CAUSAL BELIEFS ABOUT DEPRESSION AS PREDICTORS OF ESSENTIALIST THINKING AND IN NETWORK MODELS

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ABSTRACT OF THE DISSERTATION

Causal Beliefs About Depression as Predictors of Essentialist Thinking

and in Network Models

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This project includes two studies of causal beliefs about depression conducted in a U.S. adult sample (N = 319) via Amazon's Mechanical Turk platform. Study 1 tested hypotheses based on essentialist theory, guided by the theoretical framework of Leventhal's Commonsense Model (CSM) of illness cognition. Essentialist theory suggests that in the general population, biological causal beliefs about mental illnesses, including depression, frequently are associated with negative prognostic beliefs and stigmatizing attitudes. Consistent with this, findings indicated that biological causal beliefs were associated with viewing depression as more consequential and longer-lasting; contrary to hypotheses, biological attributions also predicted viewing depression as more treatable. Also counter to predictions, biological causal beliefs were *inversely* related to depression stigma; these relationships were partially mediated by beliefs about consequences and duration. Relationships between biological causal beliefs and stigma also were moderated by familiarity with depression, such that weaker biological attributions predicted higher levels of stigma specifically among participants who reported a history of depression. Study 2 used network analysis to model perceived interrelationships among putative causes of depression. Network models of causal beliefs

were generated for the full sample and for subgroups reporting high versus low confidence in their understanding of depression (illness coherence). These models varied considerably in complexity and illuminated within-sample differences in construals of stress versus depression, beliefs about mutually maintaining factors (bidirectional relationships), and the role of biological causes in the context of other factors. Together, these studies suggest refinements to essentialist theory, avenues for future research into relationships between mental illness beliefs and stigma, and guidance for psychoeducation in depression treatment and public health messaging.

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General Introduction

Essentialist beliefs about human groups attribute group differences to an 'essence' that is fixed, perceived as distinctive of group members, thought to give rise to group-defining properties, and often viewed as biologically based. In the study of lay beliefs about mental disorders, essentialist theory has been invoked to explain associations between beliefs that these disorders are biologically caused and, (1) beliefs that their prognosis is poor, and (2) stigmatizing attitudes toward affected individuals (Dar-Nimrod & Heine, 2011; Haslam, 2011; Haslam et al., 2006). The commonsense model of self-regulation (CSM; Leventhal et al., 1997; 2010) provides a conceptual framework describing the content of lay models of illness that provides a basis for extending and elaborating upon essentialist theory. The two studies we report examined causal beliefs about major depression, which ranks as the world's leading cause of disability and a leading source of health care costs (WHO, 2013; 2017). A sample of U.S. adults was recruited nationwide via Amazon's Mechanical Turk platform to complete a set of online questionnaires. Study 1 tested hypotheses derived from essentialist theories—and framed in terms of CSM constructs—concerning the relationships of biological causal beliefs about depression to prognostic beliefs and depression-stigmatizing attitudes. Study 2 used network analysis to provide a more nuanced depiction of respondents' causal models of depression. A consensus causal network was generated based on the beliefs of the full sample, and separate networks were generated and compared for sample subgroups reporting high versus low confidence in their understanding of depression.

Study 1

Advances in biomedical research have fueled public health campaigns promoting a view of mental illnesses as biomedical diseases (Pescosolido et al., 2010; Schomerus et al., 2012). One goal of these campaigns has been to reduce the stigma associated with mental health problems (Pescosolido et al., 2010; Schomerus et al., 2012). In line with this goal, research suggests that viewing mental illness as due to biological causes is associated with ascribing less blame to individuals with these conditions (Kvaale, Gottdeiner, & Haslam, 2013; Lebowitz, 2014). However, biomedical views also have been linked to more negative prognostic beliefs about mental illnesses (Kvaale, Gottdeiner, & Haslam, 2013; Lebowitz, 2014) and to increases in certain aspects of mental illness stigma, such as perceiving affected individuals as threatening and unpredictable, and responding to them with anger, fear, and social avoidance (e.g., Boysen, 2011; Mehta & Farina, 1997; Parcesepe & Cabassa, 2013; Rüsch et al., 2010a; 2010b; Speerforck et al., 2014).

Essentialist Theory

Essentialism has conceptual roots in philosophy and cognitive science; early research on psychological essentialism focused on the bases for children's conceptual distinctions between living things and man-made objects (Gelman & Hirschfield, 1999). Over subsequent decades, this line of work broadened to examine the extent to which people implicitly view various types of human differences in an essentialist way: as identity-defining, permanent, and attributable to an unseen "substance…power…or entity that *causes* other category-typical properties to emerge and be sustained" (Gelman & Hirschfeld, 1999, p. 406, emphasis in the original). Essentialist beliefs and their social

implications have been studied with regard to personality attributes and many kinds of social groups (Gelman & Hirschfield, 1999). Essentialist beliefs about social groups hold that group members share a fixed, fundamental nature (Dar-Nimrod & Heine, 2011; Haslam et al., 2006; Yzerbyt, Corneille, & Estrada, 2001; Yzerbyt & Rocher, 2002; Yzerbyt & Rogier, 2001). One of the more developed theoretical frameworks for describing essentialist beliefs about social groups suggests that these beliefs have four facets: (1) The group is *discrete* and categorically separable from other groups; (2) group-defining attributes are *immutable;* (3) these attributes are broadly *informative* about members' other characteristics; (4) these properties are manifestations of an underlying 'essence' perceived to be *biologically based*. These four facets are thought to be interrelated yet conceptually and empirically distinguishable (Haslam et al., 2006). There is some preliminary support for these assertions (e.g., Haslam & Ernst, 2002).

Biological Essentialism and Mental Illness. When essentialist beliefs are applied to social groups, the group-defining 'essence' often is construed as natural and attributed to biology (Kashima et al., 2005; Rothbart & Taylor, 1992); it is further characterized by its perceived stability and presumed causal relationship with observable features (Dar-Nimrod & Heine, 2011). The fact that these same properties often are attributed to genes—likewise viewed as biological, stable, and not directly observable yet presumed to cause distinctive, observable features—has led to the suggestion that lay beliefs about genetic influences may be especially likely to promote essentialist thinking about group differences, with genes serving as conceptual "placeholders" for essences (Dar-Nimrod & Heine, 2011). For example, lay thinking about the biological species concept tends to view species as discrete categories, each defined by an innate, shared

genetic essence that shapes and constrains members' observable attributes (Dar-Nimrod & Heine, 2011). Communications to the public about genetic research may reflect and reinforce biological essentialist thinking by presenting genes as powerfully and directly deterministic of human differences (Conrad, 2002).

One significant focus of biological essentialist research has been lay beliefs and attitudes about mental illness. Theorists have argued that lay beliefs about mental illness often incorporate an essence-like understanding of genetic (and perhaps also neurobiological) causes, and that clear links therefore can be expected between biological causal beliefs and other beliefs and attitudes about these conditions (Dar-Nimrod & Heine, 2011; Haslam, 2011). In this view, attributing mental disorders to biological causes implicitly invokes beliefs that the disorders themselves are *immutable* (i.e., chronic, difficult to treat) and highly *informative* about affected individuals, who are then viewed as a relatively homogenous, *discrete* group. This possibility has garnered some preliminary experimental support (Haslam & Ernst, 2002). Though genetic causal beliefs have been highlighted as especially compatible with essentialist beliefs, theorists have not yet made claims distinguishing between the implications of neurobiological versus genetic etiological beliefs. They have more often highlighted associations between various "biogenetic" causal beliefs and negative views of mental disorders' prognoses (e.g., Deacon & Baird, 2009; Lam & Salkovskis, 2007; Lebowitz, 2014; Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Phelan, 2005) and with stigmatizing attitudes toward affected individuals (Heine, 2011; Kvaale, Gottdiener, & Haslam, 2013; Schomerus et al., 2012).

Biological Essentialism and Mental Illness Stigma. The concept of stigma in social psychology has roots in sociological theories about the ways in which a particular

feature of a person's identity can be translated into a devalued social status such as may occur with race, ethnicity, religious affiliation, physical illness or handicap, or a behavior viewed as morally suspect, like gambling or receiving government aid (Goffman, 1963). Social disfavor associated with mental illness was fundamental to the development of the stigma concept, as individuals' 'tainted' social position after release from a psychiatric hospital served as one of sociology's foundational examples of stigmatization (Goffman, 1961; Pescosolido, 2013). Today, mental illness stigma refers to the attitudes and beliefs that lead people to fear, reject, avoid, and discriminate against individuals with mental illnesses (Corrigan & Penn, 1999). Researchers have defined and measured stigma, including mental illness stigma, using a variety constructs including emotional responses to stigmatized people (fear, anger), beliefs about responsibility and behavioral control (blame, perceived unpredictability, perceived danger), and behavioral inclinations toward stigmatized people (desire to maintain social distance; Link et al., 2004).

Essentialist beliefs are thought to have important implications for the social judgments that constitute stigma (Dar-Nimrod & Heine, 2011; Haslam et al., 2006). Holding essentialist beliefs about social categories has been described as tantamount to misconstruing socially constructed groups as 'natural kinds' (Rothbart & Taylor, 1992). This way of thinking can amplify perceived group differences by implying that social groups are bounded, discrete, and homogenous (Dar-Nimrod & Heine, 2011). Such thinking has been linked with the emergence of stereotypes and their use in explaining individuals' behavior. When a group is perceived as a stable, uniform social entity defined by shared characteristics, group membership can become the basis for inferring that the characteristics viewed as group-defining are also dispositional to individual

members. In this context, group membership can suggest internal, dispositional attributions for individual members' behavior, whenever it aligns with expectations about the group, and it can set expectations for members' future behavior (Yzerbyt, Rogeir, & Fiske, 1998). In this respect, essentialist beliefs "serve as theories that give explanatory coherence to group stereotypes and foster dispositional attributions" about individuals (Haslam et al., 2006, p. 67).

Essentialist theory, as applied to lay beliefs and attitudes about mental illness, predicts several specific negative implications of attributing these disorders to biological causes. These implications include viewing the disorder as more chronic and intractable (*immutable*; Haslam & Ernst, 2002; Haslam et al., 2006) and viewing affected individuals as both more like one another (*informative*) and more categorically distinct from others (*discrete*; Dar-Nimrod & Heine, 2011; Haslam, 2011). These predictions are based on the essentialist premise that for the lay public, biological causal beliefs about a particular disorder are inherently linked with specific negative prognostic beliefs about it (e.g., its *immutability* and *informativeness*), which, together inform the perception of the diagnostic category as more internally homogenous and distinctive from nonmembers, like a *discrete* natural kind.

Consistent with essentialist theory, biological causal beliefs about mental illness have been associated with some kinds of negative beliefs about disorders themselves in studies of the general public, including a greater degree of prognostic pessimism (Liebowitz, 2014; Liebowitz & Ahn, 2015; for a review, see Kvaale, Gottdiener, & Haslam, 2013). Evidence in the literature is mixed for essentialist predictions regarding stigma. Studies of the general population and other non-patient samples have found that

holding biological causal beliefs about metal illness is associated with *less* blaming of individuals for their disorder (Kvaale, Haslam, & Gottdiener, 2013) and, counter to essentialist predictions, with greater expressed support for affected individuals receiving treatment (Pescosolido et al., 2010). The desire to change public presumptions about individuals' responsibility for their own illnesses, and thereby reduce blame, contributed to the rationale for emphasizing biological causality in some anti-stigma campaigns (Jorm et al, 1997; Weiner, Perry, & Magnussen, 1988). However, biological attributions for mental illness also have been associated with a greater likelihood of viewing affected people as dangerous and desiring more social distance from them (reviewed in Kvaale, Gottdiener, & Haslam, 2013). Some studies of patient samples also have reported a positive correlation between holding biological causal beliefs about one's own illness and higher illness-related self-stigma on implicit measures (Rüsch et al., 2010a; 2010b).

These studies offer some support for essentialist theorists' concerns about negative correlates of holding biological causal beliefs. However, work in this area typically lacks a theoretical framework for describing illness beliefs. Instead, these studies often have examined only specific beliefs considered most central to essentialist hypotheses, measuring them with a small number of narrowly construed items (see Kvaale, Gottdiener, & Haslam, 2013; Lebowitz, 2014, for reviews). This approach has provided limited understanding of the kinds of thinking about mental illness that gives rise to these relationships. These studies also generally have not accounted for individual difference factors that would be expected to influence the relationships predicted by essentialist theory, such as participants' clinical history and other lived experience—or

lack thereof—with the disorder of interest (e.g., Baines & Wittkowski, 2013; Hale, Treharne, & Kitas, 2007; Leventhal et al., 2008).

Lay Representations of Illness and the Commonsense Model

The claims within essentialist theory that biological causal beliefs about mental disorders are inherently linked with other beliefs about these conditions may profitably be construed within the framework of Leventhal's Commonsense Model (CSM). The CSM focuses on the content and implications of individuals' multifaceted illness beliefs, and thereby anticipates some of the interests of essentialist theorists. The CSM provides a framework for describing individuals' beliefs about specific health problems in terms of five main factors: (1) the *identity* of the problem (its label and symptoms), (2) its *causes*, (3) its anticipated *consequences*, (4) its expected *duration*, and (5) its *treatability* (Leventhal, et al., 1997; 2010). Over several decades, these factors have been studied in connection with a wide variety of physical health problems (e.g., Leventhal et al., 2010) and, more recently, with a number of mental disorders as well (Baines & Wittkowski, 2013). In addition to specifying these five components of illness beliefs, the CSM posits that a lay person's beliefs about a given illness reflect the person's general knowledge of the condition (the *illness prototype*), further informed and enriched, where relevant, by personal experiences with the illness (the *illness representation*; Leventhal et al., 2010; Leventhal, Leventhal, & Contrada, 1998). The CSM's five illness belief constructs can be measured, empirically intercorrelated, and examined in association with stigmatizing attitudes and with respect to potential demographic and clinical moderators (Haggar & Orbell, 2003).

The Commonsense Model and Essentialist Theory

The clearest point of correspondence between essentialist theory and the CSM is their causal belief constructs. While the CSM broadly construes causal beliefs to include whatever the individual thinks brought about the illness, essentialist theory is particularly concerned with whether the illness is viewed as *biologically caused*. Second, the CSM's *consequences* construct corresponds, in part, to the essentialist perception that the group-defining essence (in this case a particular mental illness) is highly *informative* about affected individuals, as both constructs describe the extent to which the presence of the illness supports inferences about other aspects of the individual. Third, two distinct CSM constructs describe beliefs about whether and by what means the illness can change (improve or worsen): the illness's perceived *duration* (e.g., acute versus chronic timeline) and its *treatability.* The corresponding essentialist construct focuses on the perceived *immutability* of the illness, which could be described in the CSM's terms as beliefs that the illness has a long (potentially unlimited) duration and is difficult or impossible to treat. The fourth facet of essentialist thinking focuses on the belief that individuals with a particular diagnosis (or those with any mental health problem) are viewed as members of a *discrete* group or category, which is considered relatively homogenous and distinct from everyone else. This facet of essentialism does not correspond to a CSM construct. Rather, its relevance to this project—beyond its role within existentialist thinking—is its close relationship to the stigma concept.

Focus on Depression: Rationale and Background

The project focuses on depression, in part because this disorder represents a significant burden on affected individuals' well-being and finances (Greenberg & Birnbaum, 2005; Kessler et al., 2005; WHO, 2013, 2017). In addition, it has a sufficient

twelve-month prevalence rate in the general U.S. adult population (7%; Kessler & Wang, 2008) to enable online recruitment of participants with varying levels of familiarity with the disorder, ranging from current, clinically significant depressive symptoms and/or a history of depression diagnosis and treatment, to second-hand experience with a close other's depression, to little or no direct experience. In addition, beliefs in the general public about the causes of depression tend to include a wider variety of contributing factors (e.g., stress, life events, personal characteristics, interpersonal problems) than do causal beliefs about psychotic disorders like schizophrenia, which are more often attributed exclusively to biology (MacDuffie & Strauman, 2017; Schomerus et al., 2012)

In line with the predictions of essentialist theory, biological causal beliefs about depression have been associated with aspects of prognostic pessimism in both depressed and nondepressed samples, including viewing depression as exerting serious negative consequences, having a long duration, and being relatively unresponsive to treatment (Deacon & Baird, 2009; Lebowitz, 2014; Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Phelan, 2005). However, as noted earlier, studies examining relationships between biological attributions and other illness beliefs generally have used brief, untested belief measures, sometimes consisting of a few narrowly construed items (reviewed in Lebowitz, 2014).

A smaller number of studies focused on illness beliefs about depression have been guided by the CSM, typically using versions of the Illness Perception Questionnaire-Revised (IPQ-R; Moss-Morris et al., 2002), to measure patients' beliefs about depression's identity, cause, consequences, duration, and treatability. Most of this research has focused on outcomes such as the adoption of particular coping strategies,

treatment utilization, or adherence to antidepressant medication (e.g., Brown et al., 2001; 2007; Houle et al., 2014; O'Mahen et al., 2009; Vollman et al., 2010). Work conducted to date has not often considered relationships between IPQ scales or focused on causal beliefs (e.g., Cabassa et al., 2008; Munson, Floersch, & Townsend, 2009). In other work, biological causal beliefs were analyzed only in combination with beliefs about nonbiological causes (e.g., Brown et al., 2007).

To our knowledge, the present study is the first to examine the relationships between causal beliefs, prognostic beliefs, and stigmatizing attitudes about depression in a large lay sample, including individuals varying in levels of familiarity and experience with the disorder and employing measures to capture these individual differences. In addition, this is the first study to test key predictions of essentialist theory regarding the structure and social implications of certain lay beliefs about mental illness by applying a robust theoretical and empirical framework for describing illness beliefs, as the CSM provides.

Hypotheses

This study investigates essentialist hypotheses about the relationships between biological causal beliefs about depression, prognostic beliefs, and depression stigma, incorporating aspects of the CSM as a guiding framework. A series of hypothesis-tests was conducted in questionnaire data gathered from a sample of U.S. adults recruited online via Amazon's Mechanical Turk platform. The first set of hypotheses predicted that holding biological causal beliefs about depression would be associated with more negative prognostic beliefs about the disorder's consequences, duration, and treatability, as well as with higher levels of depression stigma. The second set of hypotheses tested the extent to

which prognostic beliefs—a key basis for out-group definition within essentialist theory—mediate the relationship between biological causal beliefs and stigmatizing attitudes. The third set of hypotheses tested the moderating effects of participants' familiarity with depression, via their own clinical history of that of a close other, on the relationship between biological causal beliefs and stigmatizing attitudes. Our expectation was that the results would inform the further development of theory to guide research on essentialist (as well as non-essentialist) beliefs about depression and the effects of these representations on stigma and other personal and social consequences of this disorder.

Effects of Causal Beliefs About Depression on Perceived Prognosis and Stigma

Based on essentialist theory and prior findings (Deacon & Baird, 2009; Lebowitz, 2014; Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Phelan, 2005), biological causal beliefs were expected to show a significant association with negative prognostic views of depression. Previous work also provides a basis for predicting that attributing depression to biological causes would show a significant positive relationship with stigmatizing attitudes toward depressed individuals (Goldstein & Roselli, 2003; Jorm & Oh, 2009; Kvaale, Gottdiener & Haslam, 2013; Lebowitz, 2014; Link et al., 2004; Pescosolido et al., 2010; Phelan, 2005; Schomerus et al., 2012).

Hypothesis 1a. The belief that depression is caused by genetic factors, and the belief that depression is caused by changes in chemicals in the brain, will each have an independent association with the belief that depression is highly consequential, has a long duration, and is difficult to treat.

Hypothesis 1b. The belief that depression is caused by genetic factors, and the belief that depression is caused by changes in chemicals in the brain, will each have an

independent association with holding stigmatizing attitudes toward depressed individuals (personal stigma), and to desiring greater social distance from depressed people. The relationship of biological causal beliefs to the third measured stigma variable—beliefs about the level of depression-stigmatizing attitudes commonly held by "most other people" (perceived stigma)—was examined on an exploratory basis.

Perceived Prognosis as a Mediator of the Effects of Causal Beliefs on Stigma

Essentialist theorists have granted biological causal beliefs a kind of primacy in their descriptions of lay thinking about mental illness (Dar-Nimrod & Heine, 2011; Racine et al., 2010). It has been suggested, for example, that "learning about genetic attributions for a particular condition *leads to* a particular set of thoughts regarding those conditions" (Dar-Nimrod & Heine, 2011, p. 800, emphasis mine). That is, the other defining facets of essentialist thinking (*immutability, informativeness, discreteness*) are described as following from biological causal beliefs. Biological causal beliefs about depression therefore are expected to evoke beliefs about its having a poor prognosis (highly consequential, long duration, unresponsive to treatment), which in turn are expected to predict depression stigma. Statistical mediation provides one way to operationalize and test this line of thinking.

Hypothesis 2. Significant relationships linking genetic and neurobiological attributions for depression to stigmatizing attitudes will be statistically mediated by relationships of those two biological beliefs with negative prognostic beliefs about its consequences, duration, and treatability. Given the potential statistical overlap between these three prognostic belief variables, and in the interest of theory development, these

variables were analyzed separately as potential mediators of the relationship of biological beliefs to stigma measures, rather than as competing mediators within the same model.

Familiarity with Depression as a Moderator of the Effects of Biological Causal Beliefs on Prognostic Beliefs and Stigma

One way this study sought to extend prior work on lay beliefs about depression was by examining the extent to which participants' familiarity with depression affects the relationships of biological causal beliefs to either prognostic beliefs or depression stigma. Though not addressed by essentialist theory (Dar-Nimrod & Heine, 2011; Haslam, 2006; 2011), the CSM recognizes lived experience as a major source of the information that shapes illness beliefs, which in turn influence illness-related emotions and behaviors (Cameron & Leventhal, 2003; Leventhal et al., 2010). Familiarity with depression was measured in terms of whether the participant believed: (1) that he or she had ever experienced an episode of clinical depression, and (2) that at least one close other had ever experienced clinical depression. No predictions were made about differential effects of first-hand versus second-hand experience; on an exploratory basis, these variables were examined both as sole and as simultaneous moderators.

Both familiarity variables were expected to moderate the relationship between biological causal beliefs and negative prognostic beliefs. Participants reporting personal experience with their own and/or close others' depression have more diverse types of information (e.g., vivid and episodic versus general and abstract) to draw on when considering depression's consequences, duration, and treatability. This is expected to inform a more varied set of prognostic beliefs among these participants (Leventhal et al.,

2015), which was expected to weaken any association between biological causal beliefs and the specific (negative) prognostic beliefs that comprise essentialist thinking.

From a social psychological perspective, the kind of individuating information about people with depression that is available to those reporting first- or second-hand experience was expected to reduce their likelihood of relying on stigmatizing heuristics or stereotypes, including those based on essentialist beliefs (Fiske, Lin, & Neuberg, 1999; Fiske & Neuberg, 1990). This prediction draws on key principles in the well-supported continuum model of impression formation (Fiske, Lin, & Neuberg, 1999; Fiske & Neuberg, 1990). This model suggests that members of stigmatized groups are more likely to be judged negatively—and with greater emphasis on stigmatized aspects of identity—when the perceiver has less access to individuating information about the target. Furthermore, the availability of such individuating information would be expected either to pre-empt or to weaken an essentialist view of the diagnostic category as a homogenous, discrete social group with fixed properties. Therefore, those who report having been depressed themselves or close to people with depression are expected to hold less stigmatizing views, due at least in part to their access to richer individuating information about people with the condition.

Hypothesis 3a. Among participants lacking first- or second-hand experience with depression, biological causal beliefs were expected to show a stronger association with negative prognostic beliefs, compared to the strength of these relationships among participants who report such experience.

Hypothesis 3b. Among participants lacking first- or second-hand experience with depression, biological causal beliefs were expected to show a stronger association with

depression stigma, compared to the strength of these relationships among participants who report such experience.

Methods

Participants

Participants in the final sample (N = 319) ranged in age from 18 to 88 years (M = 38.34, SD = 12.696); 60.8% were female. Whites/European Americans (83.7%) and Asians/Asian Americans (7.2%) were overrepresented relative to their proportions in the national population (72.4% and 4.8%, respectively; CIA, 2017), while Blacks/African Americans were underrepresented (6.6% versus 12.6% nationally) as were those of Latino/a or Hispanic ethnicity (4.7% versus 16.3% nationally; CIA, 2017). Other groups comprised a total of 4.9% of the sample: 3.1% American Indian, .9% Hawaiian or Pacific Islander, and .9% Middle Eastern or North African. (Among these seven racial/ethnic groups, participants were allowed to mark all that apply, so percentages sum to more than 100%.) Most participants worked for pay for 35 hours per week or more (64.3%), in addition to completing tasks on MTurk. Education levels in the sample ranged from a high school degree or less (8.8%) to a graduate degree (16.6%); approximately half the sample (53.9%) had completed at least a four-year college degree. Household income levels in the sample were measured using six categories in $20,000 intervals. As Table 1 shows, the majority of the sample (n = 211, 66.2%) was divided approximately equally across the three middle income categories between $20,000 and $80,000. Regarding clinical characteristics, about half the sample (48.6%) endorsed the belief that they had previously experienced an episode of clinical depression. On a brief measure of depressive symptoms experienced in the past two weeks (PHQ-9; Spitzer, Kroenke, &

Williams, 1999), 20% of respondents (n = 65) scored at or above the threshold for moderate clinical significance (PHQ-9 ≥ 10).

Measures

Illness Perception Questionnaire-Revised (IPQ-R), modified for depression.

Participants' beliefs about depression were measured by a version of the Illness Perception Questionnaire–Revised (IPQ–R; Moss-Morris et al., 2002) adapted to assess representations of depression. The IPQ-R includes five scales designed to measure the five components of illness representations in the Commonsense Model (CSM) of illness cognition (Leventhal et al., 1997): beliefs about the *identity* of the illness and its symptoms, its *causes*, its expected *duration*, *consequences*, and *treatability*. Response formats vary among IPQ-R scales; for most scales, respondents indicate their level of agreement with statements on a five-point, non-numeric Likert scale ranging from "strongly disagree" to "strongly agree." Research into the validity of the CSM's major illness belief constructs has found support across a variety of chronic physical illnesses, severe mental illness, and depression in particular (Baines & Wittkowski, 2013; Haggar & Orbell, 2003; Lobban, Barrowclough, & Jones, 2003; Lynch et al., 2011). Evidence from studies of chronic physical illness, where research is more abundant, also generally has supported the expected relationships between CSM illness beliefs. Illnesses seen as longer-lasting are, for example, viewed as more consequential for those affected, while illnesses viewed as more treatable tend to be viewed as less consequential (Haggar & Orbell, 2003).

The IPQ-R was revised for this study based on recommendations for adapting it to specific illnesses (Moss-Morris et al., 2002) and on prior adaptations to assess beliefs

about depression (e.g., Fortune, Barrowclough, & Lobban, 2004; Vollman et al., 2010). Other depression-focused revisions of the IPQ have shown moderate to good internal consistency at the scale level (α = .56 – .78; Fortune, Barrowclough, & Lobban, 2004); to our knowledge, their test-retest reliability has not been studied. The IPQ-R's standard instructions presume that the respondent has the (unnamed) illness of interest. In the present study, references to "my illness" in the instructions were replaced with "depression" and instructions were lightly edited to make them appropriate for both depressed and nondepressed respondents (e.g., "We are interested in your own personal views about depression. Please indicate how much you agree or disagree with each of the following statements"). An exception, the Cause scale, takes a slightly different format; its standard instructions were similarly revised (see Appendix).

Illness identity scale. The main Identity scale, which provides a list of common symptoms for respondents to endorse as related or unrelated to the illness in question, was omitted from this version of the IPQ-R. Instead, participants were given sufficient information about the "illness identity" of major depression to provide a minimal, common frame of reference for responding to the other IPQ-R scales (see Appendix). This choice was based on a difference between this study's sample and that of most other studies using the IPQ-R. Other studies typically have examined the beliefs of a particular population with direct experience of the target illness, such as patients or caregivers. In contrast, this study examined beliefs about major depression in a large sample of individuals with varying levels of experience and knowledge of the condition. Respondents were provided a frame of reference for depression's illness identity (e.g., symptoms, differences from normal functioning) so they could respond to the items with

the same basic type of functional problem in mind. The aim was to introduce a sufficiently consistent frame of reference to improve the interpretability of results, without over-riding participants' own beliefs about clinical depression.

Emotional representations scale. The six IPQ-R items pertaining to emotional representations of illness (e.g., illness-linked experiences of depression, anxiety, worry, or anger) were omitted because of their overlap with depressive symptoms.

Coherence scale. The five-item Identity-related subscale that pertains to illness *coherence*—the extent to which the person believes he or she clearly understands the illness—was retained for use only in Study 2 and is discussed further in that part of the dissertation.

Cause scale. The Cause scale was revised according to three goals: (a) to reflect putative causes of depression rather than causes of physical illness, (b) to more clearly distinguish key biological causal beliefs from other causal attributions in order to aid interpretation, and (c) to address an issue of data quality in prior IPQ-based depression research. Regarding the first two goals, several items that have rarely been cited as causes for depression in prior IPQ-based research and that have more face validity as causes of physical illness were omitted. These include "a germ or virus," "diet or eating habits," "pollution in the environment," and "altered immunity" (Brown et al., 2001; 2007; Fortune, Barrowclough, & Lobban, 2004; O'Mahan et al., 2008; Prins et al., 2008; Vollman et al., 2010). Causes that are potentially depression-relevant and include a biological component were reworded to sharpen the distinction between more directly "biological" effects on mood, such as the depressant effects of alcohol, as opposed to psychosocial ramifications of problem drinking. For example, "alcohol" and "smoking"

were replaced by “Problems resulting from alcohol and/or drug use.” Based on prior IPQ-R-based research on depression, the item “The death of someone close” was added to the list of potential causes (e.g., Brown et al., 2001). In addition, the item “Biological changes in the brain” was added to this scale, as conceptualizing depression as neurobiologically caused is both increasingly common and relevant to this study’s hypotheses about implications of biological causal beliefs (Pescosolido et al., 2010).

The third goal for revising the Cause scale pertained to improving the quality of information gathered about causal beliefs about depression. To that end, the item “Stress or worry” was replaced with several items specifying sources of stress. The nonspecific item “Stress or worry” has been consistently, by a large margin, the most popular attribution for depression in IPQ-based research (e.g., Brown et al., 2001; 2007; Fortune, Barrowclough, & Lobban, 2004; O’Mahan et al., 2008; Prins et al., 2008; Vollman et al., 2010). Yet, due to the breadth of the stress concept and its confounding with the nature of depressive symptoms (e.g., Sawatzky et al., 2012), this endorsement reveals little about participants’ causal beliefs. Revising this item to specify common sources of stress (e.g., “financial problems or worries,” “problems with friendships or other social relations,” and “experiencing a traumatic event”) was expected to yield clearer information about stress-related attributions for depression than has been gathered with the IPQ-R.

Consequences scale. The six-item Consequences scale assesses respondents’ beliefs about the nature and severity of depression’s effects on the lives of those who have it (e.g., “Depression has major consequences in the lives of people who have it”), with higher scores reflecting belief in more significant negative consequences (Table 2). It was retained as written for the IPQ-R, with the exception of replacing “my illness” with

"depression." In the current sample, this scale initially yielded an internal reliability measure (α =.694) that fell below the conventional threshold of α = .700 (Nunnally, 1978). Item intercorrelations were examined to determine whether any individual items that did not perform consistently with the others should be omitted. Based on this analysis, the only reverse-scored item on this scale was dropped ("Depression does not have much effect on the lives of people who have it"), increasing internal consistency to α = .766.

Timeline scale. The IPQ-R Timeline scale consists of nine items: five pertaining to beliefs about illness *duration* (permanent or temporary, long-term or short-term) and four about symptom fluctuations (the extent to which symptoms change cyclically; Table 3). These items make no reference to treatment or lack thereof, which would be expected to influence beliefs about depression's duration among individuals who view it as a treatable condition. Therefore, these items were revised in two ways, so that one nine-item set asked about the duration and fluctuations of untreated depression, while the other set asks about depression when it is treated, without specifying a treatment type. Two additional items were added to assess explicitly beliefs about long-term predisposition toward depressive episodes (following recovery from an episode either with or without treatment) as opposed to the duration of episodes. For this study's purposes, all 12 items focused on depression's *duration* (untreated and with treatment) were summed to create the Timeline scale score, which showed good internal consistency in this sample (α = .812).

Cure/Control scale. This scale includes 11 items assessing the perceived *treatability* of a given illness. Five of these items assess beliefs about treatment

effectiveness (treatment control); six items assess beliefs about the potential for someone with the illness to ameliorate it through their own actions (personal control; Table 4). Items implying that the respondent is being treated for the illness (e.g., "My treatment will be effective in curing depression") were edited to ask about treatment effectiveness generally (e.g., "Treatment is effective in curing depression"), to account for the variety of participants' experience and the fact that depression often goes untreated (Greenberg & Birnbaum, 2005). This scale differs from the other IPQ scales used in this study in that higher scores on this scale indicate a more *positive* prognostic view—that is, viewing the illness as more treatable. All 11 items were summed to create the Cure/Control scale score. Internal consistency in this sample was adequate (α = .774).

Depression Stigma Scale (DSS). Depression stigma was measured with the 18-item Depression Stigma Scale (DSS; Griffiths, Christensen, & Jorm, 2008; Griffiths et al., 2004; Table 5). The DSS measures two theoretically and empirically independent constructs reflecting aspects of depression stigma: *Personal stigma* refers to respondents' own negative attitudes toward people with depression, while *perceived stigma* reflects respondents' perceptions of the negative attitudes held by "most other people," each of which is assessed with a nine-item scale in the original instrument. The DSS was selected for its specificity to depression stigma, its design and validation based on several large general population samples, and the fact that it also has performed well among individuals expressing current psychological distress (Griffiths, Christensen, & Jorm, 2008). For this study, the item on each scale pertaining to whether "[Most people believe that] People with depression are dangerous" was revised into two items, distinguishing between danger to self and danger to others ("[Most people believe that] People with

depression are dangerous to others" or "…dangerous to themselves"). Item responses are measured on a 5-point Likert scale ranging from "strongly disagree" (0) to "strongly agree" (4), with higher values indicating higher levels of stigmatizing attitudes; each scale's set of 10 items was summed to create separate personal stigma and perceived stigma scores. In the current sample, these scales each showed high internal consistency (personal stigma: $\alpha = .864$; perceived stigma: $\alpha = .881$). They showed a correlation which, though statistically significant, was moderately small ($r = .303$, $p < .01$). They were therefore analyzed as separate variables.

Psychometric properties of the DSS have been analyzed in four large samples (N = 487–6134), in which the two scales' items loaded on separate factors (Griffiths, Christensen, & Jorm, 2008; Griffiths et al., 2004). Both scales have shown good internal consistency (personal stigma: $\alpha = .76–.82$; perceived stigma: $\alpha = .75–.82$; Griffiths, Christensen, & Jorm, 2008; Griffiths et al., 2004), and they have generally displayed adequate test-retest reliability in online data collection (personal stigma: $r = .67–.79$; perceived stigma, .63–.73, $p<.001$ for all samples; Griffiths et al., 2004).

Regarding construct validity, the personal stigma scale has been positively correlated with a measure of desired social distance ($r = .53$; Griffiths et al., 2004), a construct theorized to be a related yet distinct facet of stigma. This relationship was replicated in the current sample ($r = .556$). The perceived stigma scale, which measures the extent to which the respondent perceives stigmatizing attitudes to be widespread, previously has shown a small positive relationship with desired social distance ($r = .12$; Griffiths et al., 2004), also replicated in this sample ($r = .162$).

Depression Social Distance Scale (DSDS). This seven-item scale is a version of

a frequently used social distance measure (Link et al., 1987), modified for assessing behavioral intentions toward and discrimination against people with depression (Rusch et al., 2008; Table 6). The items assess willingness to be in specific situations with people who have depression. Items are rated on a four-point Likert scale ranging from "definitely willing" to "definitely unwilling"; higher scores reflect greater desired social distance, which is interpreted as an indication of more stigmatizing attitudes. The DSDS previously has shown good internal consistency (α = .75–.86; Kanter, Rusch, & Brondolino, 2008; Rusch et al., 2008); its performance in this sample was comparable (α = .90).

The original version of the Social Distance Scale (not modified for depression) has shown significant positive relationships with other facets of mental illness stigma, such as perceiving affected individuals as dangerous (r = .46), and making negative inferences about their traits (r = .38) and abilities (r = .30) based on descriptive vignettes (Penn et al., 1994). Test-retest reliability has not been reported for either the DSDS or the original Social Distance Scale (Link et al., 1987), though the latter is among the most widely used social distance measures in the mental illness stigma literature (Jorm & Oh, 2009).

Clinical History Questionnaire. This instrument captured basic information about participants' first- and second-hand experiences with depression, including, where relevant, experiences with diagnosis and treatment. At the beginning of this section, participants were presented with the same brief description of clinical depression that was provided at the beginning of the IPQ-R (DSM-5; APA, 2013a). This reminder was intended to once again make salient the disorder's basic defining features (illness

identity), following a series of items in which participants were asked to focus on their own opinions and beliefs. Participants were asked whether, based on this description, they believed they ever have experienced an episode of clinical depression. Those who said "yes" or that they were "unsure" received a sequence of follow-up questions including whether they were diagnosed by a professional (and if so, what type of professional); whether they received treatment (and if so, what type of treatment); and whether they believe any type of treatment that they used was effective (if so, which they found to be most and least effective). Throughout this sequence, the display logic for the questionnaire ensured that participants received items relevant to their reported experiences (e.g., indicating that one has never received a diagnosis from a professional resulted in skipping the follow-up item about the type of diagnosing professional). Participants also were asked a parallel set of questions about whether they believed someone close to them ("for example, a spouse or romantic partner, parent, sibling, or close friend") has experienced depression, and if applicable, their beliefs about that person's experiences with diagnosis and treatment.

To facilitate data analysis, all questions were closed-ended (i.e., multiple choice, including an option for "other" where a brief text response could be entered), with the option to select all that apply wherever relevant, and a "none" option where applicable. As a cursory assessment of comorbid psychopathology, respondents also were asked whether they believed they have experienced any psychological disorder other than depression, as well as whether any other psychological disorder was diagnosed by a professional (and if so, what type of professional).

Demographic Questionnaire. Demographic information about participants,

including age, gender, race/ethnicity, education level, employment status, and household income level was collected.

Procedure

Data Collection. A link to the study task was posted on MTurk's website on March 6, 2017. It was available to workers on the site for approximately 24 hours, by which point the pre-selected number of responses had been submitted (N = 351). Per eligibility criteria, the link, which read, "Quick (approx. 20 min) survey for psychology dissertation," was visible to the subset of MTurk workers with a U.S. IP address and at least a 90% approval rating for their work on the site. MTurk also displays the payment rate with each task's link. In accord with compensation rates for comparable MTurk tasks, participants were paid $0.40 to complete the survey. Individuals who clicked the link arrived at the main study page. They were informed that the study is confidential and voluntary; given contact information for the investigators; provided with basic, minimal information about the nature, topic, and duration of the study and how data would be used (i.e., reporting of aggregate results only); and offered the opportunity to provide consent or opt out by clicking designated buttons (see Appendix for full text of the study description and consent).

Consenting participants continued to the set of measures outlined earlier and summarized in Table 7. The order of instruments was held constant to ensure that all participants completed the sections of the questionnaire requesting their beliefs about depression before the sections requesting information about first- and second-hand experiences with the disorder, current symptoms, and/or demographics. The rationale was to capture participants' generally accessible beliefs about depression—albeit likely

informed by personal experiences where relevant—before making any particular personal experiences salient.

After completing the survey, participants were debriefed online with a more complete explanation of the research aims and thanked for participating. They were reminded of the researcher's contact information for questions or concerns, and were provided with the phone number for the national crisis hotline, in case any aspect of their participation was upsetting or prompted a desire for information about local mental health services. They also were provided a code entitling them to payment through MTurk.

Data Quality. To improve data quality, published recommendations for conducting research with MTurk's participant pool were implemented (Paoliacci & Chandler, 2014; Shapiro, Chandler, & Mueller, 2013). These practices included using an MTurk feature that screens for participants within a specific country based on IP addresses rather than using self-reported location, disguising the purpose of the task until participants are debriefed to minimize demand characteristics, and recruiting only participants with at least a 90% completion rate for accepted tasks and at least a 90% approval rating for their performance (Paoliacci & Chandler, 2014; Shapiro, Chandler, & Mueller, 2013). In addition, four "attention check" items instructing participants to choose a specific response, were embedded sporadically in the questionnaire. Individuals who failed or omitted more than one of these items were omitted from analyses (n = 28, 8%).

Statistical Overview. The general mode of analysis involved hierarchical multiple regression (Cohen et al., 2003). Preliminary analyses and regression diagnostics were performed to ensure that assumptions of multiple regression were met and that the

models fit the data. To determine whether variables were normally distributed, Q-Q plots were examined and histograms were checked for skewness, kurtosis, and fit to a normal curve. Independent variables were screened for multicollinearity by computing variance inflation factors (VIF) and tolerance values. Multicollinearity was evaluated using VIF of <10 and tolerance values of < .1 and was found to be inconsequential (Tabachnick & Fidell, 2001). Residual plots were examined for heteroscedasticity. Additional regression diagnostics were conducted following each analysis to make sure models were appropriate to the data. Testing for unduly influential observations based on the conventional Cook's *D* threshold (4/n; Tabachnick & Fidell, 2001) did not yield any observations for removal.

The first series of hierarchical multiple regression analyses was used to test biological causal beliefs (neurobiological, genetic) as predictors of prognostic beliefs about depression (consequences, duration, and treatability). Before undertaking these analyses, the IPQ-based measures of prognostic beliefs were examined to determine whether they were largely independent or sufficiently correlated ($r \geq 0.7$; Tabachnick & Fidell, 2001) that they should be combined for analysis. Based on their moderate associations (see Bivariate Associations), they were treated as separate variables. The second series of hierarchical regression analyses tested biological causal beliefs as predictors of depression stigma (personal stigma, perceived stigma, and desired social distance).

Predictors were entered into each analysis in the sequence summarized in Table 8. Demographic variables were entered first (age, gender, education level, and household income), followed by clinical variables (respondent's history of depression and history of

depression in a close other). The predictor of interest, the strength of either neurobiological or genetic causal beliefs, was entered third. The two biological causal beliefs initially were examined in separate analyses based on their moderate bivariate correlation, as noted above, as well their differential bivariate relationships with some prognostic beliefs and stigma measures.

For each set of predictors, the overall model R^2 was computed; for the second and subsequent sets of variables per model, ΔR^2 also was examined. These values were used to determine how much variance in prognostic beliefs, or in depression stigma, was explained by the sets of predictors. To assess whether biological causal beliefs showed an independent relationship with the outcomes of interest (i.e., prognostic beliefs, stigmatizing attitudes) within the context of a particular model, *t*-tests were performed on the relevant beta coefficients. Squared semipartial correlation coefficients (sr^2) were calculated to determine each predictor's contribution as an estimate of effect size.

Mediation Analyses. Analyses of mediation were conducted to determine whether any of the prognostic belief constructs (consequences, duration, treatability) statistically mediated the relationships between biological causal beliefs and depression stigma. Following Baron and Kenny's (1986) guidelines, initial regression analyses were performed to determine the extent to which neurobiological and genetic causal beliefs predicted these aspects of depression stigma. In these analyses, neurobiological and genetic causal beliefs served as simultaneous, competing predictors, so that each one could be examined as an independent predictor. Predicated on significant findings in these analyses (Baron & Kenney, 1986), each of three subsequent regression analyses tested both biological causal beliefs as simultaneous predictors of each one of the

prognostic beliefs (consequences, duration, treatability). Again, contingent on prior significant findings, the final three regression analyses tested whether the addition of the prognostic belief variables (mediators) significantly reduced the associations between biological attributions and depression stigma, which would provide evidence of statistical mediation. The Sobel (1982) test was then used to determine whether the pathways involving indirect effects of biological beliefs mediated by a prognostic belief were statistically significant.

Moderator Analyses. These analyses tested the moderating influence of two forms of familiarity with clinical depression, first-hand experience (whether the respondent believed he or she ever had experienced a depressive episode), and second-hand experience (whether the respondent believed someone close to him or her ever had experienced a depressive episode). In advance of these analyses, all non-dichotomous variables were mean-centered. To compute two product terms to carry information about statistical interactions for use in each analysis, each centered biological causal belief variable was separately multiplied by each of the two familiarity variables. Hierarchical multiple regression analyses then were performed, in which demographic variables were entered first, followed by three focal (main effect) predictors (one of the biological causal belief variables, respondent's own depression history, and depression history in a close other). Next, the two product terms were entered into the analyses sequentially, varying the order so that the effect of each interaction could be examined both with and without controlling for the effect of the other (Table 9). Results of the statistically significant interactions were plotted for interpretation following Cohen et al.'s (2003) recommendations.

Results

Preliminary Analyses

Analyses were performed in SPSS Statistics 24 (IBM Corporation, 2016). The initial data set (N = 351) was cleaned to remove responses from participants who failed more than 1 attention check items (n = 28, 7.98%) and to omit the records of participants who requested at the end of the survey that their data be excluded from any aggregate reporting (n = 4, 1.13%). These processes yielded a final sample (N = 319), which was analyzed for missing values. This analysis reflected little missing data in the variables of interest; the proportion of missing values was highest for the IPQ Timeline scale (n = 7, 2.2%). Most variables of interest had no missing values. Therefore, analyses were conducted using all available data; variations in degrees of freedom associated with different analyses reflect occasional missing values. Focal measures were checked at the item level for skew, kurtosis, and univariate outliers (Kline, 2005). Skewness and kurtosis were evaluated using a threshold of +/-2 and found to be acceptable (Gravetter & Wallnau, 2014). Means, standard deviations, skewness and kurtosis values are shown in Table 1.

Bivariate Associations

Relationships among the variables of interest ranged from minimal to moderate (r = .032–.556; Table 10). The two biological causal beliefs, “Biological changes in the brain” and “Heredity or genes – it runs in families” (hereafter, “neurobiological” and “genetic” causal beliefs, respectively), were significantly positively correlated (r = .363), yet the association was small enough in magnitude to warrant analyzing them separately rather than combining them into a single measure. Correlations among prognostic belief

measures ranged in strength from negligible to moderate. As expected, the view that depression has major consequences was positively associated with believing it has a long duration (r = .450). More surprisingly, viewing depression as more consequential also was significantly associated with viewing it as more treatable (r = .241). Correlations among the measures of depression stigma, which elicited respondents' own stigmatizing attitudes toward people with depression, their perceptions of these attitudes in others, and as their desired social distance from people with depression—were small to moderate in strength, significant, and positive (r = .162–.556).

Multivariate Associations

Hierarchical multiple regression analysis (Cohen et al., 2003) was used to determine the extent to which neurobiological and genetic causal belief variables, respectively, were associated with specific prognostic beliefs about depression and with depression stigma. As noted earlier, demographic covariates were entered into each analysis first, followed by clinical variables, then the biological causal belief variable (Table 8). It was hypothesized that stronger biological attributions for depression would be associated with more negative prognostic beliefs about its consequences, duration, and treatability. Each of these three dependent variables was examined in a separate analysis. Findings partially supported the hypotheses (Tables 11a-d).

Predicting Prognostic Beliefs. The strength of belief in neurobiological causes was first examined as a predictor of beliefs about depression's consequences. Table 11a shows that in this model, both the belief that a close other had experienced depression (β = .191, t = 3.465, p = .001) and stronger neurobiological causal beliefs (β = .298, t = 5.474, p < .001) were significantly associated with more negative beliefs about

consequences. No other predictors showed significant effects. The next analysis, which examined the same variables as predictors of beliefs about depression's duration, yielded similar results (Table 11a). Affirming past depression in a close other (β =.184, t = 3.279, p = .001) and holding stronger neurobiological causal beliefs (β = .285, t = 5.189, p < .001) were associated with viewing depression as having a longer duration. Again, the effects of other demographic and clinical variables were not significant. When neurobiological causal beliefs were examined as a predictor of views about depression's treatability, older age was significantly associated with viewing depression as more treatable (β = .156, t = 2.827, p = .005), as was belief in neurobiological causes (β = .258, t = 4.570, p < .001). No other variables were significantly associated with treatability beliefs.

A parallel set of analyses examined the strength of genetic causal beliefs as a predictor of the three prognostic beliefs. As Table 11b shows, holding more negative views of depression's consequences was significantly associated with older age (β = .132, t = 2.435, p = .015), with affirming a history of depression in a close other (β = .183, t = 3.228, p = .001), and with stronger endorsement of genes as a cause (β = .201, t = 3.523, p < .001). No other variables showed significant effects. More negative views of depression's duration were likewise associated with affirming a history of depression in a close other (β = .167, t = 2.980, p < .001), and with genetic causal beliefs (β = .307, t = 5.440, p < .001; Table 11b). Other predictors' effects were not significant. Finally, beliefs about depression's treatability were not significantly associated with the strength of genetic causal beliefs (β = .029, t = .476, p = .635). In this analysis, only age showed a significant relationship to treatability beliefs, such that older age was associated with

viewing depression as more treatable (β = .182, t = 3.212, p = .001). When neurobiological and genetic casual beliefs were examined as simultaneous predictors of each prognostic belief, the pattern of significant relationships was unchanged in the respective analyses of duration and treatability beliefs, whereas genetic attributions no longer significantly predicted beliefs about consequences (β = .115, t = 1.962, p = .051).

Predicting Stigma. Additional regression analyses were performed to test the hypotheses that stronger biological causal beliefs would be associated with holding more depression-stigmatizing attitudes across three measures of stigma. In the first set of analyses, neurobiological causal beliefs served as the predictor for each stigma variable; results are summarized in Table 11c. Across the stigma measures, results consistently failed to support the hypotheses linking biological attributions with greater stigma. The first analysis focused on personal stigma, a measure of respondents' own stigmatizing attitudes. Both affirming a history of depression in a close other (β = -.129, t = 2.241, p = .026) and strength of belief in neurobiological causes (β = -.238, t = -4.215, p < .001) showed significant *inverse* relationships to personal stigma. This association of stronger neurobiological causal beliefs with *lower* stigma was the opposite of the hypothesized relationship. Other demographic and clinical variables yielded no significant effects. Perceived stigma, a measure of respondents' beliefs about the level of depression stigma broadly held by others, was analyzed next. Neurobiological causal beliefs did not show a statistically significant relationship to perceived stigma (β = .102, t = 1.768, p = .078), nor did any demographic or clinical predictors. Desired social distance from people with depression served as the third stigma measure. In this analysis, older age was significantly associated with desiring more social distance (β = .189, t = 3.430, p = .001),

while affirming a close other's history of depression was associated with desiring less social distance (β = -.199, t = -3.466, p = .001). Neurobiological causal beliefs failed to show a statistically significant association with desired social distance (β = -.0102, t = -1.811, p = .071).

A parallel set of analyses examined the strength of genetic attributions as a predictor of each stigma variable. Both affirming a close other's history of depression (β = -.119, t = -2.021, p = .044) and expressing stronger belief in a genetic cause (β = -.152, t = -2.567, p = .011) showed significant inverse relationships with personal stigma (Table 11d). Other predictors' effects of were not significant. Next, genetic causal beliefs were analyzed as a predictor of perceived stigma. Here, the significant effect of gender (β = -.116, t = -2.024, p = .044) indicated that women perceived a higher level of broadly held depression stigma than men did. The relationships of genetic causal beliefs (β = .010, t = .167, p = .868) and other predictors to perceived stigma were not significant. Finally, genetic attributions were examined as a predictor of desired social distance. Older age was significantly associated with desiring more social distance (β = .180, t = 3.281, p = .001). The associations of genetic causal beliefs (β = -.084, t = -1.445, p = .149) and of the other predictors with desired social distance were not significant. When neurobiological and genetic casual beliefs were allowed to compete as predictors of each stigma variable analyzed above, neurobiological attributions retained the above-described pattern of significant relationships; genetic causal belief was no longer significantly associated with personal stigma (β = -.083, t = -1.361, p = .174).

Mediation Analyses

In the analyses described above, biological causal beliefs were associated with significantly *lower* levels of personal stigma and with a specific pattern of prognostic beliefs: viewing depression as more consequential and of a longer duration, yet also as more treatable. Mediation analyses were performed to test whether any of the significant relationships between biological causal beliefs and *lower* levels of personal stigma might be explained by participants' prognostic beliefs. These analyses were conducted using Baron and Kenny's (1986) approach described earlier. (In accordance with this procedure, perceived stigma and desired social distance were not included as outcomes in the mediation analyses, as they had shown statistically nonsignificant relationships to the biological causal belief variables of interest.) Separate mediation analyses were performed to test each of the three prognostic belief constructs—consequences, duration, and treatability—as a potential mediator of the relationship between biological causal beliefs and personal stigma.

The first component of each analysis involved determining the extent to which each of the biological causal beliefs predicted respondents' level of personal stigma. Given that neurobiological and genetic causal beliefs each showed statistically significant, inverse relationships to personal stigma when analyzed separately, the mediation analyses examined these causal belief variables as simultaneous predictors, to determine the extent to which each one was independently associated with personal stigma. Demographic covariates were entered first, followed by clinical covariates, then both of the biological causal belief variables (Table 8). When the two biological causal belief variables were entered into the analyses together, genetic causal beliefs no longer showed a significant relationship to personal stigma (β = -.083, t = -1.361, p = .174). The

inverse relationship between neurobiological causal beliefs and personal stigma remained significant (β = -.213, t = -3.581, p < .001). In this analysis, the only other predictor to show a significant relationship to personal stigma was affirming a close other's history of depression (β = -.124, t = -2.142, p = .033).

The second component of each mediation analysis involved examining the associations between biological causal beliefs and each of the three prognostic beliefs serving as potential mediators (consequences, duration, and treatability). Again, variables were entered into each analysis following the above-described sequence (Table 8). Results indicated that stronger neurobiological attributions again significantly predicted more negative beliefs about depression's consequences (β = .262, t = 4.569, p < .001). The relationship of genetic causal beliefs and to negative views of consequences approached significance (β = .115, t = 1.962, p =.05). Older age (β =.105, t = 1.990, p = .047) and affirming a close other's history of depression (β =.185, t = 3.373, p = .001) also significantly predicted more negative beliefs about depression's consequences; other predictors' effects were not significant. Stronger endorsement of neurobiological causes (β = .212, t = 3.756, p < .001) and genetic causes (β = .238, t = 4.082, p < .001) each predicted more negative beliefs about depression's duration. In this analysis, only affirming a close other's depression history (β = .170, t = 3.093, p = .002) also predicted more negative views about duration, while the effects of other predictors were not significant. Consistent with the results of the initial regression analyses, belief in neurobiological causes was significantly associated with viewing depression as more treatable (β = .281, t = 4.683, p < .001), while genetic attributions did not significantly predict treatability beliefs (β = -.070, t = -1.128, p = .260). Older age also was

significantly associated with viewing depression as more treatable (β = .155, t = 2.810, p = .005); other predictors' effects were not significant.

Subsequently, each of the three prognostic beliefs was analyzed as a potential mediator of the inverse relationship between neurobiological causal beliefs and personal stigma. These analyses provided evidence of partial mediation in all three instances, as each of the prognostic beliefs showed a significant inverse association with personal stigma (consequences: β = -.258, t = -4.411, p < .001; duration: β = -.327, t = -5.669, p < .001; treatability: β = -.132, t = -2.307, p = .020). That is, viewing depression as less consequential, of shorter duration, and less treatable, respectively, were associated with more stigmatizing attitudes. In all three analyses, the standardized coefficient for the initial path between neurobiological causes and stigma was reduced (Figures 1a-c). Follow-up Sobel tests (1982) indicated beliefs that depression has significant negative consequences (z = -3.171, p = .002) and a long duration (z = -3.130, p = .002) each significantly mediated the association between stronger neurobiological attributions and less stigmatizing views of people with depression. Views about depression's treatability did not significantly mediate this relationship (z = -1.732, p = .083). In the analysis of treatability beliefs as a mediator, affirming a close other's history of depression showed a statistically significant, inverse relationship to personal stigma (β = -.120, t = -2.074, p = .039), while no other demographic or clinical variable had a significant predictive effect.

Moderation Analyses

Further analyses were conducted to test the extent to which familiarity with depression moderated the relationships of biological causal beliefs to either negative prognostic beliefs or stigmatizing attitudes. Initially, stronger biological causal beliefs

had been hypothesized to predict higher levels of stigma, and to do so more strongly among participants less familiar with depression. Contrary to this prediction, the initial multivariate analyses reported above indicated that the strength of biological causal beliefs was *inversely* associated with personal stigma and was unrelated to either perceived stigma or desired social distance. Therefore, moderation analyses were conducted to test whether respondents' familiarity with depression moderated the relationships of stronger genetic or neurobiological causal beliefs, respectively, to *lower* levels of personal stigma.

Familiarity with depression was measured with two items. The first item asked whether the respondent believed he or she had ever experienced a depressive episode. The second item asked whether the respondent believed that a close other ("for example, a spouse or romantic partner, parent, sibling, or close friend") ever had experienced a depressive episode. Both items included the same brief description of the disorder that was presented at the beginning of the questionnaire (see Appendix). Both familiarity items offered the response options "yes," "no," and "don't know." Based on the low proportion of "don't know" responses to both items (self: $n = 36$; 11.3%; close other: $n = 24$, 7.5%) and the priority of differentiating respondents who affirmed personal experience with depression from those who did not, these variables were dichotomized for analysis by combining each item's "no" and "don't know" responses.

Results indicated that neither first-hand nor second-hand familiarity with depression significantly moderated the relationships of biological causal beliefs to prognostic beliefs. In contrast, familiarity with depression significantly moderated several relationships between biological causal beliefs and aspects of depression stigma.

Associations involving significant interaction effects are reported, starting with those involving genetic casual beliefs.

The relationship of genetic causal beliefs to personal stigma was significantly moderated by respondents' own history of depression (β = -.154, t = -2.133, p = .034). History of depression in a close other did not significantly moderate this relationship (β = -.152, t = -1.650, p = .100). Affirming a close other's history of depression did have a significant main effect, showing a significant inverse relationship to personal stigma (β = -.127, t = -2.164, p = .031). These results are summarized in Table 12. Plotting the significant interaction revealed that, among respondents who reported a history of depression, endorsing stronger genetic causal beliefs was associated with lower levels of personal stigma, compared to those who held weaker genetic causal beliefs. Among respondents who did not report a history of depression, the strength of genetic attribution was unrelated to levels of personal stigma (Figure 2).

Familiarity with depression also moderated the relationship between genetic causal beliefs and desire for social distance from people with depression (β = -.195, t = -2.168, p = .031). This was the only analysis in which a close other's depression history showed a statistically significant moderating effect, whereas the moderating effect of the respondent's own history did not reach significance (β = -.139, t = -1.962, p = .051). In this analysis, older age also was significantly associated with desiring more social distance (β = .166, t = 3.049, p = .002), and affirming a close other's history of depression significantly predicted desiring less social distance (β = -.206, t = -3.590, p < .001). No other predictors showed statistically significant effects (Table 13). Plotting the significant interaction between reporting a close other's depression history and desired

social distance showed a relationship with a similar form to that described above. Among respondents affirming a close other's history of depression, stronger belief in genetic causes was associated with less desired social distance, compared to respondents in this group making weaker genetic attributions. (Figure 3). Among respondents who did not report a history of depression in a close other, genetic causal beliefs were unrelated to desired social distance. A plot to explore the near-significant interaction between respondents' own history of depression and desired social distance (not shown) took the same form as well: Only among respondents reporting a history of depression, stronger genetic causal belief was associated with less desired social distance.

In addition, the relationship between genetic causal beliefs and levels of perceived stigma—the level of stigma presumed among "most other people"—was moderated by respondents' own history of depression (β = -.194, t = -2.660, p = .008). Close others' history of depression did not significantly moderate this relationship (β = -.048, t = -.519, p =.604). In this analysis, gender also significantly predicted perceived stigma (β = -.117, t = -2.066, p = .040), such that women perceived higher levels of broadly held depression stigma than men did. The effects of other predictors were not significant (Table 14). Plotting the significant interaction showed that among respondents reporting a history of depression, those making stronger genetic attributions endorsed lower levels of perceived stigma, compared to those making weaker genetic attributions. The inverse relationship emerged among those who did not report prior depression. Among these participants, stronger genetic causal beliefs were associated with higher levels of perceived stigma, while weaker genetic attributions were associated with lower perceived stigma (Figure 4).

These familiarity variables also were examined as moderators of the relationships between neurobiological attributions and the three stigma variables, which yielded one significant interaction. Whether respondents reported prior depression moderated the relationship between neurobiological causal beliefs and desired social distance from people with depression (β = -.177, t = 2.449, p =.015). As the interaction plot shows (Figure 5), among respondents reporting a history of depression stronger belief in neurobiological causes was associated with desiring less social distance from people with depression, compared to those making weaker neurobiological attributions. By contrast, among those who did not report prior depression, the strength of neurobiological causal belief was unrelated to desired social distance (Figure 5). Again, close others' depression history did not significantly moderate the association between neurobiological causal belief and desired social distance (β = -.129, t = -1.339, p =.181). This analysis showed a significant main effect of age, such that older participants desired more social distance (β = .182, t = 3.358, p = .001), and of affirming a close other's prior depression, which was associated with desiring less social distance (β = -.198, t = -3.475, p = .001; Table 15).

Discussion

This study aimed to improve understanding of the implications of holding biological causal beliefs about depression by testing biological essentialist hypotheses within the CSM framework. Consistent with these hypotheses, biological causal beliefs predicted negative beliefs about depression's consequences and duration; contrary to hypotheses, they also were associated with viewing depression as more treatable. Also counter to predictions, biological causal beliefs were *inversely* related to depression stigma. These inverse relationships between biological attributions and stigma were

partially mediated by beliefs about depression's consequences and duration. First- and second-hand familiarity with depression moderated some relationships between biological causal beliefs and depression stigma, and these interactions tended to take a similar form. Among individuals reporting more familiarity with depression, endorsing biological causal beliefs predicted lower levels of stigma. For participants less familiar with depression, biological causal beliefs appeared unrelated to stigma. These findings suggest refinements to essentialist theory, avenues for future research into mental health literacy, and guidance for addressing stigma within the context of depression treatment and in the general population.

Biological Causal Beliefs as Predictors of Prognostic Beliefs and Stigma

The initial regression analyses, in which neurobiological and genetic causal beliefs were analyzed separately, showed each of these biological causal beliefs to be significantly associated with more negative prognostic beliefs about depression's consequences and duration. This finding is consistent with essentialist claims that aspects of identity—including mental illness—attributed to biological causes are more likely to be viewed as stable, highly informative features of an individual (Dar-Nimrod & Heine, 2011). Inconsistent with essentialist theory, however, the hypothesized associations between these biological causal beliefs and viewing depression as *less* treatable were not in evidence. While the relationship of genetic causal beliefs to perceived treatability was not significant, neurobiological attributions were significantly associated with viewing depression as *more* treatable. Essentialist theory further predicts that unfavorable aspects of identity viewed as biologically based, stable, and highly informative or consequential are more likely to be stigmatized (Dar-Nimrod & Heine, 2011). In this sample, however,

both genetic and neurobiological attributions were associated with *lower* levels of personal stigma, and they showed nonsignificant relationships to desired social distance and perceived stigma. Though these findings contradict essentialist predictions, they align with other evidence showing biological causal beliefs about mental illness to be associated with both more negative prognostic beliefs and ascribing lower levels of blame to those affected (Kvaale, Gottdeiner, & Haslam, 2013; Lebowitz, 2014).

Taken together, these findings point to a different set of illness beliefs contributing to depression stigma than those suggested by essentialist theories. While viewing the disorder as more consequential, longer-lasting, and more treatable was associated with lower levels of stigma, considering depression to be less consequential, of shorter duration, and less treatable were associated with more stigmatizing attitudes. These distinct sets of illness beliefs each suggest different ways of understanding the behavior of people with depression. Beliefs linked with low stigma—seeing depression as a long-lasting, consequential, yet highly treatable condition— initially may seem contradictory. Yet, these beliefs are consistent with public messaging that encourages treatment-seeking, both by public health agencies and by advertisers of antidepressant medications. Views of depression as serious, long-lasting, and treatable also may be based on participants' first- or second-hand experiences with recovery from significant depression. This possibility is consistent with the associations in this sample between both first- and second-hand experience with depression and lower stigma. On the other hand, illness beliefs linked with high levels of stigma suggest viewing depression as more akin to a bad mood or bad attitude than a biologically fixed, out-group-defining property. The link between viewing depression as less treatable and endorsing greater stigma does

fit essentialist predictions—but considering this relationship alongside the association of higher levels of stigma with viewing depression as relatively brief and inconsequential casts it in a different light. The view of depression as "less treatable" may reflect, for example, beliefs that it is too mild and fleeting to warrant treatment, that it is a choice or exaggeration of normal negative emotion, that it is too integral to one's personality to be changed, or aspects of all three.

Prognostic Beliefs as Mediators of the Inverse Relationships between Biological Attributions and Personal Stigma

In the first steps of the mediation analyses, neurobiological and genetic attributions were examined as simultaneous, rather than separate, predictors of prognostic beliefs and personal stigma. As these two causal beliefs correlated significantly (r = .363, p <.001), they were allowed to compete in these analyses to determine the extent to which they showed independent relationships to prognostic and stigma-related variables. When they were analyzed as simultaneous predictors, neurobiological causal belief retained its significant associations with all three prognostic beliefs and with stigma, while genetic causal beliefs remained significantly associated only with beliefs about depression's duration. This result indicates that these biological causal beliefs shared some of the variance that they each explained in prognostic beliefs and stigma. This finding is consistent with prior research showing a positive correlation between neurobiological and genetic causal beliefs in the general public (Rüsch et al., 2010). The stronger predictive power of neurobiological explanations compared to genetics in these analyses accords with the faster rise in public acceptance of neurobiological attributions compared to genetic ones in recent decades (Schomerus et al., 2012). The independent

relationship of genetic causes to perceived duration is consistent with prior findings and essentialist theory suggesting that genetic causes are perceived as particularly stable, and that mental illness attributed to genes are likewise viewed as long-lasting (Dar-Nimrod & Heine, 2011; Phelan, 2005).

The relationships between stronger neurobiological attributions and lower levels of personal stigma were partially mediated by beliefs about depression's consequences and duration. This lends some qualified support to essentialist claims about the association of biological causal beliefs with more negative prognostic views and the potential for prognostic beliefs to influence illness stigma. However, as noted above, the association of these causal and prognostic beliefs with *lower* stigma runs counter to the concern within essentialist theory about an inherent link between, on one hand, endorsing biological attributions and a negative prognosis—and on the other hand, mental illness stigma. An alternative interpretation is that instead of being inherently linked with stigma, negative beliefs about duration and consequences—that is, about depression's severity—may have served in this instance as partial mediators of ascribing less responsibility to individuals for their depression, along with lower levels of blame (Jorm et al, 1997; Weiner, Perry, & Magnussen, 1988). This possibility is consistent with findings that, among several stigmatized conditions, those perceived as "physical" (rather than "mental-behavioral") were perceived to have a less controllable onset, and associated with ascribing less blame to affected individuals, and with emotional responses of pity, liking, low anger, and inclination to help. In contrast, this study found that "mental-behavioral" conditions were perceived as more onset-controllable, more associated with blaming affected individuals, and more likely to elicit responses of anger,

low pity, and disinclination to help (Weiner, Perry, & Magnussen, 1988). However, the stigmatized conditions selected for that study differed from depression, as they were picked to support clear distinctions between "physical" and "mental-behavioral" attributions. In addition, more recent work has underscored the complexity of emotional reactions associated with stigma, in which pity and anger are not mutually exclusive (Cottrell & Neuberg, 2005). The possibility that the current mediation effects were driven by a relationship between perceived severity of depression and lower perceived blame or responsibility is also consistent with items on the personal stigma measure that seem to tap these beliefs (e.g., "People with depression could snap out of it if they wanted; Depression is not a real illness"; Griffiths et al., 2004).

Moderating Effects of Familiarity with Depression on Relationships between Biological Attributions and Stigma

As hypothesized, reporting a history of depression or affirming a history in a close other both moderated associations between biological attributions and depression stigma. In these analyses, three out of four instances of significant moderation took a very similar form: Among respondents reporting a high degree of familiarity with depression, weaker biological attributions predicted more stigma, whereas among those with less familiarity with depression, biological attributions were unrelated to depression stigma. These findings suggest that individuals' experiences with their own and others' depression may provide important sources of information that affect their responses to public messages about the disorder. Large-scale studies of mental illness beliefs and stigma in the general public, which have yielded mixed results (reviewed in Angermeyer et al., 2011; Jorm & Griffiths, 2008; Pescosolido, 2013; Schomerus et al., 2012), therefore might be rendered

more interpretable by the addition of similar moderator analyses. Accounting for respondents' beliefs about their first- and second-hand experience with the target illness—that is, the extent to which respondents view the target social group as an ingroup or an outgroup—might reveal informative group differences that are otherwise statistically obscured or averaged out. These analyses also might suggest more effectively tailored, subgroup-specific means of reducing stigma, rather than assuming uniform effects of public messaging.

Although this study did not directly measure self-stigma, the results linking the endorsement of both prior depression and weaker biological attributions to higher stigma are consistent with some prior findings in this area and suggests caveats on others. This pattern of results suggests that personal experience with a target illness may enhance or help to explain the inverse relationship previously reported between biological attributions for mental illness and ascribing less blame to people with these diagnoses (Kvaale, Gottdeiner, & Haslam, 2013; Lebowitz, 2014). In this vein, it accords with qualitative evidence that biomedical—and specifically genetic—explanations can help reduce the effects of stigma for people with a history of depression and for those with a significant family history (Buchman, Borgelt, Whiteley, & Illes, 2013; Laegsgaard et al., 2010; Schreiber & Hartrick, 2002). On the other hand, the present findings contrast with evidence linking higher self-stigma with individuals' biological explanations for their own severe mental illness (Rüsch et al., 2010), a disparity potentially explained by diagnostic and demographic differences between the samples as well as the prior study's use of implicit, self-focused stigma measures. Together, the current findings support including biological explanations in therapeutic psychoeducation, especially in ways that

emphasize agency—via the responsiveness of biological systems to experience (e.g., Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Macduffie & Strauman, 2017). They further suggest that these kinds of messages about dynamic relationships between biology and behavior could be incorporated into broader efforts to reduce depression stigma and encourage treatment-seeking.

Other Findings

Outside the scope of the hypotheses, the variables of interest showed several associations with demographic and clinical predictors that also warrant mention. In the initial multivariate analyses, age was uniquely associated with higher levels of stigma only when stigma was measured in terms of desired social distance. The lack of relationship between older age and *personal stigma*—one's own negative or disparaging beliefs about people with depression—as well as the association of older age with viewing depression as more treatable, both suggest that the age–social distance association does not reflect an age-related tendency toward more essentialist thinking about depression. One alternative interpretation is that this pattern is consistent with socioemotional selectivity theory, which predicts that with age, adults increasingly focus their social and emotional resources on a smaller number of close, satisfying relationships, rather than on expanding their network (Cartensen, 1995; Cartensen, Isaacowitz, & Charles, 1999). In this view, older participants may have had a more resource-intensive understanding of social closeness—and less inclination to associate with the hypothetical person referenced in the desired social distance items, for whom depression is the only salient feature. Age-related differences in this sample also may reflect cohort- or period-related changes in the social acceptance of individuals with

mental illness that are not captured by the relatively brief timeframe in which large-scale longitudinal studies of U.S. public attitudes have been conducted (Angermeyer et al., 2011; Jorm & Griffiths, 2008; Pescosolido, 2013; Schomerus et al., 2012).

Female gender also emerged as the only significant predictor of perceived stigma. Being female was associated with a stronger belief that depression is stigmatized by "most other people." While female gender was significantly correlated with affirming both one's own and a close other's history of depression, these experiential variables themselves did not significantly predict perceived stigma, suggesting that the gender–perceived stigma link is not fully explained by the possibility that more women than men had experienced or observed depression stigma. Another interpretation links this finding to U.S. cultural values pertaining to gender differences in emotional expression. That is, compared to men, women may experience comparatively more negative social responses to a lack of positive emotional display, even within the range of normal affect. Women and girls are perceived to smile more than men and boys, and across many contexts these perceptions accurately reflect behavioral differences (e.g., Adams, Hess, & Kleck, 2014; LaFrance, Hecht, & Paluck, 2003), despite a consistent lack of evidence for gender differences in subjective emotional experience (LaFrance, Hecht, & Paluck, 2003; Simon & Naith, 2004). Findings that show more pronounced gender differences in smiling in contexts emphasizing gender roles or status have been interpreted as suggesting that positive emotional displays may serve "affiliative" or "social appeasement" (Adams, Hess, Kleck, 2014, p. 6) functions for women to a greater extent than for men. If women more frequently than men experience social disapproval for an unsmiling expression, this

feedback may contribute to a stronger tendency to perceive depression's persistent, pronounced negative affect to be broadly socially unacceptable.

Significance and Limitations

This study makes several theoretical and practical contributions. From a theoretical standpoint, it suggests refinements to essentialist theory as applied to lay beliefs and stigmatizing attitudes about depression. This study provided evidence that, counter to the predictions of essentialist theory, biological causal beliefs can predict *less* stigma, even when they are also associated with negative beliefs about depression's consequences and duration. The finding that treatability beliefs were inversely related to these other prognostic beliefs and to stigma also suggests that essentialist theory would benefit from focusing its predictions on specific prognostic beliefs' relationships to each other and to stigma, rather than presuming all prognostic beliefs to covary.

More generally, this study takes a step toward integrating essentialist theories with the more established Commonsense Model of lay illness representations (CSM), by using the CSM to frame essentialist hypotheses. The CSM can play an important role in guiding refinements to essentialist theory about mental illness in several respects. For one, as this study shows, the specific types of illness beliefs described by the CSM are compatible with essentialism's focal constructs. Essentialist hypotheses about the relationships of biological causal beliefs to negative prognostic beliefs, and about associations among different prognostic beliefs, can be examined as components of multifaceted illness representations. In this study, for example holding stronger biological causal beliefs was associated with viewing depression as consequential and long-lasting, yet also with viewing it as more treatable. In addition, these associations were linked more strongly with neurobiological than genetic causal beliefs. These findings indicate

that at the level of specific disorders, the broad constructs of *biological causal beliefs* and *prognostic beliefs* can obscure meaningful, unexpected relationships. Studying these relationships, and understanding their connections to stigma, therefore warrants the use of the more thorough, precise set of illness belief constructs like those afforded by the CSM. In addition, the CSM's focus on individuals' lived experience with illness as a basis for illness beliefs provides a theoretical foundation for examining experiential moderators of relationships between illness beliefs and stigma. The significance of first- and second-hand experience with depression as moderators of relationships between biological causal beliefs and stigma suggests that similar analyses might elucidate patterns in the often-mixed results of studies of mental illness stigma in nonclinical populations.

In addition to these contributions, several limitations of this study should be kept in mind. First, the use of cross-sectional, correlational data precludes any causal inferences. As noted earlier, different relationships between key variables—such as whether stigma constructs predict illness beliefs, or vice versa—are equally plausible. Biological causal beliefs served as predictors in this study because they have become a focal point in disagreements between, on one hand, public health campaigns that present biomedical explanations of mental illness as destigmatizing (Pescosolido et al., 2010) and, on the other hand, essentialist theorists and others concerned that these messages may unwittingly increase stigma. To test predictions of essentialist theory, this study's hypotheses were structured to operationalize claims that biological causal beliefs may influence other illness beliefs and related social judgments. Future work may benefit from comparisons of mediation models and other analyses with alternative structures, including testing stigma variables as predictors rather than outcomes.

Second, the convenience sample drawn from the population of MTurk workers differed in some expected ways from the national population; compared to U.S. adults, the sample overrepresented Whites and Asians, underrepresented other ethnic and racial groups, and was more highly educated. In these respects, the sample more closely resembles U.S. adult internet users than the adult population as a whole (Chandler & Shapiro, 2016). It is, however, more demographically and geographically representative of U.S. adults than traditional convenience samples (e.g., a student pool or university community sample). Moreover, the sample included members of the understudied cross-section of individuals who endorse past or current clinically significant depressive symptoms yet have not received treatment. This group, by definition, is ineligible for patient studies, whereas research on the general population generally has not enquired about participants' clinical history or current symptoms. The present sample also reported rates of a lifetime history of depression and of clinically significant depressive symptoms than exceed rates among U.S. adults as a whole (Kessler & Bromet, 2013; Kessler & Wang, 2008). These sample characteristic all need to be considered with regard to potential generalizability of the findings.

Third, this study relied on self-reported beliefs and attitudes about depression. The well-known limitations of self-report measures are common to the majority of research on illness beliefs and on mental illness stigma. Studies using implicit measures, such as the Implicit Associations Test (e.g., Rüsch et al., 2010a; 2010b), may minimize some response biases, or they may measure different stigma constructs (Fazio & Olsen, 2003). While this study remains subject to the limitations of self-report data (Chan, 2009), preliminary evidence suggests that the complete anonymity conferred by MTurk

recruitment may promote self-disclosure and reduce social desirability biases, compared to interacting with researchers in person (Joinson, 2001; Shapiro, Chandler, & Meuller, 2013).

Study 2

The first study tested predictions based on essentialist theory about the relationships of biological causal beliefs about depression to other beliefs and attitudes concerning this disorder. That aim provided the rationale for assessing the strength of specific causal beliefs and other illness beliefs and for statistically modeling their independent, unidirectional influences on other variables. However, there is sound theory (Kelly, 1983) and empirical evidence (e.g., Brogan & Hevey, 2009; Lunt, 1991) suggesting that people also think about causality in terms of multiple causal factors that may influence one another as well influencing outcomes, including the possibility of bidirectional effects. Illness cognition has been highlighted as a domain in which causal thinking often incorporates multiple, interrelated contributing factors (Sensky, 1997).

Network analysis is a quantitative method that can be used to represent beliefs about the structure of relationships among a set of interrelated causal factors. It involves measuring the perceived strength of relationships between each pair of a set of putative causal factors and between each factor and an outcome of interest. Based on the resulting correlation matrix, a network diagram can be constructed that depicts the structure, strength, and direction of the perceived relationships among the causes and between each one and the outcome. Network analysis has been applied to causal beliefs about physical health problems (e.g., obesity, myocardial infarction; Brogan & Hevey, 2009; Muncer, Taylor, & Link, 2001; French et al., 2002) and behavioral health issues (e.g., work stress;

loneliness, cigarette smoking; Muncer et al., 2001; Lunt, 1991; Lydon et al., 2016); a related approach was used to model patients' beliefs about the relationships among PTSD symptoms (Frewen et al., 2013). Causal belief networks generated for different groups also can be compared to identify structural differences. These may include the networks' relative complexity, evident in bidirectional relationships and/or instances of mediation; and the relative centrality of a particular causal factor, evident in the interconnectedness of the network nodes representing that cause. To our knowledge, network analysis has not been applied before to causal beliefs about a mental illness.

Study 2 used network analysis to model lay causal beliefs about depression, a topic well-suited for this approach. Given the high prevalence of depression and its considerable illness burden, a significant proportion of U.S. adults are likely to hold some beliefs about the nature of the problem and the factors likely to influence it, whether based on first- or second-hand experience with it, exposure to public health and advertising messages, or other portrayals of depression in the culture (Hagmayer & Engelmann, 2014; Leventhal et al., 2015; WHO, 2013; 2017). Furthermore, network representations may capture important, yet-unexamined aspects of causal thinking about depression. The disorder presents with psychological, somatic, and behavioral symptoms—some observable, others invisible—and it is associated with risk factors spanning biological, psychosocial, and socioeconomic domains (Hagmayer & Engelmann, 2014). Lay causal beliefs about depression seem to incorporate these complexities, as they include biological, psychological, environmental, and interpersonal attributions (MacDuffie & Strauman, 2017; Schomerus et al., 2012), yet the perceived structural relationships among these factors has not been examined.

Specific Aims

In contrast to Study 1's focus on biological causal beliefs, Study 2 takes a multifaceted approach to causal thinking about depression. Its first aim was to generate a consensus causal belief network based on the full sample. The second aim was to investigate whether and how *illness coherence*, a construct reflecting how well individuals believe they understand an illness (Moss-Morris et al., 2002), related to the structure and complexity of causal thinking about depression. This second aim involved examining separate network diagrams for high- and low-coherence subgroups of the original sample. These groupings were based on participants' scores on the IPQ-R Coherence scale.

Overview

First, a causal belief network model based on the full sample was generated. This model was assessed in terms of its complexity (number of links, proportion of bidirectional links, proportion of direct versus mediated links to depression), the types of causes linked directly to depression, and the relative interconnectedness of biological causes compared to other causal factors. Next, the causal networks of participants with high and low IPQ-R Coherence scale scores were constructed separately. This scale assesses how clearly the respondent believes that he or she understands the target illness (Moss-Morris et al., 2002). As such, this scale can be viewed as an expression of the respondent's level of confidence in the accuracy and completeness of his or her knowledge of the disorder.

Prior research into mental illness representations suggests that it may be helpful for providers and public health advocates to understand more about the causal

explanations associated with high versus low illness coherence. Among patients with mood disorders, high coherence has been associated with "indifference to stigma"—a relative lack of concern about the possibility of others' negative reactions to their treatment-seeking (Munson, Floersch, & Townsend, 2009). It has also been linked to willingness to stay engaged in treatment among patients receiving care (Munson, Floersch, & Townsend, 2009; Williams & Steer, 2011; reviewed in Baines, 2013).

Research into attributional complexity (Fletcher et al., 1986) suggests that individuals who provide more complex causal explanations do not differ systematically in their level of confidence in those explanations from those who provide simpler causal attributions. A prevalent interpretation of this finding is that those who make more complex attributions may assess their own understanding as relatively sophisticated, leading them to express high confidence in their knowledge, while those with simpler attributions may express confidence as well because they are less aware of the complexities missing from their understanding (Kruger & Dunning, 1999; Fletcher et al., 1986). These findings suggest that high illness coherence may be equally likely to be associated with high or low complexity causal belief networks.

Another way low coherence may affect that group's network diagram has to do with the inductive eliminative analysis (IAE) method used for generating the networks (Reser & Muncer, 2004; Muncer & Gillen, 1997). This method, described in greater detail below, includes in the network only those causal links that surpass an established threshold for "consensus" endorsement by the majority of the group (French et al., 2002; Muncer & Gillen, 1997). If the low-coherence group has fewer strong beliefs about depression's causes, the group may show relatively low consensus in rating the strengths

of causal relationships. In this case, their *consensus* network may be relatively simple due to omitting causal links that fall below the endorsement threshold. In short, the lack of individual understanding expressed by individuals in this group may result in the failure of the group's endorsements to cohere into an interpretable network. However, if a meaningful belief structure does emerge for those expressing low coherence, it will be informative to see what kinds of causal beliefs are associated with the perception that one's understanding of depression is poor. It also may be useful to examine differences between the full sample's consensus network, in which some structural relationships of interest may be obscured (averaged out), and the subgroups' models, in which structural variations may be more apparent.

Methods

Participants

The same U.S. adult sample recruited via Amazon's Mechanical Turk platform to take part in Study 1 also completed the measures for Study 2 (N = 319).

Procedure

Participants completed all instruments for Studies 1 and 2 during a single session; data collection methods described in Study 1 also apply to Study 2. The Appendix includes the complete questionnaire as it was administered.

Measures

Coherence Subscale, Illness Perception Questionnaire-Revised, modified for depression. The IPQ-R Coherence scale includes five items assessing the extent to which the respondent believes he or she clearly understands depression and its symptoms (e.g., "Depression doesn't make much sense to me"; Moss-Morris et al., 2002; Table 16).

Higher scores reflect *lower* illness coherence. The scale was administered as written for the IPQ-R, with the exception of replacing "my illness" with "depression," as described for the IPQ-R's other scales. In this sample, the Coherence scale showed good internal consistency (α = .887).

Network Analysis Questionnaire. On this 49-item instrument, which was created for this study, participants rated the perceived strength of causal associations among six potential causes of depression, and between each cause and depression itself. Each item consisted of a single causal strength rating for one of the 49 possible pairwise relationships, including separate items for both causal directions for each pair. Respondents were asked to rate the likelihood that one factor would cause another (e.g., "How likely is PHYSICAL ILLNESS to cause PROBLEMS WITH WORK OR FINANCES?), that one of these factors would cause depression ("How likely is PHYSICAL ILLNESS to cause DEPRESSION?"), or that depression would cause one of these factors ("How likely is DEPRESSION to cause PROBLEMS WITH WORK OR FINANCES?"). Ratings were provided on a three-point Likert scale (1=Never, or only rarely; 2=Possibly, or only sometimes; 3=Definitely, or most of the time). Each item was presented on the screen individually. For this instrument only, item order was randomized per participant to minimize any order effects between items.

The six causes were selected based on findings describing causal beliefs about depression in patient samples and the general public (e.g., Brown et al., 2001; 2007; Jorm et al., 1997; Lauber et al., 2003). These causes represent a subset of the items from the Cause scale of the IPQ-R, modified for depression (Study 1), chosen to include biological, psychosocial, and environmental factors, and to include acute life events (e.g.,

"experiencing a traumatic event") as well as potentially longer-term issues ("problems with work or finances"). To reduce participant burden and facilitate interpretation, some causes listed separately on the IPQ-R were combined in this assessment (e.g., "overwork," "unemployment," and "financial problems or worries" combined into "problems with work or finances"). Similarly, "biological changes in the brain" was selected over "genes/heredity" to represent biological causal beliefs in this study because neurobiological changes could be more proximally linked to the other causal factors and more plausibly involved in a variety of bidirectional relationships.

Statistical Analysis

Network analysis is used to quantify and represent relationships between interrelated causal beliefs by depicting them in a network diagram. In the diagram, each of the putative causal factors and the outcome of interest is represented as a separate node; the perceived causal relationships between nodes are represented as arcs. The network diagram is generated based on the causal strength ratings that participants assign to each of the possible pairwise relationships. The target outcome—in this case, depression—is included in the pairwise ratings to account for perceived direct causal links (Heffernan et al, 1998) and for the possibility that the outcome itself may be viewed as a causal or maintaining factor Brogan & Hevey, 2009; Reser & Muncer, 2004.

Generating networks. As noted above, participants used a Likert scale to rate the perceived strength of each pairwise causal relationship—e.g., "How likely is PHYSICAL ILLNESS to cause DEPRESSION?" The network is generated using an iterative process called inductive eliminative analysis (IAE; Reser & Muncer, 2004; Muncer & Gillen, 1997). This method, which yields a consensus representation of all links endorsed by the

majority of the sample, is considered preferable to other approaches because it strikes a balance between participant inclusivity and network parsimony (French et al., 2002; Muncer & Gillen, 1997). The process involves adding individual pairwise relationships (a pair of nodes connected by an arc) to the network hierarchically, starting with the relationship most strongly endorsed, on average, among all participants. Each subsequent link is added to the network in descending order of their mean strength ratings. As each new link is added to the diagram, the whole network is evaluated to determine the number of participants who have endorsed all existing links. For this purpose, "endorsement" is evaluated by determining the mean of the causal strength ratings for all the links in the network thus far, which must surpass a pre-selected item average criterion (IAC) value. This threshold value is selected at the outset based on the overall strength of endorsements in the data; it usually is set at or just above the midpoint of the causal strength rating scale (French et al., 2002; Muncer & Gillen, 1997). After each new link is added, the network is considered consensual if all of its links are still endorsed by the majority of respondents, at a strength equal to or greater than the IAC value (French et al., 2002; Lyden et al., 2016; Muncer & Gillen, 1997). Network generation stops when adding the next link would reduce the proportion of participants represented in the network (the proportion surpassing the IAC) to below a pre-selected percentage of the original sample. Published networks typically have used consensus thresholds of either 51% of the sample (Brogan & Hevey, 2009) or 70% of the sample (Lyden et al., 2016) surpassing the IAC, depending on the evident causal strength ratings. Based on the generally high ratings in the current study as well as the decision to prioritize network interpretability and representativeness of the sample over including additional links, the

threshold was set at 70%. Once the complete set of links in the consensus network has been established, the consensus matrix of causal strength ratings can be subjected to multidimensional scaling to determine the placement of network nodes.

Comparing networks. Networks can be compared in several ways, including their relative complexity and the perceived relationships of particular cause, such as "biological changes in the brain," to other factors. Networks' relative complexity can be assessed with several metrics, including the number and proportion of bidirectional relationships and/or feedback loops that each one includes, the number of instances of mediation that appear in the network, and the number of instances in which the outcome of interest (depression) is perceived to be a cause.

Another concept used in network analysis is centrality. In analyses representing social networks, this concept pertains to the degree of interconnectivity of a particular node, representing an individual person (Knoke & Yang, 2008). Similarly, the role of a particular casual factor (e.g., the node representing "biological changes in the brain") can be analyzed and compared between networks based on the number and relative strength of links originating from that node. This comparison accounts for causal strength ratings of this factor relative to others'. For example, a network in which a greater proportion of its nodes are "attributed" to neurobiological changes, or stronger links originate from that node than from others, would be interpreted as showing a more central role for neurobiological causes (Knoke & Yang, 2008).

Results

Consensus Network

The mean strengths of the links in the consensus network representing 70% of the full sample are presented in Table 17. Multidimensional scaling of the causal strength ratings matrix yielded a two-dimensional solution with a low level of stress (.067), indicating good fit to the data. A high proportion of variance was accounted for in the nodes' dispersion (R^2 = .98). According to published guidelines (French et al., 2002; Muncer & Gillen, 1997), the link with the strongest mean rating, *trauma causes stress* (*M* = 2.66), was entered into the network first; it was endorsed at the IAC (≥ 2) by 97.5% of the sample. Seventeen links were added to the network in descending order of mean causal strength ratings (Figure 6). Adding link 18, *depression causes biological changes in the brain*, reduced the proportion of the sample endorsing the network to below the 70% consensus threshold (67%), so network construction ended with link 17.

The network included two direct, bidirectional links with depression (*stress or worry; problems with work or finances*) and two additional, unidirectional, direct links to depression (*trauma; biological changes in the brain*). One additional bidirectional link did not include depression (*stress or worry–social relationship problems*). Based on the number of associated links, *stress or worry* was the most central node in the network with six incoming links, two of which were bidirectional; 47% of the links ran through *stress or worry*. Two causal links originated at *biological changes in the brain*, including a direct, unidirectional link to depression.

Low and High Coherence Groups

To compare networks for participants expressing high versus low levels of confidence in their understanding of depression, sample subgroups were selected based on IPQ-R Coherence scale scores (*M* = 10.34, *SD* = 4.24). Higher scores on this measure

indicated lower coherence, so participants scoring one standard deviation above the mean or higher constituted the low coherence group (n = 74), while those scoring less than or equal to one standard deviation below the mean made up the high coherence group (n = 80). First, group differences in demographic, clinical, causal belief, prognostic, and stigma-related variables were examined using chi-square tests for dichotomized variables and either *t*-tests or the Mann-Whitney *U* test for continuous variables. Table 18 shows descriptive statistics for each group for those variables showing statistically significant group differences. Chi-square tests of group differences in gender, self-reported history of depression, and depression history in a close other showed that the high coherence group included a significantly greater proportion of women ($\chi^2(1) = 9.24$, $p = .002$), individuals reporting a history of depression ($\chi^2(1) = 28.11$, $p < .001$), and those affirming a close other's prior depression ($\chi^2(1) = 17.74$, $p < .001$). Other variables of interest were screened for skewness and kurtosis in the pooled high and low coherence groups (n = 154) to determine the appropriateness of parametric versus nonparametric tests. One variable, neurobiological causal beliefs, exceeded the kurtosis threshold of +/-2 (kurtosis = 2.32; Gravetter & Wallnau, 2014), so a Mann-Whitney *U* test was used to test this variable. Results showed that the high coherence group endorsed significantly stronger neurobiological causal beliefs compared to the low coherence group (U = 1502.50, $p < .001$). The remaining variables met the assumptions for parametric tests, so the groups were compared using independent-samples *t*-tests (Bonferroni-corrected α = .004). In addition to the groups' significant difference in coherence scores (higher scores reflecting *lower* coherence: high: $M = 5.19$, $SD = .39$; low: $M = 16.51$, $SD = 2.09$; $t(152) = 47.47$, $p < .001$), the groups also differed significantly in beliefs about depression's

duration and in desired social distance. The high coherence group viewed depression as longer-lasting (M = 41.91, SD = 7.49) than the low coherence group did (M = 36.58, SD = 5.03; $t(148) = -5.07$, $p = .001$). The high-coherence group (M = 13.05, SD = 5.35) also desired significantly less social distance than the low-coherence group (M = 16.38, SD = 3.72; $t(150) = 4.43$, $p = .003$).

Low Coherence Network. The low coherence group yielded a consensus network of eight links; mean strengths are listed in Table 19. Multidimensional scaling of causal strength ratings yielded a two-dimensional solution with low stress (.004), indicating a good fit to the data, and $R^2 = .99$, indicating that the diagram accounted for a very high proportion of the variance. The most strongly endorsed link, *trauma causes stress* (M = 2.42; endorsed by 93% of the sample), was entered into the network first. A total of eight links were added using the process described above (Figure 7). Adding the ninth link, *depression causes problems with work or finances,* reduced the endorsement of the network from 70% to 69% of the sample, so network construction stopped at link eight. This group's network included a single causal link to depression (*trauma causes depression*), which was unidirectional. The network did not include any bidirectional links. Based on the number of associated links, *stress or worry* was the most central node; this node had relationships to five incoming links and no outgoing links, or 71% of the total links. *Biological changes in the brain* was the only causal factor that was not included in this network.

High Coherence Network. The high coherence group yielded a consensus network of 20 links; Table 20 shows their mean strengths. Multidimensional scaling of the causal ratings matrix produced a two-dimensional solution with low stress (.067),

indicating that it fit the data well, and R^2 = .98, indicating that it accounted for a high proportion of the variance. The most strongly endorsed link, *trauma causes stress* (*M* = 2.83; endorsed by 100% of the sample), was entered into the network first. Twenty links were added following the steps described earlier (Figure 8). Adding link 21, *stress or worry causes problems with work or finances,* reduced network endorsement from 70% of the group to 69%, so network generation ended with link 20.

Four nodes in the high coherence network had direct, bidirectional links with depression (*biological changes in the brain*; *stress or worry*; *problems with work or finances*; *social relationship problems*), and one additional node had a unidirectional link to depression (*trauma causes depression*). The network included one additional bidirectional link that did not involve depression (*stress or worry–social relationship problems*). Depression was the most central node, associated with five incoming and four outgoing links or 45% of the total links. *Biological changes in the brain* was associated with three outgoing and two incoming links, including its bidirectional connection with depression.

Discussion

Consensus Network

The central role for stress in this network is consistent with prior research into lay beliefs about depression using the IPQ-R. In this work, "stress or worry" has been by far the most frequently endorsed cause (e.g., Brown et al., 2001; 2007; Fortune, Barrowclough, & Lobban, 2004; O'Mahan et al., 2008; Prins et al., 2008; Vollman et al., 2010). However, the substantial overlap between the stress construct and the nature of depressive symptoms (e.g., Sawatzky et al., 2012) raises the possibility that attributing

depression to stress reflected a conflation of the two conditions, other circular reasoning, and/or a significant role for causes of stress not assessed in these studies. Without probing these respondents' attributions for *stress*, those findings remain difficult to interpret. By placing the belief that *stress causes depression* in the context of other causal beliefs, network analysis may shed light on the earlier findings.

In the full sample's network, *stress or worry* was the most central node. Nearly half (47%) of the links ran through it, and it was endorsed as an effect of every other causal factor. In addition to being broadly construed as an outcome of other factors, *stress or worry* also had a direct, bidirectional connection with depression, therefore serving as a mediator between every other causal factor and depression. In this respect, the network suggests a view of depression as *proximally* caused by stress which, is itself, broadly multidetermined by both biological and psychosocial factors. A few select causes—*trauma*; *biological changes in the brain*; *problems with work or finances*—were conceptualized as direct causes of depression, in addition to their stress-mediated paths.

The network also depicts a view of depression as more consequential than *stress or worry*. Both *stress or worry* and depression showed an equal number of bidirectional relationships. Apart from these, *stress or worry* was linked to four other nodes as an outcome and was never implicated as a cause. In contrast, depression was viewed as both a unidirectional outcome of various factors (like stress), and a unidirectional cause of *social relationship problems*. This difference suggests that participants may have been more inclined to view stress (when it did not lead to depression) as a manageable "endpoint" outcome, compared to depression, which was more likely to become a maintaining factor, causing new problems in its own right. This distinction suggests some

structural similarity between lay beliefs and cognitive-behavioral theories, which often highlight the potential for particular symptoms, such as problematic patterns of thought and behavior, to exacerbate existing stressors and maintain or worsen depression (e.g., Joiner, 2000). Though modeling causal beliefs as a network may inherently accentuate these kinds of relationships, different networks in this study reflected different beliefs about stress and depression, as well as the extent to which these factors were construed as causes versus outcomes.

Low and High Coherence Groups' Networks

Low Coherence Network. The low coherence group's network is better described as a picture of *stress or worry* as a multidetermined outcome than as a representation of causal beliefs about depression. Only one link to depression, from *experiencing a traumatic event*, reached the threshold for inclusion in the network, and this was the final (weakest) link to be added. Given that *experiencing a traumatic event* necessarily involves encountering a severe external stressor, its role as the network's only cause of depression seems to afford individuals who become depressed relatively low control over that development. Depression was somewhat more likely to be conceptualized as a cause in itself, leading to *stress or worry* and *social relationship problems*. These links are notable in light of this group's self-professed limited understanding of depression as well as some differences between this group and those reporting high coherence. Compared to the high coherence group, those with low coherence were significantly less likely to affirm a history of depression in a close other (or themselves); they also desired significantly more social distance from people with depression. Their network representation, in which depression is perceived as especially

problematic for social relationships, accords with their greater desire to minimize social contact with people with depression, which, in turn, is consistent with their lower likelihood of reporting a close relationship with someone who had depression.

This network also suggests a simpler and more concrete folk psychology in the low coherence group, and perhaps a more external attributional style, compared to the full sample or the high coherence group. Overall, the low coherence group's network was distinguished by its sparse links and lack of bidirectional relationships. Most nodes are conceptualized as either causes or effects. Moreover, the nodes serving as causes—*trauma; physical illness*; *problems with work or finances*—can be viewed as relatively more extrinsic to the individual or to individual control. Trauma and physical illness, for example can be interpreted as problems that befall someone, which the individual may have limited ability to prevent or mitigate. Along this line of reasoning, individual control may be likewise limited in *problems with work or finances,* which are often defined by obligations and hierarchical power relations. By comparison, the factor serving only as an outcome in the network (*stress or worry*) references a subjective state. Of the two factors that play both cause and effect roles (*depression; social relationship problems*), the first describes internal experience, while the second allocates more control to the individual than do the strictly casual factors—to the extent that "social relationships" are viewed as more voluntary, and less power-inflected than work-related problems. In this context, the omission of *biological changes in the brain* from this network appears consistent with this group's focus on the effects of external stressors on subjective experience, while giving less weight to causal influences of internal factors. This interpretation is also broadly consistent with the lower rate of self-reported depression history among

participants in the low coherence group, as the tendency to make external rather than internal attributions has been associated with lower rates of depression (Abramson, Seligman, & Teasdale, 1987; Hu & Zhang, 2015)

High Coherence Network. The network for the high coherence group was characterized by greater complexity and interconnectedness—including a higher number of links, a larger proportion of which were bidirectional—compared to the low coherence group and to the full sample. Most nodes in the high coherence network were linked with multiple other factors as both causes and outcomes, and were implicated in at least one bidirectional relationship. As exceptions, *experiencing a traumatic event* and *physical illness* served only as causes. This network also was distinguished by the central role of depression in its structure, followed by *stress and worry*, which was central to both other networks. In comparing the roles of depression and stress in the high coherence network relative to their roles in the other networks, it is notable that once again stress was linked to every other node as an outcome and was linked to depression as a cause, so it mediated between other factors and depression again, as in the other networks. Further resembling the other networks, depression was implicated as a cause more frequently than *stress and worry*, suggesting that it was perceived to be more consequential.

The distinction between the consequences of stress versus depression was even more pronounced in this network, as depression was implicated as a cause more frequently than any other node except *experiencing a traumatic event*. Unlike trauma, which served *only* as a cause, all four instances of depression as a cause were conceptualized as bidirectional links. The perception of depression as a highly likely cause and outcome in this network, and its involvement predominantly in bidirectional

relationships, may reflects, at least in part, the higher prevalence of first- and second-hand experience with depression among these participants. One interpretation of this association is straightforward: Participants in the high coherence group may have gleaned from their lived experience knowledge about a broader variety of potential causes of depression. Based on this knowledge, they may have formed a more dynamic understanding of causal interactions between life events, psychosocial problems, subjective distress, and biological influences than is reflected in the other networks, with an especially pronounced difference from the low coherence network.

An alternative interpretation, which is not exclusive of this possibility, is that this network's complexity and the centrality of depression in it may reflect differences in this group's explanatory style, which may also be associated with their report of more first- and second-hand experience with depression. Intolerance of uncertainty and a tendency to make pessimistic predictions have been studied as cognitive vulnerabilities for developing depression and maintaining factors in its chronicity (Hong & Cheung, 2015; McEnvoy & Mahoney, 2012; Miranda & Mennin, 2007). Given that the high coherence group had a higher proportion of participants reporting a history of depression, this group also may have been, on average, more predisposed to make negative predictions backed by a high degree of certainty. Though the Network Analysis Questionnaire items were designed to ask about the general likelihood that one factor causes another, the items also can be viewed as eliciting predictions about the perceived likelihood of a variety of negative outcomes. In this light, participants with a more pessimistic style would be expected to rank more of these outcomes as likely and to express more certainty, or causal strength, in their rankings. Given that the network generation process uses the

group's mean causal strength ratings, the high coherence group could generate a more complex network simply by providing ratings that generally were more certain and pessimistic, causing a larger number of links to surpass the IAC and be included in the network. One caveat, however, is that the high and low coherence groups did differ in their reports of current depressive symptoms, and most research linking these cognitive vulnerabilities to depression has focused on clinical populations (Hong & Cheung, 2015).

Significance and Limitations

The use of network analysis in this study provides a novel perspective on participants' causal beliefs about depression and related insights into the meaning of the illness coherence construct. Studying causal beliefs about depression as networks of interrelated factors expands in some important ways beyond the hypotheses of Study 1 and beyond some basic premises that Study 1 shares with most research on causal thinking about mental illness. The usual approach to this topic has involved asking participants to evaluate potential causes individually, presenting them as separable, isolated influences. In contrast, network analysis provides a systematic means of representing more of the complexity that characterizes lay causal explanations of illness when they are elicited open-endedly (e.g., French, Maissi, & Marteau, 2005; Sensky, 1997; Schreiber & Hartrick, 2002). This method also allows for quantitative comparisons, either hypothesis-driven or exploratory, to characterize group differences in causal beliefs pertaining not only to beliefs' content but to the implicit structure of perceived causal relationships.

This study also has implications regarding the construct of illness coherence as measured by the IPQ-R. The structurally distinct causal belief networks of the low and

high illness coherence groups suggest that these groups differed not only in their explicit beliefs about depression captured by the IPQ-R but in the kind of problem they construed depression to be—and that illness coherence may be an important correlate of the latter differences. In addition to the association in this sample between illness coherence and prior experiences with depression, the networks also suggest associations between illness coherence and depression's perceived prevalence (likelihood as an outcome), its role as a mutually maintaining factor in bidirectional relationships, and the breadth of its potential consequences. This third component, evident in the networks' disparity in how often depression causes other problems, is notable in light of the lack of a significant difference between these groups' IPQ-R Consequences scale scores. The Consequences scale items focus on the severity of illness effects (see Table 2) which may be a separate construct from pervasiveness. As discussed above, some implications of the illness coherence construct may be illnesses- and population-specific. Nonetheless, its association with meaningful group differences in implicit and explicit illness beliefs suggest that coherence can provide a useful complement to familiarity moderators in accounting for participants' views of their own relationship to the target group or illness (e.g., as ingroup versus outgroup; as a domain of proficiency versus confusion), which may interact with the content of their beliefs. A metacognitive measure like illness coherence may be especially relevant to studies of illness beliefs and stigma in the general population, in which wide variations in knowledge about, and even basic construal of, the target illness could be expected.

Study 2 shares some of Study 1's limitations, including its reliance on cross-sectional, correlational data, which precludes casual inferences. This issue is less

consequential for Study 2, which aimed to assess and describe group differences in belief structures, rather than testing predictive associations. However, it bears mentioning because the belief structures represented in the network diagrams depict perceived cause-effect relationships. It is important to keep in mind that they represent a snapshot—and an aggregate—of participants' causal strength ratings at one point in time, rather than any causal influences. The limitations referenced in Study 1 with respect to the validity of self-report data and the generalizability of results gleaned from a convenience sample recruited online also apply to Study 2.

In addition, several limitations specific to network analysis warrant mentioning. First, the need to include all possible pairings of putative causes with one another and with the target outcome quickly yields a large number of items. To reduce participants' burden, a balance was struck between thoroughness and parsimony by, for example, using broader categories of causation in these items compared to the IPQ-R Cause scale modified for depression. A qualitative study posing open-ended questions about perceived causes of depression could capture a broader range of causal factors, more nuance in their relationships, and a variety of individual differences left out of these consensus network models. Second, network analysis of belief structures is relatively early in its development, and best practices have yet to be established for some aspects of the technique, such as the bases for selecting the breadth of the causal strength rating scale, the item average criterion (IAC) value, and the proportion of the sample that the network represents (Brogan & Hevey, 2009; French et al., 2002; Lydon et al., 2016; Muncer & Gillen, 1997).

General Discussion

Taken together, Studies 1 and 2 suggest refinements to essentialist theory, opportunities to apply other methodological approaches to investigate relationships between mental illness beliefs and stigma, and guidance for clinicians and public health advocates in explaining the biology of depression.

Theoretical Implications

The prevalence and implications of biological causal beliefs can be expected to vary for specific biological attributions as well as across disorders. This remains a fruitful area for research, as few studies have examined how these associations differ depending on both the specific biological cause in question and the target disorder. One vignette-based study examined the differential associations of three biological causal beliefs with measures of stigma and emotional responses toward individuals with either depression, schizophrenia, or alcohol dependence. In relation to alcohol dependence, the two brain-based attributions ("brain disease," and "chemical imbalance") were associated with desiring less social distance and responding emotionally with sympathy and desire to help, whereas making these same attributions for depression or schizophrenia was associated with desiring greater social distance. "Heredity," on the other hand, was consistently unrelated to stigma measures in this study (Speerforck et al., 2014). Together with the results of the current study, these findings highlight the need to differentiate the correlates of specific biological attributions, rather than presuming that all biological explanations carry similar meanings. Such results also imply significant limits on the view that essentialist thinking about people might be a domain-general or trait-like individual difference (Bastian & Haslam, 2006), as opposed to a more domain-specific interpretation.

Along related lines, the current results suggest that the strength and direction of relationships between biological attributions and mental illness stigma depend in part on the *type of problem* being attributed to biological causes, and that a more multifaceted assessment of illness beliefs yields meaningful differences in both the content of those beliefs and their relationships to stigma. Studies 1 and 2 each provide evidence of significant variations in participants' conceptualizations of depression, related in part to their first- and second-hand experience with the disorder and to how well they believed they understood it. These views showed some consistencies with essentialist predictions, such as the associations between neurobiological attributions and viewing depression more consequential and long-lasting. However, these negative prognostic beliefs were associated with positive beliefs about treatability and *inversely* associated with personal stigma. By documenting these variations within the scope of a single diagnosis, the current findings strengthen the case for integrating essentialist theories about mental illness beliefs with a broad-based theory of illness cognition like the CSM. This move would encourage more precision in essentialist theory's use and definition of illness belief constructs. It also would place more emphasis on understanding the specific conditions—perhaps constellations of illness beliefs and experiences—that foster essentialist thinking about a particular problem, and the implications of these beliefs for forms of mental illness stigma.

The type of problem a particular mental illness is perceived to be is also informed in complex ways by cultural factors, most of which fall outside the scope of this project (Angel & Thoits, 1987; Kirmayer & Bhugra, 2009). However, with an eye toward future research, it bears emphasizing that the current sample was insufficiently diverse in terms

of race and ethnicity to include this factor as a covariate. In the U.S., racial and ethnic minority groups are significantly less likely than Whites to use services for mental health problems (Substance Abuse and Mental Health Service Administration [SAMHSA], 2015). Reasons for this disparity vary by population; with regard to depression treatment, barriers in some underserved groups include insufficient availability, affordability, and acceptability of care; stigma associated with disclosing mental health problems and receiving treatment; and/or a lack of recognition of mental health problems prior to a crisis point (Cabassa, Lester, & Zayas, 2007; Hudson et al., 2016; SAMHSA, 2015). Bridging the considerable gaps between providers and underserved patient groups therefore requires not only improving access to current forms of treatment, but also learning more about the illness beliefs associated with forms of stigma (including perceived stigma in specific communities) and with the specific treatment preferences and concerns within these cultural contexts.

The variations that emerged within this study of beliefs about a single diagnosis also caution against generalizing about the structure and implications of illness beliefs—essentialist or otherwise—across mental illnesses as a category. Depression and schizophrenia have received the bulk of researchers' attention in this area. As noted above, preliminary evidence has shown that associations between biological attributions and stigma that are evident for depression and schizophrenia may run in the inverse direction for other diagnoses (e.g., alcohol dependence; Speerforck et al., 2014), or even for depression itself, depending on sample characteristics and the particular constructs of interest. Therefore, further research is needed to understand how specific beliefs about other mental illnesses may relate to whether and how they are stigmatized.

This application of the CSM also would shift its focus from understanding *illness representations*—beliefs about one's own case of the illness—to *illness prototypes,* which refers to the set of beliefs about a given illness's typical attributes (Leventhal, Leventhal, & Contrada, 1998; Leventhal et al., 2010). The CSM has often been used to understand the illness beliefs of individuals who have not experienced the target illness themselves (e.g., caregivers); it has less often been used to study the beliefs of those without a direct connection to the target illness. If the CSM were broadly employed to study mental illness prototypes with the goal of understanding their relationships to stigma, it would be helpful to incorporate the construct of continuum beliefs. This construct refers to the extent to which an illness is viewed as existing on a continuum of symptoms, as opposed to viewing it as discrete and categorically unlike experiences of unaffected people (Schomerus, Matschinger, & Angermeyer, 2013). A common emphasis in essentialist theories and other models of mental illness stigma is the amplification of perceived group differences, in this case between affected and unaffected individuals (Dar-Nimrod & Heine, 2001; Haslam, 2011; Link & Phelan, 2001; Link et al., 2004). Recent research has linked continuum beliefs about several diagnoses, including depression, to both desiring less social distance from affected individuals and reporting more pro-social emotional and behavioral responses (Schomerus, Matschinger, & Angermeyer, 2013).

Essentialist theories posit an implicit association between biological causal beliefs and low continuum beliefs, conceptualizing the latter as group *discreteness*. These theories warn that anti-stigma messages emphasizing biological causality may unintentionally increase stigma in part by magnifying perceived differences between

those with a given diagnoses and everyone else (Dar-Nimrod & Heine, 2001; Haslam, 2011). The current results, in which biological attributions and even some negative prognostic beliefs showed an *inverse* relationship to depression stigma, suggest that causal and prognostic beliefs are less reliably linked to high discreteness (or low continuum) beliefs than essentialist theories argue. This possibility, too, can be tested in the context of beliefs about specific disorders. Other recent experimental work targeting continuum beliefs to reduce schizophrenia stigma (e.g., Corrigan et al., 2017; Thibodeau, Shanks, & Smith, 2018; Weisjahn et al., 2016), also suggests that these beliefs can play an important, distinctive role connecting illness beliefs with stigma. It is not known whether low continuum beliefs are associated with depression stigma. However, along with the above-mentioned findings, research showing that depression symptoms do occur on a continuum in the general population (Hankin et al., 2005; Tebka et al., 2018) make this prospect worth investigating.

Methodological Implications

Qualitative Studies. In pursuing a fuller understanding of the topics outlined above, alternative methods could provide an important complement to those applied in this project. Closed-ended, quantitative measures of illness beliefs like the IPQ-R facilitate data collection and analyses, yet qualitative approaches, such as semi-structured interviews, can allow researchers to explore participants' beliefs and attitudes in greater depth. By eliciting open-ended explanations, these methods allow participants the opportunity to explain perceptions of various interacting risk factors and causes, their relative importance, their implications for beliefs about treatment, and the bases for these beliefs (Bhui & Bhugra, 2002). Where biological causal beliefs are concerned, qualitative

methods allow researchers to probe related beliefs about biological mechanisms, including the extent to which biological factors are perceived as immutable versus changeable or as deviating categorically versus incrementally from a healthy baseline. The evidence from qualitative studies, which often elicit complex, multifaceted explanations for specific illnesses indicates that the convenience of closed-ended measures is not without costs (Bhui & Bhugra, 2002; Sensky, 1997). However, such measures fit the current study's aims, in which the selection of causal belief constructs and stigma variables was theory-driven, and the generation of causal belief networks emphasized parsimony over thoroughness to facilitate comparisons. In contrast, qualitative studies will be especially important to begin addressing potential relationships between illness beliefs and stigma in specific underserved groups. Where relatively little is known about specific groups' beliefs about depression and its treatment, yet these beliefs may contribute to disparities in treatment utilization, qualitative studies provide an alternative to presuming that the relevant constructs are already understood (Bhui & Bhugra, 2002). Instead, this work can guide researchers, providers, and outreach agencies toward community-relevant services as well as meaningful efforts to improve mental health literacy.

Experimental Studies. In addition to qualitative studies' role in understanding relationships among illness beliefs and links to stigma, experimental studies are needed to investigate the nature of these relationships and the effectiveness of anti-stigma interventions premised on changing illness beliefs. One area of increasing interest focuses on dispelling potentially negative implications of biological causal beliefs, like prognostic pessimism and fear, by emphasizing the malleability of biological factors. In

the first study of this sort, individuals reporting above-threshold depression symptoms watched one of two brief videos explaining depression in biological terms (Lebowitz, Ahn, & Nolen-Heoksema, 2013). One video provided a biomedical explanation, including information on heritability and neurobiological bases; the other included information about moderating environmental influences on these factors. Watching the latter video was associated with reduced prognostic pessimism and increased agency regarding symptom improvement. Yet, the study did not account for the fact that only the latter video discussed any means of symptom improvement, so the effect of information on biological dynamics was unclear. Nonetheless, it provides a model that could be enhanced by balancing the interventions' information on symptom improvement, and could be extended to the general population with the addition of stigma measures.

Studies with a similar structure have tested interventions to reduce schizophrenia stigma in the general population by either targeting continuum beliefs (Corrigan et al., 2017; Thibodeau, Shanks, & Smith, 2018) or comparing an intervention promoting continuum beliefs with one promoting biological causal beliefs (the latter did not discuss biology-environment interactions; Wiesjahn et al., 2016). In the comparative study, the continuum intervention yielded greater reductions in most baseline stigma measures, though biological causal information was associated with greater reductions in blame. One possibility that has yet to be examined is that interventions explaining the malleability of biological factors and those explaining the continuity of symptoms with normal functioning may target similar underlying, stigma-mediating beliefs about group discreteness. Teaching continuum beliefs about symptom experiences may counter these beliefs directly, while explaining biology-environment interactions may counter the same

beliefs indirectly: Understanding that some biological factors are malleable and under a degree of behavioral control may reduce the perception of biological differences as fixed and categorical. The possibility that *discreteness* beliefs may mediate both types of interventions also could be tested experimentally.

Applications

The current findings also have implications for psychoeducation, both in clinical practice and in public health campaigns against depression stigma. For clinicians and public health officials, these findings provide a counterpoint to the concerns emphasized in essentialist theory about the negative implications of biomedical views of mental illness. In Study 1, stronger biological attributions, whether genetic or neurobiological, were associated with *lower* stigma. Moderator analyses indicated that this inverse association was especially characteristic of individuals reporting both a history of depression and *weaker* biological causal beliefs, who tended to report higher stigma. In contrast, levels of stigma were lower and similar across those with no depression history and to those endorsing prior depression and stronger biological attributions. These findings run counter to essentialist theory, yet they should be considered against the background of mixed prior results which include support for essentialist predictions. In that context, these findings support the possibility that biological attributions per se are unlikely to exacerbate stigma, and that some kinds of biological explanations either may help reduce stigma or may be associated with other stigma-reducing beliefs and experiences. Either way, these results caution clinicians and educators against avoiding biomedical explanations out of concerns about stigma.

Whether particular beliefs about biological causes may be associated with different levels of stigma is outside the scope of this study. However, the results of Study 2 help to address, in a preliminary way, the question of how participants viewed biological causes in relation to depression and to other causal factors. In the network for participants with high confidence in understanding depression, the node representing neurobiological causes has a direct, bidirectional relationship with depression, and it is linked as a cause to psychosocial mediators of depression. In contrast, participants expressing low confidence singularly omitted the neurobiological cause node from their network. Interestingly, the latter group also desired significantly greater social distance from people with depression, compared to the group that was both more confident in understanding depression and conceptualized an integral, dynamic role for biological causes. These findings are consistent with a set of broad recommendations grounded in attribution theory that emphasizes teaching about depression's biological causes and mechanisms in ways that balance "retrospective" and "prospective" framings (MacDuffie & Strauman, 2017). The retrospective frame, in which biological factors are stable and uncontrollable, can help reduce blame for depression onset (e.g., "depression runs in families"). Yet these explanations should be used judiciously, within the context of "prospective" framings that emphasize the role of individual agency in the malleability and controllability of our own biological systems (e.g., "experience changes the brain"). These kinds of messages, bolstered with effective metaphors, may help to reduce stigma associated with essentialist beliefs about biology, while also increasing awareness and acceptance of treatments utilizing behavioral principles.

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Tables

Table 1

Descriptive statistics for the full sample.

Demographics	*N*	Number	%	M	SD	Min	Max	α	Skew	Kurtosis
Age	319		100	38.34	12.67	18	88		.824	.087
Gender	319		100						.445	-1.813
Female		194	60.8							
Male		125	39.2							
Race/ethnicity*	319									
White or European American		267	83.7							
Black or African American		21	6.6							
Asian or Asian American		23	7.2							
Latino/a or Hispanic		15	4.7							
American Indian		10	3.1							
Hawaiian or Pacific Islander		3	0.9							
Middle Eastern or North African		3	0.9							
Highest education level	319		100							
High school degree or less		28	8.8							
Some college		74	23.2							

Two-year college degree		45	14.1		
Four-year college degree		101	31.7		
Some grad. school		18	5.6		
Graduate degree		53	16.6		
Four-year college degree	319		100	-.158	-1.988
No		147	46.1		
Yes		172	53.9		
Employment status**	319		100		
>35h per week		205	64.3		
10-35h per week		43	13.5		
<10h per week		5	1.6		
Work for pay only via MTurk		66	20.7		
Household income	319		100	.353	-.816
$100,000 or more		39	12.2		
$80,000-$99,999		29	9.1		
$60,000-$79,999		64	20.1		
$40,000-$59,999		69	21.6		
$20,000-$39,999		78	24.5		
Less than $20,000		38	11.9		
Clinical variables					
Believe self had depression	319		100	.057	-1.988
Yes		155	48.6		
No		128	40.1		
Don’t know		36	11.3		
Believe close other had depression	319		100	-.792	-1.381

Yes		218	68.3							
No		77	24.1							
Don't know		24	7.5							
Depression symptoms (PHQ-9)	308		96.5	5.22	6.15	0	27	.92		
Biological causal beliefs (IPQ)										
Biological changes in the brain	319		100	4.30	.02	1	5		-1.252	1.871
Heredity/genes	319		100	3.94	1.00	1	5		-1.122	.965
Prognostic beliefs (IPQ)										
Consequences	314		98.4	21.57	2.70	12	25	.766	-.614	-.088
Duration	314		98.4	39.06	6.59	23	55	.812	.215	-.479
Treatability	314		98.4	24.22	5.55	11	40	.774	.227	-.121
Stigma variables										
Personal stigma	318		99.7	22.82	6.96	10	45	.864	.387	-.145
Perceived stigma	314		98.4	33.38	7.51	10	50	.881	-.759	.716
Desired social distance	316		99.1	15.04	4.66	7	28	.900	0.180	-.244

* Respondents were asked to mark all that apply, so percentages sum to more than 100.

** Hours per week working for pay, excluding MTurk

Table 2

IPQ-R Consequences scale overview and items.

Construct	Beliefs about the magnitude and extent of depression's negative consequences for those who have it
Response format	5-point Likert scale: Strongly disagree; Disagree; Neither agree nor disagree; Agree Strongly agree
Instruction	We are interested in your own personal views about depression. Please indicate how much you agree or disagree with the following statements:
Items	Depression is a serious condition.
	Depression has major consequences in the lives of people who have it.
	Depression strongly affects the way others see those who have it.
	Depression does not have much effect on the lives of people who have it.
	Depression has serious financial consequences.
	Depression causes difficulties for people close to those who have it.

Table 3

IPQ-R Timeline scale overview and items.

Construct measured	Beliefs about the duration of depression
Response format	5-point Likert scale: Strongly disagree; Disagree; Neither agree nor disagree; Agree Strongly agree
Instruction	We are interested in your own personal views about depression. Please indicate how much you agree or disagree with the following statements:
Items - Untreated Depression	When it is not treated, depression usually lasts a short time.
	Without treatment, depression is likely to be permanent rather than temporary.
	Untreated depression usually lasts for a long time.
	Depression usually passes quickly without treatment.
	People with depression who don't receive treatment generally have it for the rest of their lives.
	Without treatment, depression will improve in time
	* The symptoms of untreated depression change a great deal from day to day.
	* The symptoms of untreated depression change a great deal from day to day
	* Untreated depression goes through cycles in which it alternates between getting better and getting worse.
	* Depression that is not treated is very unpredictable.
	** Someone who has experienced depression and recovered without treatment is likely to experience additional depressive episodes in the future.
Items - Treated Depression	With treatment, depression usually lasts a short time.
	Depression is likely to be permanent rather than temporary, even when it is treated.

	Even with treatment, depression usually lasts for a long time.
	Depression usually passes quickly when it is treated.
	People with depression generally have it for the rest of their lives, even if they receive treatment.
	†Depression that is treated will improve in time.
	* For a person receiving treatment for depression, symptoms still change a great deal from day to day.
	* With treatment, depression goes through cycles in which it alternates between getting better and getting worse.
	* Depression is very unpredictable, even if a person receives treatment.
	** Someone who has been treated for depression before and recovered is likely to experience additional depressive episodes in the future

† Item was omitted from the analyzed scale score sum due to low intercorrelation with other scale items.

* Items measuring beliefs about *cyclical* change in symptoms. These items were excluded from the measure of illness duration.

* * Items distinguishing acute depressive episodes from a chronic predisposition toward such episodes. These items were added for future exploratory analyses and were not included in the current analyses.

Table 4

IPQ-R Cure/Control scale overview and items.

Construct measured	Beliefs about depression's treatability
Response format	5-point Likert scale: Strongly disagree; Disagree; Neither agree nor disagree; Agree Strongly agree
Instruction	We are interested in your own personal views about depression. Please indicate how much you agree or disagree with the following statements:
Items – Personal Control	There is a lot that people with depression can do on their own to reduce their symptoms.
	What people with depression do can determine whether their depression gets better.
	Nothing that people with depression do will affect their condition.
	The course of people's depression depends on them.
	People with depression have the power to influence their condition.
	The actions of people with depression will have no effect on whether their depression gets better.
Items – Treatment Control	There is very little that can be done to help with depression.
	Treatment is effective in curing depression.
	Nothing can be done to improve depression.
	Treatment can reduce the symptoms of depression.
	The negative effects of depression can be avoided with treatment.

Table 5

Depression Stigma Scale overview and items.

Constructs measured	(1) Personal stigma - Respondents' own stigmatizing attitudes toward people with depression; (2) Perceived stigma - Respondents' perceptions of depression-stigmatizing attitudes held by "most other people."
Response format	5-point Likert scale: Strongly disagree; Disagree; Neither agree nor disagree; Agree Strongly agree
Personal Stigma Scale	
Instruction	The following items ask for your own opinions and beliefs about depression. There are no right or wrong answers. Please indicate how much you agree or disagree with each statement.
Items	People with depression could snap out of it if they wanted.
	Depression is a sign of personal weakness.
	*People with depression are dangerous to others.
	*People with depression are dangerous to themselves.
	It is best to avoid people with depression, so you do not become depressed yourself
	People with depression are unpredictable.
	Depression is not a real illness.
	If I had depression I would not tell anyone.
	I would not employ someone if I knew they had been depressed.
	I would not vote for a politician if I knew they had been depressed.
Perceived Stigma Scale	
Instruction	The following items ask for your opinions about what MOST OTHER PEOPLE BELIEVE about depression. Again, there are no right or wrong answers. Please indicate how much you agree or disagree with each statement.
Items	Most people believe that people with depression could snap out of it if they wanted.

Most people believe that depression is a sign of personal weakness.
*Most people believe that people with depression are dangerous to others.
*Most people believe that people with depression are dangerous to themselves.
Most people believe that it is best to avoid people with depression so that you don't become depressed yourself.
Most people believe that people with depression are unpredictable.
Most people believe that depression is not a real illness
If they had depression, most people would not tell anyone.
Most people would not employ someone they knew had been depressed.
Most people would not vote for a politician they knew had been depressed

* Revised from the original DSS items (“[Most people believe] People with depression are dangerous”) to distinguish danger to self from danger to others.

Table 6

Depression Social Distance Scale overview and items.

Construct measured	Depression stigma, expressed in terms of self-reported willingness to be in specific social situations and relationships with people who have depression
Response format	4-point Likert scale: Definitely unwilling; Probably unwilling; Probably willing; Definitely willing
Instruction	Please indicate how you would feel about being in the following social situations with someone who has depression. If you are not sure, please take your best guess about how you think you would feel. There are no right or wrong answers.
Items	How would you feel about renting a room in your home to someone with depression?
	How would you feel about having someone with depression as a coworker?
	How would you feel about having someone with depression as a neighbor?
	How would you feel about having someone with depression as the caretaker of your children for a couple of hours?
	How would you feel about having one of your children marry someone with depression?
	How would you feel about introducing someone with depression to someone you are friendly with?
	How would you feel about recommending someone with depression for a job working for a friend of yours?

Table 7

List of measures for Studies 1 and 2.

Measure	Items Administered
Illness Perception Questionnaire-Revised, modified for depression	43
Consequences scale	*6*
Timeline scale	*20*
Cure/Control scale	*11*
Coherence scale (Study 2 only)	*6*
Depression Stigma Scale	20
Personal Stigma scale	*10*
Perceived Stigma scale	*10*
Depression Social Distance Scale	7
Network Analysis Questionnaire *(Study 2 only)*	49
Clinical History Questionnaire	5–19*
Demographic Questionnaire	6
Patient Health Questionnaire-9	9
Impression of Hypotheses	1
TOTAL	190–204*

* Number of clinical follow-up items was determined by individuals' responses.

Table 8

Sequence for entering predictor variables into initial multivariate and mediation analyses.

Sequence	Predictors entered
1	Demographic predictors: Age, gender, education level, household income*
2	Clinical predictors: Whether respondent reports a history of depression; Whether respondent reports a history of depression in a close other
3	IV: Strength of biological causal belief(s)**

*Race/ethnicity data were collected but not included because representation levels for minority groups were too low to support meaningful examination of group differences.

** In the initial multivariate analyses, either biological changes in the brain or genes/heredity was entered as the sole predictor. In the mediation analyses, these two predictors were entered together.

Table 9

Sequence for entering predictor variables into moderation analyses.

Sequence	Predictors entered
1	Demographic predictors: Age, gender, education level, household income*
2	Focal predictors: Strength of biological causal belief (either *biological changes in the brain* or *genes/heredity*), believe self had depression, believe close other had depression
3	First interaction term: Causal belief x first familiarity variable (Either *respondent's history of depression* or *history of depression in respondent's close other*)
4	Second interaction term: Causal belief x second familiarity variable

*Race/ethnicity data were collected but not included because representation levels for minority groups were too low to support meaningful examination of group differences.

Table 10

Correlations among selected predictor and outcome variables.

Measure	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Age	.035	-.044	-.059	-.047	.089	.089	-.002	.142*	-.034	.184**	-.006	-.020	.158**
2. Gender		.098	-.002	-.112*	-.102	-.156**	-.202**	-.127*	-.140*	-.011	.005	-.141*	.008
3. Edu. (4-year degree)			.279**	-.045	.006	-.067	.033	-.002	-.005	.033	.026	.016	.064
4. Household income				-.086	.076	-.011	.149**	.036	.008	-.024	.059	.107	.055
5. Believe self had depression (MDD)					.298**	.218**	.265**	.124*	.158**	.071	-.148**	.124*	-.164**
6. Believe close other had MDD						.089	.168**	.237**	.219**	.105	-.153**	.155**	-.203**
7. Cause: Biological changes in the brain							.363**	.335**	.312**	.270**	-.250**	.140*	-.116*
8. Cause: Heredity/ genes								.248**	.343**	.051	-.168**	.091	-.113*
9. Consequences									.450**	.241**	-.324**	.091	-.130*
10. Duration										-.050	-.387**	.111	-.207**
11. Treatability											-.193**	.077	-.032
12. Personal stigma												.303**	.556**
13. Perceived stigma													.162**
14. Desired social distance													

* Correlation is significant at the .05 level (2-tailed). ** Correlation is significant at the .01 level (2-tailed).

Table 11a

Multivariate analyses of neurobiological causal beliefs about depression as a predictor of three prognostic beliefs.

	Consequences		Duration		Treatability	
Predictors	β	sr^2	β	sr^2	β	sr^2
Demographics						
Gender	-.061	.004	-.075	.005	.028	.001
Age	.103	.010	-.070	.005	.156**	.023
Education level (4-year degree)	.023	.000	.021	.000	.059	.003
Household income	.028	.001	-.014	.000	-.033	.001
Clinical variables						
Believe self had depression	.006	.000	.032	.001	.007	.000
Believe a close other had depression	.191**	.033	.184**	.030	.077	.005
Biological causal belief						
Neurobiological causes	.298***	.081	.285***	.075	.258***	.061

β values are standardized regression coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 11b

Multivariate analyses of genetic causal beliefs about depression as a predictor of three prognostic beliefs.

	Consequences		Duration		Treatability	
Predictors	β	sr^2	β	sr^2	β	sr^2
Demographics						
Gender	-.071	.005	-.058	.003	`-.007	.000
Age	.132*	.017	-.047	.002	.182**	.032
Education level (4-year degree)	.008	.000	.004	.000	.050	.002
Household income	.004	.000	-.056	.003	-.033	.001
Clinical variables						
Believe self had depression	.019	.000	.016	.000	.052	.002
Believe a close other had depression	.183**	.030	.167**	.025	.074	.005
Biological causal belief						
Genes/heredity	.201** *	.036	.307** *	.082	.029	.001

β values are standardized regression coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 11c

Multivariate analyses of neurobiological causal beliefs about depression as a predictor of three stigma variables.

	Personal stigma		Perceived stigma		Desired social distance	
Predictors	β	sr^2	β	sr^2	β	sr^2
Demographics						
Gender	-.054	.003	-.104	.010	-.047	.002
Age	.034	.001	-.028	.001	.189**	.034
Education level (4-year degree)	-.004	.000	.005	.000	.052	.003
Household income	.064	.004	.101	.009	.062	.003
Clinical variables						
Believe self had depression	-.058	.003	.064	.003	-.072	.004
Believe a close other had depression	-.129*	.015	.111	.011	-.199**	.035
Biological causal belief						
Neurobiological causes	-.238***	.052	.102	.010	-.102	.010

β values are standardized regression coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 11d

Multivariate analyses of genetic causal beliefs about depression as a predictor of three stigma variables.

	Personal stigma		Perceived stigma		Desired social distance	
Predictors	β	sr^2	β	sr^2	β	sr^2
Demographics						
Gender	-.048	.002	-.116*	.013	-.048	.002
Age	.011	.000	-.017	.000	.180**	.032
Education level (4-year degree)	.010	.000	.000	.000	0.059	.003
Household income	.082	.006	.103	.009	.070	.004
Clinical variables						
Believe self had depression	-.070	.004	.083	.006	-.073	.005
Believe a close other had depression	-.119*	.012	.110	.011	-.194	.033
Biological causal belief						
Genes/heredity	-.152*	.020	.010	.000	-.084	.006

β values are standardized regression coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 12

Analysis of familiarity with depression as a moderator of the relationship between genetic causal beliefs and personal stigma.

	β	*t*
Demographics		
Age	0.000	0.004
Gender	-0.048	-0.846
Education	-0.012	-0.213
Household Income	0.094	1.614
Main Effects		
Genetic causal belief	0.059	0.622
Believe self had depression	-0.052	-0.878
Believe close other had depression	-0.127*	-2.164
Product Terms		
Genetic causal belief x Believe self had depression	-0.154*	-2.133
Genetic causal belief x Believe close other had depression	-0.152	-1.650

β values are standardized regression coefficients. * $p < .05$.

Table 13

Analysis of familiarity with depression as a moderator of the relationship between genetic causal beliefs and desired social distance.

	β	*t*
Demographics		
Age	0.166**	3.049
Gender	-0.047	-0.854
Education	0.035	0.625
Household Income	0.085	1.482
Main Effects		
Genetic causal belief	0.151	1.623
Believe self had depression	-0.055	-0.933
Believe close other had depression	-0.206***	-3.590
Product Terms		
Genetic causal belief x Believe self had depression	-0.139	-1.962
Genetic causal belief x Believe close other had depression	-0.195*	-2.168

β values are standardized regression coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$

Table 14

Analysis of familiarity with depression as a moderator of the relationship between genetic causal beliefs and perceived stigma.

	β	*t*
Demographics		
Age	-0.021	-0.382
Gender	-0.117*	-2.066
Education	-0.020	-0.341
Household Income	0.112	1.914
Main Effects		
Genetic causal belief	0.163	1.702
Believe self had depression	0.097	1.612
Believe close other had depression	0.112	1.895
Product Terms		
Genetic causal belief x Believe self had depression	-0.194**	-2.660
Genetic causal belief x Believe close other had depression	-0.048	-0.519

β values are standardized regression coefficients. * $p < .05$. ** $p < .01$.

Table 15

Analysis of familiarity with depression as a moderator of the relationship between neurobiological causal beliefs and desired social distance.

	β	*t*
Demographics		
Age	0.182**	3.358
Gender	-0.043	-0.783
Education	0.055	0.978
Household Income	0.075	1.331
Main Effects		
Neurobiological causal belief	0.113	1.168
Believe self had depression	-0.056	-0.963
Believe close other had depression	-0.198**	-3.475
Product Terms		
Neurobiological causal belief x Believe self had depression	-0.177*	-2.449
Neurobiological causal belief x Believe close other had depression	-0.129	-1.339

β values are standardized regression coefficients. * $p < .05$. ** $p < .01$.

Table 16

IPQ-R Coherence scale overview and items.

Construct measured	Respondents' beliefs about how clearly he or she understands depression
Response format	5-point Likert scale: Strongly disagree; Disagree; Neither agree nor disagree; Agree Strongly agree
Instruction	We are interested in your own personal views about depression. Please indicate how much you agree or disagree with the following statements:
Items	The symptoms of depression are puzzling to me
	I'm not sure I would recognize symptoms of depression.
	Depression is a mystery to me.
	I don't understand depression.
	Depression doesn't make much sense to me.
	I have a clear picture or understanding of what depression is.

Table 17

Mean strength ratings of causal links in the consensus network (N = 319).

Rank	Link entered	Mean strength	Percent at IAC ≥ 2
1	Trauma causes stress or worry	2.66	98%
2	Problems with work or finances cause stress or worry	2.60	95
3	Depression causes stress or worry	2.60	93
4	Depression causes social relationship problems	2.56	92
5	Physical illness causes stress or worry	2.50	91
6	Social relationship problems cause stress or worry	2.48	90
7	Trauma causes depression	2.47	90
8	Depression causes problems with work or finances	2.42	89
9	Stress or worry cause social relationship problems	2.38	87
10	Biological changes in the brain cause depression	2.37	84
11	Trauma causes problems with work or finances	2.35	84
12	Stress or worry cause depression	2.34	83
13	Trauma causes social relationship problems	2.34	82
14	Physical illness causes problems with work or finances	2.34	81
15	Biological changes in the brain cause stress or worry	2.32	79
16	Problems with work or finances cause social relationship problems	2.31	76
17	Problems with work or finances cause depression	2.30	75

Table 18

Key variables on which low and high coherence groups significantly differed

	Low coherence group				High coherence group			
	Number	Percent	M	SD	Number	Percent	M	SD
Gender		100				100		
Women	37	50			59	73.8		
Men	37	50			21	26.3		
Believe self had depression		100				100		
Yes	23	31.1			59	73.8		
No or Don't know	51	68.9			21	26.3		
Believe close other had depression		100				100		
Yes	34	45.9			63	78.8		
No or Don't know	40	54.1			17	21.3		
Coherence (IPQ-R)		100	13.49	2.10		100	24.81	0.39
Cause: Biological changes in the brain (IPQ-R)		100	3.82	0.93		100	4.55	0.71
Duration (IPQ-R)		97.3	36.58	5.02		97.5	41.91	7.49
Desired Social Distance		98.6	16.38	3.72		98.8	13.05	5.35

Table 19

Mean strength ratings of causal links in the low coherence group (*n* = 74).

Rank	Link entered	Mean strength	Percent at IAC ≥ 2
1	Trauma causes stress or worry	2.42	93%
2	Social relationship problems cause stress or worry	2.38	88
3	Depression causes social relationship problems	2.35	84
4	Problems with work or finances cause stress or worry	2.34	81
5	Depression causes stress or worry	2.31	77
6	Physical illness causes stress or worry	2.31	76
7	Problems with work or finances cause social relationship problems	2.30	70
8	Trauma causes depression	2.26	70

Table 20

Mean strength ratings of causal links in the high coherence group (n = 80).

Rank	Link entered	Mean strength	Percent at IAC ≥ 2
1	Trauma causes stress or worry	2.83	100%
2	Depression causes stress or worry	2.76	100
3	Problems with work or finances cause stress or worry	2.73	99
4	Trauma causes depression	2.66	99
5	Depression causes social relationship problems	2.65	98
6	Physical illness causes stress or worry	2.63	98
7	Biological changes in the brain cause depression	2.58	95
8	Depression causes problems with work or finances	2.56	95
9	Social relationship problems cause stress or worry	2.51	94
10	Depression causes biological changes in the brain	2.51	86
11	Stress or worry causes depression	2.50	85
12	Physical illness causes problems with work or finances	2.46	84
13	Problems with work or finances cause depression	2.46	81
14	Stress or worry causes social relationship problems	2.46	79
15	Biological changes in the brain cause stress or worry	2.45	78
16	Trauma causes social relationship problems	2.44	78
17	Trauma causes biological changes in the brain	2.38	75
18	Trauma causes problems with work or finances	2.35	74
19	Social relationship problems cause depression	2.35	74
20	Biological changes in the brain cause social relationship problems	2.34	70

Figures

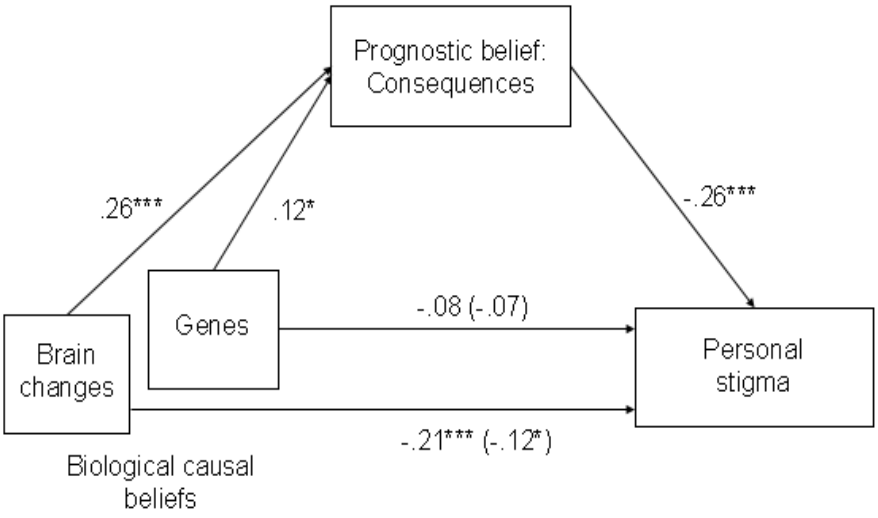

Figure 1a

Beliefs about depression's consequences as a mediator of the inverse relationship between biological causal beliefs and personal stigma.

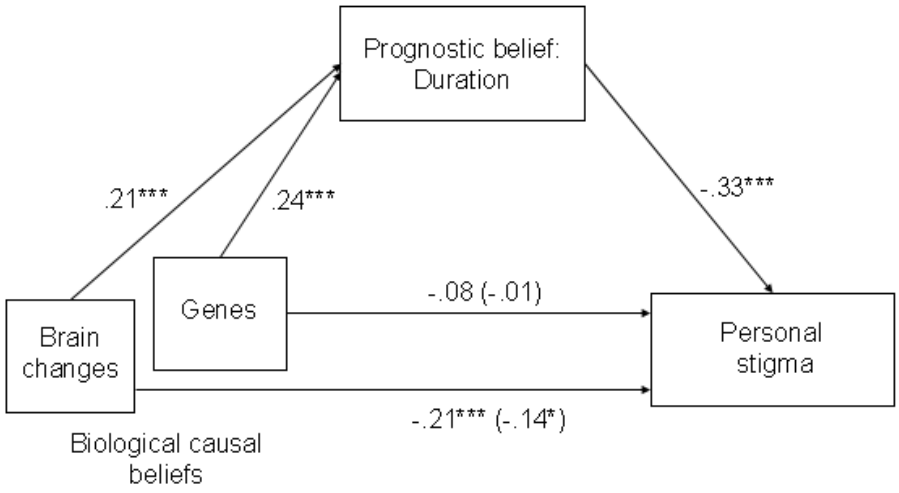

Figure 1b

Beliefs about depression's duration as a mediator of the inverse relationship between biological causal beliefs and personal stigma.

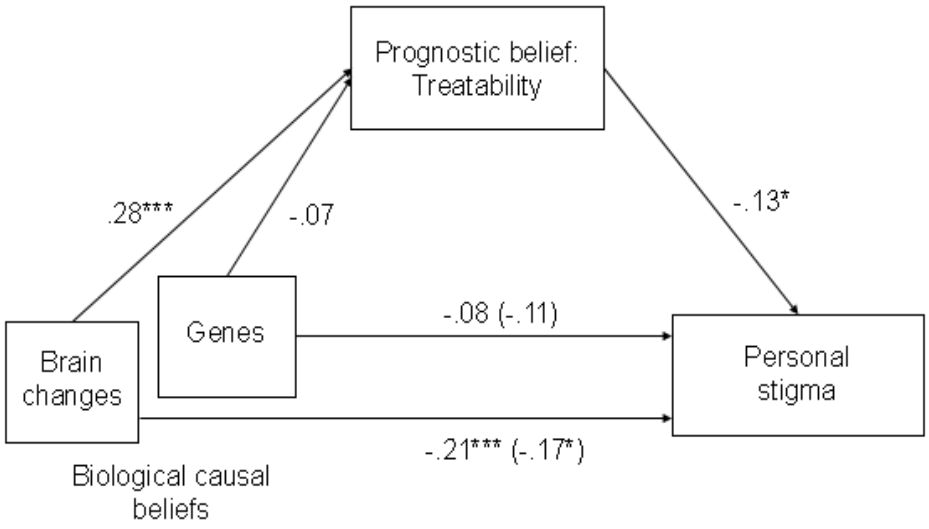

Figure 1c

Beliefs about depression's treatability as a mediator of the inverse relationship between biological causal beliefs and personal stigma.

Figures 1a–c

Mediation models. The models show standardized regression coefficients for relationships between biological causal beliefs and personal stigma, mediated by each of three prognistic beliefs about depression. Standardized regression coefficients (β) between biological causal beliefs and depression stigma, controlling for the specific prognostic belief analyzed are in parentheses. *$p < .05$, **$p < .01$, *** $p < .001$

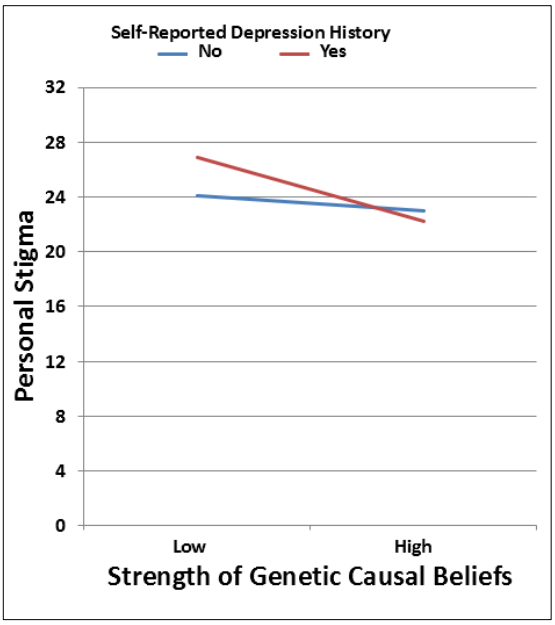

Figure 2

Self-reported history of depression as a moderator of the relationship between genetic causal beliefs and personal stigma.

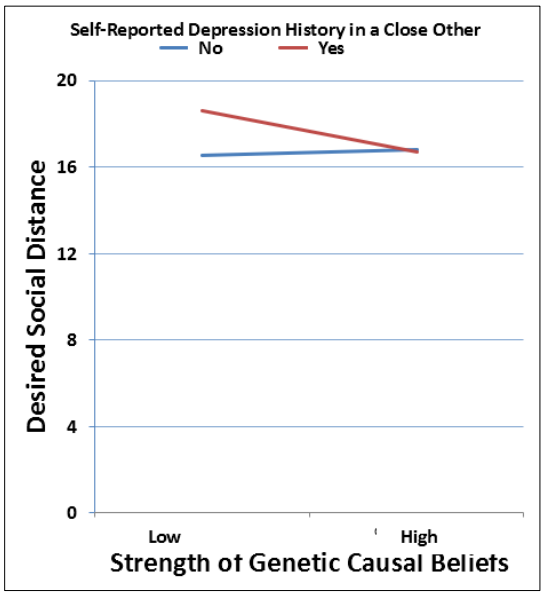

Figure 3

History of depression in a close other as a moderator of the relationship between genetic causal beliefs and desired social distance.

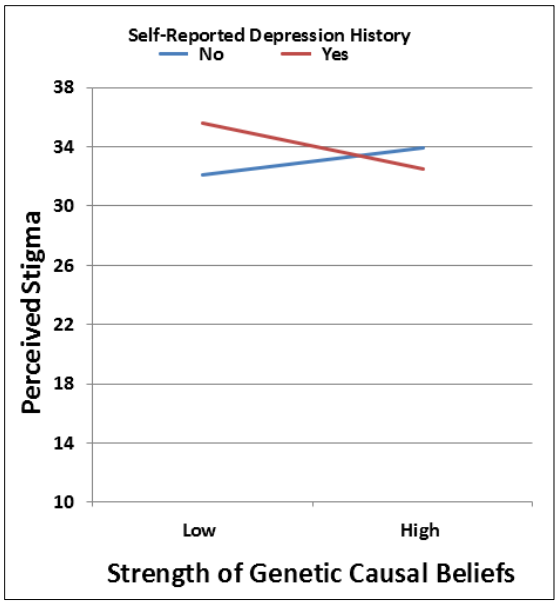

Figure 4

Self-reported history of depression as a moderator of the relationship between genetic causal beliefs and perceived stigma.

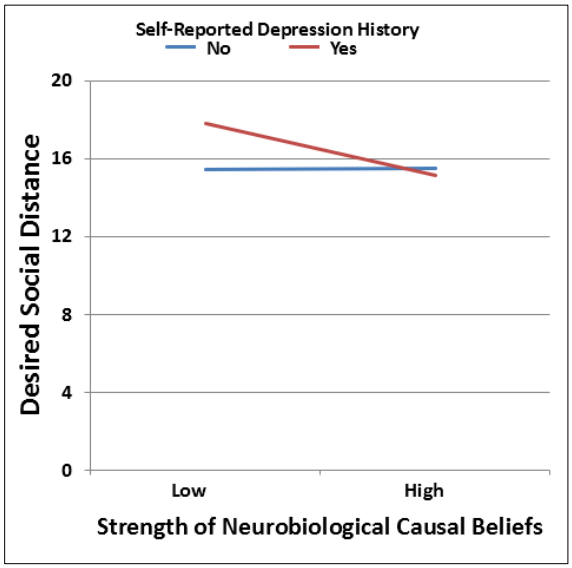

Figure 5

Self-reported history of depression as a moderator of the relationship between neurobiological causal beliefs and desired social distance.

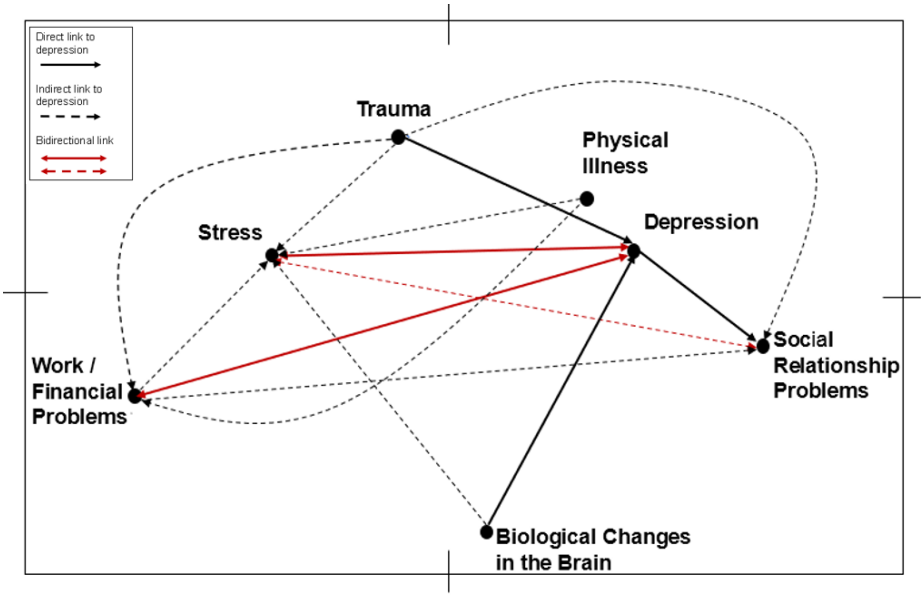

Figure 6

Consensus network of the perceived causal structure of depression.

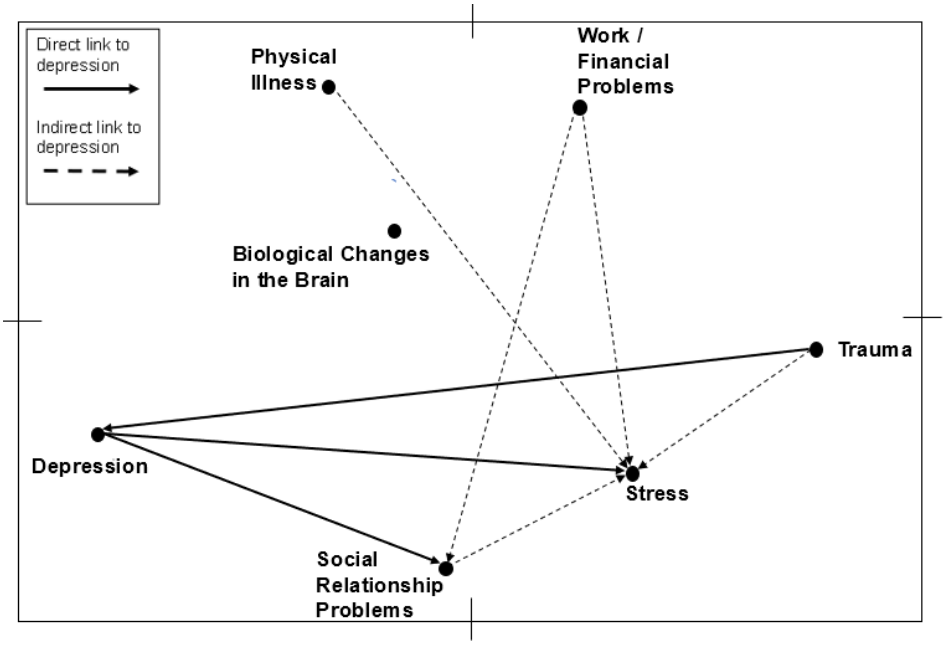

Figure 7

Network of the perceived causal structure of depression for participants with low illness coherence (n = 74).

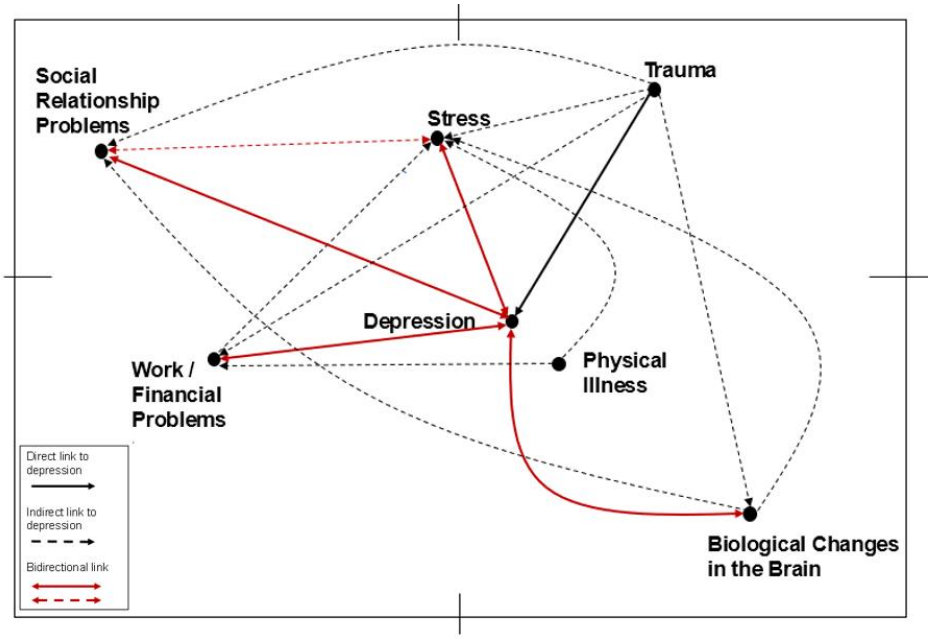

Figure 8

Network of the perceived causal structure of depression for participants with high illness coherence (n = 80).

Appendix: Survey

Text and headings appearing in square brackets below are included to aid the reader's navigation. They were not included in the survey presented to participants.

As described in the Measures section for Study 1, the order of all measures and items was held constant except for the order of the Network Analysis Questionnaire items. These were presented to each participant in random order, one item at a time.

Consent

You are invited to participate in a research study by the Rutgers University Psychology Department, conducted by Sarah Mann, M.S., under the supervision of Dr. Richard Contrada. Before you agree to participate in the study, you should have enough information about it to make an informed decision.

The purpose of the study is to understand specific beliefs people hold about mental illness. Participating in the study involves completing a questionnaire made up of rating scales and multiple-choice questions. Participation takes place in one session of approximately 10-15 minutes. The study is open to people who are at least 18 years old, are citizens or legal residents of the United States, and are proficient at reading and understanding English.

This research involves no physical or emotional risks to you, and it offers no direct benefit to you. You may withdraw from the study at any time without penalty by closing your web browser. Participants in this study are anonymous, meaning there is no record of any participant's identity associated with his or her responses to the questionnaire. The research team and the Institutional Review Board at Rutgers

University are the only parties allowed to see the data, except as may be required by law. If a report of this study is published or results are presented at a professional conference, only group results will be stated.

You will receive $0.40 (40 cents) for participating in this research. If you withdraw from the study before completing it, you will not receive compensation and there is no penalty to you. Compensation will be given within one week of participating. Participation in this study is voluntary. You many choose not to participate, and you may withdraw without penalty at any time during the study procedures.

Research designs often require that the full intent of the study not be explained prior to participation, in which case an explanation is provided later. By indicating below that you wish to take part in this project, you agree to participate with this knowledge, and agree not to share the nature of your participation with other potential participants until the study is completed.

If you have questions at any time about the study, or if you wish to receive a summary of the results, you may contact the researcher:

Sarah Mann, M.S.
53 Avenue E
Piscataway, NJ 08854
email: sarah.mann@rutgers.edu

Or her academic advisor:

Dr. Richard Contrada
53 Avenue E
Piscataway, NJ 08854
email: contrada@rci.rutgers.edu

If you have questions about your rights as a research subject, you may contact the IRB Administrator:

Arts and Sciences IRB New Brunswick

Office of Research Regulatory Affairs, Rutgers University
335 George St.
Liberty Plaza, 3rd Floor, Suite 3200
New Brunswick, NJ 08901
tel: 732-235-9806

IRB Protocol Number: E17-471
Approved 2/23/17

If you have read the procedure described above, are 18 years of age or older, are a U.S. citizen or legal resident, can read and understand English proficiently, and voluntarily agree to participate in this study, select "Yes." Otherwise, select "No." When you have made your selection, click the forward arrows to proceed.

Do you wish to participate in this study?

Yes No

[Introductory Description of Clinical Depression]

Many of the following questions ask about your thoughts and beliefs about clinical depression. As you answer them, please keep in mind that clinical depression differs from brief, passing feelings of sadness, irritability, or low mood. Here is a short description of clinical depression:

An episode of clinical depression involves several types of changes that are present for most of the day, nearly every day, for at least two weeks. These changes include:

- depressed mood (feeling sad, empty, or irritable), and/or
- loss of interest or pleasure in doing things that the person would normally find interesting or enjoyable.

People with clinical depression may also experience:

- changes in sleep,
- changes in appetite and weight,
- having less energy than usual,
- having trouble concentrating, and
- frequent thoughts about their own worthlessness, hopelessness, or guilt, and/or about wishing to no longer be alive.

[Illness Perception Questionnaire–Revised (IPQ-R), Modified for Depression]

We are interested in what you consider to be the factors that cause depression. There are no incorrect answers, because we are seeking information about <u>your own</u> thoughts and beliefs. Please respond based on <u>your own</u> views, rather than what others, including doctors or family members, may have suggested to you.

[Cause scale]

Below is a list of possible causes for depression. Please indicate how much you agree or disagree that each one is likely to be a cause of depression.

Strongly disagree —— Disagree —— Neither agree nor disagree ——Agree —— Strongly agree

---

Heredity or genes—it runs in families	Biological changes in the person's brain
Problems resulting from a physical illness or medical complications	The person's behavior
Financial problems or worries	The person's attitude - thinking about life negatively
Chance or bad luck	Family problems or worries
The death of someone close	Overwork

The person's emotional state - feeling down, lonely, or empty

Aging

Problems resulting from alcohol or drug use

Problems with friendships or social relations (other than family)

Accident or injury

The person's personality

Experiencing a traumatic event

Having a difficult childhood

Being unemployed or underemployed

[Consequences scale]

[Instructions and response format for all IPQ-R scales, unless otherwise indicated:]

We are interested in <u>your own personal views</u> about depression. Please indicate how much you agree or disagree with the following statements.

Strongly disagree —— Disagree —— Neither agree nor disagree ——Agree —— Strongly agree

---

1. Depression is a serious condition.

2. Depression has major consequences in the lives of people who have it.

3. Depression strongly affects the way others see those who have it.

4. Depression does not have much effect on the lives of people who have it.

5. Depression has serious financial consequences.

6. Depression causes difficulties for people close to those who have it.

[Timeline scale–Untreated depression]

1. When it is not treated, depression usually lasts a short time.

2. Without treatment, depression is likely to be permanent rather than temporary.

3. Untreated depression usually lasts for a long time.

4. Depression usually passes quickly without treatment.

5. People with depression who don't receive treatment generally have it for the rest of their lives.

6. Without treatment, depression will improve in time.

7. The symptoms of untreated depression change a great deal from day to day.

8. Untreated depression goes through cycles in which it alternates between getting better and getting worse.

9. Depression that is not treated is very unpredictable.

10. Someone who has experienced depression and recovered without treatment is likely to experience additional depressive episodes in the future.

[Timeline scale–Treated depression]

1. With treatment, depression usually lasts a short time.

2. Depression is likely to be permanent rather than temporary, even when it is treated.

3. Even with treatment, depression usually lasts for a long time.

4. Depression usually passes quickly when it is treated.

5. People with depression generally have it for the rest of their lives, even if they receive treatment.

6. Depression that is treated will improve in time.

7. For a person receiving treatment for depression, symptoms still change a great deal from day to day.

8. With treatment, depression goes through cycles in which it alternates between getting better and getting worse.

9. Depression is very unpredictable, even if a person receives treatment.

10. Someone who has been treated for depression before and recovered is likely to experience additional depressive episodes in the future

[Cure/Control scale]

1. There is a lot that people with depression can do on their own to reduce their symptoms.

2. What people with depression do can determine whether their depression gets better.

3. Nothing that people with depression do will affect their condition.

4. The course of people's depression depends on them.

5. People with depression have the power to influence their condition.

6. The actions of people with depression will have no effect on whether their depression gets better.

7. There is very little that can be done to help with depression.

8. Treatment is effective in curing depression.

9. Nothing can be done to improve depression.

10. Treatment can reduce the symptoms of depression.

11. The negative effects of depression can be avoided with treatment.

12. What types of treatment do you believe can be effective for depression? Please select all that apply.

Medications	Changes in diet
Psychotherapy or counseling	Reading/listening to self-help materials
Alternative medicine or naturopathy	Other treatment(s) (please specify)
Religious or spiritually based approaches	No treatment can be effective for depression
Exercise	

[Coherence scale]

1. The symptoms of depression are puzzling to me.

2. I'm not sure I would recognize symptoms of depression.

3. Depression is a mystery to me.

4. I don't understand depression.

5. Depression doesn't make much sense to me.

6. I have a clear picture or understanding of what depression is.

[Depression Social Distance Scale]

Please indicate how you would feel about being in the following social situations with someone who has depression. If you are not sure, please take your best guess about how you think you would feel. There are no right or wrong answers.

Definitely unwilling—— Probably unwilling—— Probably willing——Definitely willing

---

1. How would you feel about . . . renting a room in your home to someone with depression?
2. . . . having someone with depression as a coworker?
3. . . . having someone with depression as a neighbor?
4. . . . having someone with depression as the caretaker of your children for a couple of hours?
5. . . . having one of your children marry someone with depression?
6. . . . introducing someone with depression to someone you are friendly with?
7. . . . recommending someone with depression for a job working for a friend of yours?

[Depression Stigma Scale]

The following items ask for your own opinions and beliefs about depression. There are no right or wrong answers. Please indicate how much you agree or disagree with each

statement.

Strongly disagree —— Disagree —— Neither agree nor disagree ——Agree —— Strongly agree

---

[Personal Stigma subscale]

1. People with depression could snap out of it if they wanted.

2. Depression is a sign of personal weakness.

3. People with depression are dangerous to others.

4. People with depression are dangerous to themselves.

5. It is best to avoid people with depression so you do not become depressed yourself.

6. People with depression are unpredictable.

7. Depression is not a real illness.

8. If I had depression I would not tell anyone.

9. I would not employ someone if I knew they had been depressed.

10. I would not vote for a politician if I knew they had been depressed.

[Perceived Stigma subscale]

1. Most people believe that people with depression could snap out of it if they wanted.

2. Most people believe that depression is a sign of personal weakness.

3. Most people believe that people with depression are dangerous to others.

4. Most people believe that people with depression are dangerous to themselves.

5. Most people believe that it is best to avoid people with depression so that you don't become depressed yourself.

6. Most people believe that people with depression are unpredictable.

7. Most people believe that depression is not a real illness.

8. If they had depression, most people would not tell anyone.

9. Most people would not employ someone they knew had been depressed.

10. Most people would not vote for a politician they knew had been depressed.

[Network Analysis Questionnaire]

The following items ask you to consider how likely it is that experiencing a particular type of event (like becoming physically ill) could cause someone to experience another specific event (like having problems with work or finances). Once again, we are interested in your own beliefs about these relationships. There are no right or wrong answers.

Never, or only rarely—— Possibly, or only sometimes—— Definitely, or most of the time

---

How likely is STRESS OR WORRY to cause BIOLOGICAL CHANGES IN THE BRAIN?

How likely is STRESS OR WORRY to cause PROBLEMS WITH SOCIAL RELATIONSHIPS?

How likely is STRESS OR WORRY to cause PROBLEMS WITH WORK OR FINANCES?

How likely is STRESS OR WORRY to cause PHYSICAL ILLNESS?

How likely is STRESS OR WORRY to cause EXPERIENCING A TRAUMATIC EVENT?

How likely is STRESS OR WORRY to cause DEPRESSION?

How likely are BIOLOGICAL CHANGES IN THE BRAIN to cause STRESS OR WORRY?

How likely are BIOLOGICAL CHANGES IN THE BRAIN to cause PROBLEMS WITH SOCIAL RELATIONSHIPS?

How likely are BIOLOGICAL CHANGES IN THE BRAIN to cause PHYSICAL ILLNESS?

How likely are BIOLOGICAL CHANGES IN THE BRAIN to cause PROBLEMS WITH WORK OR FINANCES?

How likely are BIOLOGICAL CHANGES IN THE BRAIN to cause EXPERIENCING A TRAUMATIC EVENT?

How likely are BIOLOGICAL CHANGES IN THE BRAIN to cause DEPRESSION?

How likely are PROBLEMS WITH SOCIAL RELATIONSHIPS to cause STRESS OR WORRY?

How likely are PROBLEMS WITH SOCIAL RELATIONSHIPS to cause BIOLOGICAL CHANGES IN THE BRAIN?

How likely are PROBLEMS WITH SOCIAL RELATIONSHIPS to cause PHYSICAL ILLNESS?

How likely are PROBLEMS WITH SOCIAL RELATIONSHIPS to cause PROBLEMS WITH WORK OR FINANCES?

How likely are PROBLEMS WITH SOCIAL RELATIONSHIPS to cause EXPERIENCING A TRAUMATIC EVENT?

How likely are PROBLEMS WITH SOCIAL RELATIONSHIPS to cause DEPRESSION?

How likely is PHYSICAL ILLNESS to cause STRESS OR WORRY?

How likely is PHYSICAL ILLNESS to cause BIOLOGICAL CHANGES IN THE BRAIN?

How likely is PHYSICAL ILLNESS to cause PROBLEMS WITH SOCIAL RELATIONSHIPS?

How likely is PHYSICAL ILLNESS to cause PROBLEMS WITH WORK OR FINANCES?

How likely is PHYSICAL ILLNESS to cause EXPERIENCING A TRAUMATIC EVENT?

How likely is PHYSICAL ILLNESS to cause DEPRESSION?

How likely are PROBLEMS WITH SOCIAL RELATIONSHIPS to cause STRESS OR WORRY?

How likely are PROBLEMS WITH WORK OR FINANCES to cause BIOLOGICAL CHANGES IN THE BRAIN?

How likely are PROBLEMS WITH WORK OR FINANCES to cause PROBLEMS WITH SOCIAL RELATIONSHIPS?

How likely are PROBLEMS WITH WORK OR FINANCES to cause PHYSICAL ILLNESS?

How likely are PROBLEMS WITH WORK OR FINANCES to cause EXPERIENCING A TRAUMATIC EVENT?

How likely are PROBLEMS WITH WORK OR FINANCES to cause DEPRESSION?

How likely is EXPERIENCING A TRAUMATIC EVENT to cause STRESS OR WORRY?

How likely is EXPERIENCING A TRAUMATIC EVENT to cause BIOLOGICAL CHANGES IN THE BRAIN?

How likely is EXPERIENCING A TRAUMATIC EVENT to cause PROBLEMS WITH SOCIAL RELATIONSHIPS?

How likely is EXPERIENCING A TRAUMATIC EVENT to cause PHYSICAL ILLNESS?

How likely is EXPERIENCING A TRAUMATIC EVENT to cause PROBLEMS WITH WORK OR FINANCES?

How likely is EXPERIENCING A TRAUMATIC EVENT to cause DEPRESSION?

How likely is DEPRESSION to cause STRESS OR WORRY?

How likely is DEPRESSION to cause BIOLOGICAL CHANGES IN THE BRAIN?

How likely is DEPRESSION to cause PROBLEMS WITH SOCIAL RELATIONSHIPS?

How likely is DEPRESSION to cause PHYSICAL ILLNESS?

How likely is DEPRESSION to cause PROBLEMS WITH WORK OR FINANCES?

How likely is DEPRESSION to cause EXPERIENCING A TRAUMATIC EVENT?

[Clinical History Questionnaire]

Please keep in mind the features of clinical depression as you answer the questions in this section:

An episode of clinical depression involves several types of changes that are present for most of the day, nearly every day, for at least two weeks. These changes include:

- depressed mood (feeling sad, empty, or irritable), and/or
- loss of interest or pleasure in doing things that the person would normally find interesting or enjoyable.

People with clinical depression may also experience:

- changes in sleep,
- changes in appetite and weight,
- having less energy than usual,
- having trouble concentrating, and
- frequent thoughts about their own worthlessness, hopelessness, or guilt, and/or about wishing to no longer be alive.

1. Based on this description, do you believe you have experienced an episode of clinical depression

Yes, I think I've experienced an episode of clinical depression.

No, I don't think I've experienced an episode of clinical depression.

I'm not sure whether I've experienced an episode of clinical depression.

2. Have you ever been diagnosed with depression by a professional?

Yes No Don't know

3. Some types of professionals who might diagnose depression include a psychotherapist or counselor, psychiatrist or other medical doctor, social worker, or a clergy member or religious leader, among others. What type(s) of professional diagnosed you with depression? Please mark all that apply.

Psychotherapist or counselor	Social worker
Psychiatrist	Clergy or religious leader
Medical doctor who is not a psychiatrist	Other (please specify)

4. Have you ever received or used any kind of treatment for depression?

Some examples of the ways depression is treated include psychotherapy or counseling, medication, religious or spiritual approaches, alternative medicine or naturopathy, changes in diet and/or exercise, or use of self-help materials.

Definitely yes

Probably yes

Probably not

Definitely not

5. What type(s) of treatment for depression have you received or used? Please mark all that apply.

Medication	Changes in diet
Psychotherapy or counseling	Exercise
Religious or spiritual approaches	Reading/listening to self-help materials
Alternative medicine or naturopathy	Other treatment(s) (please specify)

6. Do you believe any treatment(s) you received or used for depression was/were helpful?

Definitely yes

Probably yes

Probably not

Definitely not

7. Which treatment(s) for depression do you believe were MOST HELPFUL to you? (If you used or received only one type of treatment, please skip this question.)

Medication	Changes in diet
Psychotherapy or counseling	Exercise
Religious or spiritual approaches	Reading/listening to self-help materials
Alternative medicine or naturopathy	Other treatment(s) (please specify

8. Which treatment(s) for depression do you believe were LEAST HELPFUL to you? (If you used or received only one type of treatment, please skip this question.)

Medication	Changes in diet
Psychotherapy or counseling	Exercise
Religious or spiritual approaches	Reading/listening to self-help materials
Alternative medicine or naturopathy	Other treatment(s) (please specify

9. Do you believe someone close to you has experienced an episode of clinical depression (for example, a spouse or romantic partner, parent, sibling, or close friend)?

As a reminder, the same description of clinical depression that was provided earlier is below:

An episode of clinical depression involves several types of changes that are present for most of the day, nearly every day, for at least two weeks. These changes include:

- depressed mood (feeling sad, empty, or irritable), and/or
- loss of interest or pleasure in doing things that one would normally find interesting or enjoyable.

Clinical depression may also involve:

- changes in sleep,
- changes in appetite and weight,
- having less energy than usual,
- having trouble concentrating,
- frequent thoughts about one's own worthlessness, hopelessness, or guilt,

- and/or thoughts about wishing to no longer be alive.

Yes, I believe one person close to me has had depression.

Yes, I believe more than one person close to me has had depression.

No, I don't believe anyone close to me has had depression.

I don't know whether I believe anyone close to me has had depression.

10. Thinking about the person or people close to you who may have had depression, were any of these people diagnosed with depression by a professional?

Some types of professionals who might diagnose depression include a psychotherapist or counselor, psychiatrist or other medical doctor, social worker, or a clergy member or religious leader, among others.

Yes No Don't know

11. What kind(s) of professional(s) provided a diagnosis of depression to someone close to you? (Please mark all that apply.)

Psychotherapist or counselor	Social worker
Psychiatrist	Clergy or religious leader
Medical doctor who is not a psychiatrist	Other (please specify)

12. Did anyone close to you who had depression receive/ use any type of treatment for it?

Some examples of ways that depression is treated include psychotherapy or counseling, medication, religious or spiritual approaches, alternative medicine or naturopathy, changes in diet and/or exercise, or use of self-help materials.

Yes No Don't know

13. What kinds of treatment for depression did people close to you receive or use? (Please mark all that apply.)

Medication	Changes in diet
Psychotherapy or counseling	Exercise
Religious or spiritual approaches	Reading/listening to self-help materials
Alternative medicine or naturopathy	Other treatment(s) (please specify)

14. Do you believe that any of the treatment(s) that anyone close to you received for depression was/were helpful?

Definitely yes

Probably yes

Probably not

Definitely not

15. Which type(s) of treatment for depression do you think was/were MOST HELPFUL to someone close to you? (If you indicated only one type of treatment used or received by someone close to you, please leave this item blank.)

Medication	Changes in diet
Psychotherapy or counseling	Exercise
Religious or spiritual approaches	Reading/listening to self-help materials
Alternative medicine or naturopathy	Other treatment(s) (please specify)

16. Which type(s) of treatment for depression do you think was/were LEAST HELPFUL to someone close to you? (If you indicated only one type of treatment used or received by someone close to you, please leave this item blank.)

Medication	Changes in diet
Psychotherapy or counseling	Exercise
Religious or spiritual approaches	Reading/listening to self-help materials
Alternative medicine or naturopathy	Other treatment(s) (please specify)

17. Do you believe you have ever experienced any psychological disorder other than depression?

Yes No Don't know

18. Have you ever been diagnosed by a professional with a psychological disorder other than depression?

Some types of professionals who might make a psychological diagnosis include a psychotherapist or counselor, psychiatrist or other medical doctor, social worker, or a clergy member or religious leader, among others.

Yes No Don't know

19. What kind of professional gave you a psychological diagnosis that was not depression? (Please mark all that apply.)

Psychotherapist or counselor	Medical doctor who is not a psychiatrist
Psychiatrist	Social worker

Clergy or religious leader Other (please specify)

[Demographic Questionnaire]

1. What is your gender?

Male Female

If not represented above, please specify:

2. Please use the slider below to select your current age.

[Available rage: Whole numbers between 18 and 99]

3. Which categories describe you? (You may select more than one.)

Hispanic, Latino/a, or Spanish Origin

Black or African American

Asian or Asian American

American Indian or Alaska Native

European American or White

Middle Eastern or North African

Native Hawaiian or other Pacific Islander

Other (please specify)

4. What is the highest level of education you have completed?

High school degree, GED, or did not finish high school

Some college

Two-year college degree

Four-year college degree

Some graduate school

Graduate degree

5. Which of the following best describes your current employment status (working for pay)?

Employed full-time (>35h/week) for pay, in addition to work performed on MTurk

Employed 10-35h/week for pay, in addition to work performed on MTurk

Employed <10h/week for pay, in addition to work performed on MTurk

Not otherwise employed for pay, apart from work performed on MTurk

6. In what range is your household's annual income level?

Less than $20, 000	$60,000 - $79,999	Decline to answer
$20,000 - $39,999	$80,000 - $99,999	
$40,000 - $59,999	$100,000 or more	

[Patient Health Questionnaire-9]

During the last 2 weeks, how often have you been bothered by any of the following problems?

NOTE: If you are in crisis, having thoughts of suicide, or need to access mental health services in your area, call 1-800-273-TALK (8255) at any time, 24 hours a day, 7 days a week, to reach the crisis hotline in your area.

Not at all—— Several days—— More than half the days——Nearly every day

---

1. Little interest or pleasure in doing things

2. Feeling down, depressed, or hopeless

3. Trouble falling asleep, staying asleep, or sleeping too much

4. Feeling tired or having little energy

5. Poor appetite or overeating

6. Feeling bad about yourself—or that you are a failure or have let yourself or your family down

7. Trouble concentrating on things, such as reading the newspaper or watching television

8. Moving or speaking so slowly that other people could have noticed. Or the opposite—being so fidgety or restless that you have been moving around a lot more than usual

9. Thoughts that you would be better off dead, or of hurting yourself in some way

[Impression of Hypotheses]

Based on the content of this survey, what do you think the aims or research question(s) for this project might be? Please type your brief impressions below.

[Thanks, Debriefing, Option to Exclude Data from Analyses]

Thank you for participating in our study.

In psychological research, it is sometimes necessary to conceal our hypotheses because knowing what is being studied can lead people to respond differently to many types of questions than they otherwise would.

Now that you have finished the survey, we can provide you with more information about it. The purpose of this study is to better understand how people think about the causes of depression. More specifically, we want to understand whether individuals' beliefs about the causes of depression—and about the role that biological

causes play—are related to other beliefs they hold about depression itself and about people with depression.

In order to help maintain the quality of the data we collect, please do not discuss this study with others who might participate anytime in the next year. Thank you for your cooperation.

If you have any questions about this study, the researcher, Sarah Mann, can be reached by email at sarah.mann@rutgers.edu. Her academic advisor, Dr. Richard Contrada, can be reached by phone at (732)445-3195, or by email at contrada@rci.rutgers.edu. Thank you again for your help today.

Now that you know the purpose of this study, please indicate below whether we may include your anonymous data in our research. If this item is left blank, we will include your data based on your consent provided at the beginning of the study.

Yes, you may include my data in this research.

Please exclude my data from this research.

Once again, if you are in crisis, having thoughts of suicide, or need to access mental health services in your area, call 1-800-273-TALK (8255) at any time, 24 hours a day, 7 days a week, to reach the crisis hotline in your area.