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The Effect of Biological versus Environmental Causal Explanations on the Stigmatization of Major Depression and Anorexia Nervosa

A thesis presented by

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to the Department of Psychology

in partial fulfillments of the requirements

for the degree of Bachelor of Arts

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Abstract

This study investigated how biological and environmental causal explanations affect the stigmatization of college students described to have major depression and anorexia nervosa. Participants (38 male and 87 female college students) read a vignette about a college student with either major depression or anorexia nervosa that provided either a biological or environmental causal explanation for the disorder. Then, they completed a Social Distancing Scale; a Treatment Recommendations Scale; a Personal Responsibility Beliefs, Pity, and Anger Questionnaire; a Causal Attributions Scale; a Familiarity with Mental Illness Questionnaire; and a Demographics Questionnaire. Results indicated that participants stigmatized the target with anorexia more than the target with depression, and that biological and environmental causal explanations had a different effect on stigma toward depression versus anorexia. The highest level of stigma occurred when participants read a biological causal explanation for anorexia, and the lowest level of stigma occurred when participants read a biological causal explanation for depression; participants moderately stigmatized both disorders after reading an environmental causal explanation. These findings support and extend previous research demonstrating the biological model's mixed effect on stigma and indicate that biological causal explanations can have a unique influence on the stigmatization of different mental disorders. After attaining a stronger understanding of the link between etiology and stigmatization of specific mental disorders, advocacy groups can work with people's stigmatizing responses while promoting the most accurate information about etiology.

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Introduction

Between 12-18% of college students suffer from a diagnosable mental illness, indicating that mental health is a major issue on college campuses (Mowbray et al., 2006). In 2010, 91% of college counseling center directors confirmed that the number of college students with severe mental health problems has continued to increase (National Survey of Counseling Service Directors, 2010). This upward trend has occurred because young adults are treated for mental illnesses today more than they were in the past, and therefore, an increasing number of mentally ill young adults are healthy enough to enroll in college. Lawrence Summers (2005), former President of Harvard University, recognized this change when discussing differences in the student population at a class reunion; he stated:

> One of the unsung and wonderful things that has happened in our country in the last 25 years, 35 years, due to medical research, has been a tremendous increase in the capacity to treat a whole range of conditions that affect adolescents and young adults. Conditions relating to depression, conditions relating to hyperactivity, conditions of other kinds that interfere with the learning process. It's a very important bit of progress that comes with a great obligation for all of us (Borenstein, 2005, p. 2).

Colleges are responsible for accommodating the growing population of students with mental disorders, just as they are responsible for supporting students of different races, genders, sexual orientations, and nationalities.

Colleges can improve students' mental health by fostering supportive communities. Research has indicated that an individual's social network influences his or

her treatment-seeking behaviors for all types of health problems, including mental health issues (Hefner & Eisenberg, 2009; Vogel, Wade, & Ascheman, 2009). Hefner and Eisenberg (2009) found that college students who received low levels of social support experienced mental health problems more frequently, whereas college students who received high levels of social support had a lower likelihood of experiencing depression, anxiety, suicidality, self-injury, and eating disorder symptoms. Students who differed from the majority in race or ethnicity, nationality, or socioeconomic status faced a higher risk of experiencing social isolation and distress. Measures of social support could help indicate risk among college students, and therefore, colleges should develop outreach programs that target students at risk of social isolation (Hefner & Eisenberg, 2009). As the number of students with serious mental health issues attending college increases, it becomes essential that colleges create more supportive communities (Gabriel, 2010).

Counseling services is a support system that benefits the mental health of college students. A study of 10,009 college freshmen and transfer students showed a significant positive relationship between counseling experiences and student retention; students who utilized counseling services had a higher likelihood of staying enrolled in school (Lee, Michelson, Olson, Odes, & Locke, 2009). Despite the positive outcomes associated with college counseling services, a 2-year study demonstrated limited help-seeking behaviors among college students (Zivin, Eisenberg, Gollust, & Golberstein, 2009). During the study, more than one half of the students surveyed had at least one mental health problem at either the baseline or follow-up screening. Students classified as having a mental health problem during the baseline screening had a 60% higher likelihood of having a mental health problem during the follow-up screening 2 years later than did those not so

identified, but less than one half of the students who had a mental health problem at both the baseline and follow-up received treatment. The prevalence of mental disorders in college students, combined with the limited service use among those who screened positive, indicates the necessity for colleges to reach out to at-risk students and increase the accessibility of mental health services.

Stigma

What are the barriers that prevent college students from utilizing existing support systems? Research has indicated that fear of stigma is the most commonly reported reason why individuals with mental health problems do not seek help (Corrigan, 2004). Stigma is a mark of shame attached to members of marginalized groups. Goffman (1963) defined stigma as a "deeply discrediting" attribute that diminishes an individual "from a whole and usual person to a tainted, discounted one" (Goffman, 1963, p. 3, as cited by Corrigan, 2004). Stigma invokes fear and leads to prejudice and discrimination. Stigma influences mentally ill individuals, their families and friends, mental health service providers, and the general public. Due to its many detrimental effects, stigma is a widely studied topic in the field of psychology. The primary goal of stigma research is to discover stigma reduction methods (Corrigan & Kleinlein, 2005).

According to Scheff's Labeling Theory, labeling plays a key role in producing stigma; "the *label* 'deviant' (i.e., mentally ill) leads society to treat the labeled individual as a deviant" (Corrigan & Kleinlein, 2005, p. 15). People respond to the label with fear and social distancing. Individuals with mental illness then face prejudice, which perpetuates their identities as mentally ill; subsequently, they conform to the label imposed upon them. A study by Link (1987) demonstrated the power of labeling: people

stigmatized a target described in a vignette based on a label, even in the absence of abnormal behavior (Link, 1987, as cited by Corrigan & Kleinlein, 2005).

Stigma is multifaceted, and therefore, research has examined the underlying dimensions that contribute to it. Jones et al. (1984) identified six dimensions of stigma: concealability, course, disruptiveness, aesthetics, peril, and origin. The concealability dimension addresses how noticeable the condition is to others and is determined by the ease with which someone can hide the stigmatizing mark. Course refers to the condition's reversibility; a condition that is irreversible is more stigmatized than is a reversible one. Origin, or cause of the condition, shapes how society perceives individuals' personal responsibility for their mental illness. This dimension has important legal implications; if individuals with mental illness are seen as responsible for their condition and associated actions, the law is likely to punish them, whereas if individuals with mental illness are not seen as responsible, the law is likely to exempt them. Finally, peril relates to the perceived dangerousness of the condition and can include fear of physical danger or uncomfortable feelings (Jones et al., 1984).

Stigma takes many forms and manifests itself in a variety of ways. Three commonly studied types of stigma are public stigma, self-stigma, and personal stigma. Stigma held collectively by members of society, known as public stigma, results in discriminatory behavior and places stigmatized individuals at a disadvantage in society (Eisenberg, Downs, Golberstein, & Zivin, 2009). Public stigma held by employers and landlords creates a barrier that prevents individuals with mental illness from attaining adequate jobs and housing. There are high unemployment rates among individuals with mental illness, despite their reported desire to work. Working contributes to

psychological wellbeing for everyone and can have a therapeutic effect on individuals with mental illness. In addition to facing employment difficulties, individuals with mental illness struggle to find adequate housing and are often forced to live in low-income neighborhoods with substandard housing and high crime rates. Although disparities are partly due to the disabilities associated with having a serious mental illness, they are "exacerbated by labels and stigma" (Corrigan & Kleinlein, 2005, p. 19). Additional negative outcomes that result from public stigma include less adequate healthcare, less income, and fewer social supports (Corrigan, 2004).

Perceived public stigma is extent to which an individual believes that the public stereotypes and discriminates against a stigmatized group (Golberstein, Eisenberg, & Gollust, 2008). In a correlational study, Golberstein et al. (2008) found that male students, older students, students of lower socioeconomic statuses, and students with current mental health problems reported higher levels of perceived public stigma than did students not so categorized. The researchers found a negative association between perceived public stigma and identification of a need for mental health services; however, this relationship only existed among younger students. The researchers did not find a significant association between perceived public stigma and service utilization, demonstrating that despite perceived public stigma, many students sought help (Golberstein et al., 2008).

When public stigma shapes the attitude of an individual with a mental illness and that individual identifies with the prejudice and stereotypes, self-stigma forms (Eisenberg et al., 2009). Self-stigma is an internalization of public stigma that leads stigmatized individuals to feel devalued by society. Self-stigma may cause a reduction in self-esteem,

self-efficacy, and confidence in one's future. It may affect treatment seeking because individuals want to resist the label and the prejudice that comes with it. An individual experiencing self-stigma anticipates rejection, which can often become a self-fulfilling prophecy (Corrigan & Kleinlein, 2005).

An additional type of stigma that harms college students facing mental health difficulties is personal stigma, which is defined as each individual's stereotypes and prejudices toward a stigmatized group (Eisenberg et al., 2009). Unlike self-stigma, which an individual with a mental illness feels toward him or herself, personal stigma is experienced more broadly across society. In a study of 5,555 college students from 13 different universities, Eisenberg et al. (2009) found that personal stigma served as the primary barrier preventing college students from seeking mental health treatment. Although the researchers found higher rates of perceived public stigma than personal stigma among students, only personal stigma inhibited treatment seeking. The researchers found particularly high rates of personal stigma among students who were male, younger, Asian, international, or from underprivileged families. Initiatives on college campuses to reduce personal stigma, particularly among populations who experience it the most, could help promote treatment seeking among college students facing mental health problems. Personal stigma influences help-seeking behavior recommendations, and, among the college population, peers play an important role in recommending treatments (Eisenberg et al., 2009).

Another barrier that prevents college students with mental illness from seeking help is the stigma associated with treatment seeking. Research has indicated that people stigmatize individuals who seek help more than those who do not seek treatment (Sibicky

& Dovidio, 1986). Ben-Porath (2002) found that participants perceived an individual who sought treatment for depression as less emotionally stable, interesting, and confident than an individual who sought treatment for back pain and an individual who had depression but did not seek treatment. The stigma attached to seeking treatment for a mental illness leads many individuals either to avoid seeking treatment or to decide not to follow through with treatment (Corrigan, 2004).

Stigma's effect on individuals with mental illness appears to be detrimental. Some people with mental illnesses have reported that stigma causes more problems for them than the mental illness itself does (Day, Edgren, & Eshleman, 2007). Research has indicated that even medical students experience high rates of perceived public stigma (Schwenk, Davis, & Wimsatt, 2010). Schwenk et al. (2010) found that medical students with moderate to severe depression more frequently agreed that if they disclosed their mental health problems, their peers would have less respect for their opinions and would view their coping skills as less adequate than if kept silent; they also believed that faculty members would see them as less capable of handling their responsibilities if they revealed their condition than if they concealed it. Medical students with depression expressed perceived risk about disclosing their depression to a counselor, and had a lower likelihood of seeking treatment compared to medical students with fewer depressive symptoms (Schwenk et al., 2010). This study demonstrated the universality of stigma; it is found even among the most educated members of our society. Thus, stigma reduction is a primary goal of mental health advocacy efforts because it will dramatically improve quality of life for a wide range of people.

The Effect of Biological versus Environmental Causal Explanations on Stigma

The origin dimension of stigma proposed by Jones et al. (1984) suggests that a condition's cause shapes how society perceives individuals' personal responsibility for the condition. Stigma and perceived etiology are thus closely linked; people's beliefs about the etiology of mental disorders contribute to their perceptions of individuals with mental illness. Therefore, researchers have investigated how different causal explanations for mental illness influence the stigmatization of mental disorders.

As the field of psychology has developed, perceived causes of mental health difficulties have changed, and simultaneously, the public's view of what constitutes a mental illness has evolved. Post World War II psychoanalysis promoted the belief that mental disturbances resulted from problematic relationships between parents and children. Star (1955) administered vignettes that portrayed paranoid schizophrenia, simple schizophrenia, alcoholism, anxiety neurosis, juvenile character disorder, and compulsive phobia to over 3000 Americans and found that people did not characterize many of these disorders as mental illnesses. Although 75% of participants identified paranoid schizophrenia as a mental illness, fewer participants indicated that they viewed simple schizophrenia (34%), alcoholism (29%), anxiety neurosis (18%), juvenile character disorder (14%), and compulsive phobia (7%) as mental illnesses (Star, 1955, as cited by Link et al., 1999).

Link et al. (1999) conducted a vignette experiment similar to Star's to examine how perceptions of mental illness have changed since 1955. The researchers found that most people considered schizophrenia (88%) and major depression (69%) mental illnesses, and they tended to attribute these disorders to a combination of biology and

stressful life circumstances. Comparatively fewer people regarded alcohol (49%) or cocaine (44%) abuse as mental illnesses, although still more than in 1955. Only 22% of people perceived the "troubled person" as having a mental illness. In every condition, people most commonly viewed stressful circumstances as the cause. Second to stressful circumstances, people cited a chemical imbalance in the brain as causing schizophrenia and depression, a person's upbringing as causing alcohol dependency, and a person's bad character as causing cocaine dependence. The researchers found that participants stigmatized each disorder differently. Participants perceived cocaine dependence as the most violent condition, followed by alcohol dependence, schizophrenia, and major depression. Participants desired the greatest amount of social distance from someone with cocaine dependence, followed by alcohol dependence, schizophrenia, and major depression. Participants viewed the "troubled person" as least dangerous and desired the smallest amount of social distance from the "troubled person" (Link et al., 1999).

Among professionals, the most widely held scientific belief today is that both biological and environmental factors contribute to mental illness (Butcher, Mineka, & Hooley, 2009). The diathesis-stress model suggests that individuals with mental illness are born with a biological predisposition for mental illness, and that an environmental trigger can lead to the development of a mental illness (Butcher et al., 2009). Research has explored how biological and environmental causal beliefs influence stigma differently with the goal of determining which model reduces stigma more effectively. Findings have indicated that the biological and environmental model each reduce stigma in some ways and contribute to it in others (Bennett, Thirlaway, & Murray, 2008;

Corrigan et al., 2002; Corrigan & Watson, 2004; Griffiths & Christensen, 2004; Jorm & Griffiths, 2009; Phelan, 2002).

Attribution theory insinuates that a biological etiological understanding of mental illness can help reduce stigma. According to attribution theory, the perception that an individual is capable of controlling a mental illness results in anger, whereas the perception that an individual is incapable of controlling a mental illness results in pity, sympathy, or helping behaviors toward the individual. Biological causal explanations promote the belief that individuals with mental illness are not capable of controlling their behavior, whereas environmental causal explanations promote the belief that individuals with mental illness are capable of controlling their behavior; thus, according to Attribution theory, biological explanations can help reduce stigma (Link et al., 2004). Weiner et al. (1988) created the first attributional measure of mental illnesses, which consisted of eight questions about 10 mental illnesses (Link et al., 2004). Attribution research has indicated that although perceived controllability is correlated with higher predicted rates of recovery (Weiner, 1986, as cited by Link et al., 2004), it is also associated with withholding help and supporting coercive treatment (Corrigan, 2003).

Consistent with attribution theory's suggestion that biological explanations will have a de-stigmatizing effect, several major mental health advocacy groups have endorsed a biological model for mental illness in their efforts to combat stigma (Corrigan et al., 2002). The stigma once associated with cancer has been reduced through the promotion of a biological understanding of cancer's cause; likewise, some advocacy groups hope that endorsement of a biological model will help de-stigmatize mental illness (Phelan, 2002). Research has indicated that the public tends to view people with mental

illnesses as responsible for their disorders and attributes mental illnesses to low morals or weak character (Corrigan, Markowitz, Watson, Rowan, & Kubiak, 2003). Blame toward the mentally ill leads to anger and social avoidance (Weiner, 1995, as cited by Corrigan & Watson, 2004). When people are educated about the biological explanation for mental illness, they are less likely to blame, avoid, or feel anger toward people with mental illnesses (Corrigan et al., 2002).

The National Alliance on Mental Illness (NAMI) has been at the forefront of promoting the biological model to reduce stigma, adopting the slogan that mental illness is a brain disease (Corrigan & Watson, 2004). Likewise, the Depression is Real Coalition has promoted the notion that depression is a biologically based disorder. According to the Depression is Real Coalition,

> Depression is biologically based, like diabetes. And like cancer, it can be fatal. Fortunately, it can also be treated. Which is why if you, or someone you know, are experiencing signs of depression, you should see your family physician or a qualified mental health professional for a thorough evaluation (Depression is Real Coalition, 2010).

Although the trend among advocacy organizations has been to reduce stigma by endorsing a biological model of mental illness, research has revealed that the biological model has a mixed effect on stigma (Corrigan & Watson, 2004). Evidence has shown that the biological model of mental illness has reduced blame but has increased stigma in unintended ways; therefore, campaigns focused on advocating for a biological model of mental illness might inadvertently exacerbate some aspects of stigma instead of reducing it.

In contrast to attribution theory, genetic essentialism holds that "genes are the unchangeable basis of a person's identity," and therefore, a genetic model of mental illness may exacerbate stigma by increasing desired social distance and promoting the idea that mental illnesses are serious, persistent, and dangerous (Rusch, Todd, Bodenhausen, & Corrigan, 2010, p. 328). Rusch et al. (2010) conducted a study that investigated how biogenetic explanations for stigma can increase implicit and explicit stigma among the general public and individuals with mental illness. The researchers found that biogenetic explanations had a mixed impact on the general public, decreasing perceived responsibility and implicit blame regarding individuals with mental illness, but increasing desired social distance. The authors pointed out that social distance is more closely linked to discrimination than is perceived responsibility, and thus, the negative effects of genetic models may outweigh the positive impact of such models. Among individuals with mental illness, a genetic model increased explicit self-reported responsibility, as well as implicit associations between mental illness and guilt (i.e., stronger Me-Guilty associations or self-blame) (Rusch et al., 2010).

As predicted by the genetic essentialism argument, the stigmatization of schizophrenia has increased since the medical model gained popularity (Matthias et al., 2005, as cited by Bennett et al., 2008). Increased public knowledge of the genetic factors of schizophrenia has led to a public rejection of and reluctance to form relationships with individuals with schizophrenia (Crisp, 1995, as cited by Bennett et al., 2008). One explanation for this finding is that while biological explanations have been helpful in reducing stigma for purely physical disorders, they may not have the same de-

stigmatizing effect for disorders with a behavioral component, such as schizophrenia (Bennett et al., 2008).

Social distance is an important outcome variable in the study of stigma, and research has explored how biological causal explanations have influenced desired social distance. Robert Park defined social distance as "the grades and degrees of understanding and intimacy which characterize pre-social and social relations generally," (Park, 1924, as cited by Link et al., 2004, p. 519). Bogardus (1925) created the first social distancing scale to measure social distance based on race and ethnicity, and Philips (1963) utilized the social distancing scale in a vignette experiment for the first time. Limitations to the validity of social distance scales include social desirability bias and the problem of inferring behavioral responses from self-reported intentions (Link et al., 2004). According to studies of social distancing, people who are older, poorly educated, and have never known anyone with a mental illness are more likely to desire more social distance from individuals with mental illness than are people who are younger, better-educated, and have had contact with someone who has a mental illness. In addition, individuals who perceive the mentally ill as dangerous are more likely to desire social distance than are those who do not perceive the mentally illness as dangerous (Link et al., 2004).

Griffiths and Christensen (2004) found that biological causal beliefs about mental illness (i.e., 'brain disease' and 'heredity') lead people to desire increased social distance from the mentally ill. However, like other data on the effect of the biological model on stigma, data on the biological model's impact on social distance are mixed. For example, Phelan (2002) found that the genetic explanation of mental illness had a weak influence

on ‘casual’ social distance (e.g., willingness to be a friend or a coworker) but had a strong influence on ‘intimate’ social distance (e.g., willingness to marry or date). Jorm and Griffiths (2008) found an association between classification of schizophrenia as a ‘real medical illness’ and reduced social distance; contrarily, Crisp (1995) found that as the biological model has gained popularity, desired social distance from individuals with schizophrenia has increased (Crisp, 1995, as cited by Bennett et al., 2008). Bennett et al. (2008) found that perceived cause of schizophrenia had no impact on desired social distance. Evidently, the effect of biological explanations on desired social distance is unclear.

The biological model has a less ambiguous effect on perceptions of dangerousness than on desired social distance. Dangerousness stigma, or the perception that individuals with mental illness are dangerous, generates prejudice and discrimination toward the mentally ill. Corrigan and Cooper (2006) deconstructed dangerousness stigma by examining the prevalence rates of violence committed by the mentally ill. They concluded that despite the weak association between mental illness and violence, the public perception is that the association is strong. Media portrayals of the mentally ill as violent have likely contributed to the perception that individuals with mental illness are dangerous. There are many negative outcomes associated with dangerousness stigma; among them are fear, avoidance, segregation, and support for coercive treatment (Corrigan & Cooper, 2006).

Research has shown that endorsement of a biological explanation heightens the stereotype that individuals with mental illness are dangerous. Bennett et al. (2008) conducted a study in which participants read a vignette that described an individual with

schizophrenia and offered either a genetic or environmental explanation of the disorder. Participants perceived the target described in the vignette as more dangerous when they read the genetic explanation than when they read the environmental explanation. People fear what they do not understand, and many people do not understand mental illness (Fink, 1996, as cited by Bennett et al., 2008). People often do not understand genetics either, and the pairing of two unknowns can heighten levels of stigma (Fink, 1996, as cited by Bennett et al., 2008).

Another form of stigma that is increased by endorsement of a biological model is benevolence stigma (Corrigan & Watson, 2004), which leads people to believe that individuals with mental illness are like children who need monitoring from a parental figure (Brockington et al., 1993, as cited by Corrigan & Watson, 2004). Benevolence stigma has a disempowering effect on individuals with mental illness because it implies that they are not self-sufficient and competent (Corrigan & Watson, 2004). In addition, previous research has demonstrated that people are less hopeful about recovery when they conceptualize mental disorders as biological rather than as environmental (Bennett et al., 2008; Corrigan & Watson, 2004).

Biological explanations may suggest that individuals with mental illness are fundamentally different and physically distinct, which exacerbates differences between mentally ill and “normal” people (Phelan, 2002). Taken to the extreme, the biological explanation may imply that individuals with mental illness are a different species (Corrigan & Watson, 2004). Phelan (2002) looked to history as a reminder of the fatal consequences that may result from biological explanations; she described the eugenics movement of the late 19th and early 20th centuries when genetic explanations for

stigmatized conditions resulted in policies such as marriage restrictions, sterilization, and extermination. The biological model may increase stigma toward families of individuals with mental illness, as well. Bennett et al. (2008) found that genetic attributions increased levels of associative stigma toward close relatives of individuals with schizophrenia. Society may begin to blame family members for passing on bad genes. Furthermore, new labels for family members, including "carrier" or "at risk," may emerge (Phelan, 2002). Although it is possible that the biological model of mental illness may increase stigma in certain ways, as increasing amounts of evidence support the biological explanation, experts have an ethical obligation not to hide this information from the public (Phelan, 2002).

Luchins (2004) highlighted the problems associated with efforts that aim to reduce stigma by promoting a biological explanation of mental illness, specifically a "brain disease" model. He argued that changing the label of serious mental illnesses to "brain diseases" will not reduce stigma alone, and that more fundamental changes (i.e., improving treatment and financial situations of the mentally ill) are necessary. He explained that the "brain disease" label for mental illnesses is problematic for three reasons: it is less descriptively accurate than the terms "mental illness" or "psychiatric illness", it is often not substantiated by scientific evidence, and it does not target the real cause of stigma. Luchins (2004) highlighted the difference between a mental illness and a brain disease and explained that the term "brain disease" describes neurological illnesses that involve dysfunction due to structural damage to the brain, whereas the term "mental illness" describes dysfunctional feelings, thoughts, and behaviors that do not affect brain structure. Luchins (2004) argued that neurological diseases can result in symptoms of

mental illnesses, but mental illnesses cannot result in signs of neurological diseases. Therefore, he asserted that the primary setback to the brain disease model is that it reduces mental health treatment to medication treatment; although medication alone can relieve symptoms, it cannot return an individual to normal mental health. Instead of conceptualizing mental health treatment as medication treatment, Luchins (2004) suggested that we broaden our understanding of physical illnesses to include social and psychological dimensions. The most common diseases in our society (e.g., cardiovascular disease, hypertension, diabetes, and lung cancer) are caused by life style choices, and therefore, all illnesses, not just mental illnesses, are caused by a combination of psychological, social, and biological factors.

Luchins (2004) suggested that a single perspective will not suffice in fully explaining the cause of mental illness, nor will it succeed in reducing stigma (Luchins, 2004). Advocacy groups should promote any particular view with caution and should highlight the multi-causal nature of mental illness. Also, advocacy groups should bear in mind that each mental illness has a different etiology and there are individual differences in the acquisition of mental illnesses, even for a given mental illness. Promotion of biological and environmental causal explanations will likely have a distinct effect on the stigmatization of different mental illnesses, and therefore, it is important to examine each mental illness separately.

The Effect of Perceived Etiology on Stigmatization of Major Depression

Major depression is the leading cause of disability in the United States for ages 15-44; it affects approximately 6.7% of American adults each year (National Institute of Mental Health [NIMH], 2010), and the lifetime prevalence rate for women is almost 25%

(Butcher, Mineka, & Hooley, 2010). Major depression interferes with daily life and normal functioning. Symptoms include persistent sadness, feelings of hopelessness and guilt, irritability, loss of interest in once pleasurable activities, fatigue, difficulty concentrating, sleep disruption, appetite change, thoughts of suicide, and persistent pains. Major depression is likely caused by a combination of genetic, biochemical, environmental, and psychological factors; different factors are more prominent for each individual. Major depression is usually treated with selective serotonin reuptake inhibitors (SSRIs) or other medications and/or psychotherapy, both with similar effectiveness overall. However, individuals have unique responses to different treatments, and therefore, one treatment may help some people more than it does others (NIMH, 2010).

Chemical imbalance explanations for depression arose in the mid-20th century and grew out of findings that chlorpromazine efficaciously treated psychosis and that monoamines existed within the central nervous system and acted as neurotransmitters. These early discoveries spurred the development of psychopharmacology as a field and the use of medication for treating mental disorders. The 1950s marked the creation of the first modern antidepressant drugs, iproniazid and imipramine (Healy, 1997, as cited by France, Lysacker, & Robinson, 2007). Following observations that iproniazid, initially a treatment for tuberculosis, appeared to have a euphoric effect on patients, researchers tested the effect of iproniazid on 17 inpatient psychiatric patients. The drug improved symptoms for 70% of the patients; the drug also proved to successfully treat depression in outpatients (Loomer et al., 1957, as cited by France et al., 2007). This discovery received widespread attention, and iproniazid became a popular treatment for depression until it

went off the market due to its harmful side effects (Healy, 2001, as cited by France et al., 2007). Imipramine was intended to produce a similar effect to chlorpromazine. Imipramine is derived from iminodibenzyl, a compound with a tricyclic central molecular structure similar to that of chlorpromazine, and therefore, it is known as a tricyclic antidepressant (Healy, 1997, as cited by France et al., 2007). An early large-scale study indicated that imipramine did not effectively work as an antipsychotic; however, a review of nursing notes suggested that the drug improved the moods of patients. This observation inspired a trial of imipramine on a sample of 40 depression patients, that in conjunction with clinical observations, revealed that imipramine effectively treated depression. Imipramine appeared on the market in the United States starting in 1958 (Healy, 2001, as cited by France et al., 2007).

In the 1950s, researchers in the United States also began investigating resperine, an herbal treatment used to treat hypertension and psychosis in India (Healy, 1997, as cited by France et al., 2007). Resperine had sedative and antipsychotic effects, but it never gained as much popularity as did chlorpromazine. However, resperine influenced chemical imbalance models of depression because it reduced levels of serotonin in the brain, and researchers hypothesized that its sedative (i.e., depressive-like) effect occurred because of this serotonin depletion. Research on the inactivation of adrenaline helped lead to the discovery of an enzyme called monoamine oxidase (MAO) that oxidized adrenaline, as well as the monoamines noradrenaline, dopamine, and serotonin (Healy, 1997, as cited by France et al., 2007). Researchers discovered that iproniazid blocked resperine's behavioral effects and hypothesized that this effect occurred because iproniazid decelerated the oxidation of serotonin depleted by resperine, enabling the

release of serotonin at neural synapses. These findings led to the serotonin deficit hypothesis of depression. Later research revealed that resperine appeared to deplete catecholamines, expanding the serotonin deficit model of depression to incorporate catecholamines. Previous research had suggested that low monoamine levels caused depression and that increasing monoamines could reduce depression; however, Schildkraut (1965) has received credit for the catecholamine hypothesis of depression, and Coppen (1967) has received credit for the serotonin hypothesis of depression. These hypotheses have been influential in shaping biological explanations of depression and in guiding pharmaceutical treatments for depression (France et al., 2007).

Utilization of antidepressants to treat depression has been cited as evidence that depression has biological causes; however, antidepressants are not the only treatment for depression, and antidepressants, like all medications, can produce a placebo effect. Depression is treated through medication, psychotherapy, exercise, and other treatments because it has multiple causes. The chemical imbalance explanation for depression is overly simplistic because it does not capture the multi-causal nature of depression. To this day, there are no anatomical, chemical, or other biological tests that reliably distinguish the brains of depressed and non-depressed people; however, brain-imaging technologies have indicated that on average the brains of people with depression differ from the brains of people without depression (France et al., 2007).

Despite the lack of conclusive evidence supporting the chemical imbalance model for depression, there is widespread knowledge of the chemical imbalance model in the United States. Advertisements for SSRIs that promote a biological model of depression are a major contributor to this widespread knowledge. With the exception of New

Zealand, the United States is the only developed nation that permits direct-to-consumer advertising of prescription drugs (Grow, Park, & Han, 2006, as cited by France et al., 2007). Direct-to-consumer advertising is delivered through broadcast and print advertisements, manufacturers' Web sites, and promotional products (France et al., 2007). The model adopted by SSRI advertisements is based on the assumption that attributing psychological problems to biological factors is the most effective way to fight stigma and consequently encourage people to use medication. However, stigma reduction is not the primary agenda of pharmaceutical companies. Their top priority is to encourage the use of SSRIs for treatment and promotion of a biological understanding of depression is a means to accomplishing this goal (Deacon & Baird, 2009). In a study of American laypersons, participants reported widespread exposure to the chemical imbalance explanation of depression, most often through television. Presumably as a result of this widespread exposure, many participants endorsed the chemical explanation of depression (France et al., 2007). Consistent with the absence of direct-to-consumer advertising of prescription drugs in other developed nations, laypersons outside of the United States are more likely to agree with a psychosocial explanation of depression, rather than a biochemical one (France et al., 2007).

In a study of college students' perceptions of depression's etiology, Goldstein and Rosselli (2003) found that American college students endorsed biological explanations the most, followed by environmental explanations, and then psychological explanations. The researchers found an association between endorsement of a biological model and preference for psychotherapy, as well as greater empowerment in the form of help-seeking behavior. The biological model decreased stigma by reducing the belief that

depressed people are to blame for their condition. People with depression preferred the biological label more than people without depression did because it helped to legitimize their condition. Endorsement of a psychological model predicted greater belief that depressed individuals can cope with their depression on their own and an increased perception that depressed individuals are to blame for their condition than did endorsement of a biological model. The psychological model contributed to stigma by increasing desired social distance from individuals with depression. Additionally, supporters of the psychological model expressed greater belief that depressed people have poor friendships and take more than they give in relationships. The environmental model reduced stigma by decreasing desired social distance and the perception that individuals with depression are to blame for their condition. However, the researchers found an association between endorsement of the environmental model and greater belief that depressed people are violent, although they did not provide an explanation for this finding (Goldstein & Rosselli, 2003).

Deacon and Baird (2009) also examined how different etiological explanations affected college students' perceptions of depression. The researchers asked participants to imagine that they had been diagnosed with major depressive disorder and provided them with either a biochemical or a biopsychosocial explanation of depression. Participants who received a chemical imbalance explanation experienced less self-stigma than did participants who received a biopsychosocial explanation. The chemical imbalance explanation reduced stigma by decreasing feelings of personal and moral responsibility for the depression. However, the authors pointed out that self-blame has some beneficial effects; acceptance of personal responsibility helps foster healthy behaviors including the

use of psychosocial coping techniques and lifestyle interventions. The researchers also found that, compared to participants who received the biopsychosocial explanation, participants who received the chemical imbalance explanation ranked the explanation as having less credibility, had less faith in the effectiveness of psychosocial interventions, and reported a worse expected prognosis. Perceived prognosis improved when the proposed treatment method matched the provided etiology (i.e., medication as treatment for a biologically-caused disorder). However, Deacon and Baird (2009) argued that congruence between perceived etiology and treatment method does not necessarily improve prognosis in real life. Their research demonstrated that biochemical and biopsychosocial explanations are each associated with both positive and negative beliefs, and therefore suggests a cautious approach to promoting any particular etiological view (Deacon & Baird, 2009).

Another study with important implications for stigma reduction is Angermeyer, Matschinger, and Corrigan's (2004) investigation of the stigmatization of depression and schizophrenia. This study highlighted the de-stigmatizing effect of familiarity with mental illness, as findings revealed a negative relationship between familiarity and perceptions that individuals with depression and schizophrenia are dangerous. The researchers found an association between lower perception of dangerousness and less fear of people with depression and schizophrenia, as well as an association between less fear and less desired social distance. In addition, the researchers found that, since 1990, there has been an increase in pity and a slight increase in the desire to act aggressively toward the mentally ill, but the expression of fear and desire for social distance has not changed (Angermeyer et al., 2004).

The use of antidepressants for treating major depression has served as the foundation for the chemical imbalance explanation of depression. However, the chemical imbalance explanation is limited because it has failed to account for the multiple causes of and effective treatments for depression. Despite these limitations, pharmaceutical companies have generated a widespread knowledge of the biological explanation among American laypersons through SSRI advertising (France et al., 2007). Evidently, the biological explanation for depression has a mixed effect on stigma, and future research should aim to achieve an improved understanding of this dynamic (Angermeyer et al., 2004; Deacon & Baird, 2009; Goldstein & Rosselli, 2003). By promoting a more accurate understanding of depression's etiology and its relationship to stigma, advocacy groups can reduce stigmatization of major depression more effectively.

The Effect of Perceived Etiology on Stigmatization of Anorexia Nervosa

Anorexia nervosa affects approximately 0.6% of American adults. According to the National Institute of Mental Health,

> Anorexia nervosa is characterized by emaciation, a relentless pursuit of thinness and unwillingness to maintain a normal and healthy weight, a distortion of body image and intense fear of gaining weight, a lack of menstruation among girls and women, and extremely disturbed eating behavior. (NIMH, 2010)

Women are three times more likely than are men to develop anorexia nervosa. Anorexia nervosa has an estimated mortality rate of 0.56% per year, which is 12 times higher than the annual death rate for women ages 15-24, and the highest mortality rate of any mental disorder. Treating anorexia nervosa involves bringing the person back to normal weight,

treating the underlying psychological issues related to the eating disorder, and reducing behaviors and thoughts that lead to disordered eating (NIMH, 2010).

Some research has indicated that antidepressants, antipsychotics, and mood stabilizers may help to treat anorexia nervosa, perhaps because depression often co-exists with the disorder; however, there is a lack of evidence for the effectiveness of medication for anorexia nervosa treatment (NIMH, 2010). Although this research is inconclusive, it reflects a trend among eating disorder experts toward classifying eating disorders as having some biological basis; this perspective has not yet been adopted by the general public, and the general public tends to view anorexia nervosa as a personally or environmentally induced disorder (O'Hara & Smith, 2007). Treatment of anorexia nervosa typically consists of psychotherapy or a combination of medical services and psychotherapy (NIMH, 2010).

Despite the availability of treatment, most individuals with anorexia nervosa do not seek help (Stewart, Keel, & Schiavo, 2006). Part of the reason for this tendency is that the symptoms of anorexia nervosa are not entirely unwanted; many anorectics perceive their symptoms (i.e., restriction and over-exercise) as the solution, rather than as the problem. Anorectics view weight-loss as a goal and as a means of gaining control. The eating disorder becomes part of the anorectic's identity, and as a result, it is difficult to give up (Vitousek, Watson, & Wilson, 1998). However, many individuals with anorexia nervosa want to seek help, and research has indicated that stigmatization of anorexia nervosa reduces help-seeking behaviors (Stewart et al., 2006).

O'Hara and Smith (2007) turned to the news media to investigate how the public opinion of anorexia nervosa is formed. They evaluated 1 year of coverage about eating

disorders in seven U.S. daily newspapers and examined messages about the causes, severity, and treatment of eating disorders. The researchers found that most articles about eating disorders (48%) ran in arts and entertainment sections, profiled primarily young White women, and typically cited environmental causal explanations. The portrayal of eating disorders in the news media has a large effect on the general public's view of anorexia nervosa and reinforces the perception that anorexia nervosa is a largely culturally-induced disorder. Therefore, the media may play a role in stigmatizing eating disorders and preventing individuals with anorexia nervosa from receiving proper treatment (O'Hara & Smith, 2007).

Other research has found that blame is a key contributing factor to public stigma toward anorectics. Nationwide surveys in 1998 and 2003 demonstrated that the public views people with eating disorders as responsible for their disorder. In a vignette study describing either an anorexic, depressed, schizophrenic, asthmatic, or healthy target, participants perceived the target with anorexia nervosa as most to blame for his/her condition, as best able to put him/herself together if he/she wanted to, and as using his/her disorder to attract attention. Therefore, anorexia nervosa is stigmatized through perceived responsibility (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000). Another study showed that laypersons viewed anorexia nervosa as significantly more controllable than anorectics themselves did. Both laypersons and anorexia patients attributed the disorder to psychological factors such as emotional state, personality, and mental attitudes (Holliday, Wall, Treasure, & Weinman, 2005).

Anorectics are perceived as having high self-control because the obsessive monitoring of food intake associated with anorexia nervosa appears to require high self-

control. The strong association between anorexia nervosa and self-control helps to explain why anorectics are blamed for their disorder. The view of anorexia nervosa as a self-inflicted illness decreases empathy and increases anger and distrust toward individuals with anorexia nervosa (Stewart et al., 2006). Although the perceived self-control of anorectics contributes to stigmatizing attitudes, research has indicated that there are ways in which the public views anorectics positively due to their high self-control. For example, one study showed that people sometimes respect individuals with eating disorders, such as by viewing them as ascetic (Crisp, 2005). Another study showed that although family members and friends of eating disorder patients reported concern for the patient, 50% also expressed envy for the patient's self-control (Branch & Eurman, 1980 as cited by Stewart et al., 2006). Thus, the high self-control associated with anorexia nervosa appears to have both positive and negative effects on the public's perception of the disorder.

Despite perceptions that anorectics have control over their disorder, according to O'Hara and Smith (2007), eating disorder specialists are increasingly conceptualizing anorexia nervosa as a mental illness caused by both genetic and social factors. This view contrasts with the general public's perception that anorexia nervosa is a culturally induced disorder. An initiative by the Eating Disorders Coalition to fight stigma with science attempts to combat stigmatization of eating disorders through endorsement of a biological model. Cynthia Bulik, the vice president of the Eating Disorders Coalition, has conducted research to show that both genetics and the environment contribute to eating disorders. By educating the public on the link between genetics and eating disorders, the

Eating Disorders Coalition has attempted to demonstrate that eating disorders are not a choice (Eating Disorders Coalition, 2011).

Despite these efforts, the finding that participants believed biological factors played a smaller role in the development of anorexia nervosa than in the development of schizophrenia and asthma indicated that the public does not strongly attribute anorexia nervosa to biological causes (Crisp et al., 2000). The public has not adopted a biological understanding of anorexia nervosa because the disorder is not typically treated through pharmacotherapy, and, for other mental illnesses, pharmacotherapy has been cited as evidence for biological etiology. Furthermore, because pharmacotherapy is not a common treatment for anorexia nervosa, advertising by pharmaceutical companies, which has been shown to reduce stigma and raise awareness about other mental illnesses, does not target anorexia nervosa (Stewart et al., 2006).

Stewart et al. (2008) investigated how college students stigmatized anorexia nervosa. The researchers assigned participants to one of four conditions in which they read a vignette that described an individual with anorexia nervosa, depression, schizophrenia, or mononucleosis. Each vignette appeared the same, with the exception of the classification of the illness it described. Results indicated that participants believed anorexia nervosa occurred more from biological factors and a lack of social support than poor living habits. The finding that participants attributed anorexia nervosa to biological factors countered the researchers' hypothesis that participants would primarily attribute anorexia nervosa to environmental factors. However, the researchers speculated that the nature of the sample contributed to this finding; the sample only consisted of female undergraduate students, who likely had more knowledge about eating disorders than the

general population. Participants attributed fewer positive characteristics to targets described as having anorexia nervosa than to targets described as having schizophrenia or mononucleosis. In addition, participants reported more discomfort interacting with someone who has anorexia nervosa than with someone who has depression or mononucleosis. Having previous contact with an individual with anorexia nervosa predicted more comfort interacting with the anorexic target (Stewart et al., 2008). For individuals who have anorexia nervosa, consequences of stigma include lower self-esteem, more shame, greater difficulty recovering, and higher likelihood of relapse (Stewart et al., 2006).

In a study exploring how sociocultural and biological explanations of anorexia nervosa influenced attitudes toward the disorder, Crisafulli, Von Holle, and Bulik (2008) randomly assigned nursing students to conditions in which they read information about anorexia nervosa that attributed the disorder to either sociocultural or biological factors. Participants who read the biological explanation of anorexia nervosa blamed anorectics for their condition less frequently than did participants who read the sociocultural explanation. This finding suggested that a shift toward a more biological conceptualization of anorexia nervosa can help lead to less blame toward individuals with anorexia nervosa and, subsequently, lower levels of stigma. Moreover, attribution theory implies that endorsement of the biological model of anorexia nervosa can help evoke pity and helping behaviors toward individuals with anorexia nervosa. Efforts to de-stigmatize schizophrenia over the past several decades have used a similar method of promoting the conceptualization that it is a biological disorder (Crisafulli et al., 2008). However, as previously stated, this approach can lead to unintended consequences, including greater

desire for social distance (Griffiths & Christensen, 2004), increased dangerousness stigma (Bennett et al., 2008), increased benevolence stigma (Corrigan & Watson, 2004), worse expected prognosis (Bennett et al., 2008), and the perception that individuals with anorexia nervosa are inherently different (Phelan, 2002).

Roehrig and McLean (2009) examined how eating disorders are stigmatized differently from depression and found that participants stigmatized individuals with eating disorders significantly more than they stigmatized individuals with depression. Participants read a vignette describing an individual with anorexia nervosa, bulimia nervosa, or depression. Participants perceived the targets with anorexia nervosa and bulimia nervosa as more fragile, more responsible for their disorder, and more likely to use their disorder to get attention than they did the target with depression. Most participants reported that they admired certain aspects of eating disorders and that they would want to imitate eating disordered behavior, which indicated some feelings of envy toward individuals with eating disorders (Roehrig & McLean, 2009). Roehrig and McLean (2009) did not assess desired social distance. As Rusch et al. (2010) stressed, social distance is more closely linked to discrimination than is perceived responsibility, and therefore, may serve as a better predictor of stigma than does perceived responsibility.

The Present Study

Despite the prevalence of mental health problems on college campuses, relatively few college students seek treatment for mental disorders (Zivin et al., 2009). Two strong barriers that prevent college students from seeking treatment are self-stigma (Corrigan & Kleinlein, 2005) and personal stigma (Eisenberg, 2009). A stronger understanding of the

factors that contribute to stigmatization of mental disorders affecting college students is necessary for stigma reduction efforts to be effective (Jorm & Griffiths, 2008). Previous research has indicated that environmental causal explanations for mental illness intensify stigma by increasing the perception that individuals are to blame for their condition (Corrigan & Watson, 2004; Crisafulli et al., 2008; Crisp et al., 2000). Biological causal explanations for mental illness help to alleviate blame but contribute to stigma by increasing social distancing and perceptions of dangerousness, gravity, and disparity (Bennett et al., 2008; Corrigan et al., 2002; Corrigan & Watson, 2004; Griffiths & Christensen, 2004). Few studies have directly explored how altering causal explanations can uniquely influence the stigmatization of different mental illnesses by examining multiple disorders within the same study. The present research sought to examine how biological and environmental causal explanations affect the stigmatization of college students who have major depression and anorexia nervosa.

Previous research has suggested that the two disorders that this study investigated vary in their perceived etiology. Evidence has indicated that biological factors contribute to the development of major depression, although it is a multi-faceted disorder (France et al., 2007). There is a widespread public knowledge of the biological model of depression in the United States because of treatment through SSRIs and direct-to-consumer advertising by pharmaceutical companies (France et al., 2007). On the other hand, anorexia nervosa is generally conceptualized by the general public as environmentally induced (Crisp et al., 2000), although there has been a recent shift toward adopting a more biological understanding of the disorder among professionals (O'Hara & Smith, 2007).

This research sought to advance the literature by directly exploring how biological and environmental causal explanations have different effects on the stigmatization of major depression and anorexia nervosa. Using an experimental design, participants were presented with a hypothetical female target with a diagnosis of either major depression or anorexia nervosa. These disorders were each explained in either biological or environmental terms, yielding four conditions. Responses to the vignettes assessed participants' desire for social distance (desired distance that individuals without a particular mental disorder wish to maintain from individuals who have that disorder), treatment recommendations (perceived utility of treatments), overall stigma (personal responsibility beliefs, pity, and anger targeted at individuals with mental disorders), and causal attributions (blame attributed to individuals with mental disorders for their condition). Familiarity with mental illness was also assessed to examine whether familiarity affected participants' responses to the vignettes.

Consistent with findings by Roehrig and McLean (2009), it was hypothesized (1) that participants would stigmatize the target with anorexia more than they would stigmatize the target with depression. In accordance with previous research indicating widespread endorsement of biological attributions for depression (Goldstein & Rosselli, 2003) and widespread endorsement of environmental attributions for anorexia (Crisp et al., 2000), it was hypothesized (2) that participants would endorse biological attributions more for depression than for anorexia, and would endorse environmental attributions more for anorexia than for depression. Previous research has indicated that biological explanations reduce stigma for both depression (Deacon & Baird, 2009; Goldstein & Rosselli, 2003) and anorexia (Crisafulli et al., 2008), but has not directly compared the

two disorders using the same causal explanations and the same measures. Therefore, it was hypothesized (3) that biological and environmental explanations would have different effects on stigma toward depression versus anorexia, but could not predict the nature of differences based on the existing literature.

In agreement with Stewart et al. (2008), it was also hypothesized (4) that participants would desire more social distance from the target with anorexia than from the target with depression. Consistent with Griffiths and Christensen's (2004) finding that biological causal explanations can increase desired social distance, it was hypothesized (5) that a biological explanation for both depression and anorexia would increase participants' desired social distance from the target described in the vignette. Finally, in agreement with previous research indicating that higher familiarity is associated with less desire for social distance (Angermeyer et al., 2004), the present study hypothesized (6) that participants who reported greater familiarity with mental illness would express lower levels of stigmatizing responses toward the target described in the vignette than would participants who reported less familiarity with mental illness. No specific hypotheses about treatment recommendations were made, as few researchers have explored this dimension. Treatment recommendations were examined for exploratory purposes, and were expected to reflect both stigma and causal beliefs. Previous research has rarely examined whether men versus women have different stigmatizing responses to mental illness, and thus, all analyses included gender to examine possible differences in causal attributions, social distancing, stigmatization, and treatment recommendations.

Method

Participants

A total of 125 participants participated in this study; the sample consisted of 38 males (30.4%) and 87 females (69.6%). One-hundred-and-four participants (83.2%) identified as White, 8 participants (6.4%) identified as Hispanic or Latino, 7 participants (5.6%) identified as Asian or Asian American, 3 participants (2.4%) identified as Black or African American, and 3 participants (2.4%) identified as other. All participants were enrolled in an undergraduate institution at the time of participation; 115 participants (92%) attended Connecticut College, and 10 participants (8%) attended Ithaca College. Forty-six freshman (36.8%), 40 sophomores (32%), 18 juniors (14.4%), and 21 seniors (16.8%) participated in this study. Forty-five participants (36%) reported that they were psychology or neuroscience majors, 59 participants (47.2%) reported that they had a major but it was not psychology or neuroscience, and 21 participants (16.8%) reported that they had not yet decided on a major.

Of the participants in the two major depression conditions (major depression with a biological casual explanation and major depression with an environmental causal explanation; *n*=65, 52%), 11 (16.9%) reported that they have had major depression, and of the participants in the two anorexia nervosa conditions (anorexia nervosa with a biological causal explanation and anorexia nervosa with an environmental causal explanation; *n*=60, 48%), 7 (10.8%) reported that they have had anorexia nervosa. Across all four conditions, 34 participants (27.2%) reported that they had been treated for a psychological condition, 38 participants (30.4%) reported that they had seen a counselor from Student Counseling Services for help with a psychological problem, and 57

participants (45.6%) reported that they had seen a therapist from outside Student Counseling Services for help with a psychological problem.

Materials

Materials included four versions of a vignette (see Appendix A); the Social Distancing and Treatment Recommendations Scale (see Appendix B); the Personal Responsibility Beliefs, Pity, and Anger Questionnaire (see Appendix C), the Causal Attributions Scale (see Appendix D), the Familiarity with Mental Illness Questionnaire (see Appendix E), and a demographics questionnaire (see Appendix F).

Mental Illness Vignettes. A vignette adapted from the one used by Stewart et al. (2008) described a college student named Kelly with either major depression or anorexia nervosa. The vignette included either a biological or environmental causal explanation for Kelly's disorder. The biological causal explanation stated, "Recent research emphasizes that [major depression/anorexia nervosa] is a medical illness that can result from biological factors like a chemical imbalance in the brain or a genetic predisposition"; the environmental causal explanation stated, "Recent research emphasizes that [major depression/anorexia nervosa] is a common mental illness that can result from environmental factors like stressful life experiences or relationship difficulties." Each version of the vignette featured identical descriptions of Kelly; the vignettes only differed in their classification of Kelly's disorder (either major depression or anorexia nervosa) and their description of the disorder's etiology (either biological or environmental).

The four different versions of the vignette corresponded to the four conditions of this study. The major depression with a biological causal explanation condition included 34 participants (27.2%), the major depression with an environmental causal explanation

condition included 31 participants (24.8%), the anorexia nervosa with a biological causal explanation condition included 32 participants (25.6%), and the anorexia nervosa with an environmental causal explanation condition included 28 participants (22.4%). Each condition had an approximately the same proportion of female to male participants.

Social Distancing Scale. The Social Distancing Scale measured social distance by assessing participants' willingness to make contact with Kelly through six questions. Ratings for each item ranged from 0 (definitely willing) to 3 (definitely unwilling). In the original version of the scale, the summation of items resulted in a score between 0-15, with a higher score indicating greater desire for social distance. The present study adapted Jorm and Griffiths' (2008) survey to increase its relevance to a college student population. Instead of asking about willingness to move next door to the target described in the vignette, the present study asked about willingness to be roommates with the target described in the vignette. Additionally, instead of assessing willingness to work closely on a job with the target, the present study examined willingness to work closely on a group project with the target. The present study excluded the item from the original scale that examined willingness to have the target marry into the family and added two items that assessed willingness to eat a meal with the target and willingness to elect the target to be head of a student organization. The present study's version of the scale had a total of six items, and a mean score was calculated. The revised scale had a Cronbach's alpha of .85.

Treatment Recommendations Scale. Next, participants made treatment recommendations for Kelly through six questions that were developed for the present study. These questions asked if Kelly should receive medication, receive counseling from

her college's student health center, receive therapy from a therapist outside her college, turn to a friend for support, seek support from Student Disabilities Services, or cope with her problem without seeking treatment. Participants ranked how strongly they recommended each treatment method on a Likert scale ranging from 0 (not at all) to 4 (very strongly), and a mean was calculated. The scale had only a moderate Cronbach's alpha (α = .687). Inter-item correlations revealed that without the item "cope with her problem without seeking treatment," the Cronbach's alpha would increase (α = .74), and therefore, subsequent analyses did not include this item.

Personal Responsibility Beliefs, Pity, and Anger Questionnaire. The Personal Responsibility Beliefs, Pity, and Anger Questionnaire came from Corrigan, Markowitz, Watson, Rowan, and Kubiak's (2003) Item Wording for Familiarity with Mental Illness, Personal Responsibility Beliefs, Emotional Responses, and Helping and Rejecting Responses. Corrigan et al.'s (2003) original survey asked questions about familiarity with mental illness, personal responsibility beliefs, pity, anger, fear, helping, and coercion-segregation. The present study used the personal responsibility beliefs, pity, anger, and familiarity with mental illness sections, but administered the familiarity with mental illness section later on, separate from the other parts of the questionnaire. Because the familiarity with mental illness questions asked participants about their personal experiences, and not about the specific individuals in the vignettes, it was less disruptive to the continuity of the vignette assessment to administer them at the end, directly preceding the demographics questionnaire.

The Personal Responsibility Beliefs, Pity, and Anger Questionnaire included a total of nine questions that participants responded to on a Likert scale from 1 to 9. A

mean score was calculated for each participant. In the present study, The Personal Responsibility Beliefs, Pity, and Anger Questionnaire had a Cronbach's alpha of .708. In Corrigan et al.'s (2003) study the personal responsibility subscale had a Cronbach's alpha of .70, the pity subscale had a Cronbach's alpha of .75, and the anger subscale had a Cronbach's alpha of .89. In the present study, the personal responsibility subscale had a Cronbach's alpha of .655, the pity subscale had a Cronbach's alpha of .676, and the anger subscale had a Cronbach's alpha of .842.

Causal Attributions Scale. Next, participants completed an adapted version of the Causal Attributions Scale (Jorm & Griffiths, 2008). The Causal Attributions Scale originally consisted of nine questions that measured participants' beliefs about the likelihood of possible causes of Kelly's disorder. When Jorm and Griffiths (2008) administered the Causal Attributions Scale, they asked participants to choose from a Likert Scale with the choices "very likely," "likely," "not likely," "depends," and "don't know" for each item. The present study asked participants to rank the likelihood of each cause on a Likert scale from 0 (very unlikely) to 4 (very likely) to make the response scale more parallel to response scales on other measures. Jorm and Griffith's (2008) original version of the scale included the following causal attributions: virus or other infection; allergy or reaction; day to day problem such as stress, family arguments, difficulties at work or financial difficulties; the recent death of a close friend or relative; some recent traumatic event such as bushfires threatening your home, a severe traffic accident or being mugged; problems from childhood such as being badly treated or abused, losing one or both parents when young, or coming from a broken home; inherited or genetic disorders; being a nervous person; having weakness of character.

The present study changed the question assessing the likelihood that "some recent traumatic event such as bushfires threatening your home, a severe traffic accident, or being mugged" caused Kelly's illness to "some recent traumatic event such as a house fire, a severe traffic accident, or being mugged" because house fires are more common in this region of the country than are bush fires. The present study incorporated three additional items into the Causal Attributions Scale: stressful life experiences, a chemical imbalance in the brain, and relationship difficulties. These three new items corresponded to the causal explanations utilized in the biological and environmental vignettes. The Causal Attributions Scale had already included an item ("genetics or inherited") that corresponded to the genetic predisposition explanation in the biological version of the vignette, so the present study did not add a question to match that explanation. However, the present study altered to wording on that item to "a genetic predisposition" to maintain consistency with the wording utilized in the vignette. The three items that the present study added increased the scale to a total of 12 questions.

Jorm and Griffiths (2008) only analyzed individual items, so they did not report scale reliability. The present study formed theoretically derived subscales and computed subscale reliabilities on the adapted version of this measure. Item content guided the formation of a biological subscale and an environmental/personal subscale. The biological subscale, including all biological items, had a moderate Cronbach's alpha (α = .621). After examining inter-item correlations, the patterns of findings suggested two dimensions: virus/allergy and genetic/chemical. The virus/allergy dimension consisted of the items "virus or other infection" and "allergy or reaction," and had a Cronbach's alpha of .778. The genetic/chemical subscale consisted of the items "genetic predisposition"

and "chemical imbalance in the brain," and had a Cronbach's alpha of .752. The environmental/personal subscale had a Cronbach's alpha of .764. The present study analyzed the total score, as well as the biological, genetic/chemical, and environmental/personal subscales.

Familiarity with Mental Illness. Next, participants completed the Familiarity with Mental Illness Questionnaire, taken from Corrigan et al. (2003), who took the Familiarity with Mental Illness questions from the Level of Contact Report (Holmes et al., 1999, as cited by Corrigan et al., 2003). The Familiarity with Mental Illness Questionnaire consisted of seven yes/no items that participants answered based on their own personal experiences. After coding the items (no = 0, yes = 1), Corrigan et al. (2003) formed an index ranging from 0-7. In the present study, the Familiarity with Mental Illness scale had a Cronbach's alpha of .612, which was almost identical to the original published Cronbach's alpha. The present study also examined intimate familiarity in some analyses, which included the items "I have a friend who has a severe mental illness," "I have a relative who has a severe mental illness," and "I live with a person who has a severe mental illness"; the intimate familiarity subscale had a Cronbach's alpha of .540. Both scales helped explore the relationship between familiarity and responses to mental illness.

Demographics Questionnaire. Lastly, this study included a demographics questionnaire that asked about participants' sex, race, class year, major, and personal experience with mental illness and treatment.

Procedure

Students enrolled in Psychology 101 and 102 at Connecticut College signed up to participate in a study titled "Perceptions of Mental Illness" and received course credit as compensation for their participation. To attain more male participants, additional recruitment targeted males from the participant pool at Connecticut College and from the Connecticut College Ski Team. Traditional recruitment methods, as well as these efforts to recruit more male participants at Connecticut College, yielded 115 participants (92% of the total sample). Additional efforts to recruit male participants targeted undergraduate students at Ithaca College by distributing the survey through personal contacts via e-mail. This recruitment method yielded 10 participants (8% of the total sample).

The researcher randomly assigned participants to one of the four conditions: major depression with a biological causal explanation, major depression with an environmental causal explanation, anorexia nervosa with a biological causal explanation, and anorexia nervosa with an environmental causal explanation. The researcher e-mailed participants the link to the survey on surveymonkey.com. The survey included an informed consent form on which participants electronically provided consent by clicking "I agree to participate in this study" or "I do not agree to participate in this study" (see Appendix G). After they consented to participation in this study, participants read a vignette and filled out the four surveys described earlier. Following the surveys, participants filled out a demographics questionnaire. After completing the demographics questionnaire, participants read the debriefing form that explained the goals of this research and provided information about Counseling Services at Connecticut College (See Appendix H).

Results

The Effects of Causal Explanation and Disorder Types

Preliminary Descriptive Analyses. Table 1 shows the descriptive statistics for the five primary dependent variables (social distance, biological attributions, environmental/personal attributions, total stigma, and treatment recommendations). For social distancing participants used the full range of responses (0-3), but generally reported a high rating of willingness to engage with the target. For both chemical/genetic and environmental/personal attributions participants also used the full range of responses (0-4). Participants endorsed both biological and environmental causes for the disorders presented in this study with approximately equal strength. Participants did not use the full range of responses for the total stigma scale (1-9), but their scores appeared to reveal more stigmatizing responses than the social distancing scale revealed. Participants reported moderately stigmatizing attitudes about the hypothetical individuals with mental illness in this study. For treatment recommendations, most participants endorsed the need for the hypothetical targets to receive some sort of support or treatment, but there was variation on this scale as well.

Dependent Variable	Minimum	Maximum	Mean	Standard Deviation
Social Distance	0	3	2.46	.52
Biological Attributions	0	4	3.07	.91
Environmental/Personal Attributions	1	4	2.97	.58
Total Stigma	1.89	6.11	3.89	.94
Treatment Recommendations	.20	4	2.61	.78

Table 1. Descriptive Statistics for the Primary Dependent Variables

Primary Analysis on Composite Variables. A 2 (disorder; depression, anorexia) x 2 (cause; biological, environmental) x 2 (gender; male, female) MANOVA on all of the primary composite dependent variables (social distance, biological attributions, environmental/personal attributions, total stigma, and treatment recommendations) indicated a multivariate main effect for disorder, Wilks's $\lambda = .696$, $F(5, 114) = 9.88$, $p < .001$, partial eta squared = .30**.** The MANOVA also revealed a significant interaction between disorder and cause, Wilks's $\lambda = .882$, $F(5, 113) = 3.03$, $p = .013$, partial eta squared = .12. The MANOVA did not find a multivariate main effect for cause or gender, or any interactions.

Follow-up univariate tests revealed a significant main effect for disorder on biological attributions, $F(1, 117) = 41.56$, $p < .01$, partial eta squared = .26. As predicted, participants made stronger biological attributions about the target with depression (M = 3.51) than about the target with anorexia (M = 2.54). Univariate tests also found a significant main effect for disorder on total stigma, $F(1, 117) = 11.61$, $p = .001$, partial eta squared = .09. As predicted, participants expressed more stigma toward the target with anorexia (M = 4.14) than toward the target with depression (M = 3.58).

In addition univariate tests indicated a significant interaction effect for disorder by cause on total stigma, $F(1, 117) = 9.51$, $p = .003$, partial eta squared = .08**.** Simple effects tests supported the prediction that biological versus environmental explanations would have different effects on stigmatizing responses to depression versus anorexia: Participants expressed more stigma toward the depressed target after reading an environmental causal explanation than after reading a biological causal explanation, $F(1,117) = 8.91$, $p < .001$ (see Figure 1). This effect did not emerge in the

biological/environmental comparison for the anorexic target, $F(1, 117) = 2.01$, $p = .16$, where the pattern of means appeared to reverse. When participants read a biological explanation, they stigmatized anorexia more than depression, $F(1, 117) = 22.37$, $p < .001$; when participants read an environmental explanation, they did not stigmatize the two disorders differently, $F(1, 117) = .05$, $p = .83$. The lowest level of stigma occurred after participants read about a target with depression that used a biological causal explanation for the disorder, and the highest level of stigma occurred after participants read about a target with anorexia that used a biological explanation for the disorder. Participants moderately stigmatized the targets with depression and anorexia after reading an environmental causal explanation.

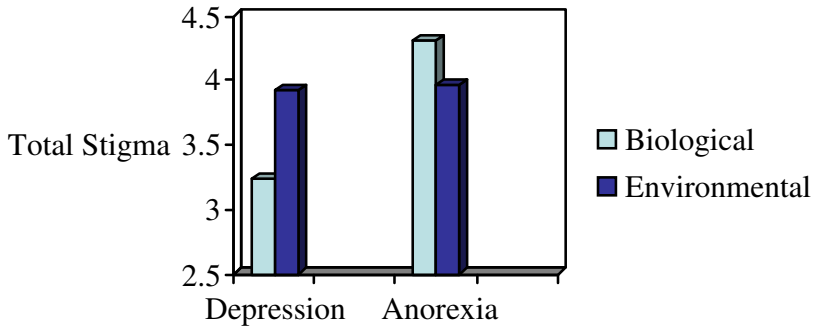

Figure 1. The effect of biological versus environmental causal explanations on stigma toward depression and anorexia

Although the MANOVA did not find a multivariate effect for causal explanation, univariate follow-up tests were examined for exploratory purposes and revealed a marginal univariate effect for social distancing, $F(1, 117) = 3.40$, $p = .068$, partial eta squared = .03. Participants desired somewhat more social distance from the target after

reading an environmental causal explanation (M = 0.65) than after reading a biological causal explanation (M = .47).

Analyses on the Facets of Social Distancing. To further explore the hypotheses that participants would desire more social distance from the target with anorexia than from the target with depression, and that a biological explanation for both depression and anorexia would increase participants' desired social distance from the target described in the vignette, a 2 (disorder; depression, anorexia) x 2 (cause; biological, environmental) x 2 (gender; male, female) MANOVA was conducted on the six social distancing items ("be roommates with Kelly," "spend an evening socializing with Kelly," "make friends with Kelly," "work closely on a group project with Kelly," "eat meals with Kelly," and "elect Kelly to be head of a student organization"). The MANOVA found a significant multivariate main effect for disorder, Wilks's λ = .862, $F(6, 112) = 2.98$, $p = .010$, partial eta squared = .14, as well as a marginal multivariate effect for cause, Wilks's λ = .91, $F(6, 112) = 1.96$, $p = .078$, partial eta squared = .10.

Follow-up univariate tests for disorder indicated a trend for socializing, $F(1, 117) = 3.11$, $p = .080$, partial eta squared = .03. As shown in Figure 2, participants reported somewhat less social distance in the form of greater willingness to socialize with an anorexic target than with a depressed target. Univariate tests for disorder also revealed a trend for eating, $F(1, 117) = 3.06$, $p = .083$, partial eta squared = .03. Participants reported somewhat less social distance in the form of greater willingness to eat with a depressed target than with an anorexic target. For cause, results indicated a significant main effect for socialize, $F(1, 117) = 4.35$, $p = .039$, partial eta squared = .04. Participants reported less social distance in the form of greater willingness to socialize with the target

after reading a biological causal explanation than after reading an environmental causal explanation. Results for cause also indicated a significant main effect for friends, $F(1, 117) = 6.74$, $p = .011$, partial eta squared = .05. Participants reported less social distance in the form of more willingness to become friends with the target after reading a biological causal explanation than after reading an environmental causal explanation. Univariate tests for cause also indicated a marginal effect for eat, $F(1, 117) = 3.06$, $p = .078$, partial eta squared = .03. Participants reported somewhat less social distance in the form of greater desire to eat with the target after reading a biological causal explanation than after reading an environmental causal explanation (see Figure 2).

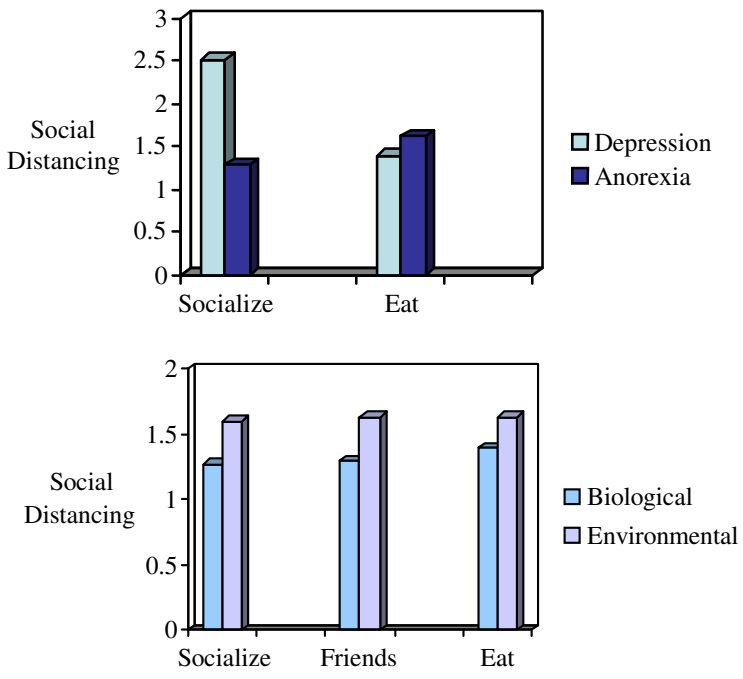

Figure 2. Differences in the facets of social distancing based on disorder (depression, anorexia) and cause (biological, environmental)

Analyses on the Facets of Biological Causes. To further examine the hypothesis that participants would make stronger biological attributions for depression than for anorexia, and to more fully examine the effect of the biological manipulation, a 2 (disorder; depression, anorexia) x 2 (cause; biological, environmental) x 2 (gender; male, female) MANOVA was conducted on the four biological causal attribution items. These items explored the range of possible biological attributions (virus, allergy, chemical, and genetic). This analysis found a significant multivariate effect for disorder, Wilks's λ = .673, $F(4, 114) = 13.86$, $p < .001$, partial eta squared = .33. The MANOVA did not find a multivariate main effect for cause or gender, or any interactions.

Univariate follow-up tests found a significant main effect for disorder on both genetic explanations, $F(1, 117) = 18.23$, $p < .001$, partial eta squared = .01, and chemical explanations, $F(1, 117) = 55.79$, $p < .001$, partial eta squared = .00. Participants endorsed genetic explanations for depression (M = 3.27) more than for anorexia (M = 2.39); likewise, participants agreed with chemical explanations for depression (M = 3.78) more than for anorexia (M = 2.68). Tests did not find significant differences for virus and allergy explanations by disorder. It is important to note that there were no multivariate or univariate main effects for causal explanation for the specific biological causes manipulated in the vignette (genetic, chemical). These findings suggest that although participants may have noticed the causal explanation for Kelly's disorder (as evidenced by other main effects and interactions involving causal explanation), they did not adopt the manipulation as a general causal explanation for the disorder.

Analyses on the Facets of Environmental and Personal Causes. A 2 (disorder; depression, anorexia) x 2 (cause; biological, environmental) x 2 (gender; male, female)

MANOVA on the seven environmental and personal causal attribution items explored whether some of the attributions combined in the large scale analyzed earlier (day to day stress, death of a loved one, stressful life experiences, trauma, problems from childhood, nervousness, or relationship problems) played a larger role than did others. This analysis found a significant multivariate effect for disorder, Wilks's $\lambda = .802$, $F(7, 111) = 3.93$, $p = .001$, partial eta squared = .20. The MANOVA did not find a multivariate main effect for cause or gender, or any interactions.

Univariate follow-up tests found a significant main effect for disorder on death of a loved one, $F(1, 117) = 21.48$, $p < .001$, partial eta squared = .16. Participants perceived death of a loved one as more likely to cause depression ($M = 3.60$) than anorexia ($M = 2.91$). Univariate ANOVAs also revealed a significant main effect for disorder on trauma, $F(1, 117) = 8.57$, $p = .004$, partial eta squared = .07. Participants viewed trauma as more likely to cause depression ($M = 3.51$) than anorexia ($M = 2.96$). Results indicated a marginal effect for disorder on problems from childhood, $F(1, 117) = 3.28$, $p = .073$, partial eta squared = .03. Participants marginally believed that problems from childhood caused depression ($M = 3.59$) more than anorexia ($M = 3.31$). Out of the causal explanations offered, participants viewed several as more likely for depression than for anorexia, but considered none of them more likely for anorexia than for depression.

Although there was no multivariate effect for causal explanation, there were significant univariate effects that are reported here to examine the effect of the causal manipulation: for loss of a loved one, $F(1, 117) = 4.34$, $p = .039$, partial eta squared = .036, and relationship problems, $F(1, 117) = 4.21$, $p = .042$, partial eta squared = .035, as well as trends for trauma, $F(1, 117) = 2.86$, $p = .093$, partial eta squared = .024, and

childhood problems, $F(1, 117) = 2.99$, $p = .086$, partial eta squared = .025. In all cases, after participants read an environmental/personal causal explanation for the target's problem, they rated these environmental/personal causes higher than they did after they read a biological causal explanation for the target's problem. This finding provides support for the causal explanation manipulation.

Analyses on the Facets of Stigma. To further explore how participants stigmatized depression versus anorexia and how causal explanation may have had an influence, a MANOVA on the three stigma subscales explored components of stigmatizing responses and found a main effect for disorder on the multivariate level, F (3, 115) = 8.39, $p < .001$, partial eta squared = .18. The MANOVA also found an interaction effect for disorder by cause on the multivariate level, F (3, 115) = 3.75, p = .013, partial eta squared = .09. Univariate tests revealed a disorder main effect for responsibility, F (1, 117) = 23.44, $p < .001$, partial eta squared = .17, and anger, F (1, 117) = 7.01, p = .009, partial eta squared = .35. Participants attributed more responsibility to the target with anorexia than to the target with depression; they also felt more anger toward the target with anorexia than to the target with depression (see Figure 3).

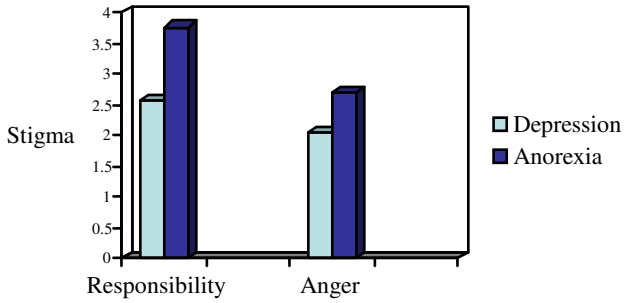

Figure 3. Responsibility and anger toward depression versus anorexia

In addition, tests revealed a significant disorder by cause interaction effect for pity, $F(1, 117) = 7.75$, $p = .006$, partial eta squared = .06. Simple effects tests revealed that participants felt more pity for the depressed target after reading the environmental causal explanation than after reading the biological causal explanation, $F(1, 117) = 5.89$, $p = .017$, partial eta squared = .05 (see Figure 4). Pity for the anorexic target did not differ over biological and environmental causes, $F(1, 117) = 2.35$, $p = .13$, and the pattern of means appeared to be opposite to the pattern for depression.

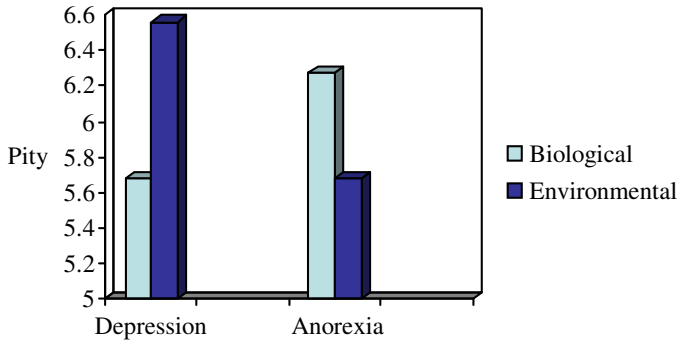

Figure 4. The effect of biological versus environmental causal explanations on pity toward a target with depression versus anorexia

Results indicated a marginal disorder by cause interaction effect for responsibility, $F(1, 117) = 3.72$, $p = .056$, partial eta squared = .03. Participants attributed more personal responsibility to the anorexic target than to the depressed target regardless of cause, but the difference was notably larger when *biological* explanations were offered ($M_{\text{anorexic}} = 3.94$; $M_{\text{depressed}} = 2.26$), $F(1, 117) = 24.34$, $p = .000$, partial eta squared = .17, compared to when environmental explanations were offered ($M_{\text{anorexic}} = 3.59$; $M_{\text{depressed}} = 2.87$), $F(1, 117) = 4.01$, $p = .048$, partial eta squared = .03. For depression, participants perceived personal responsibility as marginally higher when environmental explanations

were offered (M = 2.87) compared to biological (M = 2.26), $F(1, 117) = 3.21$, p = .076, partial eta squared = .027. Means did not differ for anorexia ($M_{biological}$ = 3.94; $M_{environmental}$ = 3.60), $F(1, 117) = .92$, $p = .339$.

Results did not point to a multivariate main effect for gender, but suggested a significant univariate effect for gender on anger, $F(1, 117) = 3.99$, $p = .048$, partial eta squared = .03. These results are discussed for exploratory purposes to help develop hypotheses for future investigations of the rarely studied topic of gender differences in mental illness stigmatization. Female participants expressed more anger (M = 2.61) than did male participants (M = 2.12) toward the target in all vignettes. The results also indicated that there was a marginal disorder by gender interaction for anger, $F(1, 117) = 3.73$, $p = .056$, partial eta squared = .03. Simple effects tests revealed that females expressed especially high anger toward the target with anorexia (M = 3.17) compared to the target with depression (M = 2.05), $F(1, 117) = 17.30$, p < .001, partial eta squared = .129, whereas males reported little difference in anger toward the target with anorexia (M = 2.21) compared to the target with depression (M = 2.03), $F(1, 117) = .18$, $p = .669$, partial eta squared = .129. Females expressed more anger than males toward anorexia, $F(1, 117) = 7.34$, $p = .008$, partial eta squared = .059, but there was no gender difference in anger toward the depressed target, $F(1, 117) = .002$, $p = .962$, partial eta squared = .000.

Analyses on the Facets of Treatment Recommendations. Although the literature did not allow for specific hypotheses about treatment recommendations, it was expected that these might differ over the different conditions and could be related to both stigma and causal attributions. To explore this possibility, a 2 (disorder; depression,

anorexia) x 2 (cause; biological, environmental) x 2 (gender; male, female) MANOVA on the full set of possible treatment recommendations (medication, college counseling, outside therapist, friend support, or student disabilities services) was conducted. Only marginal multivariate main effects were revealed for cause, Wilks's $\lambda = .913$, $F(5, 113) = 2.16$, $p = .063$, partial eta squared = .087, and for gender, Wilks's $\lambda = .912$. $F(5, 113) = 2.172$, $p = .062$, partial eta squared = .088. Univariate effects were examined for exploratory purposes but results should be interpreted cautiously.

Follow-up tests for the two marginal multivariate effects revealed a significant univariate effect for cause on medication, $F(1, 117) = 5.06$, $p = .026$, partial eta squared = .041. Participants recommended medication significantly more strongly after reading a biological causal explanation of the target's disorder ($M = 2.32$) than after reading an environmental causal explanation of the target's disorder ($M = 1.84$). Follow-up tests also revealed a significant univariate effect for gender on medication, $F(1, 117) = 3.92$, $p = .05$, partial eta squared = .032. Male participants recommended medication significantly more ($M = 2.29$) than did female participants ($M = 1.87$).

An exploration of other univariate effects revealed a variety of possible interactions with gender of participant that could be fruitful to explore with a larger sample size. There was a marginal cause by gender interaction on outside therapy, $F(1, 117) = 3.22$, $p = .075$, partial eta squared = .027. The pattern of means suggested that male participants recommended seeking treatment through outside therapy more after reading an environmental causal explanation for the disorder ($M = 3.05$) than after reading a biological causal explanation for the disorder ($M = 2.50$), whereas female participants recommended seeking treatment through outside therapy more after reading a

biological causal explanation for the disorder (M = 2.56) than after reading an environmental causal explanation for the disorder (M = 2.29).

Results also suggested a marginal disorder by cause by gender interaction for friend support, $F(1, 117) = 3.34$, $p = .07$, partial eta squared = .028. The pattern of means suggested that for depression, male participants recommended seeking support through friends marginally more after reading a biological causal explanation (M = 3.50) than after reading an environmental causal explanation (M = 2.80), whereas female participants recommended seeking support through friends marginally more after reading an environmental causal explanation (M = 3.43) than after reading a biological causal explanation (M = 3.29). For anorexia, male participants recommended seeking support through friends marginally more after reading an environmental causal explanation (M = 3.13) than after reading a biological causal explanation (M = 2.80), whereas female participants recommended seeking support from friends marginally more after reading a biological causal explanation (M = 2.96) than after reading an environmental causal explanation (M = 2.75).

Finally, these exploratory analyses suggested a trend toward a cause by gender interaction effect for medication, $F(1, 117) = 2.99$, $p = .087$, partial eta squared = .025. The pattern of means suggested that females recommended medication more for disorders attributed to biological factors (M = 2.30) compared to disorders attributed to environmental factors (M = 1.44), whereas males recommended medication to a similar degree for disorders attributed to environmental (M = 2.24) and biological (M = 2.35) factors.

In addition to these possible gender differences, exploratory univariate analyses suggested a marginal univariate effect for disorder on friend support, $F(1, 117) = 3.46$, $p = .066$, partial eta squared = .029. Participants recommended seeking support from a friend marginally more strongly for the target with depression ($M = 3.26$) than for the target with anorexia ($M = 2.91$). These analyses help highlight the value of more fully exploring gender differences in mental illness stigma and treatment recommendations.

Analyses on the Facets of Familiarity with Mental Illness. To test the hypothesis that participants who reported greater familiarity with mental illness would express less stigmatizing responses toward the target described in the vignette, correlational analyses on mean familiarity with mental illness were conducted. This hypothesis was only weakly supported by the significant positive correlation between familiarity with mental illness and less social distancing in the form of willingness to be roommates with the target in the vignette, $r = .236$, $p = .008$. Participants who scored higher in familiarity reported greater willingness to be roommates with the target. The effects of familiarity may have varied by disorder type or explanation, and therefore, familiarity groupings were formed based on a median split and analyzed next. A 2 (disorder; depression, anorexia) x 2 (cause; biological, environmental) x 2 (familiarity; high, low) MANOVA on all of the main composite variables collapsed over participant gender revealed no effect for overall familiarity group.

To further explore the effects of familiarity with mental illness, items that corresponded to intimate familiarity were identified ("I have a friend who has a severe mental illness," "I have a relative who has a severe mental illness," and "I live with a person who has a severe mental illness") and combined. Correlational analyses examined

relationships between intimate familiarity and stigmatizing responses to the mental illness vignettes. Consistent with the correlational analyses on general familiarity, the analyses of intimate familiarity found a significant positive relationship between familiarity and willingness to be roommates with the target, $r = .258$, $p = .004$. The correlational analysis also found a trend toward intimate familiarity and scoring low on social distancing, $r = -.153$, $p = .089$, and a trend toward intimate familiarity and less social distance in the form of willingness to socialize with the target, $r = .153$, $p = .089$. As with general familiarity, a 2 (disorder; depression, anorexia) x 2 (cause; biological, environmental) x 2 (intimate familiarity; high, low) MANOVA on all of the main composite variables collapsed over participant gender revealed no effect for the intimate familiarity group.

Discussion

The present study sought to investigate whether biological and environmental causal explanations have different effects on the stigmatization of major depression and anorexia nervosa. Results indicated that participants viewed depression and anorexia differently and had different assumptions about the causes of the two disorders. Biological and environmental causal explanations also had a different effect on stigma toward depression versus anorexia. The lowest level of stigma occurred after participants read about a target with depression that used a biological causal explanation for the disorder, and the highest level of stigma resulted after participants read about a target with anorexia that used a biological causal explanation for the disorder. Participants moderately stigmatized the target with both depression and anorexia after reading an environmental causal explanation. Findings about treatment recommendations were complex, and were influenced by both disorder and causal explanations. These findings

support and extend previous research demonstrating the biological model's mixed effect on stigma (Corrigan & Watson, 2004) and indicate that biological causal explanations can have a unique influence on the stigmatization of different mental disorders.

Stigma toward Major Depression versus Anorexia Nervosa

Results supported that hypothesis that participants would stigmatize the target with anorexia more than they would stigmatize the target with depression. This finding is consistent with Roehrig and McLean's (2009) research, which found that participants stigmatized individuals with eating disorders (anorexia nervosa and bulimia nervosa) more than they stigmatized individuals with depression. Like the results of Roehrig and McLean (2009), the present study found that participants attributed more responsibility to the target with anorexia than to the target with depression. Furthermore, the present study found that participants felt more anger toward the target with anorexia than toward the target with depression. This finding connects with previous research that has demonstrated a link between the view that anorexia is a self-inflicted illness and expressions of anger toward individuals with anorexia (Stewart et al., 2006). Reports of higher stigma toward anorexia, relative to depression, are consistent with attribution theory, which predicts that the perception that an individual is capable of controlling a mental illness results in anger toward that individual (Link et al., 2004).

Social Distance toward Major Depression versus Anorexia Nervosa

Social distance is an important outcome variable in the study of stigma (Link et al., 2004), and results indicated that despite higher levels of stigma toward individuals with anorexia than individuals with depression, participants desired somewhat less social distance from individuals with anorexia than from individuals with depression. This

finding contrasted with the prediction that participants would desire more social distance from the target with anorexia than from the target with depression. It also conflicted with Stewart et al.'s (2008) finding that participants desired more social distance from a target with anorexia than from a target with depression.

An analysis of the items on the social distancing scale indicated that participants reported somewhat more willingness to socialize with the target that had anorexia, but reported somewhat more willingness to eat with the target that had depression. Participants' willingness to socialize with the anorexic target may reflect the envy component of anorexia (Stewart et al., 2006). Most people want to socialize with someone who is socially attractive and appealing (Fiske, Gilbert, & Lindzey, 2010). It could be argued that anorectic individuals stereotypically have more socially desirable qualities (e.g., thinness and an appearance of perfection), whereas depressed individuals may lack some important socially desirable qualities (e.g., positive affect and sociability). The finding that participants would rather eat with the depressed target than with the anorexic target is logical given the nature of anorexia nervosa. Participants may feel comfortable associating with anorectics in social settings but may not feel comfortable eating with anorectics because anorexia nervosa is centered on disordered eating. Participants may have imagined feeling most uncomfortable being around anorectics at the time that their disorder is most apparent.

Causal Explanations

Consistent with previous research indicating widespread endorsement of biological explanations for depression (Goldstein & Rosselli, 2003) and widespread endorsement of environmental explanations for anorexia (Crisp et al., 2000), results

confirmed the prediction that participants would make stronger biological (i.e., genetic and chemical) attributions for depression than for anorexia. A related hypothesis, that participants would make stronger environmental attributions for anorexia than for depression, was not supported. In fact, there was some evidence that participants made stronger environmental attributions for depression than for anorexia. Participants perceived death of a loved one and trauma as more likely to cause depression than anorexia. Results also suggested that participants perceived problems from childhood as somewhat more likely to cause depression than anorexia. Together, these results indicated that people saw the causes of depression as varied, viewing biological, personal, and environmental factors as contributing to depression. In contrast, participants had a more narrow view of causes for anorexia, as they did not endorse any of the provided explanations more for anorexia than for depression. Likely, participants would have viewed items such as "desire for control" and "low body image" as more likely to cause anorexia than depression; however, the Causal Attributions Scale did not include these items.

The Effect of Causal Explanations on Stigma

Results supported the prediction that biological and environmental explanations would have different effects on stigma toward depression versus anorexia. Consistent with previous research (Deacon & Baird, 2009; Goldstein & Rosselli, 2003), the present study found that participants stigmatized depression less after reading a biological causal explanation than after reading an environmental causal explanation. The finding that biological causal explanations reduced stigma toward depression is consistent with attribution theory's implication that biological causal explanations can help reduce stigma

because they promote the belief that individuals with mental illness are not capable of controlling their behavior (Link et al., 2004). Furthermore, this finding supports the endorsement of a biological model among advocacy groups such as the National Alliance on Mental Illness and the Depression is Real Coalition in their efforts to reduce stigma (Corrigan et al., 2002).

Contrary to the stigma reducing effect that biological causal explanations had on depression, biological causal explanations seemed to increase stigma toward anorexia. Participants' overall stigma ratings were higher for anorexia after reading a biological causal explanation than after reading an environmental causal explanation. Although these means were not significantly different, their direction was the opposite of the direction of the means for depression. Furthermore, participants stigmatized anorexia with a biological explanation more than biological depression, whereas they did not view anorexia with an environmental explanation differently from environmental depression. Biological explanations can in some cases increase stigma because they imply that individuals with mental illnesses are fundamentally different, which exacerbates perceived differences between mentally ill and "normal" people (Phelan, 2002). However, this effect did not occur for depression, and future research should investigate why biological explanations had a different effect on stigma toward anorexia compared to stigma toward depression.

The finding that biological causal explanations did not reduce stigma toward anorexia conflicted with previous research by Crisafulli et al. (2008), which found that nursing students who read a biological and genetic explanation for anorexia nervosa blamed anorectics for their condition less than did nursing students who read a

sociocultural explanation. There are differences between the present study and Crisafulli et al.'s (2008) study that may account for this discrepancy. The present study's causal explanation consisted of only one sentence describing either a biological or environmental etiology of the disorder. Contrarily, Crisafulli et al. (2008) provided participants with a page of information about either biological and genetic or sociocultural information about anorexia nervosa. In the present study, it is possible that participants responded with reactance to the biological causal explanation for anorexia. Participants made stronger biological attributions for depression than for anorexia, and the causal explanation manipulation did not influence their attributions. Participants did not commonly endorse biological explanations for anorexia, and therefore, they may have responded to the biological causal explanation they read more negatively because it violated their expectations. Perhaps if the biological causal explanation had included more extensive information and current research regarding biological components of anorexia, similar to Crisafulli et al.'s (2008), participants' causal attributions would have been more affected by the manipulation, and their stigmatizing responses would have been reduced rather than enhanced.

Future research should clarify the effect that biological causal explanations have on stigma. The present study's finding that a brief biological causal explanation of anorexia does not decrease, and may serve to increase, stigma suggests that the Eating Disorders Coalition and other advocacy groups should not promote a biological model for anorexia without considering people's stigmatizing responses to the explanation. Watson and Corrigan (2005) suggested that people attend more to information that confirms their stereotypes and require encouragement to consider stereotype-disconfirming information.

A biological explanation for anorexia nervosa defies people's stereotypes, and therefore, people might not attend to or fully integrate that information as much as they would attend to a cultural or personal explanation of anorexia nervosa. This effect is exacerbated when the explanation is brief when there is no opportunity to really educate and challenge the pre-existing belief (Watson & Corrigan, 2006).

Results indicated an interaction effect for pity, in which participants felt more pity for the depressed target after reading the environmental explanation than after reading the biological explanation, but appeared to feel more pity for the anorexic target after reading the biological explanation than after reading the environmental explanation. The biological/environmental difference was only observed for depression, but the overall pattern of means suggested the same reversal observed in the overall stigma result. Thus, biological information had an impact on participants' impressions of anorexia, but a complex one. One explanation for the pity findings is that the biological explanation of anorexia and the environmental explanation for depression conflicted with participants' pre-existing beliefs about the etiology of each disorder, creating a sense of surprise. The unexpected etiology may have caused participants to feel worse for the target, as if she had been dealt a bad hand. Most researchers consider pity to be part of a stigmatizing response to mental disorders, so at that level, the impact of biological explanations on perceptions of the target with anorexia had some coherence.

The Effect of Causal Explanations on Social Distance

The hypothesis that biological causal explanations would increase participants' desired social distance from the target described in the vignette was not supported. Instead, results suggested that biological causal explanations somewhat decreased

participants' desired social distance. An analysis of individual items on the social distancing scale indicated that participants reported less social distance in the form of greater willingness to socialize with and become friends with the target after reading a biological causal explanation than after reading an environmental causal explanation. Additionally, results indicated that participants reported somewhat less social distance in the form of greater desire to eat with the target after reading a biological causal explanation than after reading an environmental causal explanation. It is possible that participants desired less social distance from biologically based disorders than from environmentally based disorders because they perceived biologically based disorders as more predictable and/or treatable. It is likely that the present study's educated sample had an awareness of treatments for biological disorders. Participants may have also viewed biologically based disorders as less likely to involve other problems, compared to environmentally based disorders caused by a difficult childhood or trauma.

The present study's findings about social distance conflict with Griffiths and Christensen's (2004) findings that biological causal beliefs about mental illness lead people to desire increased social distance from the mentally ill. However, the literature on the biological model's impact on social distance is mixed. For example, consistent with the present study's findings regarding social distance, Jorm and Griffiths (2008) found that biological explanations helped reduce desired social distance from individuals with schizophrenia. The effect of biological causal explanations on social distance is unclear, and more research is needed to clarify this effect.

The Effect of Mental Illness Familiarity on Social Distance

It was predicted that participants who reported greater familiarity with mental illness would express lower levels of social distancing toward the target described in the vignette than would participants who reported less familiarity with mental illness. This hypothesis was not supported. However, results suggested that participants with higher familiarity scored somewhat lower on social distancing than did those with lower familiarity. An analysis of individual items on the social distancing scale indicated that participants with higher familiarity reported greater willingness to be roommates with the target in the vignette than did those with lower familiarity. Additionally, results suggested that participants with higher familiarity reported somewhat greater willingness to socialize with and be friends with the target than did those with lower familiarity. These findings support previous research that has shown that people who have never known anyone with a mental illness are more likely to desire social distance from individuals with mental illness than are people who have had contact with someone who has a mental illness (Link et al., 2004).

The analysis of intimate familiarity yielded findings similar to the analysis of familiarity. Participants with higher intimate familiarity reported somewhat lower social distancing overall and greater willingness to be roommates with the target. These findings indicated that the level of intimacy in participants' experiences with individuals with mental illness only modestly influenced their stigmatizing responses.

The Effect of Causal Explanations on Treatment Recommendations

Participants did not make significantly different treatment recommendations after reading a biological versus an environmental causal explanation. However, an assessment

of individual items on the Treatment Recommendations Scale indicated that participants recommended medication significantly more strongly after reading a biological explanation of the target's disorder than after reading an environmental explanation of the target's disorder. This finding suggests that participants attended to the causal manipulation, even though it may not have had the intended effect on stigma. This finding is also consistent with the model adopted by SSRI advertisements, which is based on the assumption that attributing psychological problems to biological factors is the most effective way to encourage people to take medication (Deacon & Baird, 2009). Participants recommended seeking friend support marginally more for depression than for anorexia. The perception that anorexia is a self-induced illness, and that individuals with anorexia could put themselves together if they wanted to, helps account for this finding (Crisp et al., 2000).

Gender Differences

Various tentative gender differences emerged in treatment recommendations. Male participants recommended seeking treatment through outside therapy somewhat more after reading an environmental causal explanation than after reading a biological causal explanation, whereas female participants recommended seeking treatment through outside therapy somewhat more after reading a biological causal explanation than after reading an environmental causal explanation. This pattern was especially clear for judgments of anorexia and may relate to earlier findings of blame and anger in women. It is possible that the biological explanation reduced female participants' blame toward the target and therefore increased their treatment recommendations. For depression, male participants were especially likely to make treatment recommendations under biological

conditions and especially unlikely to make them under environmental conditions. This difference might relate to men's socialization around the notion of "toughing out" difficult circumstances. Overall, male participants recommended medication for treatment more than did female participants. Results indicated a trend toward females recommending medication more after reading a biological causal explanation than after reading an environmental causal explanation. It is important for future research to further explore gender differences in treatment recommendations, and to investigate the extent to which treatment recommendations are influenced by the recipient's gender.

Results indicated several additional gender differences. Female participants expressed more anger than did male participants toward the target in all vignettes. This effect may have occurred because the vignette described a female, and females are more likely to express anger toward other females, especially those seen as vulnerable or weak. Goldberg's (1968) finding that women are biased against other women confirms this phenomenon. Had the vignette described a male target, it is likely that female participants would not have expressed more anger toward the target than did male participants.

The findings pointed to a disorder by gender interaction on anger, as well. Females expressed especially high anger toward the target with anorexia compared to the target with depression, whereas males reported little difference in anger toward the target with anorexia compared to the target with depression. The anger that females attributed to the anorexic target likely may have related to their feelings of envy toward individuals with eating disorders (Roehrig & McLean, 2009). The thin ideal is not as valued by men in our society as it is by women, and therefore, envy may not have had as strong an influence on male participants' expression of anger toward the anorexic target as it had

on female participants' expression of anger. It is important for future research to further explore why women may express more anger toward women with mental illness, and especially anorexia, than men do.

Limitations

This study had several limitations. The sample did not have equal gender representation. Results indicated several marginal gender differences, which likely would have been more pronounced had more males participated in this study. The sample had minimal variability in race, a variable that is important to examine in future research because of cultural differences in perceived etiology and stigmatization of mental illness. In addition, the sample primarily consisted of psychology students, who may have different and more informed perceptions of mental illness than does the general population. As Watson and Corrigan (2005) pointed out, people with a stronger understanding of mental illness stigmatize individuals with mental illness less. Additionally, the causal manipulation did not have a strong effect. The manipulation had some influence, but was not strong or detailed enough to shift people's prior causal beliefs. Another limitation to the present study is the social desirability bias effect. Participants were reluctant to admit their own stigma, particularly on the Social Distancing Scale. Furthermore, self-reported responses to questionnaires assessing stigma do not always predict real-world behaviors (Link et al., 2004). It is useful to also assess stigma through other methods that are less affected by the social desirability bias and more predictive of real-world behaviors.

Future Directions

Future research could explore the stigmatization of different mental illnesses through the Implicit Association Task, for which social desirability bias would not skew participants' responses. Future research could also directly observe behavior by examining whether participants are willing to sit near or choose to work with an individual with mental illness after learning about different etiological explanations. In addition, future research could expand the biological or environmental causal explanation provided and could include additional information and recent research about the disorder. Future research could use a more detailed causal explanation, and could provide more extensive educational training on the etiology of mental illness. Future research could also compare short-term and long-term effects of learning biological versus environmental causal explanations. In addition, future research could examine the discrepancy between people's endorsed biological explanation and the causal explanation they received to determine whether people with smaller discrepancies shifted more toward the provided causal explanation and people with larger discrepancies responded to the provided causal explanation with more reactance.

Conclusion

This research demonstrated that biological and environmental causal explanations have unique effects on the stigmatization of depression versus anorexia. This finding has important implications for stigma reduction efforts. As etiological understandings of mental illnesses are strengthened, advocacy groups are ethically obliged to promote this knowledge. With a stronger understanding of the link between perceived etiology and stigmatization of specific mental disorders, advocacy groups can work with people's

stigmatizing responses while promoting evidence about etiology. Advocacy groups that seek to reduce stigma for specific mental disorders can particularly benefit from this research because it is evident that different etiological explanations have a unique effect on the stigmatization of different mental disorders.

The present study examined perceptions of college students regarding a mentally ill college student, and therefore, has particular implications for stigma among college students. Research has indicated that stigma is one of the primary barriers preventing college students from seeking mental health treatment (Eisenberg, 2009), and therefore, stigma reduction among the college student population is an important goal. The present study helped shed light on the factors that contribute to stigmatization of mental disorders among college students. This knowledge is a key step to accommodating the needs of the growing population of mentally ill college students.

References

Angermeyer, M., & Matschinger, H. (2004). Public attitudes to people with depression: Have there been any changes over the last decade? *Journal of Affective Disorders, 83,* 177-182. doi. 10.1016/j.jad.2004.08.001

Angermeyer, M., Matschinger, H., & Corrigan, P. (2004). Familiarity with mental illness and social distance from people with schizophrenia and major depression: Testing a model using data from a representative population survey. *Schizophrenia Research, 69*, 175-182. doi. 10.1016/S0920-9964%2803%2900186-5

Bennett, L., Thirlaway, K., & Murray, A. (2008). The stigmatising implications of presenting schizophrenia as a genetic disease. *Journal of Genetic Counseling, 17,* 550-559. doi. 10.1016/S0920-9964%2803%2900186-5

Ben-Porath, D. (2002). Stigmatization of individuals who receive psychotherapy: An interaction between help-seeking behavior and the presence of depression. *Journal of Social and Clinical Psychology, 21,* 400. doi. 10.1016/S0920-9964%2803%2900186-5

Borenstein, J. (2005). Harvard president discusses impact of psychiatric treatment. *The New York State Psychiatric Bulletin, 46,* 2.

Butcher, J., Mineka, S., & Hooley, J. (2010). *Abnormal psychology.* Boston, MA: Pearson Education, Inc.

Corrigan, P. (2003). An attribution model of public discrimination towards persons with mental illness. *Journal of Health and Social Behavior, 44,* 162-179. doi. 10.2307/1519806

Corrigan, P. (2004). How stigma interferes with mental health care. *American Psychologist, 59,* 614-625. doi. 10.1037/0003-066X.59.7.614

Corrigan, P. W., & Cooper, A. E. (2006). Mental illness and dangerousness: Fact or misperception, and implications for stigma. In P.W. Corrigan (Ed.) *On the stigma of mental illness: Practical strategies for research and social change* (pp. 165-179). Washington, DC: American Psychological Press.

Corrigan, P.W., & Kleinlein, P. (2005). "The impact of mental illness stigma." In P. W. Corrigan (Ed.) *On the stigma of mental illness: Practical strategies for research and social change* (pp. 11-44). Washington, DC: American Psychological Press.

Corrigan, P., Markowitz, F., Watson, A., Rowan, D., & Kubiac, M. (2003). An attribution model of public discrimination towards persons with mental illness. *Journal of Health and Social Behavior, 44,* 162-179. doi. 10.2307/1519806

Corrigan, P., & Watson, A. (2004). Stop the stigma: Call mental illness a brain disease. *Schizophrenia Bulletin, 30,* 477-479.

Corrigan, P., Rowan, D., Green, A., Lundin, R., River, P., Uphoff-Wasowski, K., White, K., & Kubiak, M.. (2002). Challenging two mental illness stigmas: Personal responsibility and dangerousness. *Schizophrenia Bulletin, 28,* 293-309.

Crisafulli, M., Von Holle, A., & Bulik, C. (2008). Attitudes towards anorexia nervosa: The impact of framing on blame and stigma. *International Journal of Eating Disorders, 41,* 333-339. doi. 10.1002/eat.20507

Crisp, A. (2005) Stigmatization of and discrimination against people with eating disorders including a report of two nationwide surveys. *European Eating Disorders Review, 13,* 147-152. doi. 10.1002/erv.648

Crisp, A., Gelder, M., Rix, S., Meltzer, H., & Rowlands, O. (2000). Stigmatisation of people with mental illnesses. *British Journal of Psychiatry, 177,* 4-7. doi. 10.1192/bjp.177.1.4

Day, E., Edgren, K., & Eshleman, A. (2007). Measuring stigma toward mental illness: Development and application of the Mental Illness Stigma Scale. *Journal of Applied Social Psychology, 37,* 2191-2219. doi. 10.1111/j.1559 1816.2007.00255.x

Deacon, B., & Baird, G. (2009). The chemical imbalance explanation of depression: Reducing blame at what cost? *Journal of Social and Clinical Psychology, 28,* 415-435. doi. 10.1521/jscp.2009.28.4.415

Depression is Real Coalition. (2011). *Depression is real.* Retrieved February 27, 2011, from: http://depressionisreal.org/.

Eating Disorders Coalition. (2011). *Eating disorders: Fighting stigma with science.* Retrieved March 28, 2011, from: http://www.eatingdisorderscoalition.org/February2008.htm.

Eisenberg, D., Downs, M., Golberstein, E., & Zivin, K. (2009). Stigma and help seeking for mental health among college students. *Medical Care and Research Review, 66,* 522-541. doi. 10.1177/1077558709335173

Fiske, S., Gilbert, D., & Lindzey, G. (2010). *Handbook of Social Psychology.* Hoboken, NJ: John Wiley & Sons, Inc.

France, C., Lysacker, P., & Robinson, R. (2007). The 'chemical imbalance' explanation for depression: Origins, lay endorsement, and clinical implications. *Professional Psychology: Research and Practice, 38,* 411-420. doi. 10.1037/0735-7028.38.4.411

Gabriel, T. (2010, December 10). Mental health needs seen growing at colleges. *New York Times,* p. A1.

Gallagher, R., Zhang, B., & Taylor, R. (2010). *National survey of counseling center directors.* Pittsburgh, PA: International Association of Counseling Services, Inc. Retrieved April 4, 2010 from: http://www.iacsinc.org/NSCCD%202010.pdf.

Golberstein, E., Eisenberg, D., & Gollust, S. (2008). Perceived stigma and mental health care seeking. *Psychiatric Services, 59,* 392-399. doi. 10.1176/appi.ps.59.4.392

Goldberg, P. (1968). Are women prejudiced against women? *Transaction, 5,* 28-30. doi. 10.1007/BF03180445

Goldstein, B., & Rosselli, F. (2003). Etiological paradigms of depression: The relationship between perceived causes, empowerment, treatment, preferences, and stigma. *Journal of Mental Health, 12,* 551-563. doi. 10.1080/09638230310001627919

Griffiths, K., & Christensen, H. (2004). Commentary on 'The relationship between public causal beliefs and social distance toward mentally ill people'. *Australian and New Zealand Journal of Psychiatry, 38,* 355-357. doi. 10.1111/j.1440-1614.2004.01374.x

Hefner, J., & Eisenberg, D. (2009). Social support and mental health among college students. *American Journal of Orthopsychiatry, 79,* 491-499. doi. 10.1037/a0016918

Holliday, J., Wall, E., Treasure, J., & Weinman, J. (2005). Perceptions of illness in individuals with anorexia nervosa: A comparison with lay men and women. *International Journal of Eating Disorders, 37,* 50-56. doi. 10.1002/eat.2005

Jones, E., Farina, A., Hastorf, A., Marcus, H., Miller, D., & Scott, R. (1984). *Social stigma: The psychology of marked relationships.* New York, NY: Freeman and Company.

Jorm, A., & Griffiths, K. (2008). The public's stigmatizing attitudes towards people with mental disorders: How important are biomedical conceptualizations? *Acta Psychiatrica Scandinavia, 118,* 315-321. doi. 10.1111/j.1600-0447.2008.01251.x

Lee, D., Michelson, S., Olsen, E., Odes, E., & Locke, B. (2009). The effects of college counseling services on academic performance and retention. *Journal of College Student Development, 50,* 305.

Link, B., Phelan, J., Bresnahan, M., Stueve, A., & Perscosolido, B. (1999). Public conceptions of mental illness: Labels, causes, dangerousness, and social distance. *American Journal of Public Health, 89*, 1328-1333. doi. 10.2105/AJPH.89.9.1328

Link, B., Yang, L., Phelan, J., & Collins, P. (2004). Measuring mental illness stigma. *Schizophrenia Bulletin, 30,* 511-541.

Luchins, D. (2004). Will the term brain disease reduce stigma and promote parity for mental illnesses? *Schizophrenia Bulletin, 30,* 1043-1048.

Mowbray, C., Megivern, D., Mandiberg, J., Strauss, S., Stein, C., Collins, K., Kopels, S., Curlin, C., & Lett, R. (2006). Campus mental health services: Recommendations for change. *American Journal of Orthopsychiatry, 76,* 226-237. doi. 10.1037/0002- 9432.76.2.226

National Institute of Mental Health. (2010). *Depression.* Retrieved April 29, 2010, from: http://www.nimh.nih.gov/health/topics/depression/index.shtml.

National Institute of Mental Health. (2010). *Anorexia Nervosa.* Retrieved May 1, 2010, from: http://www.nimh.nih.gov/health/publications/eating-disorders/anorexia-nervosa.shtml.

O'Hara, S., & Smith, C. (2007). Presentation of eating disorders in the news and media: What are the implications for patient diagnosis and treatment? *Patient Education and Counseling, 68,* 43-51. doi. 10.1016/j.pec.2007.04.006

Phelan, J. (2002). Genetic bases of mental illness – a cure for stigma? *TRENDS in Neurosciences, 25,* 430-431. doi. 10.1016/j.pec.2007.04.006

Roehrig, J., & McLean, C. (2009). A comparison of stigma toward eating disorders versus depression. *International Journal of Eating Disorders, 43*, 671-674. doi. 10.1002/eat.20760

Rusch, N., Todd, A., Bodenhausen, G., & Corrigan, P. (2010). Biogenetic models of psychopathology, implicit guilt, and mental illness stigma. *Psychiatry Research, 179,* 328-332. doi. 10.1016/j.psychres.2009.09.010

Schwenk, T., Davis, L., & Wimsatt, L. (2010). Depression, stigma, and suicidal ideation in medical students. *Journal of the American Medical Association, 304,* 1181-1190. doi. 10.1001/jama.2010.1300

Sibicky, M., & Dovidio, J. (1986). Stigma of psychological therapy: Stereotypes, interpersonal reactions, and the self-fulfilling prophecy. *Journal of Counseling Psychology, 33,* 148-154. doi. 10.1037/0022-0167.33.2.148

Stewart, M., Keel, P., & Schiavo, R. (2006). Stigmatization of anorexia nervosa. *International Journal of Eating Disorders, 39,* 320-325. doi. 10.1002/eat.20262

Stewart, M,. Schiavo, R., Herzog, D., & Franko, D. (2008). Stereotypes, prejudice and discrimination of women with anorexia nervosa. *European Eating Disorders Review, 16,* 311-318. doi. 10.1002/erv.849

Vitousek, K., Watson, S., & Wilson, G. (1998). Enhancing motivation for change in treatment resistant eating disorders. *Clinical Psychology Review, 18*, 391-420. doi. 10.1016/S0272-7358%2898%2900012-9

Vogel, D., Wade, N., & Ascheman, P. (2009). Measuring perceptions of stigmatization by others for seeking psychological help: Reliability and validity of a new stigma scale with college students. *Journal of Counseling Psychology, 56,* 301-308. doi. 10.1037/a0014903

Watson, A., & Corrigan, P. (2005). Challenging public stigma: A targeted approach. In P. Corrigan (Ed.), *On the stigma of mental illness: Practical strategies for research and social change* (pp. 281-295). Washington, DC: American Psychological Press.

Zivin, K., Eisenberg, D., Gollust, S., & Golberstein, E. (2009). Persistence of mental health problems and needs in a college student population. *Journal of Affective Disorders, 117,* 180-185. doi. 10.1037/a0014903

Appendix A

Mental Illness Vignettes

Vignette with biological causal explanation:

Kelly is an eighteen-year-old girl who has [major depression/anorexia nervosa]. Recent research emphasizes that [major depression/anorexia nervosa] is a medical illness that can result from biological factors like a chemical imbalance in the brain or a genetic predisposition. After receiving some treatment Kelly appears to be doing fairly well, although she still has issues from time to time. Kelly is clean and well groomed. She is now a freshman at a four-year residential college. She gets along well with her peers, eats her meals in the dining hall, and spends time working in the library. Kelly completes her assignments carefully and finishes each task before moving on to another. This tendency might slow Kelly down a little, but she is never criticized for the quality of her work. Socially, Kelly is interested in meeting and dating people in the college community, and is considering joining student organizations.

Vignette with environmental causal explanation:

Kelly is an eighteen-year-old girl who has [major depression/anorexia nervosa]. Recent research emphasizes that [major depression/anorexia nervosa] is a common mental illness that can result from environmental factors like stressful life experiences or relationship difficulties. After receiving some treatment Kelly appears to be doing fairly well, although she still has issues from time to time. Kelly is clean and well groomed. She is now a freshman at a four-year residential college. She gets along well with her peers, eats her meals in the dining hall, and spends time working in the library. Kelly completes her assignments carefully and finishes each task before moving on to another. This tendency might slow Kelly down a little, but she is never criticized for the quality of her work. Socially, Kelly is interested in meeting and dating people in the college community, and is considering joining student organizations.

Appendix B

Social Distancing and Treatment Recommendations Scale

Please rank your willingness to do the following…

1. Be roommates with Kelly

0	1	2	3
Definitely unwilling			Definitely willing

2. Spend an evening socializing with Kelly

0	1	2	3
Definitely unwilling			Definitely willing

3. Make friends with Kelly

0	1	2	3
Definitely unwilling			Definitely willing

4. Work closely on a group project with Kelly

0	1	2	3
Definitely unwilling			Definitely willing

5. Eat meals with Kelly

0	1	2	3
Definitely unwilling			Definitely willing

6. Elect Kelly to be head of a student organization

0	1	2	3
Definitely unwilling			Definitely willing

How strongly would you recommend that Kelly…

1. Receives medication for her [major depression/anorexia nervosa]?

0	1	2	3	4
Not at all				Very strongly

2. Receives counseling for her [major depression/anorexia nervosa] from her college's counseling center?

0	1	2	3	4
Not at all				Very strongly

3. Receives therapy for her [major depression/anorexia nervosa] from a therapist outside her college?

0	1	2	3	4
Not at all				Very strongly

4. Turns to a friend for support for her [major depression/anorexia nervosa]?

0	1	2	3	4
Not at all				Very strongly

5. Seeks support for her [major depression/anorexia nervosa] from Student Disabilities Services?

0	1	2	3	4
Not at all				Very strongly

6. Copes with her [major depression/anorexia nervosa] without seeking treatment?

0	1	2	3	4
Not at all				Very strongly

Appendix C

Personal Responsibility Beliefs, Pity, and Anger Questionnaire

Please read the following statements and questions carefully and indicate the answer that best describes your opinion.

1. I would think that it were Kelly's own fault that she is in the present condition.

1	2	3	4	5	6	7	8	9
No, not at all								Yes, absolutely

2. I would feel pity for Kelly.

1	2	3	4	5	6	7	8	9
None at all								Very much

3. I would feel aggravated by Kelly.

1	2	3	4	5	6	7	8	9
Not at all								Very much

4. How controllable is the cause of Kelly's present condition?

1	2	3	4	5	6	7	8	9
Not at all under personal control								Completely under personal control

5. How much sympathy would you feel for Kelly?

1	2	3	4	5	6	7	8	9
None at all								Very much

6. How angry would you feel at Kelly?

1	2	3	4	5	6	7	8	9
Not at all								Very much

7. How responsible is Kelly for her present condition?

1	2	3	4	5	6	7	8	9
Not at all responsible								Very much responsible

8. How much concern would you feel for Kelly?

1	2	3	4	5	6	7	8	9
None at all								Very much

9. How irritated would you feel by Kelly?

1	2	3	4	5	6	7	8	9
Not at all								Very much

Appendix D

Causal Attributions Scale

The next few questions are about possible causes of [major depression/anorexia nervosa] in anybody. How likely do you think each of the following is to be a cause of [major depression/anorexia nervosa]?

1. A virus or other infection

0	1	2	3	4
Very unlikely				Very likely

2. An allergy or reaction

0	1	2	3	4
Very unlikely				Very likely

3. A genetic predisposition

0	1	2	3	4
Very unlikely				Very likely

4. Day to day problems such as stress, family arguments, difficulties at work or financial difficulties

0	1	2	3	4
Very unlikely				Very likely

5. The recent death of a close friend or relative

0	1	2	3	4
Very unlikely				Very likely

6. Stressful life experiences

0	1	2	3	4
Very unlikely				Very likely

7. Some recent traumatic event such as a house fire, a severe traffic accident, or being mugged

0	1	2	3	4
Very unlikely				Very likely

8. A chemical imbalance in the brain

0	1	2	3	4
Very unlikely				Very likely

9. Problems from childhood such as being badly treated or abused, losing one or both parents when young, or coming from a broken home

0	1	2	3	4
Very unlikely				Very likely

10. Being a nervous person

0	1	2	3	4
Very unlikely				Very likely

11. Relationship difficulties

0	1	2	3	4
Very unlikely				Very likely

12. Having weakness of character

0	1	2	3	4
Very unlikely				Very likely

Appendix E

Familiarity with Mental Illness Questionnaire

Please indicate the answer that best describes your personal experiences.

1. My job involves providing services/treatment for persons with mental illness.
   - Yes
   - No

2. I have observed, in passing, a person I believe may have had a severe mental illness.
   - Yes
   - No

3. I have observed persons with a severe mental illness on a frequent basis.
   - Yes
   - No

4. I have worked with a person who had a severe mental illness at my place of employment.
   - Yes
   - No

5. I have a friend who has a severe mental illness.
   - Yes
   - No

6. I have a relative who has a severe mental illness.
   - Yes
   - No

7. I live with a person who has a severe mental illness.
   - Yes
   - No

Appendix F

Demographics Questionnaire

Please read each question carefully and provide the answer that best applies.

1. What is your sex?
    - Male
    - Female

2. How would you describe your race?
    - Hispanic or Latino
    - Black or African American
    - Asian or Asian American
    - American Indian
    - Hawaiian or other Pacific Islander
    - Non-Hispanic White
    - Other

3. What college do you go to? _____

4. What is your class year?
    - Freshman
    - Sophomore
    - Junior
    - Senior

5. What is your major? _____

6. Have you ever had [major depression/anorexia nervosa]?
    - Yes
    - No

7. Have you ever been treated for a psychological problem?
    - Yes
    - No

8. Have you ever seen a counselor from Student Counseling Services for help with a problem?
    - Yes
    - No

9. Have you ever seen a therapist outside Student Counseling Services for help with a problem?
    - Yes
    - No

Appendix G

Informed Consent

The goal of this research is to examine attitudes about people experiencing mental health difficulties. The following survey will ask you questions about your perception of an individual described in a vignette that you will read.

You have the right to decline and withdraw from this study at any point in time without penalty. If you decide to participate, you will consent to the publication of the results, as long as your identity remains anonymous. The information you provide on the questionnaires will be kept confidential.

Participation in this study should take about 30 minutes, and will involve reading a vignette, filling out four surveys, and filling out a demographics questionnaire. This study will enhance previous research on this topic. There are no known risks involved in participating in this study.

The results will be available to look at after the study is done. If you would like to see the results, please contact the researcher, Briana Borenstein (646-734-3348; bborenst@conncoll.edu).

If you have any further questions about this study, please contact Briana Borenstein or the chair of the Connecticut College Institutional Review Board, Jason Nier, at 860-439-5057. Thank you for your participation.

I am at least 18 years of age and I voluntarily consent to participate in this study on attitudes about mental illness.

- I agree to participate in this study.
- I do not agree to participate in this study.

Appendix H

Debriefing Statement

First off, thank you for participating in this research dealing with attitudes about mental illness. This research is examining how biological and environmental explanations for major depression and anorexia nervosa have a differential effect on stigmatization of each disorder.

Previous research has indicated that environmental causal explanations for mental illness intensify stigma by increasing perceptions that individuals are to blame for their condition (Corrigan & Watson, 2004; Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000; Crisafulli, Von Holle, & Bulik, 2008). Biological causal explanations for mental illness help to alleviate blame but contribute to stigma by increasing social distancing and perceptions of dangerousness, gravity, and disparity (Bennett, Thirlaway, & Murray, 2008; Corrigan et al., 2002; Corrigan & Watson, 2004; Griffiths & Christensen, 2004). Previous researchers have not directly compared and contrasted how biological and environmental explanations influence stigmatization of different mental illnesses. The present study is designed to examine how perceived etiology affects the stigmatization of college students who have major depression and anorexia nervosa.

Please do not share information with peers until the end of the academic year when the study is completed. If you are interested in this topic and want to read the literature in this area, please contact the researcher, Briana Borenstein (bborenst@conncoll.edu). Any concerns about how the study was conducted can be addressed to Professor Nier, Chair of the IRB (860-439-5057). If participating in this study caused you distress and you would like to talk to someone at Student Counseling Services, please call 860-439-4587.

Listed below are three sources you may want to consult to learn more about this topic:

Corrigan, P., & Watson, A. (2004). Stop the stigma: Call mental illness a brain disease. *Schizophrenia Bulletin, 28,* 293-309.

France, C., Lysacker, P., & Robinson, R. (2007). The 'chemical imbalance' explanation for depression: Origins, lay endorsement, and clinical implications. *Professional Psychology: Research and Practice, 38,* 411-420.

Stewart, M., Schiavo, S., Herzog, D., & Franko, D. (2008). Stereotypes, prejudice and discrimination of women with anorexia nervosa. *European Eating Disorders Review, 16,* 311-318.