The Blame Game:

Lay causal theories and familiarity with mental illness

Margaret A. Martinez

Senior Thesis in Psychology

Advisor: Susan Nolen-Hoeksema

April 19, 2010

Abstract

The present study focuses on lay theories of causality in mental illness, specifically whether or not etiological theories vary as a function of one's exposure to populations with mental health problems. Individuals who knew someone who received treatment for a mental health problem, or who had personally received treatment, were more likely to endorse biological theories of causality than those individuals without this familiarity to mental health problems. This tendency to endorse biological theories of causality was correlated with the perceived severity of the other person's mental health problem and with the amount of distress caused by that person's mental health problem. While this study was unable to clarify the reasons behind the association between familiarity with mentally unhealthy individuals and endorsement of biological etiologies, it has important implications for interventions aimed at reducing the stigma of the mentally ill. Most significantly, I suggest that behavioral contact may be necessary to prevent biological theories of causality from being associated with stigmatizing attitudes and behaviors.

The Blame Game:

Lay causal theories and familiarity with mental illness

People have a wide variety of theories of what leads to good and bad health. However, most of the literature on lay theories of health focuses on physical health, rather than mental health (see Calnan, 1987). This literature attempts to answer questions such as: why do people think they get sick? How do different individuals experience illness? How do they believe they will get better? Do they believe medical professionals can help them improve? (Nettleton, 2006). In the last two decades there has been a significant effort to extend this research to include mental health as well and answer these same questions as they relate to mental health and illness.

As a conceptual framework for lay theories of mental illness, Jorm and colleagues (1997) coined the term "mental health literacy," defined as "knowledge and beliefs about mental disorders which aid their recognition, management or prevention" (Jorm, 2000). The construct of mental health literacy is multi-faceted and incorporates several components of lay theories of mental illness: the recognition of symptoms; knowledge and beliefs about risk factors and causes; knowledge and beliefs about self-help interventions; knowledge and beliefs about professional interventions; attitudes that facilitate seeking help when help is necessary; and knowledge of how to obtain information regarding mental health. The present study focuses on one facet of mental health literacy, lay theories of causality, and investigates whether these theories vary as a function of people's familiarity with mental illness.

Causal Theories Held by the General Public

Studies of the general public suggest that lay theories of the causes of mental illness are, for the most part, "elaborate, consensual, and moderately accurate with respect to academic theories" (Kim & Ahn, 2002, p. 34). Jorm (2000) reports that psychosocial stress is the favored cause for mental illness, whereas biological factors are less frequently endorsed. Psychosocial stressors commonly cited as causal in the development of psychopathology include life events (such as the death of a loved one), relationship problems, and financial or work-related strain. These psychosocial stressors are commonly endorsed as causal for both depression and schizophrenia. Biological causes are implicated less frequently for both disorders, although they are more frequently endorsed in lay causal theories of schizophrenia than depression (Angermeyer & Dietrich, 2005; Angermeyer & Matschinger, 2005; Jorm, Christensen, & Griffiths, 2005; Jorm, 2000). It is possible that psychosocial causes are endorsed more frequently because they are better understood by the lay public, whereas biological theories might seem more abstract and are therefore endorsed less frequently. Alternatively, the lay public might truly believe that the psychosocial plays a greater role in causing mental illness than the biological, for all disorders.

This proclivity for psychosocial theories of causation over biological ones is not a uniquely western phenomenon; similar preferences have been demonstrated in studies using non-Western samples as well. For example, Swami, Furnham, Kannan, and Sinniah (2008) demonstrated that Malaysians prefer social-environmental causes for schizophrenia to biological causes for the same disorder. Adewuya and Makanjuola (2008) performed a similar study with a Nigerian sample. The authors demonstrated a strong preference for supernatural explanations of mental illness; subjects also frequently

believed mental illness to be the result of misuse of psychoactive substances. This pattern was especially pronounced for older individuals living in rural areas who were unfamiliar with the concept of "mental illness." Taken together, these studies demonstrate the similarities – and discrepancies – between different cultures' lay causal theories.

Mental health professionals, on the other hand, more frequently conceptualized psychopathology in terms of the medical model (Link et al., 1999; Jorm et al, 1997b; Jorm, 2000; Angermeyer and Dietrich, 2006). Furnham and Bower (1992) state that the biological model of schizophrenia is "the dominant…conceptual model for the understanding of [schizophrenia]" (p. 202); the authors claim that mental health professionals prefer the medical model to the moral-behavioral, psychoanalytic, social, and conspiratorial models of schizophrenia (the conspiratorial model being that "schizophrenia" is a stigmatizing term applied to people whose behavior is radical or eccentric, not actually a disorder). However, they found that lay participants did not share the same beliefs as professionals. Lay respondents more often attributed the etiology of schizophrenia to stress than to biomedical causes, and on the whole they preferred the psychoanalytic to the medical model. They also borrowed from other models, including the social and conspiratorial models, when asked about the appropriate treatment for schizophrenia. These results demonstrate that lay people often do not have one coherent theory regarding schizophrenia. Rather, they pick and choose from the variety of existing theories.

The public's understanding of biological causes seems to be changing in the wake of the introduction of Direct-to-Consumer (DTC) advertising in the United States,

according to a study by France, Lysaker, and Robinson (2007). DTC advertising, introduced in the U.S. in 1997, allows pharmaceutical companies to market their products directly to consumers in the form of television and magazine advertisements or promotional products such as pens or notepads. DTC advertisements for antidepressants in particular often portray depression as a strictly biological disease that their products can treat. Although 91.6% of the participants in this study reported being familiar with the "chemical imbalance" theory of depression propagated by pharmaceutical companies, they were still more likely to endorse certain psychosocial stressors as causal over biological ones. These results suggest that the public is increasingly becoming aware of the role of biological factors in mental illness but still favors an understanding that places a premium on psychosocial and environmental forces. An identical preference for psychosocial factors over the biological has been demonstrated among people in countries where DTC advertising is not legal (Jorm, Christensen, & Griffiths, 2005; Holzinger et al., 2003; Read & Harré, 2001).

The Role of Familiarity on Lay Causal Theories

Familiarity with mental illness does seem to play an important role in lay theories of causality. Angermeyer and Matschinger (1996) report that the relatives of people with schizophrenia are more likely than the lay public to endorse biological or constitutional factors in the development of the disorder; this tendency was most pronounced in German (as opposed to Austrian) mothers who were the primary caretaker of their suffering child. The authors speculate that this could be a result of their increased exposure to mental health professionals or it could be a coping mechanism that minimizes the guilt they feel at seeing their child suffer. In an earlier study by

Angermeyer and Klusmann (1988), mothers of the mentally ill favored biological causes over upbringing and other environmental causes, however they still ranked recent psychosocial stressors and personality factors over both biology and upbringing.

Again we can see the influence of culture, however, as evidenced by a study by Kurihara, Kato, Reverger, and Tirta (2006). This study, conducted in Bali, demonstrated a preference for supernatural causal explanations among relatives of patients with schizophrenia. Those relatives who endorsed supernatural, as opposed to natural, causes were more likely to be older, less educated, and have family members who were never treated for their disorder. This research suggests that a preference among family members of the mentally ill for biological theories of causation may be a Western phenomenon.

Limited research has been done within the United States that looks at causal lay theories as a function of experience with or exposure to mental illness. What little has been done focuses on particular demographic groups, such as Esterberg and Compton's work with the African-American community. Esterberg and Compton (2006) surveyed family members of urban African-American inpatients hospitalized for schizophrenia. They found that three of the top five most frequent causes most frequently endorsed were biological in nature, and the average number of endorsements was 8.3 (that is on average, respondents endorsed 8.3 causes of schizophrenia, rather than selecting just one). This suggests that relatives of African-Americans suffering from schizophrenia hold causal beliefs that are primarily biological and multi-factoral. The authors speculate that the increased valued placed on biological causes is a result of the family's exposure to the mental health system.

Even less work has been done that examines how individuals understand the causes of their own mental illness. In one of the few studies to ask this question, Angermeyer and Klusmann (1988) found that patients, like professionals, made multiple causal attributions for their illness. However, they endorsed recent psychological factors, personality issues, and family causes over biological causes. There was a tendency for those patients suffering from affective psychoses to endorse biological causes more frequently than those suffering from schizophrenia or schizoaffective disorder. To my knowledge, there has been no work investigating the progression of patient causal theories in the wake of DTC advertising in the United States.

This research hoped to address the following questions: are people with first-hand knowledge of mental illness (i.e. people who know someone who had a mental health problem) more likely to endorse biological causes for mental health problems? Does this tendency vary by the distress caused by the other person's poor mental health? Do causal endorsements vary with the perceived severity of the other person's mental health problem? Are primary consumers (i.e. those individuals who have personally received treatment for a mental health problem) more likely to attribute their own mental health issues to biological causes? My goal was to help bridge the gaps in this rather disparate literature. Whereas the majority of the work on lay causal theories has been done with populations from Europe, Australia and New Zealand, I utilize American data from the General Social Survey (GSS) in an attempt to generalize the results of previous studies. Furthermore, where the literature considers lay causal theories of family members it focuses primarily on schizophrenia and depression. Because the GSS dataset does not differentiate between disorders, this study considered a wider range of disorders. Finally,

because I examined patients' (also referred to as primary consumers) lay causal theories, this study has the potential to further the research on how patients think about their own disorders.

In answer to these questions, I hypothesized that: (1) participants who know someone with a mental health problem will be more likely to endorse biomedical causes than participants who do not report the same personal experience with mental disorders. Those participants with no reported personal contact with mental health problems should be more likely to endorse other causes, including psychological causes or forces outside the individual's control. I further hypothesize that (2) participants who report having been more distressed by their personal experience with mental health problems will be more likely to endorse biological causes than participants who report less distress, since they will have a greater need to displace blame onto an external factor; and (3) participants who report personally having experienced a mental health problem will be more likely to endorse biological causes than those individuals who have not experienced and received treatment for a mental health problem. I believe that biological causal factors such as genetics or brain disease serve an important function for individuals who know someone with a mental health problem or who have personally experienced a mental health problem: attributing the cause of the mental health problem to a biological factor places it outside of their control and thus displaces blame from the sufferer and his or her loved ones.

Methods

The data for this study come from the General Social Survey (GSS), a publicly available dataset that has tracked societal trends from 1972 to the present. The GSS is

part of the National Data Program for the Social Sciences, started in 1972 by the National Opinion Research Center (NORC), a social science research center at the University of Chicago. The complete dataset is available online and grants researchers access to "a standard 'core' of demographic, behavioral, and attitudinal questions, plus topics of special interest" (GSS Website, http://www.norc.org/GSS+Website). The questions pertaining to mental health (considered a "topic of special interest") were of interest for the purposes of this study.

Methods of GSS Data Collection

The data that make up the GSS dataset are collected via interview. According to the GSS Codebook (2009, NORC), over 53,000 interviews have been conducted to date. Until 2004, participants were an independently drawn sample of English-speaking adults living in non-institutionalized settings across the United States; starting in 2006, Spanish-speaking adults with the same living arrangements were included in the sample.

Subset of Data

This study used GSS data collected in 2006, specifically data from the 2006 mental health topical module. The data analyzed here were collected using full probability sampling. Variables considered in the analyses include: whether the respondent knew someone who had received treatment for a mental health problem (to assess closeness to an individual with a mental illness); whether that person's problem was caused by bad character, bad luck, a genetic problem, God's will, brain disease, stress, or upbringing (to assess causal theories); how severe the individual's mental health problem was, as determined by the respondent; the amount of distress this individual's mental health problem caused the respondent; and whether the respondent had personally

experienced and received treatment for a mental health problem. Participants' endorsement of the causal factors was assessed on a four-point Likert scale, with one representing "very likely" and four representing "not at all likely." The other continuous variables (severity of other's mental health problem and amount of distress caused by other's mental health problem) were likewise assessed on 4-point Likert scales, where lower numbers indicated higher levels of severity and distress and larger numbers indicated lower levels of severity and distress. The precise wording of each question can be found in Appendix A. The number of participants who answered each question varied from question to question: 1417 individuals answered either yes or no to the question, "have you personally ever known someone who has received treatment for a mental health situation?" Of these, between 941-961 individuals answered that they knew someone who had been treated for a mental health problem and were included in the analyses. With regard to personal mental health problems, 1413 individuals answered either yes or no to the question, "have you personally ever received treatment for a mental health problem?" Of these, between 204-212 answered that they had personally received treatment for a mental health problem and were included in the analyses.

Participants

Among those participants who answered yes or no to the question "have you personally ever known someone who has received treatment for a mental health situation?," 47% were males (N=662) and 53% were females (N=755). Seventy-six percent identified as white (N=1079), 15.1% as Black (N=214), and 8.8% as other (N=124). With regard to age, 1.3% were 18-19 (N=19), 14.4% were in their 20s (N=203), 14.1% were in their 30s (N=278), 23% were in their 40s (N=324), 18.1% were

in their 50s (N=256), 11.8% were in their 60s (N=168), 7.1% were in their 70s (N=101), and 4.2% were 80 or older (N=60). Forty-nine percent were married (N=694), 7.4% were widowed (N=105), 17% were divorced (N=240), 3.3% were separated (N=46), and 23.2% had never married (N=328). The majority of them had completed high school (54%, N=762); 12.9% had completed some high school (N=182), 7.4% attended junior college (N=105), 16.5% had a bachelor's degree (N=234), and 9.3% had a graduate degree of some kind (N=132). Seven percent of participants reported a total family income of $9,999 a year or less (N=100), 10.9% reported an income of between $10,000-$19,999 (N=147), 12.2% of between $20,000-$29,999 (N=166), 10.7% of between $30,000-$39,999 (N=146), 9.5% of between $40,000-$49,999 (N=129), 7.5% of between $50,000-$59,999 (N=102), 14.9% of between $60,000-$89,999 (N=203), 17% of between $90,000-$149,999 (N=160), 5.3% of over $150,000 (N=73), and 10.2% refused (N=139).

Among those participants who answered yes or no to the question "have you personally ever received treatment for a mental health problem?," 47% were males (N=658) and 53% were females (N=755). Seventy-six percent identified as white (N=1077), 15% as Black (N=212), and 8.8% as other (N=124). With regard to age, 1.4% were 18-19 (N=19), 14.4% were in their 20s (N=203), 19.6% were in their 30s (N=277), 23.1% were in their 40s (N=323), 18% were in their 50s (N=252), 12.1% were in their 60s (N=169), 7.1% were in their 70s (N=101), and 4.3% were 80 or older (N=61). Forty-nine percent were married (N=692), 7.4% were widowed (N=105), 17% were divorced (N=239), 3.3% were separated (N=47), and 23.2% had never married (N=327). The majority of them had completed high school (54%, N=762); 12.9% had completed some

high school (N=182), 7.4% attended junior college (N=105), 16.4% had a bachelor's degree (N=232), and 9.3% had a graduate degree of some kind (N=131). Seven percent of participants reported a total family income of $9,999 a year or less (N=100), 10.4% reported an income of between $10,000-$19,999 (N=147), 11.7% of between $20,000-$29,999 (N=166), 10.7% of between $30,000-$39,999 (N=146), 9.4% of between $40,000-$49,999 (N=128), 7.6% of between $50,000-$59,999 (N=103), 14.9% of between $60,000-$89,999 (N=203), 11.7% of between $90,000-$149,999 (N=160), 5.2% of over $150,000 (N=71), and 10.1% refused (N=138).

Results

Multivariate analysis of variance was used to compare the causal endorsements of those who knew someone who had received treatment for a mental health problem to the causal endorsements of others without this experience. The test suggested a significant difference between these groups, $F(7,1302) = 5.205$, $p<.001$ (see Table 1). Follow-up univariate analyses revealed that the groups differed significantly on their endorsements of bad character, $F(7,1302)=10.348$, $p=.001$, brain disease $F(7, 1302)=11.018$, $p=.001$, and upbringing $F(7, 1302)=5.285$, $p=.022$. The direction of these between-group differences were such that those individuals who knew someone who had received treatment for a mental health problem were significantly more likely to endorse brain disease and upbringing and less likely to endorse bad character than those individuals who did not know someone who had received treatment for a mental health problem.

Next, Pearson's correlations were used to examine whether the causal theories endorsed varied with the severity of the other person's mental health problem (see Table 2). All seven causal theories were considered (bad luck, God's will, bad character, stress,

way raised, genetic problem, brain disease; all seven of these will be included in all of the following analyses). The tests revealed that those respondents who found the other person's mental health problem to be more serious were significantly less likely to believe the individual's problem was a result of their bad character (N=956, r=-.096, p=.003), more likely to believe the other individual's problem was the result of a genetic problem (N=945, r=.080, p=.014), and less likely to believe it was the result of God's will (N=946, r=-.078, p=.016). There were no significant correlations between perceived severity and endorsements of bad luck, brain disease, stress, or upbringing. Again, it appears that as an individual is exposed to more severe forms of mental illness, s/he is more likely to endorse biological theories and less likely to blame the victim.

A second series of Pearson correlations examined the relationship between the amount of distress experienced by the respondent as a result of this other person's mental health problem and causal theories (see Table 2). These tests revealed that those respondents who experienced more distress were significantly less likely to believe the other person's mental health problem was a result of his or her bad character (N=957, r=-.111, p=.001). They were also significantly more likely to believe the other person's problem was the product of a brain disease (N=943, r=.065, p=.047). There were no significant correlations between the amount of distress experienced by the respondent and endorsements of bad luck, genetics, God's will, stress, or upbringing. As with the severity of the other person's mental health problem, increased levels of distress were thus associated with an increased likelihood to endorse biological causes and a decreased likelihood to attribute the person's suffering to his or her bad character.

An additional multivariate analysis of variance compared the causal endorsements of respondents who had personally received treatment for a mental health problem to the causal endorsements of those respondents who had not (see Table 3). The overall significance suggested variance between these two groups, $F(7, 1297)=3.585$, $p=.001$. Follow-up univariate analyses of the causal endorsements revealed significant between-group differences on endorsements of bad character ($F(7, 1297)=14.999$, $p=.000$), genetics ($F(7, 1297)=6.321$, $p=.012$), and brain disease ($F(7, 1297)=4.181$, $p=.041$). The direction of the between-group differences indicated that individuals who had personally been treated for a mental health problem were more likely to endorse genetics and brain disease and less likely to endorse bad character than were those individuals who had not been treated for a mental health problem.

Finally, paired means t-tests compared within-group causal endorsements of biological versus other causes. Those who knew someone who had been treated for a mental health problem were significantly more likely to endorse genes than bad character, $t(943)=25.22$, $p=.000$; brain disease than bad character, $t(939)=18.13$, $p=.000$; stress than genes, $t(945)=-8.71$, $p=.000$; stress than brain disease, $t(943)=-14.37$, $p=.000$; and stress than bad character, $t(952)=-37.75$, $p=.000$. Those who had personally been treated for a mental health problem were significantly more likely to endorse stress than genes, $t(204)=-2.25$, $p<.05$; stress than brain disease, $t(203)=-5.61$, $p=.000$; genes than brain disease, $t(199)=-3.67$, $p=.000$; genes than bad character, $t(204)=15.32$, $p=.000$; brain disease than bad character, $t(203)=10.97$, $p=.000$; and stress than bad character, $t(208)=-19.04$, $p=.000$.

Discussion

These results demonstrate that respondents who knew someone who had been treated for a mental health problem or who had been treated for a mental health problem of their own are more likely to attribute their mental health problems and those of their loved ones to biological causes, including genetics and brain disease, than respondents with no personal exposure to mental health problems. Those respondents who knew someone who had received treatment for a mental illness were more likely to endorse brain disease as the cause for that person's difficulty than respondents who did not know someone. As the severity of the other's mental health problem (as perceived by the respondent) increased, so did the likelihood that respondents would endorse genetics as the cause of their loved one's problems. Furthermore, the more distress caused by the other person's mental health problem, the more likely the respondent was to endorse brain disease as a causal factor. Finally, respondents who had themselves received treatment for a mental health problem were more likely to endorse both genetics and brain disease as causal compared to respondents who had never received treatment for a mental health problem.

Respondents were significantly more likely to endorse biological factors than factors that might place blame on the patient, such as that person's bad character. Those respondents who knew someone who had received treatment for a mental heath problem were less likely than those respondents who did not know someone who had received treatment to endorse bad character as the cause of the individual's problems; these same respondents also felt that it was less likely that God's will caused the other person's problems. Decreased likelihood of endorsement of causal factors that "blame the victim," such as bad character, was also observed as the perceived severity and amount of

distress increased. That is, as the perceived severity of the other's mental health problem and the amount of distress experienced by the respondent as a result of the other's mental health problem increased, the respondents' tendency to endorse bad character as a causal factor decreased. Likewise, respondents who had received mental health treatment were less likely to endorse their own bad character as the cause of their problems.

Those respondents who knew someone treated for a mental health problem were more likely than those respondents who did not know someone to endorse the individual's upbringing as playing a causal role in their illness. This finding suggests that the lay public considers both biological and environmental factors causal in the development and maintenance of mental disorders, as others have suggested (Angermeyer & Dietrich, 2005; Angermeyer & Matschinger, 2005; Jorm, Christensen, & Griffiths, 2005; Jorm, 2000). The results of the current study suggest that the public considers multiple factors as causal in mental health problems, including stress, biological factors and environmental variables.

The present study clarifies existing literature with regard to lay causal theories as a function of one's exposure to mental illness. By utilizing recent data collected from a large and diverse U.S.-based sample, these results demonstrate the extent to which biological explanations of mental illness have gained favor in the last several decades. Whereas Angermeyer and Klusmann (1988b) showed that mothers of the mentally ill ranked psychosocial stressors and personality characteristics over biological and familial factors, the present results demonstrate that eighteen years later those who know someone being treated for a mental health problem were more likely to endorse biological causes than psychosocial, personality, or familial causes. In this regard, these results are more in

accordance with those of Angermeyer and Matschinger (1996), who demonstrated that relatives of individuals with schizophrenia are more likely to endorse biological or constitutional causal factors than the lay public. These results establish that, ten years later, the between-group differences amongst individuals who do and do know someone who has been treated for a mental health problem still exist with regard to biological causal theories: the former group endorses biological causes as more likely than does the latter group. The recent work of Esterberg and Compton (2006), in combination with the results reported here, further supports this group difference. Esterberg and Compton discovered that their sample of African-American relatives of inpatients hospitalized for schizophrenia was more likely to endorse biological causes. The current results demonstrate a similar preference in a demographically more diverse sample.

With regard to theories of causality held by individuals who have themselves experienced a mental illness, the present study provides a much-needed follow-up to the work of Angermeyer and Klusmann (1988a). Angermeyer and Klusmann's study demonstrated that patients, like their relatives, endorsed recent psychosocial, personality, and familial causes over biological explanations of their illnesses. In contrast, the present study shows that patients have adopted a more biological understanding of the causes of their illnesses, as have those who know them. Personality characteristics such as bad character now seems less likely than biological factors in the minds of those who have experienced a mental health problem. Interestingly enough, psychosocial stressors still seem most likely in the minds of these individuals, a trend that has not changed with time. These results demonstrate a fundamental difference in the way people who have

first-hand knowledge and people without this familiarity think about the causes of mental health problems.

One may attempt to make sense of these results in several ways. As suggested by some (Angermeyer & Matschinger, 1996; Esterberg & Compton, 2006), the tendency for those who know someone who has received treatment for a mental health problem to endorse biological causes more frequently than those who do not have the same experiences might reflect an increased exposure to the mental health system. As discussed earlier, mental health professionals frequently conceptualize mental illness in terms of biological causes (Furnham & Bower, 1992; Link et al., 1999; Jorm et al, 1997b; Jorm, 2000; Angermeyer & Dietrich, 2006). Mental health professionals might pass on their etiological theories to their patients; this could explain why those who have exposure to the mental health system, be it direct or indirect, are more likely to display causal endorsements similar to those of mental health professionals.

Additionally, higher endorsement of biological causes may reflect the effect of direct-to-consumer advertising on patients and their relatives. France, Lysaker, and Robinson (2007) showed that the general public was still more likely to endorse psychosocial stressors over biological causes despite being familiar with the "chemical imbalance" theory of depression put forth by the pharmaceutical companies; these results suggest that patients and their friends and family members may have been affected by DTC advertising differently. For those individuals for whom mental illness is an issue of some relevance, such as the respondents in the current study, DTC promotions of biological causes may have made them seem more likely than character or environmental factors. If this is true, DTC marketing does not seem to have been powerful enough to

overcome the public's view that stress causes mental health problems, as stress was rated as a more likely cause than biological causes in both the France, Lysaker and Robinson study and the present study.

Thirdly, we might speculate that knowing an individual who has received treatment for a mental illness (or receiving treatment oneself) might allow the respondents to rule out certain etiological theories by direct observation. That is, knowing a patient's history and personality might provide respondents with the information to negate bad character or upbringing as causal factors in their mental health problems. Having eliminated these factors, biological etiologies might seem more likely.

Finally, greater endorsement of biological etiologies among those who know someone who has received treatment for a mental health problem and among those who have themselves received treatment for a mental health problem may indicate a protective function of biological theories of causality. This explanation has been tentatively suggested by Angermeyer and Matschinger (1996), and the current study strengthens this theory as a plausible explanation. It is possible that viewing the etiology of mental illness as biological helps the mentally ill and those who know them displace the blame onto a more neutral source: rather than blaming one's upbringing or character for one's mental health problems, biological theories of causality allow individuals to explain suffering in terms of factors outside of one's control. In short, biological causes allow the respondents in this study to "blame it on biology," which may be viewed as more out of the individual's control than personality factors.

While the use of a large and diverse American sample is a strength of this study, the study also has limitations. First and foremost, this work is purely correlational; since

no experimental manipulations were done, we are limited in our ability to make causal conclusions about the patterns observed in these data. In other words, it is impossible to conclude from these results that exposure to mentally ill individuals causes individuals to endorse biological etiologies more strongly.

Secondly, the use of an existing dataset placed constraints on the questions that could be asked and the ways in which they could be answered. Most notably, I was restricted to the seven causal variables included by the authors of the GSS (bad luck, God's will, bad character, stress, way raised, genetics, and brain disease); while these seven are generally representative of the variables used in the wider literature, it is plausible that the results would have come out differently if the respondents had been able to answer freely. Additionally, the questions asked about "mental health problems" in the abstract and did not differentiate between disorders; thus, it is unclear whether the "problems" the respondents had in mind correspond to legitimate psychiatric diagnoses. By considering mental health problems as a general category, this wording has the potential to allow us to look at disorders other than schizophrenia and major depression, the two disorders considered most frequently in the literature. However, because of the wording of the questions it is impossible to ascertain whether lay causal theories differ by diagnosis.

Finally, the nature of the dataset did not give us information as to whether one variable was preferred over another. While the paired means t-tests allowed us to compare one variable to another directly, respondents were never asked whether they preferred cause *x* to cause *y*. Thus, we can make no conclusions as to whether biological

factors are preferred over psychosocial or environmental factors per say, only that they are endorsed more frequently by these particular populations.

This work has critical implications for research on reducing the stigma faced by mentally ill individuals. The present study forces us to reconsider how best to minimize this stigma and the mechanisms by which stigma might successfully be diminished. Over the last several decades, many researchers have attempted to address the question of how we might reduce stigma of the mentally ill. Corrigan and Penn (1999) group interventions designed to reduce the stigma associated with mental illness into three categories: demonstrations against prejudice; public education; and behavioral contact. Of these three, contact with a mentally ill individual appears to be the most effective in reducing stigmatizing beliefs held by the lay public, or so suggest Corrigan and Penn (1999) and Hinshaw and Cicchetti (2000). Indeed, a meta-analysis by Kolodziej and Johnson (1996) demonstrated the efficacy of behavioral contact in reducing the stigmatizing attitudes of mental health employees and of students (see also Read & Law, 1999; Read & Harré, 2001). Taken together with the present results, one might conclude that individuals who know people with mental health problems – such as the respondents in the current study – hold less stigmatizing attitudes towards the mentally ill. These results demonstrate that their causal beliefs in particular tend towards the biological and might suggest that biological theories of causality are associated with reduced stigma. One should bear in mind that comments regarding the implications of this research on stigmatizing attitudes are purely speculative, given that the present study did not measure stigma directly.

Unfortunately, the picture is not so simple. A growing body of literature suggests that biological etiological theories – that is, viewing mental illness in terms of the disease

model of illness – does not in fact reduce stigmatizing attitudes and behaviors. In their 1997 study, Mehta and Farina demonstrated that individuals' mental schemas of mental illnesses affected their behavior towards an individual with mental illness. Those participants (all male) who were led to believe that the confederate's "mental illness" had biological origins gave shocks of greater intensity and duration to the confederate than did those men in the psychosocial or control conditions, even while they said nice things about the confederate and did not believe they were hurting him. Additional work by Read and colleagues has shown correlations between biological and genetic causal beliefs and negative attitudes towards "mental patients" (Read & Law, 1999; Read & Harré, 2001). This line of research suggests that viewing the etiology of mental disorders in biological terms may not be effective in reducing stigmatizing attitudes and behaviors.

Thus, the present results are situated at the opposition of two seemingly contradictory bodies of literature: that which suggests that behavioral contact is the best way to reduce stigma; and that which suggests that biological theories of causality are in fact correlated with stigmatizing attitudes and behaviors. In this study, individuals who knew someone who had received treatment for a mental health problem (or who had themselves received treatment for such a problem) more strongly endorsed biological theories of causality, such as genetics or brain disease, the very same etiological theories that have been associated with increased stigma. The work of Mehta and Farina (1997), Read and Law (1999), and Read and Harré (2001) suggests that their biological etiological theories make them more prone to stigmatizing attitudes and behaviors. However, because of their direct contact with persons with mental health problems,

Kolodziej and Johnson (1996), Corrigan and Penn (1999), Hinshaw and Cicchetti (2000) would say they should display fewer stigmatizing attitudes and behaviors.

Perhaps the maladaptive, stigmatizing effects of biological theories of mental illness are moderated by personal relationships with the mentally ill: they are only stigmatizing to those who do not have personal exposure to this population. That is to say, biological theories of causality may have different effects for different groups of people. Individuals who do not know people with mental illness but who believe mental illness has biological origins may be biased against the mentally ill, thinking that they are defective or dangerous. These biases may manifest themselves as the stigmatizing attitudes and behaviors described earlier by Mehta and Farina (1997), Read and Law (1999), and Read and Harré (2001). However, individuals who hold the same etiological beliefs but do know people with mental health problems may not be prone towards the same stigmatizing attitudes and behaviors. Future research is necessary to clarify the precise mechanisms of this relationship between behavioral contact with a person with a mental health problem and biological etiological theories.

References

Adewuya, A. O., & Makanjuola, R. O. A. (2008). Lay beliefs regarding causes of mental illness in Nigeria: pattern and correlates. *Social Psychiatry & Psychiatric Epidemiology, 43,* 336-341.

Angermeyer, M. C., & Dietrich, S. (2006). Public beliefs about and attitudes towards people with mental illness: a review of population studies. *Acta Psychiatrica Scandinavica, 113*: 163-179.

Angermeyer, M. C., & Klusmann, D. (1988a). The causes of functional psychoses as seen by patients and their relatives: I. The patients' point of view. *European Archives of Psychiatry and Neurological Sciences, 238*, 47-54.

Angermeyer, M. C., & Klusmann, D. (1988b). The causes of functional psychoses as seen by patients and their relatives: II. The relatives' point of view. *European Archives of Psychiatry and Neurological Sciences, 238*, 55-61.

Angermeyer, M. C., & Matschinger, H. (1996). Relatives' beliefs about the causes of schizophrenia. *Acta Psychiatrica Scandinavica, 93*: 199-204.

Angermeyer, M. C., & Matschinger, H. (2005). Causal beliefs and attitudes to people with schizophrenia: Trend analysis based on data from two population surveys in Germany. *British Journal of Psychiatry, 186,* 331-334.

Calnan, M. (1987). *Health and Illness: the Lay Perspective*. London: Tavistock Publications Ltd.

Corrigan, P. W., & Penn, D. L. (1999). Lessons from social psychology on discrediting psychiatric stigma. *American Psychologist, 54,* 765-776.

Esterberg, M. L., & Compton, M. T. (2006). Causes of schizophrenia reported by family members of urban African American hospitalized patients with schizophrenia. *Comprehensive Psychiatry, 47,* 221-226.

Furnham, A., & Bower, P. (1992). A comparison of academic and lay theories of schizophrenia. *British Journal of Psychiatry, 161,* 201-210.

Jorm, A. F. (2000). Mental health literacy: public knowledge and beliefs about mental disorders. *British Journal of Psychiatry, 177*: 396-401.

Jorm, A. F., Christensen, H., & Griffiths, K. M. (2005). Public beliefs about causes and risk factors for mental disorders: Changes in Australia over 8 years. *Social Psychiatry & Psychiatric Epidemiology, 40,* 764-767.

Jorm, A. F., Korten, A. E., Jacomb, P. A., Christensen, H., Rodgers, B., & Pollitt, P. (1997a). "Mental health literacy": a survey of the public's ability to recognise mental disorders and their beliefs about the effectiveness of treatment. *Medical Journal of Australia, 166*: 182-186.

Jorm, A. F., Korten, A. E., Jacomb, P. A., Christensen, H., Rodgers, B., & Pollitt, P. (1997b). Public beliefs about causes and risk factors for depression and schizophrenia. *Social Psychiatry & Psychiatric Epidemiology, 32,* 143-148.

France, C. M., Lysaker, P. H., Robinson, R. P. (2007). The "chemical imbalance" explanation for depression: origins, lay endorsement, and clinical implications. *Professional Psychology: Research and Practice, 38(4)*: 411-420.

Hinshaw, S. P., & Cicchetti, D. (2000). Stigma and mental disorder: Conceptions of illness, public attitudes, personal disclosure, and social policy. *Development and Psychopathology, 12*, 555-598.

Holzinger, A., Kilian, R., Lindenbach, I., Petscheleit, A., & Angermeyer, M. C. (2003). Patients' and their relatives' causal explanations of schizophrenia. *Social Psychiatry & Psychiatric Epidemiology, 38,* 155-162.

Kim, N. S., & Ahn, W-K. (2002). The influence of naïve causal theories on lay concepts of mental illness. *American Journal of Psychology, 115(1)*: 33-65.

Kolodziej, M. E., & Johnson, B. T. (1996). Interpersonal contact and acceptance of persons with psychiatric disorders: A research synthesis. *Journal of Consulting and Clinical Psychology, 64,* 1387-1396.

Kurihara, T., Kato, M., Reverger, R., & Tirta, I. G. R. (2006). Beliefs about causes of schizophrenia among family members: A community-based survey in Bali. *Psychiatric Services, 57(12),* 1795-1799.

Link, B. G., Phelan, J. C., Bresnahan, M., Stueve, A., & Pescosolido, B. A. (1999). Public conceptions of mental illness: labels, causes, dangerousness, and social distance. *American Journal of Public Health, 89(9):* 1328-1333

Mehta, S., & Farina, A. (1997). Is being "sick" really better? Effect of the disease view of mental disorder on stigma. *Journal of Social and Clinical Psychology, 16(4)*, 405-419.

Nettleton, S. (2006). The Sociology of Health and Illness (2nd Ed.). Cambridge, UK: Polity Press.

Read, J., & Harré, N. (2001). The role of biological and genetic causal beliefs in the stigmatisation of 'mental patients.' *Journal of Mental Health, 10,* 223-235.

Read, J., & Law, A. (1999). The relationship of causal beliefs and contact with users of mental health services to attitudes to the 'mentally ill.' *International Journal of Social Psychiatry, 45(3)*, 216-229.

Swami, V., Furnham, A., Kannan, K., & Sinniah, D. (2008). Beliefs about schizophrenia and its treatment in Kota Kinabalu, Malaysia. *International Journal of Social Psychiatry, 54,* 164-179.

Table 1: Mean scores for causal theories for participants who knew someone who received treatment for a mental health problem and for those who did not

	Ps who knew someone who received tx for mental health problem	Ps who did not know someone who received tx for mental health problem
Bad Luck	Mean=3.32 N=959 SD=.825	Mean=3.30 N=434 SD=.843
God's Will	Mean=3.33† N=950 SD=.930	Mean=3.23 N=434 SD=.980
Bad Character	Mean=3.13*** N=959 SD=.861	Mean=2.97 N=431 SD=.940
Stress	Mean=1.88† N=961 SD=.715	Mean=1.94 N=434 SD=.839
Way Raised	Mean=2.85* N=950 SD=.881	Mean=2.97 N=427 SD=.905
Genetic Problem	Mean=2.17 N=948 SD=.744	Mean=2.23 N=426 SD=785
Brain Disease	Mean=2.39*** N=945 SD=.959	Mean=2.60 N=421 SD=.955

*= p<.05; **= p<.01; ***=p<.001; †=p is marginal

Table 2: Correlations between causal theories and severity of other person's mental health problem and distress caused by that mental health problem

	Bad Character	Genetics	Brain Disease	Upbringing	Bad Luck	Stress	God's Will
Severity of Others' Problem	N=956 r=-.096**	N=945 r=.080*	N=941 r=.061	N=946 r=-.042	N=955 r=-.037	N=957 r=-.025	N=946 r=-.078*
Distress Caused by Other's Problem	N=957 r=-.111**	N=946 r=.027	N=943 r=.065*	N=948 r=-.019	N=957 r=-.042	N=959 r=-.008	N=948 r=-.060

*= $p<.05$; **= $p<.01$

Table 3: Mean scores for causal theories for participants who personally had received treatment for a mental health problem and for those who did not

	Ps who had received tx for mental health problem	Ps who had not received tx for mental health problem
Bad Luck	Mean=3.37 N=210 SD=.797	Mean=3.31 N=1179 SD=.836
God's Will	Mean=3.34 N=207 SD=.971	Mean=3.30 N=1173 SD=.939
Bad Character	Mean=3.29*** N=212 SD=.814	Mean=3.05 N=1173 SD=.897
Stress	Mean=1.89 N=209 SD=.810	Mean=1.90 N=1182 SD=.745
Way Raised	Mean=2.95 N=206 SD=.885	Mean=2.88 N=1167 SD=.891
Genetic Problem	Mean=2.06* N=205 SD=.745	Mean=2.21 N=1165 SD=.756
Brain Disease	Mean=2.33* N=204 SD=.976	Mean=2.48 N=1157 SD=.958

*= $p<.05$; ***=$p<.001$

Appendix A: Variables

Variable Used to Assess:	Mnemonic	Question Text	Variable Type and Measurement
Whether R knows someone who has received treatment for mental health	MHTRTOT2	Leaving yourself aside, have you personally ever known someone who has received treatment for a mental health situation?	Dichotomous, Categorical: Y/N
Severity of other's mental health problem	MHSEROTH	How serious would you say (his/her) mental health problem was?	Continuous: 4-pt Likert scale (1 very serious, 2 somewhat serious, 3 not very serious, 4 not at all serious)
Amount of distress this person caused R	MHDISOTH	How much distress did this person's mental health problem cause you?	Continuous: 4-pt Likert (1 a great deal, 2 quite a bit, 3 a little, 4 not at all)
Whether R has received treatment for mental health problem	MHTRTSLF	Have you personally ever received treatment for a mental health problem?	Dichotomous, Categorical: Y/N
Extent to which R endorses bad character as a causal theory	MHCHRCTR	Is it very likely, somewhat likely, not very likely, or not at all likely that [NAME]'s situation is caused by his/her own bad character?	Continuous: 4-pt Likert (1 very likely, 2 somewhat likely, 3 not very likely, 4 not at all likely)
Extent to which R endorses bad luck as a causal theory	MHLUCK	Is it very likely, somewhat likely, not very likely, or not at all likely that [NAME]'s situation is caused by bad luck?	Continuous: 4-pt Likert (1 very likely, 2 somewhat likely, 3 not very likely, 4 not at all likely)
Extent to which R endorses genetics as a causal theory	MHGENES	Is it very likely, somewhat likely, not very likely, or not at all likely that [NAME]'s situation is caused by a genetic or inherited problem?	Continuous: 4-pt Likert (1 very likely, 2 somewhat likely, 3 not very likely, 4 not at all likely)

God's will	MHGOD	Is it very likely, somewhat likely, not very likely, or not at all likely that [NAME]'s situation is caused by bad luck?	Continuous: 4-pt Likert (1 very likely, 2 somewhat likely, 3 not very likely, 4 not at all likely)
Brain disease	MHBRAIN	Is it very likely, somewhat likely, not very likely, or not at all likely that [NAME]'s situation is caused by God's will?	Continuous: 4-pt Likert (1 very likely, 2 somewhat likely, 3 not very likely, 4 not at all likely)
Stress	MHSTRESS	Is it very likely, somewhat likely, not very likely, or not at all likely that [NAME]'s situation is caused by stress?	Continuous: 4-pt Likert (1 very likely, 2 somewhat likely, 3 not very likely, 4 not at all likely)
Way R was raised	MHRAISED	Is it very likely, somewhat likely, not very likely, or not at all likely that [NAME]'s situation is caused by the way he/she was raised?	Continuous: 4-pt Likert (1very likely, 2 somewhat likely, 3 not very likely, 4 not at all likely)