Published in final edited form as:
Psychiatry Res. 2010 October 30; 179(3): 328–332. doi:10.1016/j.psychres.2009.09.010.

Biogenetic models of psychopathology, implicit guilt, and mental illness stigma

Nicolas Rüsch[a,b,*], **Andrew R. Todd**[c], **Galen V. Bodenhausen**[c], and **Patrick W. Corrigan**[a]
[a]Illinois Institute of Technology, Chicago, Illinois

[b]Department of Psychiatry and Psychotherapy, University of Freiburg, Germany

[c]Department of Psychology, Northwestern University, Evanston, Illinois

Abstract

Whereas some research suggests that acknowledgment of the role of biogenetic factors in mental illness could reduce mental illness stigma by diminishing perceived responsibility, other research has cautioned that emphasizing biogenetic aspects of mental illness could produce the impression that mental illness is a stable, intrinsic aspect of a person ("genetic essentialism"), increasing the desire for social distance. We assessed genetic and neurobiological causal attributions about mental illness among 85 people with serious mental illness and 50 members of the public. The perceived responsibility of persons with mental illness for their condition, as well as fear and social distance, were assessed by self-report. Automatic associations between Mental Illness and Guilt and between Self and Guilt were measured by the Brief Implicit Association Test. Among the general public, endorsement of biogenetic models was associated with less perceived responsibility, but also greater social distance. Among people with mental illness, endorsement of genetic models had only negative correlates: greater explicit fear and stronger implicit self-guilt associations. Genetic models may have unexpected negative consequences for implicit self-concept and explicit attitudes of people with serious mental illness. An exclusive focus on genetic models may therefore be problematic for clinical practice and anti-stigma initiatives.

Keywords

prejudice; illness models; causality; genetics; prejudice; social distance; fear

1. Introduction

Extensive research on the etiology of psychiatric disorders has vastly expanded our understanding of the role played by genetic and neurobiological factors in mental illnesses (Kendler and Prescott, 2006). Mental illness stigma remains a major burden for people with mental illnesses (Corrigan, 2005; Thornicroft, 2006; Hinshaw, 2007), but there is widespread optimism that public understanding of the biogenetic aspects of psychopathology will alleviate this stigma by reducing the tendency to hold persons experiencing disorders responsible for their illness. For example, a leading mental health

[*]Send all correspondence to Nicolas Rüsch, Department of Psychiatry and Psychotherapy, University of Freiburg, Hauptstr. 5, 79104 Freiburg, Germany. nicolas.ruesch@uniklinik-freiburg.de; Tel. +49 761 2706501; Fax. +49 761 2706619.

advocacy group, the National Alliance on Mental Illness (www.nami.org), promotes a biogenetic model of mental illness in order to reduce stigma and blame against individuals with mental illness. Mental health care providers and people with mental illness argue that use of functional brain imaging in the diagnosis of mental illness will diminish stigma and self-blame among people with mental illness (Illes et al., 2008). Genetic counseling for people with mental illness is also expected to decrease stigma (Hill and Sahhar, 2006; Austin and Honer, 2007). Thus, there is a common expectation that adoption of a biogenetic view of psychiatric disorders will produce benefits in social attitudes about mental illness.

Optimism about the stigma-reducing effects of biogenetic views of mental illness is not universal. Indeed, prominent theories lead to diverging predictions (Phelan, 2005; Spriggs et al., 2008). On one hand, attribution theory (Weiner et al., 1988) generates the optimistic expectations about the influence of biogenetic models on stigma just described. According to attribution theory, anger and blame are mitigated when personal responsibility is perceived to be low. Because a genetic model implies that persons with mental illness are not responsible for their condition, endorsement of this model should diminish the blame attached to them (Phelan et al., 2002; Corrigan et al., 2003). On the other hand, “genetic essentialism” implies that genes are the unchangeable basis of a person's identity (Nelkin and Lindee, 1995), and such essentialism is associated with increased prejudice (Keller, 2005). Supporting this view, a stronger endorsement of biogenetic causes for mental illness has been associated with increased social distance (Lauber et al., 2004; Angermeyer and Matschinger, 2005), with perceptions of mental illness as more persistent, serious (Phelan, 2005), and dangerous (Jorm and Griffiths, 2008), and with more pessimistic views about treatment outcomes (Phelan et al., 2006; Lam and Salkovskis, 2007). However, because most of this research has been conducted with members of the general public, very little is known about the correlates of the endorsement of a biogenetic perspective among people experiencing mental illness.

The question of whether biogenetic models of mental illness will be associated with reduction or enhancement of stigma is further complicated by recent evidence suggesting that important components of stigma may operate in an implicit, automatic manner that is not necessarily directly aligned with explicit beliefs (Teachman et al., 2006). Attitude researchers have become increasingly interested in automatic aspects of cognition and have documented many dissociations between rapid, automatic reactions and more thoughtful, deliberative ones (Gawronski and Bodenhausen, 2006). A member of the public may, for example, automatically associate ‘mental illness’ with ‘guilt’ and thus implicitly harbor a guilt-related negative stereotype about mental illness. Persons with mental illness, on the other hand, might develop implicit-automatic guilt-related self-associations, and thus implicit self-blame, which may operate outside their awareness or control. Automatic and deliberative aspects of stigmatizing attitudes can predict different kinds of cognitive, affective, and behavioral tendencies (Dovidio et al., 2002; Greenwald et al., 2009) and may respond differently to attempts to reduce stigma (Stier and Hinshaw, 2007; Lincoln et al., 2008). In the present research, we thus examined not only explicit, deliberate aspects of mental illness stigma but also its more automatic, implicit components.

Within a biogenetic explanatory framework for mental illness, genetic and neurobiological models are related (Kendler and Prescott, 2006) but have different foci. Genetic models stress heritability and may be seen as implying immutability, whereas neurobiological models focus on the body (specifically, the brain) as the locus of the disorder, analogous to physical illnesses. We therefore investigated the link between endorsing genetic or neurobiological models of mental illness on the one hand and two key types of reactions to people with mental illness on the other hand: decreased responsibility and blame versus increased fear and avoidance. We examined both explicit and implicit manifestations of

these reactions, in both a sample of the general public as well as people experiencing a mental illness.

2. Methods

2.1. Participants

Eighty-five persons with serious mental illness were recruited from outpatient mental health centers in the Chicago area in the context of a larger study on mental illness stigma (Rüsch et al., 2009a, 2009b, 2009c, 2009d). The project was advertised as a study on attitudes toward people with mental illness, using flyers in mental health service centers. An eighth grade reading level as assessed by the Wide Range Achievement Test (Wilkinson and Robertson, 2006) was required. Fifty members of the general public—matched for age, gender, and ethnicity, and screened for any lifetime or current axis I disorder—were recruited, using flyers in the community and on a university campus. Physical disabilities were an exclusion criterion to avoid confounds in the implicit measures (described below) that used physical disability as a comparison category for mental illness. After a detailed description of the study procedures, all participants gave written informed consent. The study was approved by the institutional review boards of the Illinois Institute of Technology and the collaborating organizations.

Participants with mental illness were, on average, about 45 years old, and about two-thirds were male. More than half were African American, about a third Caucasian, while a few reported Hispanic, mixed, or other ethnicities (Table 1). Axis I diagnoses were made using the Mini-International Neuropsychiatric Interview (Sheehan et al., 1998) based on DSM-IV criteria. Twenty-three (27%) participants had schizophrenia, 22 (26%) schizoaffective disorder, 30 (35%) bipolar I or II disorder, and 10 participants (12%) had recurrent unipolar major depressive disorder. In addition, in the entire sample 33 subjects (39%) had comorbid current alcohol- or substance-related abuse or dependence. On average, participants with mental illness were first diagnosed about 15 years ago (M=14.9, SD=10.2) and had been hospitalized in psychiatric institutions about nine times (M=9.2, SD=13.1).

2.2. Self-report measures

Following an introductory question (“What do you think about the causes of mental illness?”), participants responded to two items measuring endorsement of genetic (“Mental illness is caused by genetic and hereditary factors”) and neurobiological (“Mental illness is a brain disorder, caused by biological changes in brain metabolism”) causes of mental illness. Both items were scaled from 1 to 7, with higher scores indicating stronger agreement.

The belief that people with mental illness are responsible for their condition as well as the fear of people with mental illness were assessed using the Attribution Questionnaire (Corrigan et al., 2003). Following a short vignette about Harry, a man with schizophrenia, participants responded to three items indicating perceived responsibility (e.g., ‘I would think that it was Harry's own fault that he is in the present condition’) and to three fear items (e.g., ‘Harry would terrify me’). Separately for fear and responsibility, the three respective items (scored from 1 to 9) were summed to yield two subscale scores, with higher scores indicating greater perceived responsibility and fear, respectively.

The desire to avoid people with mental illness was assessed by the Social Distance Scale (Link et al., 1999). Following a short vignette about John, a man with psychotic symptoms, respondents rated five items (e.g., ‘How willing would you be to make friends with John?’), scored from 1 to 6, with higher mean scores indicating stronger desire for social distance.

2.3. Implicit measures

We used a computer-based response-latency measure, the Brief Implicit Association Test (BIAT; Sriram and Greenwald, 2009), to assess automatic aspects of stigma; this shorter version of the full-length IAT (Greenwald et al., 1998) was selected because we expected more participants to complete the task. It was recently used to measure implicit attitudes toward psychiatric medication among the same participants (Rüsch et al., 2009e). During the BIATs, participants classified a series of words into superordinate categories. In the BIAT measuring the association between mental illness and guilt, the target categories were "Mental Illness" versus "Physical Disability," and the attribute categories were "Guilty" versus "Innocent." For the BIAT measuring implicit guilt-prone self-concepts (used in the diagnosed group only), the target categories were "Me" versus "Not Me", and the attribute categories were again "Guilty" versus "Innocent."

During the BIAT, a series of words was presented at the center of the screen that either did or did not belong to one of two categories represented on the top of the screen (e.g., Me/Guilty). Participants' task was to press a right-hand response key if the word belonged to either of the two categories and a left-hand response key if it belonged to neither category. Thus, for example, if the categories were Me and Guilty, participants should respond with a right-key press to the word "myself" or "blameworthy," but they should respond with a left-key press to the word "them" or "innocent." The logic of the task is that verbal stimuli are classified more quickly when the target and attribute category pairings (e.g., Me/Guilty) match respondents' automatic associations with the target categories versus during the other block when the target and attribute category pairings do not belong together in the respondents' eyes (e.g., Me/Innocent).

In the full-length IAT, all four categories remain on the screen in both blocks (e.g., Mental Illness, Physical Disability, Guilty, Innocent). The BIAT is different in that only two categories are shown on the screen at any one time (e.g., Mental Illness and Guilty in one block; Mental Illness and Innocent in the other block); thus, three focal categories are employed within a given BIAT), whereas one category (e.g., Physical Disability) is never shown on the screen and therefore is referred to as a non-focal category (Sriram and Greenwald, 2009). Physical disability was the non-focal category in our Mental Illness-Guilty BIAT, Innocent in the Me-Guilty BIAT. This design has the advantage of focusing participants' attention on the three focal categories, such that implicit associations with the non-focal category (e.g., Physical Disability) become less relevant. BIAT scores are therefore more straightforward to interpret because they reflect associations between focal categories and are less confounded by associations with the non-focal category than in the full-length IAT.

There were two blocks of 20 trials each, and from each block the first four practice trials were excluded from analyses (for details, see Sriram and Greenwald, 2009). BIAT data with more than 30% errors were excluded from analyses (Teachman and Woody, 2003), leaving 78 Mental Illness-Guilty and 79 Me-Guilty BIATs valid in the diagnosed group and 46 Mental Illness-Guilty BIATs valid in the non-diagnosed group. We used the following stimuli for each category: Mental Illness (mentally disturbed, mental illness, mentally unbalanced, mentally ill), Physical Disability (physically impaired, physical disability, physically disabled, physically handicapped), Me (me, my, mine, myself), Not Me (not me, they, them, their), Guilty (blameworthy, guilty, guilt, blame) and Innocent (faultless, innocence, innocent, guiltless). The order of BIATs and of blocks within each BIAT was counterbalanced across participants. BIAT scores were calculated using the improved scoring algorithm, resulting in a *D*-measure (Greenwald et al., 2003). More positive values represent a stronger association between Mental Illness and Guilty or between Me and Guilty, respectively.

3. Results

3.1. Endorsement of biogenetic causation in both groups

On average, genetic and neurobiological causes were endorsed by both people with mental illness and the general public sample to similar degrees (Table 1). While endorsements of genetic and neurobiological causes were positively correlated in both groups (general public: r=0.57, p<0.001; diagnosed: r=0.22, p=0.04), the difference between the two correlations was significant (Z=2.32, p=0.02; Krishnamoorthy and Yanping, 2007). Given the differential degree of association of these beliefs across the two groups, we examined results separately for neurobiological versus genetic illness beliefs.

We also examined differences in endorsement of genetic or neurobiological causes between the four groups of subjects with schizophrenia, schizoaffective disorder, bipolar disorder and unipolar depression. Analyses of variance did not indicate significant group effects for genetic (F[3,81]=2.50, p=0.07; all post-hoc Scheffé tests non-significant with p-values >0.15) or neurobiological causality (F[3,81]=0.34, p=0.80; all post-hoc Scheffé tests non-significant with p-values >0.80). Subjects with versus without comorbid substance- or alcohol related disorders in the entire sample did not differ in terms of endorsement of genetic or neurobiological causes (p-values >0.20).

3.2. Endorsement of stigmatizing beliefs in both groups

Items assessing fear and social distance were endorsed by people with mental illness and members of the general public to similar degrees (Table 1). Similarly, automatic associations between Mental Illness and Guilty did not differ between the groups. However, compared to members of the public, the diagnosed group judged people with mental illness as significantly more responsible for their condition.

3.3. Endorsement of biogenetic causes and stigma in the general public sample

Among members of the general public, endorsement of biogenetic models was related to self-reported aspects of mental illness stigma in a manner confirming the double-edged nature of such beliefs. As shown in Table 2, endorsement of the genetic model was associated with lower perceptions of personal responsibility (i.e., decreased stigma) but also with increased desire to avoid persons with mental illness (i.e., greater stigma). In contrast, endorsement of a neurobiological model did not predict any explicit stigma-related reactions. At the implicit level, endorsement of both models was associated with weaker automatic mental illness-guilt associations, but this pattern was only marginally significant (p=0.09) for the genetic model.

3.4. Endorsement of biogenetic causes and stigma in the diagnosed group

Of particular interest was the question of how endorsement of biogenetic causes would be related to the tendency to endorse stigma among people experiencing mental illness. For these participants, endorsement of biogenetic causes was unrelated to explicit feelings of personal responsibility (Table 3). However, self-reported feelings of fear regarding other people with mental illness were higher among diagnosed participants who endorsed a genetic model of mental illness. At the implicit level, endorsement of the genetic model was associated with significantly stronger me-guilty associations. Thus, among persons with mental illness, endorsement of a genetic model was associated with more stigmatizing reactions both in terms of explicit fear and implicit guilt. Endorsing a neurobiological model, on the other hand, was not associated with explicit or implicit reactions in the diagnosed group. The implicit and explicit measures were not significantly correlated in either group (p-values >.15; Appendix 1).

4. Discussion

The present study examined the link between endorsing genetic or neurobiological causes and reactions toward people with mental illness, focusing on key components of stigma that might be predicted by these causal models. Biogenetic beliefs appear to constitute a decidedly mixed bag with regard to the reactions of the general public. On the positive side, among members of the general public endorsement of genetic models was associated with reduced perceived responsibility and weaker implicit blame regarding people with mental illness, as attribution models would predict, but it was also associated with a preference for greater social distance, as suggested by notions of genetic essentialism. Considering these mixed consequences of genetic models, the desire for social distance is a closer proxy of discriminating behavior than perceived responsibility; furthermore, behavioral consequences are likely to be more harmful for stigmatized individuals than personally held views about responsibility. Therefore, the current findings suggest that among members of the general public the negative effects of endorsing genetic models seem to outweigh the positive.

A noteworthy contribution of the current study was the investigation of these issues within a sample of clinically-diagnosed individuals. In general, little is known about the etiological beliefs of people with mental illness (Lobban et al., 2003). It is interesting that, although members of the general public evinced a strong positive correlation between endorsements of genetic and neurobiological causes, consistent with past research (Kendler and Prescott, 2006), endorsements of these beliefs were significantly more weakly associated in the diagnosed group. This suggests that people with serious mental illness may espouse causal beliefs about their condition that substantially differ from the public view. We can only speculate that for people with mental illness genetic models, with their focus on heritability and seeming immutability, may have quite different personal implications compared to neurobiological models.

Empirically, endorsement of the neurobiological model had no significant correlates among the diagnosed sample, but endorsement of the genetic model predicted some noteworthy patterns. First, unlike the general public, there was no evidence that endorsement of genetic causation was associated with lower explicit feelings of responsibility or weaker automatic associations between mental illness and guilt. Instead, the negative implications of a genetic-essentialist view seemed to be evident, in that both greater explicit fear toward persons with mental illness and stronger automatic me-guilty associations were found among diagnosed participants who endorsed the genetic model. Perhaps because genetic qualities can be seen as deeply defining (indeed, constituting a person's essence; Keller, 2005), individuals who view their psychiatric disorder as genetic in origin may develop an irrational sense of implicit guilt because the disorder comes to be seen as a fundamental, and perhaps immutable, identity-defining trait. For stigmatized individuals, a dominant genetic view of their illness may make it difficult to develop their own narrative taking into account non-genetic factors in a more balanced fashion (Lysaker et al., 2007).

Unfortunately, these findings confirm a pessimistic prediction of the genetic essentialism perspective, in that endorsement of the genetic model was associated with increased desire to avoid persons with mental illness among the general public, and with greater explicit fear and implicit guilt among the diagnosed sample. On the other hand, endorsing neurobiological causes was not associated with negative consequences in either group, and it was associated with weaker implicit mental illness-guilt associations among the general public. Neurobiological factors may appear less immutable and essence-defining than genetic factors and therefore induce less fear or avoidance. However, although we did not find evidence for negative effects of neurobiological models on reactions toward mental

illness, our findings do not support the hypothesis (Illes et al., 2008) that they might be helpful to reduce stigma.

It is noteworthy that people with mental illness did not show more positive reactions than members of the general public in implicit and explicit measures. On the contrary, in terms of perceived responsibility of people with mental illness for their condition, the diagnosed group held even more negative views. While the reasons remain unclear, it is interesting to note that a high level of contact with consumers --usually a powerful anti-stigma tool (Corrigan and Penn, 1999) that can be taken for granted in our diagnosed group, recruited from mental health care settings --apparently did not reduce this facet of stigma among people with mental illness toward their own group. It could further be speculated that the high rate of comorbid substance-related disorders, stereotypically associated with strong responsibility, may have contributed to this finding in our study.

Some limitations of this study should be noted. First, our data are correlational and do not allow firm conclusions regarding causality. Second, we focused on perceptions of responsibility/blame and fear/distance, but other aspects of stigma warrant inclusion in future studies. Third, we assessed endorsements of causality of mental illness in general and did not differentiate between, for example, schizophrenia and depression. Fourth, our diagnosed sample was not representative of people with mental illness in general and factors such as ethnic minority status, male gender, and serious psychiatric disorders were over-represented; the same applies to the general public sample which was matched for gender, age, and ethnicity to the diagnosed group. Fifth, the level of contact with people with mental illness that may influence both perceived causality and attitudes should be investigated in future studies. Finally, other causal attributions such as life events, stress, or family upbringing should be examined along with biogenetic models.

Our findings highlight potential benefits and risks of promoting biogenetic models in attempts to reduce stigma. As far as the general public is concerned, the good news is that these models may indeed decrease blame at explicit and implicit levels; the bad news is that genetic models may lead to increased desire for social distance. In terms of reducing public stigma, the important task of developing effective anti-stigma strategies thus faces challenges, and more research is required to understand the role that biogenetic causal models should play in such strategies (Corrigan and Penn, 1999; Thornicroft et al., 2008). With respect to self-stigma in people with mental illness, biogenetic illness models could also lead diagnosed individuals to implicitly feel fundamentally flawed and guilty for their condition. This is consistent with previous findings that persons with schizophrenia prefer psychosocial over biogenetic explanations for their condition (Holzinger et al., 2001, 2003). People with mental illness possibly prefer psychosocial models because, among other reasons, these models are less associated with implicit guilt or fear of their ingroup members. Biogenetic models of mental illness have further been criticized by proponents of a recovery-oriented approach because they do not reflect subjective experience or meaning, central features of living with a mental illness, and are often deficit-oriented, thereby facilitating stigma (Slade, 2009). Consumers, relatives, and mental health professionals alike should therefore be cautious in promoting explanations that exclusively focus on biogenetic factors when trying to help consumers to cope better with their mental illness.

Acknowledgments

We thank Karen Batia, Whitney Key, Norine McCarten, Patrick Michaels, Karina Powell, Anita Rajah, Abigail Wassel, Sandra Wilkniss, and staff of Thresholds and Heartland Alliance in Chicago for assistance with recruitment and data collection. We thank N. Sriram and Tony Greenwald for their help in designing and implementing the Brief Implicit Association Tests, and four anonymous reviewers for their thoughtful comments. This work was

supported by a Marie Curie Outgoing International Fellowship of the European Union to Nicolas Rüsch and by funding from NIAAA, NIMH, and the Fogarty International Center to Patrick W. Corrigan.

Appendix 1

Correlations between implicit and explicit attitude measures, for the diagnosed group (lower left triangle of the table, in bold font; n between 78 and 85) and members of the general public (upper right triangle, n between 46 and 50)

	Implicit 'Me-Guilty' association *(a)*	**Implicit 'Mental Illness-Guilty' association** *(a)*	**Fear** *(b)*	**Social Distance** *(c)*	**Responsibility** *(b)*
Implicit 'Mental Illness-Guilty' association *(a)*	**0.13**		0.08	-0.19	-0.01
Fear *(b)*	**-0.01**	**0.05**		0.35 *	0.23
Social Distance *(c)*	**-0.09**	**0.03**	**0.41** **		-0.01
Responsibility *(b)*	**-0.16**	**0.06**	**-0.05**	**0.09**	

* $p < 0.0$

** $p < 0.01$ (two-tailed)

(a) Brief Implicit Association Test, higher scores indicate stronger implicit Mental Illness-Guilty or Me-Guilty associations

(b) Attribution Questionnaire (Corrigan et al., 2003)

(c) Social Distance Scale (Link et al., 1999)

References

Angermeyer MC, Matschinger H. Causal beliefs and attitudes to people with schizophrenia. Trend analysis based on data from two population surveys in Germany. British Journal of Psychiatry. 2005; 186:331–334. [PubMed: 15802691]

Austin JC, Honer WG. The genomic era and serious mental illness: a potential application for psychiatric genetic counseling. Psychiatric Services. 2007; 58:254–261. [PubMed: 17287384]

Corrigan, PW. On the stigma of mental illness: Practical strategies for research and social change. American Psychological Association; Washington DC: 2005.

Corrigan PW, Markowitz FE, Watson A, Rowan D, Kubiak MA. An attribution model of public discrimination towards persons with mental illness. Journal of Health and Social Behavior. 2003; 44:162–179. [PubMed: 12866388]

Corrigan PW, Penn DL. Lessons from social psychology on discrediting psychiatric stigma. American Psychologist. 1999; 54:765–776. [PubMed: 10510666]

Dovidio JF, Kawakami K, Gaertner SL. Implicit and explicit prejudice and interracial interaction. Journal of Personality and Social Psychology. 2002; 82:62–68. [PubMed: 11811635]

Gawronski B, Bodenhausen GV. Associative and propositional processes in evaluation: An integrative review of implicit and explicit attitude change. Psychological Bulletin. 2006; 132:692–731. [PubMed: 16910748]

Greenwald AG, McGhee DE, Schwartz JL. Measuring individual differences in implicit cognition: The Implicit Association Test. Journal of Personality and Social Psychology. 1998; 74:1464–1480. [PubMed: 9654756]

Greenwald AG, Nosek BA, Banaji MR. Understanding and using the implicit association test: I. An improved scoring algorithm. Journal of Personality and Social Psychology. 2003; 85:197–216. [PubMed: 12916565]

Greenwald AG, Poehlman TA, Uhlmann E, Banaji MR. Understanding and using the Implicit Association Test: III. Meta-analysis of predictive validity. Journal of Personality and Social Psychology. 2009; 97:17–41. [PubMed: 19586237]

Hill MK, Sahhar M. Genetic counselling for psychiatric disorders. Medical Journal of Australia. 2006; 185:507–510. [PubMed: 17137456]

Hinshaw, SP. The mark of shame: Stigma of mental illness and an agenda for change. Oxford University Press; Oxford; 2007.

Holzinger A, Kilian R, Lindenbach I, Petscheleit A, Angermeyer MC. Patients' and their relatives' causal explanations of schizophrenia. Social Psychiatry & Psychiatric Epidemiology. 2003; 38:155–162. [PubMed: 12616314]

Holzinger A, Löffler W, Müller P, Priebe S, Angermeyer MC. Vorstellungen schizophrener Patienten über die Ursachen ihrer Erkrankung. Psychotherapie Psychosomatik Medizinische Psychologie. 2001; 51:328–333.

Illes J, Lombera S, Rosenberg J, Arnow B. In the mind's eye: Provider and patient attitudes on functional brain imaging. Journal of Psychiatric Research. 2008; 43:107–114. [PubMed: 18423669]

Jorm AF, Griffiths KM. The public's stigmatizing attitudes towards people with mental disorders: How important are biomedical conceptualizations? Acta Psychiatrica Scandinavica. 2008; 118:315–321. [PubMed: 18759807]

Keller J. In genes we trust: The biological component of psychological essentialism and its relationship to mechanisms of motivated social cognition. Journal of Personality & Social Psychology. 2005; 88:686–702. [PubMed: 15796668]

Kendler, KS.; Prescott, CA. Genes, environment, and psychopathology: Understanding the causes of psychiatric and substance use disorders. Guilford Press; New York: 2006.

Krishnamoorthy K, Yanping X. Inferences on correlation coefficients: One-sample, independent and correlated cases. Journal of Statistical Planning and Inference. 2007; 137:2362–2379.

Lam DC, Salkovskis PM. An experimental investigation of the impact of biological and psychological causal explanations on anxious and depressed patients' perception of a person with panic disorder. Behavior Research & Therapy. 2007; 45:405–411.

Lauber C, Nordt C, Falcato L, Rössler W. Factors influencing social distance toward people with mental illness. Community Mental Health Journal. 2004; 40:265–274. [PubMed: 15259631]

Lincoln TM, Arens E, Berger C, Rief W. Can antistigma campaigns be improved? A test of the impact of biogenetic versus psychosocial causal explanations on implicit and explicit attitudes to schizophrenia. Schizophrenia Bulletin. 2008; 34:984–994. [PubMed: 18056110]

Link BG, Phelan JC, Bresnahan M, Stueve A, Pescosolido BA. Public conceptions of mental illness: labels, causes, dangerousness, and social distance. American Journal of Public Health. 1999; 89:1328–1333. [PubMed: 10474548]

Lobban F, Barrowclough C, Jones S. A review of the role of illness models in severe mental illness. Clinical Psychology Review. 2003; 23:171–196. [PubMed: 12573669]

Lysaker PH, Buck KD, Roe D. Psychotherapy and recovery in schizophrenia: A proposal of key elements for an integrative psychotherapy attuned to narrative in schizophrenia. Psychological Services. 2007; 4:28–37.

Nelkin, D.; Lindee, MS. The DNA mystique: The gene as a cultural icon. Freeman; New York: 1995.

Phelan JC. Geneticization of deviant behavior and consequences for stigma: The case of mental illness. Journal of Health and Social Behavior. 2005; 46:307–322. [PubMed: 16433278]

Phelan JC, Cruz-Rojas R, Reiff M. Genes and stigma: The connection between perceived genetic etiology and attitudes and beliefs about mental illness. Psychiatric Rehabilitation Skills. 2002; 6:159–185.

Phelan JC, Yang LH, Cruz-Rojas R. Effects of attributing serious mental illnesses to genetic causes on orientations to treatment. Psychiatric Services. 2006; 57:382–387. [PubMed: 16524997]

Rüsch N, Corrigan PW, Powell K, Rajah A, Olschewski M, Wilkniss S, Batia K. A stress-coping model of mental illness stigma: II. Emotional stress responses, coping behavior and outcome. Schizophrenia Research. 2009a; 110:65–71.

Rüsch N, Corrigan PW, Todd AR, Bodenhausen GV. Implicit self-stigma in people with mental illness. Journal of Nervous and Mental Disease. 2009b in press.

Rüsch N, Corrigan PW, Wassel A, Michaels P, Olschewski M, Wilkniss S, Batia K. A stress-coping model of mental illness stigma: I. Predictors of cognitive stress appraisal. Schizophrenia Research. 2009c; 110:59–64.

Rüsch N, Corrigan PW, Wassel A, Michaels P, Olschewski M, Wilkniss S, Batia K. Ingroup perception and responses to stigma among persons with mental illness. Acta Psychiatrica Scandinavica. 2009d10.1111/j.1600-0447.2009.01403.x

Rüsch N, Todd AR, Bodenhausen GV, Weiden PJ, Corrigan PW. Implicit versus explicit attitudes toward psychiatric medication: Implications for insight and treatment adherence. Schizophrenia Research. 2009e; 112:119–122.

Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, Hergueta T, Baker R, Dunbar GC. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. Journal of Clinical Psychiatry 59 Suppl. 1998; 20:22–33.

Slade, M. Personal recovery and mental illness: A guide for mental health professionals. Cambridge University Press; Cambridge: 2009.

Spriggs M, Olsson CA, Hall W. How will information about the genetic risk of mental disorders impact on stigma? Australian & New Zealand Journal of Mental Health Nursing. 2008; 42:214–220.

Sriram N, Greenwald AG. The Brief Implicit Association Test. Experimental Psychology. 2009; 56:283–294. [PubMed: 19439401]

Stier A, Hinshaw SP. Explicit and implicit stigma against individuals with mental illness. Australian Psychologist. 2007; 42:106–117.

Teachman BA, Wilson JG, Komarovskaya I. Implicit and explicit stigma of mental illness in diagnosed and healthy samples. Journal of Social and Clinical Psychology. 2006; 25:75–95.

Teachman BA, Woody SR. Automatic processing in spider phobia: implicit fear associations over the course of treatment. Journal of Abnormal Psychology. 2003; 112:100–109. [PubMed: 12653418]

Thornicroft, G. Shunned: Discrimination against people with mental illness. Oxford University Press; Oxford; 2006.

Thornicroft G, Brohan E, Kassam A, Lewis-Holmes E. Reducing stigma and discrimination: Candidate interventions. International Journal of Mental Health Systems. 2008; 2:3. [PubMed: 18405393]

Weiner B, Perry RP, Magnusson J. An attributional analysis of reactions to stigmas. Journal of Personality & Social Psychology. 1988; 55:738–748. [PubMed: 2974883]

Wilkinson, GS.; Robertson, GJ. Wide Range Achievement Test (WRAT4). Psychological Assessment Resources; Lutz: 2006.

Table 1

Demographic variables and endorsement of genetic and neurobiological models of mental illness among 85 persons with mental illness and 50 members of the general public

	Persons with mental illness	Members of the general public	*t* or χ^2 *(a)*	*p*
Age (years; *M, SD*)	44.8 (9.7)	45.0 (8.1)	0.11	0.91
Gender (% female)	32%	30%	0.05	0.83
Ethnicity (% African-American / Caucasian / Hispanic / Other or Mixed)	58 / 34 / 5 / 4	60 / 32 / 6 / 2	0.42	0.94
Endorsement of genetic model (scale-midpoint: 4, range 1-7)	4.7 (1.6)	4.7 (1.6)	-0.14	0.89
Endorsement of neurobiological model (scale-midpoint: 4, range 1-7)	5.6 (1.4)	5.4 (1.3)	-0.60	0.55
Fear *(b)* (scale-midpoint: 15, range 3-27)	7.4 (4.9)	8.4 (5.6)	-1.11	0.27
Responsibility *(b)* (scale-midpoint: 15, range 3-27)	11.2 (4.9)	9.4 (3.9)	2.12	0.04
Social Distance *(c)* (scale-midpoint: 3.5, range 1-6)	3.7 (1.2)	3.9 (1.2)	-0.81	0.42
Mental Illness - Guilty BIAT, *D*-score *(d)*	0.15 (0.44)	0.19 (0.46)	-0.45	0.65
Me - Guilty BIAT, *D*-score *(d)*	0.25 (0.49)	-	-	-

(a) Comparisons are χ^2 tests for proportions, or *t*-tests for means across each row (two-sided)

(b) Attribution Questionnaire (Corrigan et al., 2003), higher scores indicating more fear or perceived responsibility

(c) Social Distance Scale (Link et al., 1999)

(d) Brief Implicit Association Test. Higher scores indicate stronger implicit Mental Illness-Guilty or Me-Guilty associations, respectively

Table 2

Correlations between causal attributions and explicit and implicit reactions among 50 members of the general public

	Fear [a]	Social Distance [b]	Responsibility [a]	Implicit 'Mental Illness-Guilty' association [c]
	(*n*=50)	(*n*=50)	(*n*=50)	(*n*=46)
Endorsement of genetic model	0.09	0.29*	−0.37**	−0.25
Endorsement of neurobiological model	−0.08	0.10	−0.19	−0.40**

*$p < 0.05$

**$p < 0.01$ (two-tailed)

[a] Attribution Questionnaire (Corrigan et al., 2003)

[b] Social Distance Scale (Link et al., 1999)

[c] Brief Implicit Association Test, higher scores indicate stronger implicit Mental Illness-Guilty association

Table 3

Correlations between causal attributions and explicit and implicit reactions among 85 persons with mental illness

	Fear *(a)*	Social Distance *(b)*	Responsibility *(a)*	Implicit 'Mental Illness-Guilty' association *(c)*	Implicit 'Me-Guilty' association *(c)*
	(*n*=85)	(*n*=85)	(*n*=85)	(*n*=78)	(*n*=79)
Endorsement of genetic model	0.28**	0.15	-0.16	0.03	0.22*
Endorsement of neurobiological model	−0.04	-0.05	0.05	−0.08	0.14

* $p < 0.05$

** $p < 0.01$ (two-tailed)

(a) Attribution Questionnaire (Corrigan et al., 2003)

(b) Social Distance Scale (Link et al., 1999)

(c) Brief Implicit Association Test, higher scores indicate stronger implicit Mental Illness-Guilty or Me-Guilty associations