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When do psychosocial explanations of psychiatric problems increase stigma? Self-report and implicit evidence

Ryan Thibodeau
St. John Fisher University, rthibodeau@sjf.edu

Gabrielle L. Amberger
St. John Fisher University, gla06309@sjf.edu

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Abstract

Background and objectives

Biomedical explanations of psychiatric problems, compared to psychosocial explanations, may amplify psychiatric stigma. One limitation of existing research is the measurement of almost exclusively self-reported stigma. This study evaluated the stigma-related effects of biomedical versus psychosocial explanations of schizophrenia using conventional self-report and two other measurement approaches that may tap more deeply held attitudes.

Methods

One hundred three undergraduates listened to a vignette describing a man with (1) schizophrenia of biomedical origin, (2) schizophrenia of psychosocial origin, or (3) diabetes. They then completed an Implicit Association Test, conventional self-report stigma measures, and *projected other* measures that captured perceptions of most other people's likely impressions. *Results*: Participants were more likely to attribute stigmatizing views to others compared to themselves. The projected other measurement, but not the conventional self-report measurement, predicted implicit attitudes. We obtained no evidence that the psychosocial causal explanation of schizophrenia led to decreased stigma compared to the biomedical causal explanation. In fact, the psychosocial causal explanation increased stereotyped attitudes.

Limitations

The absence of a schizophrenia control group complicates interpretation of biomedical versus psychosocial group comparisons.

Conclusions

Further research is needed to evaluate discrepancies between the present findings and other published evidence pertaining to psychosocial causal explanations of psychiatric problems.

Keywords

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Disciplines

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Comments

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Abstract

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Results: Participants were more likely to attribute stigmatizing views to others compared to themselves. The projected other measurement, but not the conventional self-report measurement, predicted implicit attitudes. We obtained no evidence that the psychosocial causal explanation of schizophrenia led to decreased stigma compared to the biomedical causal explanation. In fact, the psychosocial causal explanation increased stereotyped attitudes.

Limitations: The absence of a schizophrenia control group complicates interpretation of biomedical versus psychosocial group comparisons.

Conclusions: Psychosocial causal explanations that portray people as subject to numerous, severe stressors may evoke the cultural stereotype of the "ticking time bomb" from whom others seek safe distance.

Keywords: psychiatric stigma; schizophrenia; implicit attitudes; biomedical; psychosocial

When Do Psychosocial Explanations of Psychiatric Problems Increase Stigma?

Self-Report and Implicit Evidence

Ryan Thibodeau & Gabrielle L. Amberger

St. John Fisher College

1. Introduction

Individuals with psychiatric problems are commonly devalued, avoided, and rejected (see Hinshaw & Stier, 2008, for a review). Development and implementation of stigma reduction programming to improve the life prospects of individuals with psychiatric problems is a critical priority (Hogan, 2003; World Health Organization, 2001).

One common approach to stigma reduction intervention targets beliefs about the causes of psychiatric problems. Promotion of a starkly biomedical etiology has been a particularly prominent element of anti-stigma messaging. Anti-stigma messaging commonly encourages the view that psychiatric problems are "diseases like any other" (Pescosolido et al., 2010) attributable to "no fault brain disorders" (e.g., McEvoy, Scheifler, & Frances, 1999). The foundational assumption that motivates these efforts is that public stigma will diminish when psychiatric problems are attributed to circumstances beyond individuals' control.

Psychiatric problems can also be conceived as understandable responses to stress, trauma, difficult life circumstances, and other psychosocial factors (Coyne & Downey, 1991; Dohrenwend & Dohrenwend, 1981; Pearlin, 1999). Promotion of psychosocial etiology as a route to stigma reduction could also be fruitful. Insofar as everybody experiences life stress and can identify with its damaging effects, promotion of psychosocial etiology could discourage outgroup categorization of individuals with psychiatric problems, a process that fuels stigma (Link & Phelan, 2001).

There is now a sizeable empirical literature that directly compares the stigma-related consequences of experimentally manipulated biomedical and psychosocial causal explanations of psychiatric problems. In their meta-analysis of this literature, Kvaale, Haslam, and Gottdiener (2013) found that promotion of biomedical etiology, compared to psychosocial etiology, is

mostly damaging. That is, it decreases appraisals of individuals' blameworthiness for their problems but increases prognostic pessimism and amplifies the dangerousness stereotype (see also Haslam & Kvaale, 2015). Essentialism, or the belief that social categories reflect immutable "natural kinds," help explain the pernicious stigma-related consequences of biomedical causal explanations (Haslam & Ernst, 2002).

These findings have been generated mostly on the basis of conventional self-report measurement (but see Lincoln, Arens, Berger, & Rief, 2008; Mehta & Farina, 1997). One priority for research on psychiatric stigma is the documentation of key effects using measures that could circumvent social desirability biases which may be especially troublesome in the context of self-report stigma measurement (Stier & Hinshaw, 2007). Psychiatric stigma scholars dating back at least to the early 1980s have encouraged use of alternative measurement tools that tap "deep" attitudes (Link & Cullen, 1983) that may more accurately illuminate psychiatric stigma but that are difficult to capture using conventional self-report measures.

Two alternative measures are pertinent here. First, measures of implicit attitudes (Greenwald, Nosek, & Banaji, 2003) capture automatic, covert impressions that mostly bypass the self-presentational motives that threaten the validity of self-report measures of psychiatric stigma. Measurement of implicit attitudes is rare in research on psychiatric stigma, but there is evidence that implicit and explicit measures of psychiatric stigma are nearly uncorrelated (Teachman, Wilson, & Komarovskaya, 2006; but see Thibodeau & Finley, 2017). Only one study has evaluated the effect of experimentally manipulated biomedical versus psychosocial causal explanations of psychiatric problems on implicit attitudes (Lincoln et al., 2008). This study found no evidence that the manipulation of causal explanation affected implicit attitudes. More research is needed to corroborate this finding.

Second, self-report scales that require respondents to indicate how they think most others would evaluate a person with psychiatric problems (Link & Cullen, 1983) are regularly administered. A common assumption is that this *projected other* response format gives respondents "tacit permission to express highly stigmatizing attitudes" (Ritsher & Phelan, 2004, p. 260) that they would otherwise censor in completing conventional measures. Yet, surprisingly, research that systematically examines the characteristics of these measures vis-à-vis conventional self-report measures is rare (but see Link & Cullen, 1983; Werner, 2015). How large are effects of stigma-relevant experimental manipulations for conventional response formats versus the projected other response format? Which response format more strongly predicts measures known to tap personally held "deep" attitudes, such as implicit attitudes?

In our experiment, undergraduates read an ostensible scientific article that highlighted biomedical causes of schizophrenia, psychosocial causes of schizophrenia, or that identified causes of diabetes, a medical control condition. They then listened to an audio-recorded vignette describing "Dennis," whose history with schizophrenia or diabetes reinforced the causal explanations embedded in the articles. Participants then completed three kinds of stigma measures: conventional self-report scales, the same scales written with a projected other response format, and an Implicit Association Test (IAT; Greenwald et al., 2003) that measured implicit attitudes.

Hypotheses regarding the experimental manipulation (biomedical vs. psychosocial vs. diabetes control) were as follows:

(1) Schizophrenia should be associated with greater stigma than diabetes regardless of the causal explanation (biomedical or psychosocial) invoked.

(2) A psychosocial explanation of the causes of schizophrenia should lead to lesser

stigma than a biomedical explanation (cf. Kvaale et al., 2013).

Hypotheses regarding self-report response format (conventional vs. projected other) were as follows:

(1) Participants should attribute greater psychiatric stigma to others compared to themselves (cf. Link & Cullen, 1983), given the powerful psychological tendency for people to evaluate themselves more positively and/or less negatively than others (Alicke, Klotz, Breitenbecher, Yurak, & Vredenburg, 1995).

(2) The effects of our experimental manipulation should be larger for the projected other response format than for the conventional response format. This is expected on the basis of the common assumption that the projected other response format permits the candid expression of negative impressions that respondents censor in completing conventional measures (Ritsher & Phelan, 2004).

(3) If the projected other response format taps "deep," personally held views that conventional measures obscure, than this response format should facilitate stronger links to a measure of "deep," implicit attitudes measured via an IAT.

2. Material and Methods

2.1 Participants

Participants were recruited from four sections of an introductory psychology course. Of the 122 total students enrolled in the course, 110 signed up to participate via SONA participant management software (SONA Systems, Ltd., Tallin, Estonia). One hundred three students (64 women, 82.5% Caucasian, *M* age = 18.8, *SD* = 1.6) eventually participated in exchange for course credit. The first-author allocated participants to the biomedical (*n* = 34), psychosocial (*n* = 35), or diabetes control (*n* = 34) conditions using computer-generated random sequences that

yielded blocks fixed at size 3.

2.2 Measures

2.2.1 Self-report measures. Self-reported stigma was indexed using three tools. The Social Distance Scale (SDS; Link, Cullen, Frank, & Wozniak, 1987) includes seven items that measured willingness to engage, at varying degrees of closeness (e.g., co-worker, neighbor, roommate) with "Dennis," the subject of the vignettes (see below). Responses were recorded on four-point scales (1 = definitely willing, 4 = definitely unwilling).

A 10-item measure of emotional reactions (Schomerus, Matschinger, & Angermeyer, 2013) was administered. Items were grouped into fear (e.g., insecure, fear), anger (e.g., angry, annoyed), and prosocial (e.g., sympathy, the need to help) categories. Responses were recorded on five-point scales (1 = strongly disagree, 5 = strongly agree).

A 12-item semantic differential (Olmsted & Durham, 1976) was administered to measure stereotyped attitudes. Respondents rated both "Dennis" and "Average Man" on seven-point scales anchored by bipolar adjectives (e.g., safe-dangerous, predictable-unpredictable) pertinent to psychiatric stigma. Difference scores for all 12 items were computed by subtracting ratings for "Average Man" from ratings for "Dennis." The 12 items were then averaged to form an overall index of stereotyped attitudes.

We carried out two separate administrations of the three scales. In the conventional administration, participants' responses reflected their personal thoughts and feelings about Dennis. In the projected other administration, participants' responses reflected their view of how "most people" would think and feel about Dennis.

To evaluate the effects of the experimental manipulation, we administered a six-item scale that measured participants' endorsement of biomedical (e.g., chemical imbalances) and

psychosocial (e.g., stressful life events) causes of schizophrenia. Responses were recorded on four-point scales (1 = definitely disagree, 4 = definitely agree).

2.2.2 Implicit measure. An IAT (Greenwald et al., 2003) was administered via E-Prime (Psychology Software Tools, Inc.; Sharpsburg, PA) to measure participants' implicit evaluations of Dennis. Stimuli were (a) 10 color photographs from the Karolinska Directed Emotional Faces (KDEF; Lundqvist, Flykt, & Öhman, 1998) depicting, at five different angles, "Dennis" (KDEF faces BM25NEFL, BM25NEFR, BM25NEHL, BM25NEHR, BM25NES) and "Somebody else" (KDEF faces BM31NEFL, BM31NEFR, BM31NEHL, BM31NEHR, BM31NES) and (b) words capturing the concepts of "safe" and "dangerous." Participants sorted these stimuli into categories combining "Dennis" or "Somebody else" with the concepts "safe" or "dangerous." We computed *D* scores following accepted procedures for the scoring and analysis of IAT data (Greenwald et al., 2003).

2.3 Procedure

First, participants provided informed consent to complete the study, which was approved by the local institutional review board. Second, participants randomized to the biomedical and psychosocial groups were given a bogus but authentic looking article from an ostensible scientific journal devoted to schizophrenia research. The first page of the article included highlighted material that participants' were asked to carefully read; namely, the title and a short abstract-like section called "Highlights," which contained bulleted information on causes of schizophrenia. In the biomedical condition, participants read material that summarized evidence attesting to biomedical causes of schizophrenia. In the psychosocial condition, participants read material that summarized evidence attesting to psychosocial causes of schizophrenia. Participants randomized to the diabetes control condition read a short excerpt from an

informational document published by the National Institute of Diabetes and Digestive and Kidney Diseases (2013). All texts from this element of the manipulation are included in Appendix A. The experimenter administered the six-item manipulation check subsequent to participants' reading of the manipulation texts.

Third, participants listened to one of three audio-recorded vignettes (see Appendix B). Both the biomedical and psychosocial vignettes (portions of which were adapted from Phelan, 2005) started by describing a 31 year-old man, Dennis, who began experiencing symptoms of a psychotic episode one year ago. The biomedical vignette explained that medical testing revealed the biological basis (i.e., dysfunctional neurotransmission, abnormal brain structure, and genetic abnormalities) of these symptoms. The psychosocial vignette identified several stressful life events (i.e., the recent death of both Dennis's parents, major interpersonal conflicts at work, and severe financial hardship) that explained psychosis onset. The diabetes control vignette described symptoms and causes of type-II diabetes. The vignettes were audio-recorded by the first author, embedded into an E-Prime stimulus presentation file, and delivered to participants via Sennheiser HD 202 headphones. A color photograph of Dennis exhibiting a neutral expression against a plain beige backdrop (KDEF face BM25NES) was shown on the computer display concurrent with narration of the vignettes.

Fourth, participants completed both versions (conventional and projected other) of the three self-report scales back-to-back, in an order counterbalanced across participants. Finally, participants were debriefed and dismissed.

2.4 Data Analyses

Manipulation check data and IAT *D* scores were analyzed using one-way analyses of variance (ANOVA) with group (biomedical vs. psychosocial vs. diabetes control) entered as a

single between-subjects factor. The self-report data were analyzed using separate mixed ANOVAs with group as a between-subjects factor and response format (conventional vs. projected other) as a within-subjects factor. In the self-report analyses, two kinds of planned comparisons were executed to follow up significant omnibus group effects. First, the psychiatric stigma effect was evaluated by comparing the average of the biomedical and psychosocial means to the diabetes control mean. Second, our primary hypothesis regarding a stigma reduction effect of psychosocial (compared to biomedical) causal views was evaluated by directly comparing the biomedical and psychosocial means.

3. Results

3.1 Preliminary Analyses

A series of initial analyses were undertaken to determine whether random assignment yielded experimental groups that were balanced with respect to demographic and other key variables. No significant group differences emerged for any demographic variables: age: $F(2,100) = 2.11$, $p = .13$; sex: $\chi^2(2) = 1.01$, $p = .60$; ethnicity (proportion of Caucasian vs. other participants): $\chi^2(2) = 1.07$, $p = .59$. There were also no group differences in the proportion of participants with friends or family members with psychiatric problems, $\chi^2(2) = 0.87$, $p = .65$.

3.2 Effectiveness of the Experimental Manipulation

One-way ANOVAs indicated that the three groups differed with respect to biomedical causal beliefs, $F(2,100) = 15.87$, $p < .001$, $\eta_p^2 = .24$, and psychosocial causal beliefs, $F(2,100) = 35.16$, $p < .001$, $\eta_p^2 = .41$ (see Table 1). All pairwise comparisons in both models were significant (all ps $< .01$).

3.3 Effects of Biomedical and Psychosocial Causal Beliefs on Psychiatric Stigma

Table 1 displays effects of the experimental manipulation (biomedical vs. psychosocial

vs. diabetes control) and response format (conventional vs. projected other) on our primary stigma measures.

3.3.1 Implicit evaluations. There was no evidence that the experimental manipulation affected IAT *D* scores, $F(2,100) = 0.87$, $p = .42$, $\eta_p^2 = .02$.

3.3.2 Social distance. The mixed ANOVA for social distance yielded a significant effect of response format, $F(1,100) = 130.77$, $p < .001$, $\eta_p^2 = .57$, whereby participants indicated that others would seek more distance from Dennis than they themselves would. The model also yielded a significant effect of group, $F(2,100) = 55.01$, $p < .001$, $\eta_p^2 = .52$. The first planned contrast indicated that more distance was desired in the schizophrenia conditions compared to the diabetes condition, $F(1,100) = 108.68$, $p < .001$, $\eta_p^2 = .52$. The second planned contrast indicated that the biomedical and psychosocial groups did not differ with respect to social distance, $F(1,100) = 1.14$, $p = .29$, $\eta_p^2 = .01$. The group by response format interaction was not significant, $F(2,100) = 1.02$, $p = .36$, $\eta_p^2 = .02$.

3.3.3 Fear. The mixed ANOVA for fear yielded a significant effect of response format, $F(1,100) = 127.56$, $p < .001$, $\eta_p^2 = .56$, whereby participants indicated that others would be more afraid of Dennis than they themselves would. The model also yielded a significant effect of group, $F(2,100) = 16.04$, $p < .001$, $\eta_p^2 = .24$. The first planned contrast indicated that more fear was reported in the schizophrenia conditions compared to the diabetes condition, $F(1,100) = 32.08$, $p < .001$, $\eta_p^2 = .24$. The second planned contrast indicated that the biomedical and psychosocial groups did not differ with respect to fear, $F(1,100) = 0.01$, $p = .93$, $\eta_p^2 = .00$. A significant group by response format interaction, $F(2,100) = 5.11$, $p = .01$, $\eta_p^2 = .09$, indicated that the group effect was larger when participants reported projected others' ($\eta_p^2 = .30$) versus their own impressions ($\eta_p^2 = .08$).

3.3.4 Anger. The mixed ANOVA for anger yielded a significant effect of response format, $F(1,100) = 140.15$, $p < .001$, $\eta_p^2 = .58$, whereby participants indicated that others would feel more anger than they themselves would. The group effect approached significance, $F(2,100) = 3.05$, $p = .052$, $\eta_p^2 = .06$. The first planned contrast indicated that more anger was reported in the schizophrenia conditions compared to the diabetes condition, $F(1,100) = 4.15$, $p = .04$, $\eta_p^2 = .04$. The second planned contrast indicated that the biomedical and psychosocial groups did not differ with respect to anger, $F(1,100) = 2.00$, $p = .16$, $\eta_p^2 = .02$. The group by response format interaction was not significant, $F(2,100) = 1.59$, $p = .21$, $\eta_p^2 = .03$.

3.3.5 Prosocial emotion. The mixed ANOVA for prosocial emotion yielded a nonsignificant effect of response format, $F(1,100) = 0.61$, $p = .44$, $\eta_p^2 = .01$. The model yielded a significant effect of group, $F(2,100) = 5.23$, $p = .01$, $\eta_p^2 = .10$. The first planned contrast indicated that more prosocial emotion was reported in the schizophrenia conditions compared to the diabetes condition, $F(1,100) = 10.02$, $p = .002$, $\eta_p^2 = .09$. The second planned contrast indicated that the biomedical and psychosocial groups did not differ with respect to prosocial emotion, $F(1,100) = 0.41$, $p = .53$, $\eta_p^2 = .00$. The group by response format interaction was not significant, $F(2,100) = 2.84$, $p = .06$, $\eta_p^2 = .05$.

3.3.6 Stereotyped attitudes. The mixed ANOVA for stereotyped attitudes yielded a significant effect of response format, $F(1,100) = 60.57$, $p < .001$, $\eta_p^2 = .38$, whereby participants indicated that others would more strongly endorse stereotyped attitudes than they themselves would. The model also yielded a significant effect of group, $F(2,100) = 24.10$, $p < .001$, $\eta_p^2 = .33$. The first planned contrast indicated that stereotyped attitudes were more strongly endorsed in the schizophrenia conditions compared to the diabetes condition, $F(1,100) = 42.62$, $p < .001$, $\eta_p^2 = .30$. Notably, the second planned contrast indicated that the psychosocial group showed a

stronger embrace of stereotyped attitudes than the biomedical group, $F(1,100) = 5.33$, $p = .02$, $\eta_p^2 = .05$. The group by response format interaction was not significant, $F(2,100) = 3.06$, $p = .051$, $\eta_p^2 = .06$.

3.4 Conventional versus Projected Other Self-Report Measurements – Correlations with Implicit Attitudes

For both the conventional and projected other measurements, we computed correlations between the safe-dangerous item of the semantic differential tool and IAT *D* scores. Only the safe-dangerous item was subjected to analysis because it was the only item, across all self-report scales administered, that mapped directly onto the sorting categories we employed in the IAT (i.e., participants sorted stimuli into categories that combined "Dennis" or "Somebody else" with words reflecting the concepts "safe" or "dangerous"). Thus, variation in the size of correlations of conventional and projected other measurements with IAT *D* scores would reflect only method variance – our central concern here – and not the additional influence of construct-related variance. Moreover, we analyzed data only for the two schizophrenia groups (i.e., biomedical and psychosocial; $n = 69$) because of our overriding interest in schizophrenia stigma and because of low variability in appraisals of the dangerousness of the individual with diabetes. Consistent with our predictions, the projected other measurement was significantly correlated with IAT *D* scores, $r = .28$, $p = .02$, whereas the conventional measurement was not, $r = .07$, $p = .55$.

4. Discussion

Our primary prediction surrounding the effects of biomedical versus psychosocial causal explanations was not supported. We obtained no evidence that a psychosocial causal explanation of schizophrenia decreased stigma compared to a biomedical explanation. In fact, findings for stereotyped attitudes demonstrated that the psychosocial causal explanation amplified stigma.

These results stand in stark contrast to the findings of a recent meta-analysis (Kvaale et al., 2013), which demonstrated that psychosocial causal explanations led to decreased stigma across a variety of key dimensions. Possible reasons for the discrepancy between the present and other published findings merit some discussion.

In the published literature, there is substantial variability in the size of effects that could be attributable to numerous factors (e.g., sample characteristics, measurement differences) that may be operational in the current study. However, the manner in which especially psychosocial manipulations are carried out could be especially consequential. In one approach, vignettes make general reference to psychosocial causes that are vague and mostly unspecified. In another approach, vignettes make specific reference to psychosocial causes that are carefully delineated and described in great detail. Our study executed this second kind of manipulation. That is, we identified and offered a vivid description of several very severe stressors with which Dennis was struggling.

The kind of manipulation undertaken – vague and unspecified psychosocial causes, on the one hand, and specific and highly detailed stressors, on the other – may predict variability in effect sizes. For example, in Kvaale and colleagues' (2013) meta-analysis, the largest stigma reduction effect of psychosocial manipulation was associated with a study (Bennett, Thirlaway, & Murray, 2008) that utilized a vignette that referred only in passing to a single, albeit severe stressor: the sudden death of a spouse. The second largest stigma reduction effect was associated with a study (Lam, Salkovskis, & Warwick, 2005) that manipulated psychosocial causal explanation only by stating that "research suggests that... psychologically based problems may be the result of environmental risk factors" (p. 457). In contrast, a handful of studies in the meta-analysis reported a stigma enhancing effect of psychosocial manipulation. Most of these studies

(e.g., Lebowitz, Rosenthal, & Ahn, 2016; Lincoln et al., 2008) utilized vignettes that included highly detailed descriptions of multiple, severe stressors. Our speculation is that this latter approach could amplify psychiatric stigma because it evokes the provocative cultural stereotype of the "ticking time bomb" who explodes under the weight of extraordinary stress. Indeed, this kind of imagery pervades popular films, television, and real-world tragedies that are highly accessible in the news media.

In light of this reasoning, one possible implication is that stigma reduction programming that invokes psychosocial stress should avoid describing distressed individuals as subject to a laundry list of very extreme stressors. Of course, this recommendation is based on a post hoc interpretation that must be considered highly tentative. Follow-up research that directly compares the two kinds of psychosocial manipulations is clearly needed to evaluate the viability of our "ticking time bomb" speculations.

The absence of an effect of causal explanation on implicit attitudes mirrors the finding documented by Lincoln and colleagues (2008). Other recent research (Thibodeau, Shanks, & Smith, 2018) failed to document an effect of continuum belief intervention (see, e.g., Schomerus et al., 2013; Thibodeau, 2017) on an index of stigma-relevant behavior. Continued failure to demonstrate effects of stigma reduction intervention on implicit attitudes and stigma-related behavior should motivate some soul-searching on the part of researchers and other professionals who endeavor to combat stigma. Interventions that positively affect explicit attitudes may do little to affect important behavioral dimensions of stigma or implicit attitudes, the latter of which are almost certainly important drivers of the rejection and avoidance that define stigma. Taken together, these results suggest that studies of stigma reduction interventions should employ implicit and behavioral measures more routinely than they presently do. This would enable

researchers to address important questions that are precluded by the use of strictly self-report measurement. For example, how valid are implicit measures of psychiatric stigma? That is, what is the nature and strength of the relationship between implicit attitudes and explicit stigma, stigma-relevant behavior, or other stigma-related processes not fully accessible to conscious awareness? How tractable are implicit attitudes toward individuals with mental illness? Do stigma reduction strategies that positively impact explicit evaluations (e.g., contact; Couture & Penn, 2003) also positively impact implicit evaluations and/or behavioral measures?

Three observations surrounding findings for self-report response format are worth highlighting. First, we obtained consistent support across measures for our prediction that participants would attribute greater psychiatric stigma to others compared to themselves Second, our prediction that the projected other response format would lend itself to larger group effects was supported only for fear. This modest support notwithstanding, it is clear that participants were quite willing to endorse psychiatric stigma using the conventional response format. This may be attributable, in part, to our rich description of a condition, schizophrenia, which is widely considered the most stigmatized of all psychiatric problems. Our description of very obvious discrediting attributes (i.e.., acute psychosis) may have compelled participants to acknowledge negative impressions that are undeniable and widely endorsed. Future research might evaluate effects of response format for less obviously stigmatized psychiatric conditions, such as depression or anxiety. Projected other measurements may be more sensitive indicators of stigma especially in the context of less extreme conditions that generate negative impressions that people can effortlessly disavow using the conventional response format.

Third, perhaps the most novel finding is that the projected other measurement of dangerousness, but not the conventional measurement, predicted implicit attitudes. This finding

lends some support to the widespread assumption that projected other measures offer "tacit permission" to endorse negative evaluations that are obscured by the self-presentational biases inherent to conventional measures (Link & Cullen, 1983; Ritsher & Phelan, 2004).

Our study was subject to several important limitations. First, two of the three stressors cited in the psychosocial condition – interpersonal conflict and financial hardship – are probably not unambiguously situational. Indeed, interpersonal conflict could be seen as arising from a durable character flaw (e.g., high trait hostility). Moreover, financial hardship could evoke poverty-related stigma which is powerful in its own right (Hanson, Bourgois, & Drucker, 2014; Reutter et al., 2009). Second, our use of the diagnostic label *schizophrenia* in both the biomedical and psychosocial groups may have unintentionally weakened the causal distinction that our manipulations sought to accentuate. Put together, these two limitations underscore the difficulties in achieving a "clean" manipulation that facilitates straightforward interpretation of biomedical versus psychosocial group differences. For example, it is possible that the increased stereotyped attitudes in the psychosocial condition are not attributable to a "ticking time bomb" effect but instead to appraisal of a kind of "double whammy." That is, participants may have attributed the target person's struggles to the joint salience of biological defect (implied in our use of the diagnostic term *schizophrenia*) *and* profound psychosocial stress.

Third, our undergraduate sample limits the generalizability of our results. Moreover, although we collected no data pertaining to our participants' mental health, a great deal of recent scholarship points to a sharp increase in psychological distress among college students (e.g., Center for Collegiate Mental Health, 2018). It is possible that our undergraduate sample possessed an idiosyncratic set of beliefs about mental illness that may have, in turn, given rise to idiosyncratic intervention outcomes. Future research in this area should recruit samples more

representative of the general population and evaluate intervention effects as a function of participants' pre-intervention causal beliefs about mental illness.

Fourth, our design did not employ a schizophrenia control group. This condition would have aided interpretation of biomedical versus psychosocial group comparisons. For example, for stereotyped attitudes, a psychosocial causal explanation increased stigma *relative to* a biomedical causal explanation. But addressing the question of whether it increases stereotyped attitudes in an *absolute* sense would require reference to a schizophrenia vignette that is agnostic with respect to causal explanation. This same limitation would render untenable the conclusion that a biomedical causal explanation decreases stereotyped attitudes. Indeed, a growing literature on essentialist belief (Haslam & Ernst, 2002) indicates that biomedical causal explanations are problematic in their own right. Finally, the critical comparison at the heart of our study – biomedical versus psychosocial causal explanations – invokes a false dichotomy. Biopsychosocial models of most psychiatric problems accommodate both biological causes and environmental stressors. Studies that have examined the stigma-related consequences of biopsychosocial or otherwise integrative explanations for psychological problems have yielded mixed results (Boysen & Gabreski, 2012; Deacon & Baird, 2009; Walker & Read, 2002).

In sum, the current study obtained no evidence that a psychosocial causal explanation of schizophrenia led to decreased stigma compared to a biomedical causal explanation. In fact, the psychosocial causal explanation even increased endorsement of damaging schizophrenia stereotypes. Additional research is needed to evaluate the tentative interpretation we advanced regarding the possibly damaging consequences of describing people as "ticking time bombs" subject to numerous, overwhelming stressors.

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Table 1

Effects of the Experimental Manipulation (Biomedical, Psychosocial, Diabetes Control) on the Manipulation Check, IAT D *Scores, and Self-Report (Conventional vs. Projected Other) Measures of Psychiatric Stigma*

	Biomedical	Psychosocial	Diabetes Control
Manipulation Check			
Biomedical Causal Beliefs	3.4 (0.4)	2.7 (0.7)	3.0 (0.4)
Psychosocial Causal Beliefs	2.2 (0.7)	3.5 (0.6)	2.9 (0.6)
IAT *D* scores	0.07 (0.5)	0.15 (0.5)	0.23 (0.5)
Social Distance			
Conventional	2.7 (0.4)	2.8 (0.5)	1.8 (0.7)
Projected Other	3.3 (0.3)	3.4 (0.4)	2.3 (0.6)
Fear			
Conventional	2.5 (0.8)	2.5 (0.9)	2.0 (0.7)
Projected Other	3.8 (0.7)	3.7 (0.8)	2.6 (1.0)
Anger			
Conventional	1.9 (0.6)	1.8 (0.7)	1.7 (0.6)
Projected Other	2.9 (0.8)	2.6 (0.7)	2.4 (0.7)
Prosocial Emotion			
Conventional	3.7 (0.7)	3.8 (0.7)	3.0 (1.0)
Projected Other	3.4 (0.7)	3.5 (0.9)	3.3 (1.0)
Stereotyped Attitudes			
Conventional	1.0 (0.7)	1.5 (0.9)	0.4 (0.6)
Projected Other	2.0 (1.2)	2.4 (1.1)	0.9 (1.1)

Note. Means (SDs) reflect the original measurement scales. Manipulation check (1 = definitely disagree, 4 = definitely agree). IAT *D* scores (greater positive values reflect greater implicit association of “Dennis” with “safe”). Social distance (1 = definitely willing, 4 = definitely unwilling). Fear, anger, prosocial emotion (1 = strongly disagree, 5 = strongly agree). Stereotyped attitudes (-6 = maximum stereotyped attitudes for “Average Man” versus “Dennis,” +6 = maximum stereotyped attitudes for “Dennis” versus “Average Man”).

Appendix A

BIOMEDICAL CONDITION

Article Title: The Neurobiology of Schizophrenia: A Review of Research
Highlights:

- This paper reviews evidence that neurobiological factors cause schizophrenia.
- Research dating back to the 1960s consistently points to several functional and structural abnormalities of the brains of individuals with schizophrenia.
- Three abnormalities appear to be most common – chemical imbalance of the neurotransmitter dopamine, reduced volume of the prefrontal cortex, and enlarged ventricles.
- A large body of genetic evidence also suggests that schizophrenia is a genetic disorder.
- Treatment of schizophrenia must involve drug interventions or other therapies aimed at correcting the neurobiological problems that cause the disorder.

PSYCHOSOCIAL CONDITION

Article Title: Schizophrenia and Stressful Life Events: A Review of Research
Highlights:

- This paper reviews evidence that stressful life events cause schizophrenia.
- Research dating back to the 1960s consistently demonstrates that stressful life events precede and cause episodes of schizophrenia.
- Commonly reported stressful life events include the death or major medical illness of a loved one; military combat; sudden, unexpected unemployment; and severe problems in relationships.
- In addition to their role in schizophrenia onset, stressful life events worsen symptoms of ongoing episodes of schizophrenia.
- Treatment of schizophrenia must involve interventions aimed at helping individuals manage the severe stressors that cause the disorder.

DIABETES CONTROL CONDITION

What is Diabetes? (National Institute of Diabetes and Digestive and Kidney Diseases, 2013)

Diabetes is when your blood glucose, also called blood sugar, is too high. Blood glucose is the main type of sugar found in your blood and your main source of energy. Glucose comes from the food you eat and is also made in your liver and muscles. Your blood carries glucose to all of your body's cells to use for energy.

Your pancreas—an organ, located between your stomach and spine, that helps with digestion—releases a hormone it makes, called insulin, into your blood. Insulin helps your blood carry glucose to all your body's cells. Sometimes your body doesn't make enough insulin or the insulin doesn't work the way it should. Glucose then stays in your blood and doesn't reach your cells. Your blood glucose levels get too high and can cause diabetes or prediabetes.

Over time, having too much glucose in your blood can cause health problems.

Appendix B

BIOMEDICAL CONDITION

This is Dennis. Dennis is a 31-year-old man. About a year ago, Dennis started thinking that people around him were spying on him and trying to hurt him. He became convinced that people could hear what he was thinking. He also heard voices when no one else was around. These voices told him what to do and what to think. Sometimes he thought people on TV were sending secret messages intended only for him. His thoughts also became very jumbled and confused; in fact, other people began having a difficult time understanding what Dennis was saying. After living this way for several weeks, Dennis visited a **doctor**, who told him that he had a **disease** called schizophrenia.

Dennis underwent some medical testing that revealed the underlying causes of his schizophrenia. His **doctor** determined that Dennis had dysfunctional levels of dopamine and glutamate, important brain chemicals that scientific evidence has linked to schizophrenia. Brain scans also determined that Dennis has an unusual brain structure; for instance, the part of his brain that controls the ability to form complex thoughts is dramatically reduced in size. Finally, genetic testing indicated that Dennis has four genes shown to relate to schizophrenia. Additional evidence of faulty genes is that Dennis's maternal grandmother and maternal uncle both had schizophrenia. So, it's clear that the disease runs in Dennis's family.

PSYCHOSOCIAL CONDITION

This is Dennis. Dennis is a 31-year-old man. About a year ago, Dennis started thinking that people around him were spying on him and trying to hurt him. He became convinced that people could hear what he was thinking. He also heard voices when no one else was around. These voices told him what to do and what to think. Sometimes he thought people on TV were sending secret messages intended only for him. His thoughts also became very jumbled and confused; in fact, other people began having a difficult time understanding what Dennis was saying. After living this way for several weeks, Dennis visited a **counselor**, who told him that he had a **problem** called schizophrenia.

Dennis had some discussions with his **counselor** that revealed the underlying causes of his schizophrenia. Dennis shared that his parents died seven weeks apart earlier in the year. This was very upsetting to Dennis because he had a close relationship with both of them. Dennis also indicated that he was under a great deal of stress at work. After a fight with his boss and three coworkers, he was demoted and had his salary cut in half. Dennis began struggling to make ends meet; for example, several times he had to obtain food at a food pantry. Dennis's **counselor** explained that these kinds of severe stressors have been shown to cause schizophrenia.

DIABETES CONTROL CONDITION

This is Dennis. Dennis is a 31-year-old man. About a year ago, Dennis started experiencing a variety of physical symptoms. He began feeling tired all of the time. He also started feeling unusually thirsty, and he began having to urinate more than he had been used to. Dennis's weight began to go up and down even though he hadn't changed anything about his diet. He started feeling tingling in his hands and feet, and although it wasn't extremely painful, he found it very

distracting. Finally, Dennis started developing tender gums that sometimes made eating and brushing his teeth unpleasant. After living this way for several weeks, Dennis visited a doctor, who told him that he had a disease called type-II diabetes.

Dennis had some discussions with his doctor that revealed the underlying causes of his type-II diabetes. His doctor explained that type-II diabetes is when your blood glucose, also called blood sugar, is too high. He explained that an organ called the pancreas releases a hormone called insulin into the blood. Insulin helps blood carry glucose to all the body's cells. Sometimes the body doesn't make enough insulin or the insulin doesn't work the way it should. Glucose then stays in the blood and doesn't reach the body's cells. Dennis's doctor said that his blood glucose levels got too high and caused his type-II diabetes.