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the moderating effects of first- and second-hand 
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Biological causal beliefs and depression stigma: the moderating effects of first- and second-hand experience with depression

Sarah L. Mann and Richard J. Contrada
Department of Psychology, Rutgers, The State University of New Jersey—New Brunswick, New Brunswick, NJ, USA

ABSTRACT
Background: Essentialist theory (ET) links biological attributions for mental illnesses to pessimistic prognostic beliefs and stigma. The commonsense model (CSM) provides a nuanced framework for studying illness beliefs as shaped by experience.
Aims: ET-informed hypotheses linking causal and prognostic beliefs and stigmatizing attitudes concerning depression were tested using CSM constructs with a focus on the moderating effects of self-reported experience with this disorder.
Methods: U.S. adults (N = 319) completed online questionnaires assessing depression-related beliefs, attitudes and experience. Multiple regression analysis focused on predictive effects of neurobiological and genetic attributions. Potential mediators (prognosis) and moderators (experience) of the biological attribution-stigma link also were tested.
Results: Neurobiological attributions predicted viewing depression as more consequential, longer lasting, and unexpectedly, more treatable. Neurobiological attributions were inversely related to stigma, a link partially mediated by beliefs about depression’s consequences and duration. However, both biological attributions’ relationships to stigma were moderated by experience. Stronger biological attributions predicted less stigma specifically among participants reporting first- or second-hand experience with depression.
Conclusion: Experience with depression may shape the relationships of specific causal and prognostic beliefs with depression stigma. Psychoeducation in clinical and public health contexts may be informed by further research using CSM constructs.

Introduction

Essentialist theory (ET) highlights lay beliefs about biological causes of mental illness as major drivers of mental illness stigma. A broader theory of illness cognition, the commonsense model (CSM), provides a more structured, nuanced view of key ET belief constructs and emphasizes the role of lived experience in shaping illness beliefs. In this study, CSM-based measures were adapted to assess beliefs about major depression, a prevalent, costly disorder and leading cause of disability (Kessler & Bromet, 2013; World Health Organization, 2013). The focal outcome was depression stigma, which promotes shame, self-blame and social isolation in depressed individuals, and is associated with symptom severity, treatment nonengagement and nonadherence (Byrne, 2000; Pyne et al., 2004; Sirey et al., 2001). Guided by ET and the CSM, we tested hypotheses linking biological attributions for depression, prognostic beliefs and depression-stigmatizing attitudes, with a focus on the moderating effects of self-reported experience with depression.

Essentialist theory

According to ET (Haslam et al., 2006), people believe that some social categories, including mental health diagnoses, reflect an underlying biological “essence” that reinforces the perceived immutability and informativeness of the illness and perceived discreteness of the affected group (Darnimrod & Heine, 2011; Haslam, 2011). Immutability may imply that group membership is uncontrollable, potentially reducing blame and arousing sympathy (Haslam & Kvaale, 2011). However, essentialist thinking can also lead to stigmatization, prejudice and discrimination, depending on stereotype content (Corrigan & Penn, 1999).

Nevertheless, some mental health anti-stigma campaigns have emphasized biological causality, aiming to increase public acceptance and decrease blame (Jorm et al., 1997; Schomerus et al., 2012). Mixed evidence for such approaches (e.g. Lebowitz, 2014; Pescosolido et al., 2010) has raised concerns that they may instead foster essentialist beliefs about affected individuals, exacerbating anger, fear and avoidance (Speerforck et al., 2014; reviewed in Kvaale et al., 2013).
Beyond stigma, ET links biological attributions for mental illnesses to negative prognostic beliefs (Kvaale et al., 2013; Lebowitz, 2014), also potentially discouraging treatment seeking.

**The commonsense model of illness cognition: a framework for studying essentialist beliefs**

According to the CSM, people understand illnesses in terms of their: (1) *identity* (label, symptoms), (2) *causes*, (3) *consequences*, (4) *duration* and (5) *treatability* (Leventhal et al., 1997). General knowledge and illness-related experience shape illness prototypes comprising these beliefs, which, during an illness episode, give rise to *illness representations* (Leventhal et al., 1998). Whereas ET focuses on biological attributions, often construed as a single biological *cause* construct (e.g. Deacon & Baird, 2009; Lam & Salkovskis, 2007; Lebowitz, 2014; Phelan, 2005), the CSM’s open framework encourages examining individual, precisely specified causal beliefs (e.g. “neurobiological” versus “genetic”). The CSM’s *consequences* construct anticipates and refines ET’s proposition that the perceived group-defining essence is highly informative about affected individuals. The CSM parses the essentialist *immutability* construct into *duration* (e.g. acute, chronic) and *treatability*. This study used the more nuanced CSM constructs to test hypotheses linking depression-related beliefs to stigma.

The CSM also foregrounds the influence of lived experience on illness beliefs (Cameron & Leventhal, 2003; Leventhal et al., 1998). Experiencing depression or observing that of a close other yields vivid, individuating information, which was expected to reduce individuals’ reliance on depression-stigmatizing heuristics or stereotypes, including those based in essentialism. This prediction draws on the continuum model of impression formation, in which stigmatized group members receive more negative, stigma-tinted judgments when perceivers lack individuating information (Fiske et al., 1999; Fiske & Neuberg, 1990). Therefore, additional hypotheses tested first- and second-hand experience with depression as separate potential moderators of the relationships of two biological attributions with prognostic beliefs and stigma.

**Hypotheses**

Data gathered from U.S. adults recruited online were used to test three hypotheses: (1) biological attributions for depression were expected to predict prognostic pessimism (i.e. viewing depression as more consequential, of longer duration, and less treatable) and more stigmatizing attitudes toward depressed individuals. (2) Pessimistic prognostic beliefs were expected to statistically mediate the relationship between biological attributions and stigma. (3) Self-reported first- and second-hand experience with depression each was expected to weaken the relationships of biological attributions to prognostic pessimism and stigma.

**Methods**

**Participants**

Participants were recruited online via Amazon’s Mechanical Turk (MTurk) platform (*N* = 319), 60.8% female, aged 18–88 years (*M* = 38.34, *SD* = 12.70). Participants were predominantly White (83.7%); smaller proportions were Asian (7.2%), Black (6.6%) or Latino/a or Hispanic (4.7%). Most were employed 35+ hours weekly (64.3%). Approximately half (53.9%) completed at least a college degree. Nearly half (48.6%) reported prior depression.

**Measures**

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

The IPQ-R (Moss-Morris et al., 2002) was used to measure CSM *cause, duration, consequences and treatability* constructs, which map onto and refine ET’s biological *cause*, *immutability* and *informativeness*. Responses used a five-point Likert scale. Instructions focused on depression without presuming the respondent was depressed. A brief description of depression was provided.

*Cause.* Two items measured the focal biological attribution predictors: “Heredity or genes—it runs in families” and “Biological changes in the brain.” The latter was added, among other revisions to adapt the scale for depression, guided by prior research (e.g. Brown et al. 2001; Mann, 2018).

*Consequences.* This six-item measure of beliefs about depression’s negative impact (e.g. “Depression is a serious condition”) initially showed low internal consistency (*α* = 0.69). Based on inter-item correlations, a reverse-scored item was removed, yielding *α* = 0.77.

*Duration.* Five timeline scale items pertain to illness *duration* and four to symptom fluctuations. The original items do not reference treatment, which was expected to influence perceived duration when depression is considered treatable. Therefore, *duration* items were revised to ask about treated and untreated depression separately. Two items were added to assess beliefs about the likelihood of recurrence after a treated or untreated episode. Summing these and the 10 *duration* items (treated, untreated) yielded a duration score (*α* = 0.81).

*Treatability.* The cure/control scale assesses perceived *treatability*. Five items concern treatment effectiveness (e.g. “Treatment is effective in curing depression”); six concern effects of the ill person’s actions (e.g. “The course of people’s depression depends on them”). These were summed to create a treatability score (*α* = 0.77).

**Depression Stigma Scale (DSS)**

This instrument measures personal stigma (respondents’ own attitudes) and perceived stigma (perceptions of “most other people’s” views; Griffiths et al., 2004) using five-point Likert scales. Each scale’s “dangerousness” item was revised into two items distinguishing self- from other-directed
danger. Summing each scale’s 10 items yielded personal (z = 0.86) and perceived (z = 0.88) stigma scores.

**Depression Social Distance Scale (DSDS)**
This seven-item measure uses a four-point Likert scale to assess willingness to be in specific situations with people with depression (e.g. recommend for a job, introduce to friends; Rusch et al., 2008; z = 0.90).

**First- and second-hand experience and demographics**
After re-reading the description of depression, participants were asked whether they believed they had experienced depression and, in a separate question, whether they believed a “spouse, romantic partner, parent, sibling or close friend” had. Age, gender, race/ethnicity, education, employment status and household income information was collected.

**Procedure**

**Data collection**
The survey link and task payment ($0.40) were posted to MTurk workers with a U.S. IP address and ≥90% approval ratings until receiving the pre-specified number of responses (N = 351), approximately 24 hours (3/6/17). Individuals who clicked the link were informed of Rutgers University IRB’s approval (protocol E17-471) and that participation was voluntary and anonymous. Investigators’ contact information and study parameters were specified, followed by links to consent or opt out; clicking the designated link provided consent. Participants read a debriefing statement online afterward and were provided mental health hotline numbers and payment codes.

**Statistical overview**
Data were omitted for participants failing more than one of four attention checks (items asking for a specified response; n = 28, 7.98%) or requesting data exclusion (n = 4, 1.13%). The final sample (N = 319) included minimal missing values, hence all data were analyzed, yielding minor variations in degrees of freedom between analyses. Distributions were acceptable for skew, kurtosis and univariate outliers. Preliminary analyses confirmed assumptions of multiple regression, and diagnostics indicated no unduly influential cases (Cohen et al., 2003).

Biological changes in the brain (hereafter “neurobiological” causes) and heredity/genes (“genetic” causes) were first examined as predictors of prognostic beliefs (consequences, duration, treatability), then as predictors of depression stigma. Other predictors were: (1) demographics (age, gender, education, income) and (2) experience variables (respondent’s depression history, depression in a close other). Statistical mediation analysis tested whether prognostic beliefs accounted for associations between biological attributions and stigma.

The moderating effects of each of the two familiarity variables (self-reported depression history, affirming a close other’s history) on the relationships of biological attributions to prognostic beliefs and stigma were tested separately. Non-dichotomous variables were mean-centered. Interactions were represented by product terms computed by multiplying each biological attribution variable by each experience moderator. Predictors entered in the first step included demographics, both focal predictors (genetic, neurobiological attribution), and one experience moderator (respondent, close other). Product terms (one experience variable × genetic attribution; one experience variable × neurobiological attribution) were entered in the second step.

**Results**

**Bivariate associations**
As Table 1 shows, neurobiological and genetic attributions showed a small, significant association (r = 0.36). Correlations among the three prognostic beliefs (consequences, duration, treatability) were negligible to moderate (r = −0.05−0.45). The three stigma measures showed small to moderate associations (r = 0.16−0.56).

**Main effects analysis**

**Prognostic beliefs**
Stronger neurobiological attribution (β = 0.26, t = 4.57, p < 0.001), prior depression in a close other (β = 0.19, t = 2.13, p = 0.035), and affirmative personal depression experiences (β = 0.20, t = 2.15, p = 0.033) were all significantly associated with greater willingness to be in specific situations with depressed people. Main effects of gender, age, race/ethnicity, education, and employment were not significant.

**Mediation**

The moderating effects of each of the two familiarity variables were tested separately. Affirming a close other’s history showed a small, significant association (β = 0.33, t = 2.63, p = 0.009). As Table 1 shows, neurobiological and genetic attributions showed a small, significant association (r = 0.36). Correlations among the three prognostic beliefs (consequences, duration, treatability) were negligible to moderate (r = −0.05−0.45). The three stigma measures showed small to moderate associations (r = 0.16−0.56).

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Only analyses involving significant interactions are displayed. Neurobiological ($\beta = 0.21, t = 3.76, p = 0.002$) and genetic attributions ($\beta = 0.24, t = 4.08, p < 0.001$) and affirming a close other’s depression history ($\beta = 0.17, t = 3.28, p = 0.001$) each predicted viewing depression as long-lasting. Neurobiological attribution ($\beta = 0.28, t = 4.68, p < 0.001$) and older age ($\beta = 0.16, t = 2.81, p = 0.005$) each was associated with viewing depression as more treatable. There were no other significant predictors of social distance ($\beta = -0.20, t = 3.40, p = 0.001$). No other predictor showed a significant relationship with personal stigma. There were no significant effects for perceived stigma.

### Mediation analyses

Because neurobiological attributions unexpectedly showed an inverse association with personal stigma, mediation analyses (Baron & Kenny, 1986) tested the extent to which the relationship between neurobiological attributions and lower personal stigma could be explained by prognostic beliefs. Consequences, duration and treatability beliefs were examined as separate mediators, each of which showed a significant inverse association with personal stigma (consequences: $\beta = -0.26, t = -4.41, p < 0.001$; duration: $\beta = -0.33, t = -5.67, p < 0.001$; treatability: $\beta = -0.13, t = -2.31, p = 0.02$). In each instance, the standardized coefficient for the initial path between neurobiological causes and stigma was reduced, reflecting partial mediation. Sobel (1982) tests

### Stigma

Stronger neurobiological attribution ($\beta = -0.21, t = -2.14, p < 0.001$) and prior depression in a close other ($\beta = -0.12, t = 2.14, p = 0.033$) each was inversely associated with personal stigma (Table 3, top). Older age predicted desiring greater social distance ($\beta = 0.19, t = 3.42, p = 0.001$); affirming a close other’s depression history predicted desiring less personal stigma ($\beta = 0.12, t = 2.31, p = 0.02$) and older age ($\beta = 0.09, t = 2.81, p = 0.005$) each was associated with viewing depression as more treatable. There were no other significant predictors of personal stigma.

<table>
<thead>
<tr>
<th>Table 2. Main effects of biological causal beliefs about depression on three prognostic beliefs.</th>
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</thead>
<tbody>
<tr>
<td><strong>Consequences</strong></td>
</tr>
<tr>
<td><strong>$\beta$</strong></td>
</tr>
<tr>
<td>Demographics</td>
</tr>
<tr>
<td>Age</td>
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<tr>
<td>Gender (male)</td>
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<tr>
<td>Education (4-year degree)</td>
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<td>Household income</td>
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<tr>
<td>Experience variables</td>
</tr>
<tr>
<td>Depression experience: personal</td>
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<tr>
<td>Depression experience: close other</td>
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<tr>
<td>Biological attributions</td>
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<tr>
<td>Neurobiological</td>
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<td>Genetic</td>
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</table>

*values are standardized regression coefficients; sr$^2$ values are squared semipartial correlation coefficients, which quantify the magnitude of each predictor’s unique contribution.

* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

<table>
<thead>
<tr>
<th>Table 3. First- and second-hand experience with depression as moderators of associations between biological attributions and stigma variables.</th>
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<tbody>
<tr>
<td><strong>Personal stigma</strong></td>
</tr>
<tr>
<td><strong>$\beta$</strong></td>
</tr>
<tr>
<td>Demographics</td>
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<td>Age</td>
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<td>Household income</td>
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<tr>
<td>Experience and attribution variables</td>
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<tr>
<td>Depression history: personal</td>
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<tr>
<td>Depression history: close other</td>
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<tr>
<td>Neurobiological attribution</td>
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<td>Genetic attribution</td>
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<td>Experience $\times$ attribution interactions</td>
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<td>Depression history: personal $\times$ genetic attribution</td>
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<td>Depression history: personal $\times$ neurobiological attribution</td>
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<tr>
<td>Depression history: close other $\times$ genetic attribution</td>
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<tr>
<td>Depression: close other $\times$ neurobiological attribution</td>
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</tbody>
</table>

Only analyses involving significant interactions are displayed. $\beta$ values are standardized regression coefficients; sr$^2$ values are squared semipartial correlation coefficients, which quantify the magnitude of each predictor’s unique contribution. All moderator analyses included main effect terms for demographics and both biological attributions. Main effects and product terms representing interactions involving the two familiarity variables (depression history: personal, depression history: close other) were examined in separate models.

* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$. 


indicated that views of depression as more consequential ($z = -3.17, \ p = 0.002$) and longer-lasting ($z = -3.13, \ p = 0.002$), each significantly mediated the inverse relationships between neurobiological attribution and personal stigma. The mediating effect of viewing depression as more treatable was nonsignificant ($z = -1.73, \ p = 0.08$).

**Moderation analyses**

Stronger neurobiological and genetic attributions each was expected to predict more stigmatizing attitudes more strongly among participants reporting less experience with depression. Unexpectedly, analyses summarized above showed the strength of neurobiological attributions to be inversely associated with personal stigma. However, these analyses examined only main effects of each biological attribution in the full sample, which potentially obscured variations in the strength or direction of these relationships between subgroups differing in experience with depression. Further analyses tested whether experience moderated the relationships of neurobiological and/or genetic attributions to personal stigma.

Both experience items assessed self-reported depression history and history in a close other (see Measures) using “yes,” “no,” and “don’t know” responses. “No” and “don’t know” responses were combined because “don’t know” responses were few (self: $n = 36, 11.3\%$; other: $n = 24, 7.5\%$) and distinguishing those who affirmed prior depression was a priority. Neither experience variable significantly moderated relationships between biological attributions and prognostic beliefs. In contrast, experience significantly moderated several relationships between biological attributions and stigma (Table 3, bottom panel).

**Personal stigma**

Self-reported depression history moderated the association between genetic attribution and personal stigma ($\beta = -0.15, \ t = -1.20, \ p = 0.047$). Among participants reporting prior depression, stronger genetic attribution was associated with less personal stigma. Among those not reporting prior depression, genetic attribution and personal stigma were unrelated (Figure 1(a)).

**Social distance**

Affirming a close other’s depression history moderated the relationship between genetic attribution and desired social distance ($\beta = -0.19, \ t = -2.01, \ p = 0.045$). Among participants affirming a close other’s history, stronger genetic attribution was associated with desiring less social distance (Figure 1(b)). Among those not reporting prior depression in a close other, stronger genetic attribution was associated with desiring more social distance.

Self-reported depression history moderated the relationship of neurobiological attribution to desired social distance ($\beta = -0.16, \ t = -2.13, \ p = 0.03$). Among participants reporting prior depression, stronger neurobiological attribution was associated with less desired social distance. Among those not endorsing prior depression, these variables were unrelated (Figure 1(c)).

**Perceived stigma**

Self-reported depression history also moderated the relationship between genetic attribution and perceived stigma ($\beta = -0.22, \ t = -2.87, \ p = 0.004$). Among participants affirming prior depression, stronger genetic attribution predicted less perceived stigma. In contrast, among those not reporting prior depression, stronger genetic causal belief predicted greater perceived stigma (Figure 1(d)).

**Discussion**

This cross-sectional study used CSM-based measures to test ET-informed hypotheses in a convenience sample recruited online. Hypotheses focused on two biological attributions for depression—“biological changes in the brain” and “heredity or genes”—as predictors of prognostic expectations and depression stigma. Attributing depression to neurobiological changes was associated with viewing it as severely consequential and long-lasting, consistent with ET’s notions of informativeness and immutability, respectively (Haslam et al., 2006; Haslam & Kvaale, 2015). Unexpectedly, neurobiological attribution also was associated with viewing depression as more treatable. Genetic attribution predicted viewing depression as long-lasting but was unrelated to beliefs about consequences and treatability. Neurobiological, but not genetic, attribution was significantly associated with stigma in the full sample. That is, unexpectedly and counter to ET, stronger endorsement of neurobiological attributions predicted lower levels of depression stigma. This effect was partially statistically mediated by beliefs about depression’s severe consequences and long duration.

Most importantly, and in accord with predictions, associations between biological attributions and stigma measures differed depending on whether respondents affirmed a history of depression and/or history in a close other. Only among individuals affirming either of these two experiences, endorsing neurobiological or genetic causes of depression predicted less stigma. For those lacking such experiences, biological attributions either were unrelated to stigma or predicted higher levels. Experience did not moderate any relationships between biological attributions and prognostic beliefs. The reported associations were generally modest in magnitude, consistent with most findings in this area. While this study was not designed to investigate the clinical significance of depression-related beliefs and stigma, the findings have potentially important theoretical and practical implications.

**Biological causal beliefs as predictors of prognostic beliefs and stigma**

Genetic and neurobiological attributions were positively correlated as in previous research (e.g. Rüsch et al., 2010). Yet they performed differently as predictors. The greater
predictive power of neurobiological attribution compared to genetics in the main effects analyses is interesting given the faster rise in public acceptance of brain-related compared to genetic attributions for mental illnesses (Schomerus et al., 2012).

Consistent with ET, stronger neurobiological and genetic attribution for depression each was associated with viewing it as long-lasting. Neurobiological attribution was additionally linked to viewing it as severely consequential. Of note, where stronger neurobiological attributions predicted perceiving severe consequences and long duration, second-hand experience also emerged as a significant predictor (Table 2), suggesting that knowledge gained from observing a close other’s struggle with depression may have informed this view. In contrast to these ET-consistent associations, additional findings linking neurobiological attributions with viewing depression as more treatable and endorsing lower personal depression stigma countered ET predictions. Overall, neurobiological attributions showed a pattern of associations with prognostic beliefs and stigmatizing attitudes that was consistent with prior findings linking biological attributions with viewing mental illness as more severe and affected individuals as less blameworthy (Haslam & Kvaale, 2015; Kvaale et al., 2013). Regarding specific measures, this relationship may have involved personal stigma but not social distance because some personal stigma items may also tap depression severity beliefs (e.g. People with depression could snap out of it if they wanted; Depression is not a real illness; Griffiths et al., 2004).

The observed inverse relationship between neurobiological attributions and stigma contrasts with findings from a large, representative German sample, linking “biogenetic”
attributions for depression to more desired social distance, mediated by perceived difference and dangerousness (Schomerus et al., 2014). Different methodologies, biological attribution constructs, and stigma measures likely contribute to the divergent findings. Marked cultural differences in attitudes toward and use of antidepressants between samples also may be relevant (Schomerus et al., 2014), as neurobiological attributions and treatability beliefs were significantly linked in our sample and unrelated in the German sample.

The prognostic beliefs associated with lower depression stigma may seem incompatible: consequential, long-lasting, yet highly treatable. They are consistent, however, with public health and pharmaceutical advertising messages encouraging treatment seeking. In this vein, prior studies have highlighted mental illness-specific “controllability” beliefs—included in the treatability scale—as a key inverse predictor of stigma (Krendl & Freeman, 2017). In contrast, beliefs linked with greater stigma—less consequential, less persistent, less treatable—suggest a view of depression more resembling a bad mood than a biologically fixed, social out-group-defining construct. In this context, “less treatable” may reflect beliefs that depression is too mild to warrant treatment, a willful exaggeration of negative emotion, or more a personality trait than an illness.

Prognostic beliefs as mediators of the inverse relationships between biological attributions and stigma

Relationships between stronger neurobiological attributions and lower personal stigma were partially mediated by beliefs about depression’s consequences and duration. As noted, the association of these causal and prognostic beliefs with lower stigma runs counter to essentialist theorists’ concern about the potential, under certain conditions, for biological attributions to evoke mental illness stigma. In this study, neurobiological attributions, along with negative beliefs about duration and consequences—that is, about depression’s severity—may have lowered stigma by reducing perceived responsibility and blame (Corrigan, 2000; Weiner et al., 1988). This possibility is consistent with findings showing that stigmatized conditions perceived as “physical” were considered less controllable and blameworthy than “mental-behavioral” conditions and elicited more positive emotional responses (Weiner et al., 1988).

Moderating effects of experience with depression on relationships between biological attributions and stigma

In several significant interactions, individuals who reported first- or second-hand experience with depression, who also more strongly endorsed biological attributions, expressed lower depression stigma than those lacking such experience. In the context of prior mixed results, this pattern suggests a complex relationship between biological attributions and stigma, in which these attributions may reduce stigma for some segments of the population, under some conditions (Lebowitz & Ahn, 2015). For example, the present findings linking biological attributions to lower stigma, specifically among participants reporting prior depression, accords with qualitative evidence that biomedical explanations can reduce self-stigma for people with a personal or family depression history (Buchman et al., 2013; Laegsgaard et al., 2010; Schreiber & Hartrick, 2002). Interestingly, the present analyses implicated genetic attributions in three of the four interactions in which biological attributions predicted lower stigma specifically among those reporting depression experience. To understand the potentially stigma-reducing role of biological attributions in this population, family history beliefs and their relationships to experience moderators may prove worthy of more investigation.

The present findings also fit attribution theory-based recommendations for reducing self-stigma in clinical and psychoeducational contexts by explaining depression’s biological mechanisms using both “retrospective” (blame-reducing) and “prospective” (agency-promoting) frames (MacDuffie & Strauman, 2017). Such explanations may reduce both biological essentialist beliefs and stigma by conveying the idea that human physiology is responsive to experience. The present findings contrast with evidence linking individuals’ biological explanations for their own severe mental illness to greater self-stigma (Rüsch et al., 2010); demographic, clinical and methodological differences may explain these studies’ disparities.

The observed moderating effects of experience suggest that accounting for respondents’ first- and second-hand familiarity with a target illness—that is, the extent to which they view affected individuals as an ingroup or an outgroup—might clarify some mixed results of nonclinical studies examining mental illness beliefs and stigma (reviewed in Pescosolido, 2013; Schomerus et al., 2012). Notably, a prior study examining experiential moderators found second-hand familiarity with “any mental illness” fell short of predicting stigma or moderating its association with belief “that people can change” (Lyndon et al., 2016, p. 2), suggesting the importance of examining disorder-specific experience as a moderator of disorder-specific illness belief–stigma relationships (Schomerus et al., 2014).

Limitations

One limitation of this study is its use of cross-sectional, correlational data, which precludes causal inferences. Second, the MTurk convenience sample differed in several ways from the national population (Chandler & Shapiro, 2016), such as overrepresenting those reporting prior depression (Kessler & Wang, 2008), women, Whites, Asians and college-educated individuals, relative to national samples. Therefore, while online recruitment improved demographic and geographic diversity over traditional convenience samples (e.g. student pool, university community), the sample was not nationally representative, limiting generalizability. A third limitation, use of self-reported data (Chan, 2009) including some single-item measures, remains common in research on illness beliefs and stigma, yet raises questions
about construct validity and reliability. Potentially offsetting some self-report limitations, preliminary evidence suggests that MTurk’s complete anonymity may promote self-disclosure and reduce social desirability biases compared to in-person interactions (Shapiro et al., 2013).

Conclusion
In an online sample, biological attributions for depression showed significant associations with depression-related prognostic beliefs and stigma. Findings linking neurobiological attribution to the perception of depression as severely consequential and long-lasting aligned with ET predictions. Contrary to predictions, stronger neurobiological attribution was associated with a view of depression as more treatable and with endorsement of lower levels of stigma. This inverse relationship to stigma was partially mediated by beliefs about depression’s severe consequences and long duration. Of note, inverse associations between biological attributions and stigma in the full sample were qualified by the moderating effects of first- or second-hand experience with depression and stigma. Specifically, among individuals reporting such experience, stronger biological attributions predicted lower stigma. This inverse relationship to stigma was partially mediated by beliefs about depression’s severe consequences and long duration.

Beyond its empirical findings, this study demonstrates the utility of CSM illness belief constructs for refining such ET constructs as biological causal beliefs and illness immutability, which may obscure meaningful, unexpected relationships. Additionally, the CSM’s focus on lived experience informing illness beliefs provides a theoretical foundation for examining the influence of experiential factors, like one’s own or a close others’ depression, on the strength and direction of links between specific illness beliefs and social-evaluative processes.

Disclosure statement
The authors have no potential conflicts of interest to report.

Data availability
The data that support this study’s findings are available from the corresponding author, SLM, for a specified purpose upon reasonable request.

References


