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COMPARING TREATMENT RELEVANT ETIOLOGICAL EXPLANATIONS FOR DEPRESSION AND SOCIAL ANXIETY: EFFECTS ON SELF-STIGMATIZING ATTITUDES

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This study examines the impact of three different etiological explanations on self-stigmatizing attitudes among individuals with clinical symptom levels of either major depressive disorder (MDD; n = 144) or social anxiety disorder (SAD; n = 175). Participants were randomly assigned to view an audiovisual presentation describing their symptoms as being caused by biological (BIO) or cognitive-behavioral (CB) factors, or their combination (BIO+CB). Self-blame and perceived helplessness were significantly greater in the CB condition compared to the BIO condition. There was no significant difference in prognostic pessimism, perceived dangerousness, and unpredictability between the CB and BIO conditions. All

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self-stigmatizing attitudes were worse in the BIO+CB condition compared to the BIO condition. The MDD group endorsed significantly higher levels of self-stigmatizing attitudes compared to the SAD group. The present findings suggest that cognitive-behavioral etiological explanations may increase self-stigma compared to biological-only explanations. Combined biological and cognitive-behavioral explanations appear to have a particularly negative impact on self-stigmatizing attitudes among people with clinical levels of MDD or SAD symptoms. Individuals with symptoms of MDD generally endorse more self-stigmatizing attitudes compared to those with symptoms of SAD, even when controlling for general hopelessness. The clinical implications of these findings are discussed.

Keywords: major depressive disorder, social anxiety disorder, cognitive-behavioral, attribution, stigma

The general public increasingly attributes mental disorders to biological causes such as a chemical imbalance or inherited genes (Schnittker, 2008; Schomerus et al., 2012). Yet, a growing body of research suggests that biological attributions have mixed effects on public attitudes toward people with mental health problems. For example, consistent with the predictions of attribution theory (Weiner, Perry, & Magnusson, 1988), biological casual explanations for mental health conditions may reduce blame directed toward people with mental health problems (Kvaale, Haslam, & Gottdiener, 2013). However, research suggests that biological attributions may promote negative stereotypes that people with mental health disorders are unpredictable, dangerous to themselves or others, unlikely to recover, and helpless or disabled (Jorm, Reavley, & Ross, 2012; Lam, Salkovskis, & Warwick, 2005; Phelan, 2005; Schomerus, Matschinger, & Angermeyer, 2014).

In contrast to public stigma, self-stigma reflects the negative mental health-related attitudes and beliefs internalized by persons with mental health conditions (Corrigan & Shapiro, 2010). Self-stigma is associated with reduced treatment seeking rates (Aromaa, Tolvanen, Tuulari, & Wahlbeck, 2011; Bathje & Pryor, 2011), increased rates of treatment discontinuation, and higher psychiatric symptom severity (Livingston & Boyd, 2010). A small body of research suggests that biological attributions have similar effects on public and self-stigmatizing attitudes. For example, a recent review concluded that biological casual explanations may lead to less blame for symptoms but greater prognostic pessimism regarding symptom course (Lebowitz, 2014). However, it

remains unclear whether biological causal beliefs promote other important self-stigmatizing attitudes such as individuals' views of themselves as unpredictable, dangerous, and helpless.

ALTERNATIVE ETIOLOGICAL EXPLANATIONS

Existing experimental studies have compared the effects of biological explanations of mental disorders on self-stigma to either biopsychosocial or no etiological information. However, given the negative effect of biological explanations on affected individuals' prognostic expectations, it is imperative to examine the effect of other theoretically viable etiological explanations. Previous research has demonstrated substantial empirical support for cognitive-behavioral conceptualizations of depression and anxiety disorders that emphasize maladaptive patterns of thinking and behavior (e.g., Clark & Beck, 2010), and a large body of evidence supports the effectiveness of cognitive-behavioral therapies for the treatment of depression and anxiety disorders (Butler, Chapman, Forman, & Beck, 2006; Olatunji, Cisler, & Deacon, 2010). However, there is also widespread agreement that the causes of many mental disorders are multi-faceted and include biological, psychological, and environmental components (Kendler, 2008). Consequently, research is needed to compare the impact of biological explanations for mental disorders with etiological explanations that emphasize cognitive-behavioral factors as well as the combination of biological and cognitive-behavioral factors on self-stigmatizing attitudes.

MENTAL HEALTH SYMPTOMS

Previous research has consistently demonstrated a positive association of depressive symptom severity with self-stigma (Livingston & Boyd, 2010; Ritsher & Phelan, 2004). However, no studies to date have compared self-stigma among persons with symptoms of other prevalent and impairing mental disorders such as depressive and anxiety disorders. Further, the effect of etiological beliefs on self-stigma may vary across different mental health conditions. For example, different etiological explanations (e.g., biological, cognitive-behavioral) may have varying

effects on self-stigmatizing attitudes among people with predominantly depressive symptoms as compared to those with symptoms of an anxiety disorder. Yet, no studies to date have simultaneously compared the effects of different casual explanations on self-stigma among people with different symptom presentations. Disorder-specific differences, if evident, could inform interventions aimed at reducing self-stigma and improving prognostic expectations.

PRESENT STUDY

The purpose of this study was to examine the effects of biological and cognitive-behavioral etiological explanations, and their combination, on self-stigma among individuals with clinical levels of depressive or social anxiety symptom severity. The five major components of self-stigma that were assessed in the present study were self-blame, prognostic pessimism, perceived dangerousness, unpredictability, and helplessness. Based on findings from previous work examining public and self-stigma, we hypothesized that, relative to the cognitive-behavioral and combined etiological explanations, biological explanations would produce lower self-blame, but greater prognostic pessimism, perceived dangerousness, unpredictability, and helplessness. Finally, it was hypothesized that, given the relationship of depressive symptom severity to self-stigma, individuals with depressive symptoms would report greater self-stigmatizing attitudes relative to those with social anxiety symptoms.

METHODS

PARTICIPANTS

Participants were recruited using Amazon.com's Mechanical Turk (M-Turk), an online crowd-sourcing marketplace where respondents complete brief tasks in exchange for small amounts of monetary compensation. Compared to university-based samples, M-Turk provides better sampling diversity and representation of the U.S. population and at least equivalent validity of data (Buhrmester, Kwang, & Gosling, 2011; Paolacci, Chandler, & Ipeirotis, 2010). A total of 1,820 individuals completed screen-

ing measures for major depression (MDD) and social anxiety (SA) symptoms. Participants endorsing current treatment for either disorder were excluded from the study. The total sample (N = 319) was comprised of a MDD (n = 144) and a SAD (n = 175) symptom group. The sample was predominantly male (n = 171, 53.6%) and White (n = 261, 81.8%), with ages ranging from 18 to 61 years (M = 27.1, SD = 8.52).

MEASURES

Screening Measures. The Patient Health Questionaire-9 (PHQ-9) is a 10-item self-administered measure which was used to screen for major depression (Kroenke, Spitzer, & Williams, 2001). Using the diagnostic algorithm, participants who endorse 5 out of 8 symptoms, including depressed mood or anhedonia, as occurring more than half the days meet criteria for a current major depressive episode (Kroenke & Spitzer, 2002). In order to increase the diagnostic validity of the screening procedure, only participants who met these criteria and rated symptoms difficulties as 1 (somewhat difficult) or greater met the inclusion criteria for the MDD symptom group. The PHQ-9 has demonstrated convergent validity (Martin, Rief, Klaiberg, & Braehler, 2006) and good internal reliability in previous work (Kroenke et al., 2001) as well as in the current study (α = .85). The Center for Epidemiologic Studies–Depression Scale (CES-D) was used to characterize the severity of depressive symptoms in the MDD symptom group (Radloff, 1977). Higher CES-D scores indicate greater depressive symptom severity. The CES-D has demonstrated convergent validity (Weissman, Sholomskas, Pottenger, Prusoff, & Locke, 1977) and good internal reliability in previous research (Roberts, 1980) as well as in the current study (α = .84).

The Social Anxiety Disorder Questionnaire is a four-item measure that was created for the present study to screen for SAD criteria. Similar to the PHQ-9, each of the items on the Social Anxiety Disorder questionnaire correspond to DSM-IV-TR criteria for SAD. Participants were asked to respond Yes or No to each item. For example, participants were asked, "Do you fear doing some things in front of other people (e.g., speaking, writing, or eating) because you think that you will act in a way that will

be humiliating or embarrassing?" Participants who responded Yes to each of the four items met inclusion criteria for the SAD symptom group. The Social Interaction Anxiety Scale (SIAS) was used to characterize the severity of social anxiety symptoms in the SAD symptom group (Mattick & Clarke, 1998). Higher scores are indicative of greater social anxiety symptoms. The SIAS has demonstrated good convergent validity with other measures of social anxiety symptom severity (Heimberg, Mueller, Holt, Hope, & Liebowitz, 1993) and shown excellent internal consistency in previous work (Mattick & Clarke, 1998) and in the current sample ($\alpha = .90$).

Study Variables. Immediately following the manipulation of etiological information, participants were presented with six different etiological factors, three of which were biological (chemical imbalance, genetics, abnormal brain functioning) and three of which were cognitive-behavioral (avoidance behaviors, negative thinking, past life events). Using a 1 (not at all) to 7 (very much) scale, participants rated the likelihood that each factor was responsible for the cause of the disorder. The sum of the scores for the three cognitive-behavioral items was subtracted from the sum of the scores for the three biological items to generate a single score with more positive scores indicating greater biological beliefs and greater negative scores indicating greater cognitive-behavioral beliefs.

Components of self-stigma were measured by creating subscales derived from the Self-Stigma of Mental Illness Scale-Short Form (Corrigan et al., 2012). Specifically, five 3-item subscales measuring self-blame, prognostic pessimism, perceived dangerousness, unpredictability, and helplessness were created to assess components of self-stigma. Subscale items assessed stereotype agreement, stereotype internalization, and harm to self-esteem across each component of self-stigma (Brohan, Slade, Clement, & Thornicroft, 2010). For example, the self-blame subscale was comprised of the following items: "I think most persons with [disorder] are to blame for their problems," "Because I may have [disorder], I am to blame for my problems," and "I currently respect myself less because I am to blame for my problems." Participants indicated their agreement with each item on a 1 (I strongly disagree) to 9 (I strongly agree) scale. Higher scores on

these component scales indicate greater self-stigmatizing attitudes. Internal consistencies for these scales ranged from acceptable to good: self-blame ($\alpha = .76$), prognostic pessimism ($\alpha = .75$), perceived dangerousness ($\alpha = .84$), perceived unpredictability ($\alpha = .80$), and helplessness ($\alpha = .84$).

The Beck Hopelessness Scale (BHS) was used to assess general hopelessness (Beck, Weissman, Lester, & Trexler, 1974). Each item represents an attitudinal statement (e.g., My future seems dark to me), and participants indicate whether the statement is True or False of their attitude during the past week. Higher BHS scores are indicative of greater pessimism. The BHS has demonstrated convergent validity as well as good internal consistency in previous work ($\alpha = .88$; Steed, 2001) and in the study sample ($\alpha = .87$). The BHS was treated as a covariate in this study.

PROCEDURE

After providing informed consent, all participants completed the screening measures as well as several items assessing MDD and SAD diagnostic and treatment history. Only those participants who met study criteria for either MDD or SAD and who did not endorse current treatment for either disorder were invited to participate in the study. Based on their responses to the screening measures, participants were informed that they "may meet criteria for [MDD or SAD]." Participants were then randomly assigned to one of three etiological conditions in which different information was presented about the causes of either MDD or SAD: a biological (BIO) condition, a cognitive-behavioral (CB) condition, and a combined (BIO + CB) condition. The three etiological conditions were standardized in length and differed only in the content of information that was provided to participants in a brief audiovisual presentation. After viewing the presentation, participants completed all measures before being debriefed and compensated. All study procedures were approved by an Institutional Review Board.

Biological (BIO) Condition. Participants assigned to the BIO condition viewed an audiovisual presentation describing either MDD or SAD (depending on their symptom group) as being caused by biological factors, including neurotransmitter imbalances, brain

structure abnormalities, and genetic contributions. To illustrate, the presenter explained that, "[disorder] results from a chemical imbalance in the brain involving a neurotransmitter called serotonin."

Cognitive-Behavioral (CB) Condition. Participants assigned to the CB condition viewed an audiovisual presentation describing either MDD or SAD as being caused by maladaptive patterns of thinking and behaving. For example, it was explained that "people are at increased risk for developing [disorder] if they frequently avoid important life activities," and "people with [disorder] often report worrying about negative outcomes in the future as well as dwelling upon past failures."

Combined (BIO + CB) Condition. Participants assigned to the BIO+CB condition viewed an audiovisual presentation describing either MDD or SAD as being caused by a combination of biological and cognitive-behavioral factors. The content of the information presented was derived equally from the etiological information presented in the previous two conditions. For example, participants were told that "[disorder] is a problem that results from a combination of biological factors, such as chemical imbalances in the brain, and psychological factors, such as avoidant patterns of thinking and behaving."

DATA ANALYSIS

A polynomial contrast was used to assess the linear trend between etiological information condition (BIO = 1, BIO + CB = 2, CB = 3) and participants' etiological beliefs. A 2 (MDD vs. SAD) × 3 (BIO vs. CB vs. BIO + CB) ANOVA was used to examine the main effects of symptom group and etiological information condition on each component of self-stigma. A set of planned contrasts was used to test the hypothesized effects of biological (BIO) and cognitive-behavioral (CB) etiological information on each of the five self-stigma components. Another set of planned contrasts was used to compare differences in self-stigma components between the biological (BIO) and combined (BIO + CB) conditions. These analyses were repeated using ANCOVA to control for participants' scores on the BHS. This measure was

TABLE 1. Unadjusted Descriptive Statistics, Inferential Tests, and Effect Sizes by Causal Explanation Condition for Self-Stigma Component Variables, *N* = 319

	Etiological Condition					
	BIO	CB	BIO + CB	*F*	*p*	η_p^2
Self-Stigma Components						
Self-blame	9.23 (5.60)	11.67* (5.74)	11.79* (6.22)	7.07	.001	.043
Prognostic pessimism	9.72 (5.52)	10.33 (6.53)	11.59* (6.40)	2.84	.060	.018
Dangerousness	5.45 (3.62)	5.18 (3.83)	6.44* (4.89)	3.38	.035	.021
Unpredictability	7.93 (5.23)	7.83 (5.16)	8.97 (5.07)	1.81	.166	.011
Helplessness	8.18 (5.81)	9.84* (6.26)	9.70* (6.40)	3.08	.047	.019

BIO = Biological condition; CB = Cognitive-behavioral condition *Significantly different than the BIO condition, $p < .05$

taken to examine the specific effect of the etiological information and symptom group on mental health related self-stigma while accounting for variance due to general hopelessness and pessimism. Analyses were performed using SPSS version 22.0. All tests were two-tailed with alpha set at .05.

RESULTS

PRELIMINARY ANALYSES

There was a significantly higher rate of prior diagnoses in the MDD symptom group relative to the SAD group, $\chi^2(1, 318) = 5.83$, $p = .016$. No other significant differences in demographic variables were observed between symptom groups or etiological conditions, suggesting that the randomization was successful. Within the MDD group, the average CES-D score ($M = 36.39$, $SD = 9.09$) exceeded the suggested cut-off score of 16 recommended for detecting depression (Radloff, 1977). Similarly, the mean SIAS score ($M = 45.29$, $SD = 12.66$) for the SAD group was greater than a score of 36 used to discriminate between SAD and other anxiety disorders (Peters, 2000). These results indicate that the majority of the sample experienced clinical levels of symptom severity, in addition to meeting criteria for MDD or SAD.

There was a significant linear relationship between etiological explanation condition and etiological beliefs, $F(1, 317) = 4.80$, $p < .001$, such that the BIO condition showed the highest endorsement of biological attributions and the CB condition showed the

highest level of cognitive-behavioral attributions. This result indicates that the experimental manipulation successfully influenced participants' causal beliefs about their mental health problems in the intended manner.

SELF-STIGMA

Table 1 presents descriptive and inferential statistics for the self-stigmatizing attitudes by etiological explanation condition. Multivariate tests demonstrated significant differences among conditions for self-blame, perceived dangerousness, and perceived helplessness. However, there were no significant differences in prognostic pessimism or perceived unpredictability among etiological conditions.

As predicted, planned contrasts revealed that self-blame was significantly greater in the CB condition than in the BIO condition, $p = .002$, $d = .43$. However, contrary to prediction, perceived helplessness was significantly greater in the CB condition compared to the BIO condition $p = .030$, $d = .27$. There were no significant differences in prognostic pessimism, perceived unpredictability, or perceived dangerousness between the BIO and CB conditions.

Contrary to our hypothesis, the BIO + CB condition produced significantly worse effects on nearly all self-stigma variables than the BIO condition, including higher self-blame, $p = .001$, $d = .43$, prognostic pessimism, $p = .019$, $d = .31$, perceived dangerousness, $p = .039$, $d = .23$, and perceived helplessness, $p = .038$, $d = .25$. There was no significant difference in perceived unpredictability between the BIO and BIO+CB conditions, $p = .096$, $d = .20$. All multivariate effects and contrasts comparing etiological conditions remained significant ($p < .05$) when controlling for BHS scores.

Table 2 presents descriptive and inferential statistics for the self-stigma variables by symptom group. Multivariate tests showed significant effects of symptom group across all self-stigma components. As hypothesized, self-blame, prognostic pessimism, perceived unpredictability, perceived dangerousness, and perceived helplessness were all greater in the MDD group

TABLE 2. Unadjusted Descriptive Statistics, Inferential Tests, and Effect Sizes by Symptom Group for Self-Stigma Component Variables, *N* = 319

	Symptom Group				
	MDD	SAD	*F*	*p*	*d*
Self-Stigma Components					
Self-blame	12.37 (6.10)	9.64 (5.59)	18.76	<.001	.47
Prognostic pessimism	11.86 (6.13)	9.52 (6.04)	12.17	.001	.38
Dangerousness	7.08 (4.87)	4.62 (3.18)	29.05	<.001	.60
Unpredictability	9.99 (5.42)	6.87 (4.54)	31.27	<.001	.62
Helplessness	11.26 (6.19)	7.54 (5.68)	32.57	<.001	.63

MD = Major Depressive Disorder; SA = Social Anxiety Disorder

relative to the SA group. Symptom group differences in prognostic pessimism became nonsignificant when controlling for BHS scores. This finding suggests the observed difference in prognostic pessimism between symptom groups was due to higher levels of hopelessness in the MDD group relative to the SAD group. All other differences between symptom groups remained significant when controlling for BHS scores. There was no significant interaction of etiological condition by symptom group on any self-stigma components.

DISCUSSION

The present study is the first to our knowledge to compare the effect of biological and cognitive-behavioral explanations, as well as their combination, on self-stigmatizing attitudes among individuals with clinical levels of depression or social anxiety. It was hypothesized that, compared to the two conditions containing cognitive-behavioral etiological information, the biological condition would lead to lower levels of self-blame but greater prognostic pessimism as well as increased perceptions of unpredictability, dangerousness, and helplessness. Further, it was predicted that all self-stigmatizing attitudes would be worse among participants in the MDD rather than SAD symptom group. The current findings partially supported this hypothesized pattern of results.

Relative to cognitive-behavioral etiological information, biological information yielded lower levels of self-blame. This effect is consistent with previous findings suggesting biological attri-

butions for mental disorders leads to lower levels of self-blame compared to either biopsychosocial or no etiological information (Deacon & Baird, 2009; Lebowitz, Pyun, & Ahn, 2014). However, the present findings are the first to show that cognitive-behavioral etiological information yielded greater self-blame compared to a biological explanation among a sample of individuals with clinical levels of depression or social anxiety.

As previous work has noted, providing patients with some level of personal control over the development and maintenance of symptoms may be an important mechanism by which cognitive-behavioral therapies effect change in maladaptive patterns of thinking and behavior (Deacon & Baird, 2009). Indeed, an individual's perceived agency over maladaptive patterns of thinking and behaving may be necessary for effective cognitive-behavioral treatment of depression and social anxiety. Consequently, it may be important for providers of cognitive-behavioral therapy to distinguish between self-defeating forms of self-blame and recognition of personal factors that contribute to the symptoms of depression and social anxiety. Future research is needed to clarify this potentially important difference.

Contrary to our hypothesis, there were no differences in prognostic expectations between the cognitive-behavioral and biological information conditions. This finding was unexpected, as other studies have shown that biological explanations are associated with increased prognostic pessimism (Kemp, Lickel, & Deacon, 2014; Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Lebowitz et al., 2014). However, important methodological differences may account for these incongruent findings. For example, a previous study examined the relationship between pre-existing etiological beliefs and prognostic expectations (Lebowitz et al., 2013). Compared to pre-existing beliefs in the biological etiology of mental disorders, experimentally manipulated biological explanations may have a comparatively smaller impact on prognostic pessimism. Two other studies in this area used an experimental design that compared the effects of biological explanations to no etiological information (Kemp et al., 2014; Lebowitz et al., 2014). Thus, it is possible that both biological and cognitive-behavioral messages elicit greater prognostic pessimism compared to no etiological information.

Also contrary to our hypothesis was the finding that cognitive-behavioral etiological information led to greater perceived helplessness compared to biological information. To date, no studies of which we are aware have examined the effect of casual attributions on perceived helplessness among people with mental health conditions. However, this effect is inconsistent with findings from at least one prior study showing that biological explanations yield greater public perceptions of mental disorders as highly disabling compared to environmental explanations (Lam et al., 2005). Participants may have interpreted cognitive-behavioral attributions for their symptoms as evidence for their own helplessness. Further research is needed to corroborate this finding. However, this result may suggest that patients could benefit from providers' attempts to directly address any untoward implications associated with cognitive-behavioral etiological conceptualizations. For example, providers might emphasize patients' control over cognitive and behavioral factors that maintain symptoms while simultaneously providing psychoeducation about patients' lack of culpability for their symptoms.

Extant literature suggests that greater endorsement of biological etiological beliefs is associated with greater perceptions of people with mental health conditions as dangerousness and unpredictable (Jorm et al., 2012). However, our results showed no differences in participants' perceptions of personal dangerousness or unpredictability between biological and cognitive-behavioral etiological conditions. This discrepancy may reflect inaccuracies in public attitudes toward people with mental disorders. For example, the general public greatly overestimates the threat posed by people with mental health conditions, including depression and anxiety disorders (Angermeyer & Dietrich, 2006; Pescosolido, Monahan, Link, Stueve, & Kikuzawa, 1999; Reavley & Jorm, 2011). Conversely, people who experience symptoms of depression or social anxiety may, based on personal experiences, more accurately determine that they are neither unpredictable nor dangerous. Framed in the context of Corrigan's progressive model of self-stigma (Corrigan, Rafacz, & Rüsch, 2011), people with symptoms of depression or social anxiety may not internalize inaccurate stereotypes regarding dangerousness or unpredictability. Consequently, the predicted effect, which was based

on findings from studies examining public attitudes, appears not to have emerged in the current study.

To our knowledge, this is the first study to compare the effects of biological information only versus combined biological and cognitive-behavioral etiological information on self-stigma. Interestingly, all self-stigma variables were significantly greater (i.e., worse) in the combined etiological information condition compared to the biological information condition. This pattern of findings is particularly worrisome given that combined etiological explanations may be most consistent with current scientific research (Kendler, 2008). It is plausible that multiple sources of etiology may imply that mental health symptoms are more serious and consequently, more stigmatizing. Further research is needed to understand the mechanisms by which combined etiological information may contribute to greater self-stigmatizing attitudes.

The present study is one of the first to compare self-stigma among individuals with different mental health symptoms. As expected, all self-stigma variables were significantly greater in the MDD group relative to the SAD group. These findings suggest that, compared to individuals with SAD, individuals with MDD may have higher levels of self-blame for symptoms, poorer prognostic expectancies as well as greater perceptions of themselves as dangerous, unpredictable, and helpless. This pattern of results is supported by cognitive models of depression that emphasize the role of negative self-schemas inherently associated with depressive symptoms (Beck & Dozois, 2011).

Symptom group differences in prognostic pessimism became nonsignificant when controlling for BHS scores, suggesting that the more negative beliefs about symptom course observed in the MDD group were driven by general hopelessness. However, all other symptom group differences in self-stigma (i.e., self-blame, perceived dangerousness, unpredictability, and helplessness) held while controlling for BHS scores, suggesting that symptom group differences in these specific self-stigmatizing beliefs may exist independent of general hopelessness and pessimism associated with depression. Future research should examine the efficacy of supplemental interventions aimed at reducing these self-stigmatizing attitudes, particularly among individuals with depression. Interventions that reduce self-stigma may improve

treatment adherence (Sirey et al., 2001) and ultimately increase individuals' likelihood of recovery.

LIMITATIONS

Findings from this study are qualified by several limitations. First, given the web-based nature of the study, we could not confirm participants' diagnoses of either MDD or SAD. However, symptom severity scores were substantially higher than the suggested cutoffs for both the CES-D and SIAS (Mattick & Clarke, 1998; Weissman et al., 1977). The majority of the sample had never been diagnosed with either disorder. However, diagnostically naïve participants may have fewer preconceptions about the causes of their symptoms and may therefore be particularly well-suited for studies assessing the effects of various etiological explanations on the self-stigma of mental disorders. Second, this study did not use a no-information control group. Consequently, it was not possible to compare the effects of any etiological information to no information. Future work could address this limitation. Third, although we used a previously validated instrument to measure self-stigmatizing attitudes, we modified the scoring scheme such that scale scores were calculated for the five attitudinal components of self-stigma as opposed to three different stages of the self-stigmatization process that has been described in previous literature. More research is needed to determine the effects of various etiological explanations on self-stigma and prognostic expectations among people with mental disorders. Future work in this area has the potential to strengthen efforts at reducing self-stigma among people with mental disorders.

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