., 2005). There were a total of 246 women participating at the start of the study, with a relatively even split between DSM-IV criteria for anorexia nervosa at intake (55%) and bulimia nervosa (45%). There was a 7% attrition throughout the course of the study, with 229 participants remaining at the end of the last follow-up. Researchers utilized and modified the Schedule for Affective Disorders and Schizophrenia—Lifetime Version to assess psychiatric history at baseline, and used the Longitudinal Interval Follow-Up Evaluation throughout follow-up assessments (Keel et al., 2005). Use of individual outpatient psychotherapy, clinician-led group psychotherapy (inpatient and outpatient) fluoxetine treatment, and inpatient treatment were additionally measured on a weekly basis.

Across AN and BN, approximately one-third of the women who recovered eventually relapsed. Interestingly, those who recovered from AN frequently relapsed into BN, whereas those who recovered from BN did not relapse into AN (Keel et al., 2005). With regard to the women who met diagnostic criteria for anorexia, only a minority achieved remission over the course of observation (N = 42, 31%), in comparison to a majority of women with bulimia nervosa (N = 83, 75%) (Keel et al., 2005). Predictors of relapse across both disorders included cognitive features of AN and BN, including body image disturbance. However, due to the normative discontent surrounding body image across the female population as a whole, it is difficult to discern whether this is a valid association. Perhaps focused work on body image may allow AN and BN patients to achieve lasting recovery, but data surrounding ongoing therapy in this study did not reveal the content of that treatment (Keel et al., 2005). Worse psychosocial function

predicted relapse in both anorexia and bulimia, with it posing a significant factor for BN relapse. With such drastic differences in coping responses between anorexic and bulimic individuals, those who suffer from bulimia nervosa may be less equipped to cope with stressors due to their tendency to be impulsive, thereby leading them to relapse.

While various eating disorder relapse studies account for psychological abnormalities that may contribute to a greater likelihood of maladaptive behavior following recovery, there are few genetic studies pointing toward specific genes that could prompt this heightened risk. However, a 2011 study by Bloss et al. suggested that GABA receptor SNPs (single-nucleotide polymorphisms) may be correlated with eating disorder recovery. 1,878 female participants (1,201 cases and 677 controls) were selected from the Anorexia Nervosa Affected Relative Pair Study, the Bulimia Nervosa Affected Relative Pair Study, and the Anorexia Nervosa Trios Study, and inclusion criteria was in part based on whether individuals had 1) restrictive AN, 2) AN with purging but no binging, 3) AN with binging, 4) a lifetime history of both AN and BN, and 5) subthreshold BN (Bloss et al., 2011). Due to the fact that this study is relatively dated (i.e., 11 years old), it is important to note that the diagnostic criteria for these disorders are now different; hence, the inclusion criteria may vary from how it once did in this study. Recovery was defined as having no eating disorder symptoms (based off of DSM-IV criteria) for at least one year. The researchers created 3 cohorts: 1) the "discovery cohort" (> 25 years old; lifetime diagnosis of AN/BN/EDNOS; data available for the presence or absence of ED symptoms); 2) the "replication cohort" (who met the same criteria for the discovery cohort, but were < 25 years old); and 3) the "follow-up cohort" (consisted of those whose recovery status was unknown, as well as the control group

individuals). By breaking down the large sample (n = 1,878) into 3 groups, the authors were able to control for age, as well as recovery status, in their research analysis.

The researchers proceeded to target traits in their phenomic association analysis, which were largely selected based on past linkage studies; these included 1) age at menarche, 2) anxiety, and 3) perfectionism. Anxiety and perfectionism were measured by the Trait Anxiety Scale of the Spielberger State-Trait Anxiety Inventory Form Y and the Multidimensional Perfectionism Scale (MPS), both of which demonstrated adequate reliability and validity (Hewit et al., 1991; Vitasari et al., 2011). Candidate genes were based both on group consensus and findings from previous literature studies, which may pose a hindrance in the event that there are genes implicated that were not highlighted in previous studies. The list was further narrowed through selecting genes with 1) evidence of brain gene expression, 2) genes with estrogen responsiveness in microarray studies, and 3) group consensus to match the genotyping budget (Bloss et al., 2011). While this approach certainly benefitted participants within the context of this study, as they were all female, it is difficult to generalize the results to both genders. In total, 182 candidate genes and 5,151 single-nucleotide polymorphisms were selected.

Results of the genetic association analyses showed that, consistent with previous findings, ill individuals show a lower current BMI, higher trait anxiety, and higher concern over mistakes (i.e., higher aspects of perfectionism) (Bloss et al., 2011). While there were 25 statistically significant SNPs found in the analysis, intronic SNP rs17536211 (located in *GABRGI* on chromosome 4) had the strongest statistical evidence of association, with a p-value of 4.63×10^{-6}. The odds ratio (i.e., the measure of association between exposure and an outcome) of this GABA receptor SNP was 0.46; in

other words, possessing copies of the minor allele would be protective from long-term chronic ED illness (vs. if OR was > 1, it would increase the occurrence of long-term illness). Hence, it can be reasonably inferred that a lack of this minor allele may increase the risk of a long-term chronic eating disorder. In addition to this finding, 10 of the 25 statistically significant SNPs were involved in GABA genes; moving forward, it would behoove researchers to further examine the role of GABA in the onset, recovery, and relapse of eating disorders. The researchers proceeded to conduct phenomic association analyses of SNP rs17536211 with the 3 targeted traits (age at menarche, anxiety, perfectionism); a nominally significant association with trait anxiety was found (p = 0.049) was found, but the other 2 traits were not found to be correlated with the SNP (Bloss et al., 2011). Due to the very marginal statistically significant value of p (i.e., it is on the borderline of not being significant), the extent to which the SNP and trait anxiety are correlated should be interpreted with caution. Nevertheless, this study may prompt new research in the years following with regard to GABA SNPs, their associations with trait anxiety, and how those relationships may influence eating disorder outcomes.

Environmental Implications

This is not to say that environmental factors do not also play a partial role in eating disorder relapse, however. A 6-year study conducted by Grilo et al. (2012) suggested that stressful life events (SLE) could potentially trigger relapse for remitted bulimia nervosa, as well as eating disorders not specified (EDNOS). The NIMH-funded naturalistic study enrolled 117 female participants, 35 of which met the diagnostic criteria for bulimia nervosa and achieved remission. With respect to all of the participants, there was a mean age of 31.2 (SD = 8.1) years, 81% were Caucasian (N = 95), and 80% (N =

94) received some form of college education (Grilo et al., 2012). Additionally, there was a mean of 1.96 (SD = 1.4) additional lifetime Axis I psychiatric disorders, suggesting high comorbidity with other mental illnesses (as supported by other studies). Baseline diagnoses were established through using the Structured Clinical Interview for DSM-IV Axis I Disorders-Patient Version (SCID-I/P), as well as the Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV), to determine Axis I psychiatric disorders and PD diagnoses respectively (Grilo et al., 2012). In the years following, the Longitudinal Interval Follow-up Evaluation (LIFE) was utilized to follow the course of the patients' BN or EDNOS diagnosis. One potential imitation of these assessment instruments is that the LIFE and DIPD-IV are comprised of semi-structured interviews, which may lead to varied discussions between interviewers and participants; there is far less standardization than the widely-used SCID-I/P ensures. In the study, Grilo et al. defined remission as 8 consecutive weeks with PSR ratings on the LIFE less than 2, and relapse was considered to be 8 consecutive weeks with PSR ratings of 2+ (that is, ED symptoms which were clinically-meaningful and caused the patient impairment).

With regard to bulimia nervosa specifically, results of the study showed that 46% (N = 16) of the participants subsequently relapsed throughout the 6-year time frame, with Cox PHREG analyses revealing that the number of stressful life events significantly predicted one's time to relapse (hazard ratio (HR) = 1.52, $p < 0.04$) (Grilo et al., 2012). More specifically, prominent stressors seemed to generally surround work occupation (HR = 3.04, $p < 0.01$) as well as social stressors (HR = 3.13, $p < 0.01$). Surprisingly, covariate variables including psychiatric comorbidity and eating disorder duration were not statistically significant in determining eating disorder relapse; higher proportions of

borderline personality disorder (BPD) and lower proportions of obsessive-compulsive personality disorder (OCPD) increased the speeds at which one relapsed, but not to statistically significant extents (Grilo et al., 2012). Nevertheless, the correlation between personality disorders and the rate at which the individual relapsed suggests that further research is warranted in order to determine whether there is, in fact, a significant relationship between the two variables.

An inevitable environmental factor that must be considered in this particular day and age is the Internet and its impact on members of society. Individuals with body dysmorphia, a history of eating disorders, and low self-esteem issues along with other factors may be particularly susceptible to unhealthy dieting fads, unrealistic body expectations, and more. A scoping review by Chung et al. (2021) aimed to examine adolescent influence on eating behaviors through social media; in this study, social media encompassed any social networking site enabling interactive, user-generated content allowing for the sharing of images, ideas, videos, music, or commentary. Various databases including PubMed, ERIC, PsycINFO, and Web of Science were utilized to conduct a literature search; inclusion criteria for the articles in this review were that the study 1) have a sample including adolescents 10-19 years old; 2) examined a social media app; 3) was cross-sectional, qualitative, observational, and experimental; 4) had a social media component; 4) examined adolescent peer communication in a social media environment; and 5) examined eating behaviors (Chung et al., 2021). Exclusionary criteria included studies that 1) described the impact of social media on body image; 2) pertained to gastric bypass; or 3) were conducted with animals as opposed to humans. By eliminating studies that simply focused on the correlation between social media and body

image, it helped to tease apart participants with low self-esteem (relating to body image) and those who truly struggled with eating disorder behaviors. Only 6 studies met all of the inclusion criteria required by this review and focused on 4 central themes: 1) visual appeal, content dissemination, 3) socialized digital connections, and 4) adolescent marketer influencers (Chung et al., 2021).

Results of the scoping review revealed that sharing food amongst social media was popular among adolescents, and may reflect behaviors that they either want to adopt or promote (Chung et al., 2021). However, on the opposing side, unrealistic food imagery might promote an unrealistic mindset toward thinness and lead to maladaptive eating behaviors. Additionally, the messages behind these posts may adversely impact one's mental health, thereby instigating unhealthy thoughts and decisions. Of course, these online communities may have both positive and negative consequences pertaining to eating disorder tendencies; on one hand, peer support aided in relapse prevention of anorexic behaviors among adolescents in the United Kingdom (Chung et al., 2021). However, it can be reasonably inferred that unhealthy community forums could hinder teenagers just as much as the media could help them. Still, this scoping review provides very limited research with regard to eating disorder relapse, and more so touches on the potentially positive effects of social media and eating disorder behavior. Further research is warranted to support the implication that it can be harmful for relapsed individuals to engage in online forums and similar outlets.

Sociocultural Implications

Unfortunately, there is very limited research with regard to eating disorder relapse from a cultural perspective; i.e., many studies are conducted in the United States and

European countries, and either do not disclose ethnic differences or have a disproportionately high percentage of Caucasian individuals. Society wrongly suggests that eating disorders primarily affect white, middle-to-upper class individuals in Westernized countries. More research is essential in discovering eating disorder course and outcomes in low-SES communities and must also account for different cultures. Perhaps eating disorder symptoms are manifested differently depending on the region that the individual resides in.

The conceptualization that eating disorders are "culture-bound syndromes" that mainly afflict Caucasian adolescent and young adult women in high-income, industrialized communities has been discredited by literature, particularly in recent years. Pike et al. (2014) conducted a literature review that explored cultural trends in eating disorders and discovered an important sociocultural transition that fails to be touched on by other recent studies. In Asia, the increasing prevalence of eating disorders is typically correlated with the countries' increasing growth and modernization; they first appeared in Japan in the 1970s, followed shortly after by industrializing countries such as Hong Kong, Malaysia, and South Korea (Pike et al., 2014). Less industrialized countries such as China, Thailand, and Taiwan did not report cases of EDs until their own advancements in the 1990s and early 2000s. Across Asia as a whole, there are lower prevalence rates compared to the West; China is the exception to this argument, as their prevalence rates are on par with age-matched Western samples. Additionally, there was a significant increase in underweight girls born after 1991; this population was positively correlated with SES. Moreover, factors associated with heightened ED risk (e.g., body dissatisfaction and dieting) have not only grown more widespread in Asian cultures, but

in certain areas have even surpassed levels reported in the West; groups such as Singaporean and South Korean women are included in this subpopulation. Asian men may also be more vulnerable to EDs than those in Western countries, although they are still less common than women populations (Pike et al., 2014). In Arab regions, there is a widespread "thin ideal" that is compounded by increasing body dissatisfaction, dieting practices; these trends have resulted in more subclinical and clinical cases of eating disorders, as well as higher rates of disordered eating behaviors. Still, this literature review, like many other studies, does not discern whether these heightened ED rates are due to genetic factors, environmental factors, or a combination of the two (i.e., diathesis–stress). With eating disorders being just as prevalent in Asian and Middle Eastern countries as the United States, their relapse rates are bound to be similar as well. However, current literature unfortunately does not cover the problem of eating disorder relapse in these geographic regions; thus, research in these areas is crucial.

Even in the United States, research is lacking among minority ethnic groups; many research studies have disproportionately high rates of Caucasian, female participants. This may be largely due to the fact that other ethnicities are correlated with lower SES, and often do not receive the same adequate treatment that white, middle-to-upper class individuals do. The gap in professional care is not due to a lower prevalence, however; rather, recent evidence suggests that the rate of clinical EDs in racial minorities has been rising (Pike et al., 2014). Not only are ED rates comparable amongst Latino, Black American, and non-Latino Caucasian groups, but a 2011 study found that BN rates (particularly in males) may be significantly higher in the Latino and Black American groups than Caucasians (Marques et al., 2011). In these populations, the "thinness ideal"

is far less prevalent; that is, the two aforementioned cultures generally prefer curvaceous figures. Hence, body satisfaction among these groups is typically higher, but BN rates are nevertheless higher than in Caucasian groups (Pike et al., 2014). Thus, further research must be conducted to examine why certain EDs are more prominent in minority groups that outwardly express more body acceptance. Environmental and genetic factors need to be disentangled, but aforementioned data in this chapter suggests that bulimia nervosa likely has genetic implications.

While culturally inclusive research surrounding eating disorder relapse and recovery is lacking, it is not entirely inexistent. Musolino et al. (2018) conducted case studies in South Australia to more deeply understand the relationship between culture and ambivalence toward seeking eating disorder treatment. Although 1) Australia is significantly more westernized than some of the above-mentioned geographic regions, and 2) case studies should be interpreted warily due to the inability to generalize their findings, this study nevertheless provides insight as to how individuals may approach EDs when accounting for sociocultural aspects, as well as the concept of desire. Australia has a similar mindset to the United and other westernized countries when it comes to the "ideal body"; i.e., Australians place a heavy emphasis on the value of thin bodies and restrictive, diet-heavy food practices (Musolino et al., 2018). With in-patient clinical treatment plans placing such a heavy emphasis on weight restoration, the Australians' fixation on anti-obesity makes it difficult to fully embrace recovery; perhaps this sociocultural pressure plays a role in relapse. The ongoing obsession with a need to be thin brings in this concept of "desire," which the authors hypothesize is produced by ED triggers. 28 female participants ranging from 19 to 52 years old met recruitment criteria

for the study: they 1) were over 16 years old and had not seen a health professional for disordered eating; 2) had not been given an ED diagnosis; 3) had been diagnosed with an ED but delayed seeking treatment; or 4) did not wish to pursue ED treatment (Musolino et al., 2018). One drawback of these criteria is the fact that it encompasses a multitude of eating disorder behaviors which may go beyond the scope of AN and BN, which the scope of this paper focuses on; still, the limited research on ED relapse and its relation to sociocultural factors gives this study immense value. Researchers utilized the EDE (Eating Disorder Examination) to collect data, although the semi-structured nature of this interview left room for leeway throughout the course of the conversation.

An immediate drawback of this study is that the results section touches on only two participants' interviews, as opposed to analyzing and connecting the conversations from all 28 participants. The first participant, Kelly, was a self-proclaimed "healthy anorexic" who went through phases of "bingeing up" and "starving down" (Musolino et al., 2018). Her phases were often dependent on her environment; that is, triggers such as relationships (e.g., a social hangout) or sociocultural factors (e.g., reading about a dieting fad) could impact whether she binged or starved. However, Kelly was also a victim of sexual abuse (another trigger that led to starvation); hence, she was somewhat unrepresentative of the greater ED population. The other case study, Charlotte, struggled with AN for the majority of her adult life and used food desires to manage everyday triggering; she categorized "safe" and "risky" foods by utilizing information that was disseminated in her own society (Musolino et al., 2018). In both examples, the authors emphasize that Australian women (similar to other westernized cultures) are expected to control their appetites, manage their bodies, and deny their needs and pleasures.

Musolino et al. also describes desire as “generative and never-ending” (2018); perhaps this is a major factor in one’s predisposition toward relapse and ambivalence toward recovery.

CHAPTER FOUR

A Comparison Between Full Recovery and Relapse Rates

There seems to be an inherent fundamental difference between the individuals that reach long-term recovery from AN and BN in comparison to those who achieve remission but then suffer from later relapse. Although environmental factors may certainly play a factor in provoking their relapse, prior findings seem to suggest that there is a major genetic component that is associated with recovery, or lack thereof, of eating disorders. The following studies and their respective analyses will aim to compare the individuals who achieve long-term remission against those who are victims of eventual ED relapse.

A study by Dobrescru, et al. published in 2020 examined the 30-year outcomes of AN individuals and suggested that good outcome was predicted by 1) later adolescent onset (i.e., being diagnosed at a later age) and 2) premorbid perfectionism. In 1985, researchers screened 4,291 eighth-graders in Gothenburg, Sweden for AN; of these, 51 participants (48 girls, 3 boys) met the DSM-III-R and DSM-IV diagnostic criteria for anorexia nervosa, with a mean age at onset of 14.3 years. Additionally, a comparison group comprised of 51 same-age, same-gender individuals with no history of an eating disorder was carefully selected as a control group. Of all the 102 participants in the study, 4 individuals (all in the AN group) declined participation at the end, resulting in a total drop-out rate of 4%. Researchers used the Mini-International Neuropsychiatric Interview (MINI 6.0) to ensure standardization, although 2 participants opted to only participate in a short interview and refused to follow this structure (Dobrescru et al., 2020). This

interview by itself was considered too brief with regard to the eating disorder module, and was therefore used alongside the Structural Clinical Interview for DSM-IV (SCID-I) and a DSM-5 checklist for feeding and eating disorders. The Global Assessment of Functioning (GAF) was used to assess general outcome, and the widely-known Morgan-Russell scales were used to calculate a composite score surrounding concerns of body weight, dieting, mental state, social relationships, etc.; the latter of the two is perhaps one of the most high-validity instruments in AN research (Dobrescru et al., 2020). Akin to other studies evaluated in this paper, full remission was defined as having no diagnostic criterion for at least 8 consecutive weeks, although in this study the individuals were required to be free of symptoms for a minimum of 6 months (Dobrescru et al., 2020). Predictive factors in the study primarily surrounded premorbid and childhood data, including body mass index (BMI), perinatal factors, social class, household problems, major life events, obsessive-compulsive traits, and perfectionism.

Results at the last follow-up found that 19% (N = 9) of individuals in the AN group met the diagnostic criteria for a current eating disorder, with 3 of the individuals retaining AN (2 of which were in partial remission) (Dobrescru et al., 2020). 64% (N = 30) of participants in the AN study group were considered fully recovered, leaving 17% (N = 8) not yet achieving full normality, but also not necessarily victims of an eating disorder at the time that the follow-up was conducted. The study does note that between the fourth and fifth (final) follow-up, 17% (N = 8) of participants fulfilled AN diagnostic criteria, and 32% (N = 15) experienced any type of eating disorder at some point; however, those who met AN criteria at study 4 no longer had an eating disorder of any type at the next follow-up (Dobrescru et al., 2020). In other words, those who had AN at

the final follow-up had either 1) relapsed within the last few years, or 2) were originally categorized under a different eating disorder and transitioned into an AN diagnosis. Considering that the mean duration of aggregated episodes of AN was 4.9 years (s.d. 5.1) in the study (Dobrescru et al., 2020), the latter option seems more likely. Psychiatric comorbidity seemed to be common among those struggling from eating disorders at the last follow-up; 37.8% of the AN group were diagnosed (using DSM-IV criteria) with any psychiatric disorder excluding eating disorders, as opposed to just 11.8% of the comparison group suffering from mental disorders (Dobrescru et al., 2020). These findings seem to indicate that there is, in fact, an innate psychological problem with those who have (or previously had) AN in the study, particularly with regard to the individuals who eventually relapsed; 55% (N = 5) of the relapsed AN participants had struggled with another psychiatric disorder at some point, whereas only 31% (N = 12) of the 38 AN participants who either achieved full recovery or were in remission had psychiatric comorbidity. While these percentage differences are significant and may allude to an underlying psychological problem, it is difficult to determine whether these other psychiatric disorders developed as a result of the participants' eating disorders, or were present before their onset.

With respect to predictors of good outcome, linear regression analysis showed that a later onset of AN and perfectionism prior to the onset of participants' eating disorders resulted in a higher likelihood of positive outcome. The first of the two predictors is consistent with other outcome studies of childhood-onset AN, in which illness typically takes a chronic course with higher rates of morbidity (Dobrescru et al., 2020). However, it is difficult to determine whether an earlier onset is more often caused

by earlier drastic environmental events, higher psychological disturbance, or perhaps both. Additionally, although premorbid perfectionism has oftentimes been stated to be a risk factor for developing anorexia nervosa, this study found that it actually helped with having favorable outcomes. While some eating disorder therapies (e.g., CBT) have been utilized to reduce clinical perfectionism, it has contrastingly persisted in AN individuals who have achieved full recovery (Dobrescru et al., 2020). Thus, perhaps this psychological trait may serve as both a benefit and hindrance in recovery.

Another study published in 2017 by Eddy et al. similarly observed the recovery from both AN and BN over the course of 22 years. 246 women participants over age 12 were recruited from Boston-area outpatient eating disorder services and met the criteria for DSM-III-R AN or BN diagnosis, with no evidence of organic brain syndrome or terminal illness (Eddy et al., 2017). Researchers applied DSM-IV criteria to intake eating disorder data, and 55% (N = 136) met anorexia nervosa criteria while the remaining 45% (N = 110) met bulimia nervosa criteria, leading to a relatively equal distribution of participants among the two eating disorder subtypes. Of the 246 participants, 176 remained at the 22-year follow-up (100 AN, 76 BN) due to deaths since the study entry and declination to participation; thus, this relatively high attrition rate (29%) is something to be cognizant of while evaluating the study. In Wave 1 of the study, participants were interviewed every 6-12 months for a mean of 9.1 years; in Wave 2, participants were contacted between 20-25 years after study entry for one follow up (Eddy et al., 2017). Researchers utilized the Longitudinal Interval Follow-Up Evaluation of Eating Disorders (LIFE-EAT-II) throughout both waves of the study, which yielded weekly psychiatric status rating (PSR) scores (i.e., symptom scale scores for AN/BN). Additionally, through

Wave 2, online self-report questionnaires (including 1) the Eating Disorder Examination Questionnaire and 2) World Health Organization Quality of Life) were used to cover subjects including restraint, eating concern, weight concern, health, and environment (Eddy et al., 2017). Due to the fact that these are both close-ended questionnaires with high internal consistency, there was little room for flexibility and error. Eating disorder recovery was defined differently in this study, with AN and BN requiring a PSR score of ≤ 2 for 52 consecutive weeks (1 year); this is profoundly different from the definition of remission in other studies, which only consists of 8 weeks without symptoms.

When comparing AN recovery and BN recovery, the study results showed that 31.4% of anorexic participants and 68.2% of bulimic participants recovered throughout Wave 1 (i.e., over the course of approximately 9 years). One drawback of this study is that the median recovery time for AN in Wave 1 could not be calculated due to the small percentage of recovered individuals (< 50%); however, BN recovery time was determined to be approximately 3.8 years (Eddy et al., 2017). With researchers concluding that AN participants were slower to recover than those with BN, it can be reasonably inferred that the median time to AN recovery was < 3.8 years. Wave 2 of the study produced interesting findings, with 62.8% of participants with an initial AN diagnosis achieving full recovery; this included 50.2% of women who were not recovered at Wave 1, but 10.5% of individuals who had recovered during the first wave were no longer deemed recovered (i.e., they relapsed). With respect to BN participants, 62.8% of the women were again found to be recovered; however, this percentage comprised of 44.1% of individuals who had not been recovered at Wave 1, as well as 20.5% who had relapsed by Wave 2 (Eddy et al., 2017). It is interesting that such a high percentage of

anorexic individuals were fully recovered by the second wave follow-up; perhaps this alludes to implications for a slower, yet nevertheless successful, recovery. Additionally, it is peculiar that a higher percentage of BN participants were fully recovered by the first-wave follow-up, yet a significantly higher percentage (compared to AN participants) had relapsed by the Wave 2. This alludes to the possibility that although BN participants initially recover at a faster rate, AN individuals may be more well-equipped to achieve full recovery, yet at a slower pace, for the long term.

Additionally, the relationship between early and long-term recovery was found to be somewhat more significant for AN individuals than those with BN. Participants with anorexia who were recovered by Wave 1 were 10.5 times more likely to be recovered at Wave 2, as opposed to those who did not achieve recovery by Wave 1 (Eddy et al., 2017). Contrastingly, early recovery for BN individuals was not associated with an increased likelihood of recovery at Wave 2; however, far more bulimic individuals were recovered by the first wave, in comparison to anorexic participants. Nevertheless, perhaps it would be fruitful to focus on earlier symptom recovery specifically for anorexia nervosa individuals moving forward.

In both the AN and BN participant groups, recovery was correlated with normal levels of eating disorder pathology (Eddy et al., 2017). In other words, perhaps the severity of the eating disorder (i.e., how many symptoms are displayed, the gravity of those symptoms, etc.) may play a role in whether individuals relapse at a later point. Specifically regarding AN, it seems as though earlier recovery from the disorder is crucial to ensuring long-term success and preventing the risk of relapse. It would thus be conducive to focus on relieving patients of their symptoms earlier on in anorexia

treatment to avoid the recurrence of maladaptive behaviors. Still, regardless of whether participants were fully recovered by Wave 1, it was still possible that they would achieve remission by Wave 2. The findings were different for bulimia nervosa; recovery rates did not increase nearly as much over time, implying that early recovery is essential for BN (Eddy et al., 2017).

Nevertheless, there were several drawbacks of the study that require its readers to interpret its findings with caution. For one, the infrequent follow-ups throughout the longitudinal course of the study left it difficult to track life events; that is, did an individual suffer from something in her environment that may have caused her to relapse? Another disadvantage of the study was that it was comprised of predominantly white (95%) individuals, leaving essentially no room to compare women of different ethnic backgrounds. Additionally, the researchers lacked specific treatment data of the participants, so it impossible to decipher whether quicker and long-term recovery was in part influenced by certain aspects of intervention (Eddy et al., 2017).

Thirdly, a systematic review and meta-analysis published by Berends et al. (2018) examined possible implications for relapse with respect to anorexia nervosa, specifically. The review analyzed sources from databases including PubMed, PsychINFO, and CINAHL and excluded studies that 1) had a sample size of less than 40 participants, 2) combined AN and BN patients, and 3) used the same/duplicate study sample; with these filters applied, 16 articles were included in the present research (Berends et al., 2018). The mean age of participants was relatively diverse (ranging from 15.4 to 26.9 years), as well as the geographic range in which the studies were conducted (USA, Canada, Denmark, Germany, the Netherlands, Italy, and Israel) (Berends et al., 2018). However,

the geographic "diversity" was limited to Western regions which predominantly consisted of Caucasians; thus, this analysis likely fails to take into account ethnic differences. The study examined three aspects of relapse: 1) rate, 2) timing, and 3) factors associated with relapse.

Across the studies, results found that the approximate relapse rate was 31%, regardless of the participants' age; this goes against the findings in Dobrescru et al. (2020), which suggested that a later age of eating disorder onset might be correlated with a higher likelihood of positive outcome; thus, further research on whether or not age does play a significant role in recovery is warranted. With regard to timing of relapse, the systematic review found that the highest risk was within the first year after discharge (Berends et al., 2018); however, this risk could continue (though at a lower rate) in the years following. Hence, it may be conducive to continue eating disorder treatment for a longer period of time than those outlined in Chapter 2 suggest. The meta-analysis also identified several factors associated with relapse across the studies, which seemed to be predominantly genetic-based rather than environmental; however, these must be interpreted with caution due to the variability in procedures and measurement within the studies (Berends et al., 2018). Such factors included shape and weight concerns, obsessions, and compulsions. These findings suggest that individuals who are more susceptible to relapse are more cognitively impaired, as they have a more negative self-image as well as comorbid OCD tendencies. This implication is compounded by the fact that individuals who 1) receive longer or more intensive treatment and 2) have a longer duration of the illness are more likely to relapse (Berends et al., 2018). Still, potential

environmental events that may have impacted the patients' courses of outcome were not discussed in this study; thus, its findings should be analyzed carefully.

CHAPTER FIVE

Conclusion

With approximately one-third of individuals suffering from anorexia nervosa and bulimia nervosa relapse, it is imperative that more research is conducted to explore ways to minimize this risk of relapse. Unfortunately, most of the available research is limited in that it focuses on Caucasian, middle-class females residing in the United States and other westernized countries. Nevertheless, existing findings suggest promising methods to achieve eating disorder remission and promote long-lasting, positive outcomes in individuals. Such methods include altered approaches to treatment and novel methods of ED treatment, such as deep brain stimulation. Despite these promising findings, there is a multitude of shortcomings in ED relapse research, particularly with regard to sample biases.

Suggestions for Prevention

Research conducted by Cockell et al. (2004) examined the factors that support and hinder eating disorder recovery by interviewing 32 women in a 6-month follow-up assessment after being released from eating disorder treatment. This sample group comprised of 21 women who initially met diagnostic criteria for anorexia nervosa, along with 11 women who met criteria for an eating disorder not otherwise specified (EDNOS). Researchers utilized the Eating Disorders Examination to assess diagnostic status, and then administered in-depth interviews with each individual. Although a set of predetermined questions guided the conversation, the participants were not limited in

what they said; thus, the open-ended nature of the study, as well as the high level of individualization in each interview, is something to be mindful of while analyzing these results. Results found that there were 3 general categories of supportive factors that assisted eating disorder recovery: 1) maintaining connections with social supports, 2) applying cognitive and affective skills learned in the program, and 3) focusing beyond the eating disorder (Cockell et al., 2004). Building personal relationships with peers, family members, and professionals helped to provide validation, individualized support, and opportunities to be honest. Additionally, applying the knowledge and skills that were taught in the eating disorder program, such as challenging distorted thinking, undoubtedly aided in maintaining recovery behaviors and reducing the risk of relapse. Furthermore, focusing on meaningful aspects of life outside of mental illness may help one to realize that there is more to one's being than weight or shape.

In contrast, Cockell et al. (2004) also identified three factors that may impede one's recovery: 1) losses, 2) self-defeating beliefs, and 3) dealing with real life. "Losses" was quite general in scope, but referred in part to a loss in structure (i.e., the transition from a rigid schedule in treatment to having freedom in the real world), a loss of professional support, and a loss of understanding; while there was an abundance of empathy in group treatment, it was not mirrored in individuals' friends and family outside of the eating disorder realm (Cockell et al., 2004). Secondly, certain beliefs that patients adopt following recovery seem to play a significant factor in detrimental behaviors and relapse. Such mentalities included having unrealistic expectations about recovery (e.g., being overconfident), as well as the overwhelming need to be in control. Thus, shifting one's mindset both throughout the process of recovery and in the months following

seems to be crucial in minimizing relapse behaviors. Lastly, dealing with stressors of the real world seemed to impede many individuals; this included witnessing diet culture on social media, as well as being faced with challenges regarding work, health, and relationships (Cockell et al., 2004). Individuals in the study admitted to adopt more negative behaviors in these stressful situations, and occasionally slipped into negative habits as a coping mechanism. Hence, this study supported the fact that both psychological and environmental factors may play significant roles in eating disorder recovery and relapse.

Also with respect to environmental factors, the Internet (as mentioned in Chapter 3) may serve as both a hindrance and advantage in eating disorders and their recoveries, depending on how it is utilized. An internet-based study conducted by Keski-Rahkonen and Tozzi in 2005 sought to understand the process of eating disorder recovery through victims' own words. The researchers examined an unmoderated eating disorder discussion forum maintained by a private clinic that was accessible to the general public, and attempted to understand what sufferers meant by the word "recovery" (Keski-Rahkonen & Tozzi, 2005). Results of the study showed that more participants actually found the Internet discussion to be more unhelpful, rather than helpful, throughout their recovery journey; it seemed to serve as a disadvantage specifically in the final stages of their remission. Factors that were particularly viewed as unhelpful in the discussion were 1) focusing on food and 2) competition (e.g., comparing height and weight). Contrastingly, participants generally found group support to be beneficial in the earlier stages of their recovery (Keski-Rahkonen & Tozzi, 2005). Moving forward, it may behoove those in eating disorder remission to seek out Internet peer support in the

beginning, but find alternative means later on, to minimize the risk of relapse and ensure long-term recovery.

As successful treatments for AN and its long-term recovery continue to be sought, a relatively new technique of deep brain stimulation in the subgenual cingulate cortex has emerged. Previous functional neuroimaging studies have shown alterations in corticostriatal pathways in both active and recovered anorexics, finding areas of increased activity in the thalamus, amygdala-hippocampal complex, caudate, and lateral inferior frontal regions (Israël et al., 2010). In particular, the anterior cingulate cortex has been thought to play a role in body image perception, the hedonic properties of food, and the pathophysiology of depression and OCD (Israël et al., 2010). The aim of deep brain stimulation for anorexia treatment, therefore, aims to decrease this activity in cortico-striato-thalamo-cortical circuits. Through diminishing these areas, deep brain stimulation shows promise in not only lessening eating disorder symptomatology, but also comorbid mental disorders such as those aforementioned.

A 2013 study by Wu et al. describes the process of deep brain stimulation for anorexia, in which DBS electrodes were implanted with magnetic resonance imaging (MRI)-guided stereotactic surgery. While the patient is under sedation, bilateral scalp incisions are made, burr holes are carefully placed, and electrodes are implanted at target coordinates (Wu et al., 2013). To verify the electrode coordination and potential side effects, macrostimulation is used as confirmation (Wu et al., 2013). Although this treatment technique may be criticized for its extreme invasiveness in comparison to treatment alternatives, the surgery is reversal and shows promise for positive, long-term outcomes. Of the four female patients evaluated in this DBS clinical trial study, all of

them no longer met AN criteria at the 38-month follow up. However, this sample was quite small and thus unrepresentative of the general population; hence, although the researchers gathered from this that deep brain simulation is an up-and-coming treatment, it must continue to be studied in larger populations.

Limitations

Moving forward, it is essential to have a standardized definitions with regard to eating disorders; this includes (but is not limited to) terms such as recovery, remission, and relapse. The previous chapters demonstrated that different studies had varying criteria for what was considered full remission, making it difficult to compare patients across studies and draw more general conclusions. For example, in Chapter 3, Olmsted et al. (1994) defined remission as a maximum of one binge eating/purging episode during the last 4 weeks of treatment; in contrast, Keel et al. (2005) described it as having minimal or no eating disorder symptoms for 8 consecutive weeks. This 4-week difference in the definition of remission is significant, and makes it impossible to universally determine what constitutes recovery. Similarly, relapse was defined by Carter et al. (2013) as a BMI of less than or equal to 17.5 for 3 consecutive months of at least one episode of binging-purging behavior per week for 3 consecutive months during the 1-year follow-up period (this definition specifically pertained to AN); Keel et al. (2005) more generally defined the same term as returning full disorder criteria after a period of remission, and Grilo et al. (2012) termed relapse as 8 consecutive weeks with PSR ratings of 2+. As we can see, the above definitions vary substantially in terms of 1) the amount of time that has passed for the individual to have relapsed, and 2) the number of eating disorder symptoms present. The time frame ranges from 2 to 3 months of symptoms

contingent on the study, and symptoms could either depend on PSR ratings or a return to full disorder criteria. Such differences will continue to pose hindrances until consistent definitions of these terms are adopted by researchers.

A review by Bardone-Cone et al. (2018) acknowledges this "broken record syndrome" in which the importance of universal definitions is repeatedly acknowledged and abandoned, and makes suggestions for future directions surrounding this ongoing issue. The researcher asserts that the eating disorder field has generally taken two approaches in acquiring a standard definition of recovery: 1) the quantitative approach, in which criterion are based on established conventions, critical literature reviews, and empirical validity; or 2) qualitative methodology, which focuses on the patient's own experience of recovery (Bardone-Cone et al., 2018). The researcher implies that contemporary literature advocates for eating disorder recovery to be measured holistically: that is, physical, behavioral, and psychological/cognitive criteria should all be taken into account. However, this implication is not supported by the studies outlined in the aforementioned chapters; rather, definitions seem to be heavily based on physical factors (e.g., BMI) and the reappearance of recurring eating disorder symptoms over a specified amount of time. Furthermore, the wide variety of assessment instruments used to measure behavior makes it difficult to draw a general conclusion across multiple studies. Bardone-Cone et al. emphasizes the need for a consensus regarding a universal description of eating disorder recovery, as well as other terms including partial recovery, remission, and relapse. Additionally, the authors argue that it would be more advantageous to have standard definitions across all eating disorder subtypes, rather than tailoring recovery criterion to AN or BN specifically; this would make it easier to make

comparisons across studies (Bardone-Cone et al., 2018). Without this universality of terminology, eating disorder research will continue to face obstacles with regard to comparisons, and the question will stand as to what truly constitutes recovery.

Another major critique of the literature at hand is the fact that the sample groups across studies overwhelmingly disregard differences in gender, socioeconomic status, race, ethnicity, and geographic region. As seen in the aforementioned studies, the majority of participants (if not all) were females. Even in studies that examined both genders, it is impossible to gather fruitful information that can be generalized to the male population; the 30-year AN follow-up by Dobrescu et al., for example, consisted of 48 females and just 3 males (2020). Hence, this resembled more of a case study with regard to the men included; additionally, the study results did not even differentiate between the two genders' long-term anorexia nervosa outcomes. Other studies consisted of hundreds of women participants and no men; it is difficult to differentiate whether this is simply due to an overall lower prevalence, or due to the negative stigma in western societies that surrounds men with eating disorders. Additionally, these same study samples did not take into account socioeconomic status when evaluating their participants; it would be conducive for researchers to more heavily examine wealth disparities moving forward in order to highlight the disparities in treatment and relapse rates. In terms of race and ethnicity, participant groups across the board either 1) did not specify ethnicity/ race, or 2) were disturbingly composed of Caucasians; the study by Carter et al., for example, was comprised of 94% white individuals (2012). Geographically speaking, an overwhelming number of the studies were conducted in westernized countries; such regions included the United States, Canada, Sweden, and Australia. Future studies should focus on untapped

areas including the Middle East, Africa, and South America. More importantly, further research must encompass individuals of all backgrounds, including both sexes, people of all socioeconomic backgrounds, and a diversity of ethnic groups.

An additional hindrance based on the current literature review is the outdated nature of most of the studies at hand. The majority of research articles pertaining to eating disorder relapse date back to as early as the 1990s, and limited findings date later than 2013 (i.e., after the DSM-5 was published). Hence, many of the studies that were evaluated in this paper have discrepancies with regard to DSM disorder definitions; for example, a study conducted by Olmstead et al. (1994) required participants to meet DSM-3 criteria for bulimia nervosa, whereas a study carried out by Zerwas et al. (2013) consisted of a cohort comprised of individuals meeting DSM-4 criteria for anorexia nervosa. As a result, the significant gap between when these studies were conducted may lead to discrepancies across DSM eating disorder symptomatology; that is, participants who may not have qualified for one of the earlier experiments may have qualified for a later one due to the changes in the DSM over the years. Consequently, there is a lack of consistency across the studies with regard to which criterion ED individuals must meet to qualify for AN or BN.

Discussion

Despite the aforementioned shortcomings in this literature review, there undoubtedly stands the argument that the roles of 1) genetics and 2) the extent of psychological disturbance are both essential components in evaluating one's risk of eating disorder relapse. While environmental factors do seem to play a role in the context of stressful events and sociocultural pressures, far more studies support the former (as

opposed to latter) findings. Findings repeatedly showed that factors such as body image disturbance and anxiety levels were positively correlated with higher rates of ED relapse (Keel et al., 2005; Bloss et al., 2011). Still, with genetic and environmental factors being so inevitably intertwined, it would be conducive for researchers to make greater efforts moving forward to disentangle the nature-nurture debate.

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