Geneticization of Deviant Behavior and Consequences for Stigma: The Case of Mental Illness*

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One likely consequence of the genetics revolution is an increased tendency to understand human behavior in genetic terms. How might this “geneticization” affect stigma? Attribution theory predicts a reduction in stigma via reduced blame, anger, and punishment and increased sympathy and help. According to “genetic essentialist” thinking, genes are the basis of human identity and strongly deterministic of behavior. If such ideas are commonly accepted, geneticization should exacerbate stigma by increasing perceptions of differentness, persistence, seriousness, and transmissibility, which in turn should increase social distance and reproductive restrictiveness. I test these predictions using the case of mental illness and a vignette experiment embedded in a nationally representative survey. There was little support for attribution theory predictions. Consistent with genetic essentialism, genetic attributions increased the perceived seriousness and persistence of the mental illness and the belief that siblings and children would develop the same problem. Genetic attribution did not affect reproductive restrictiveness or social distance from the ill person but did increase social distance from the person’s sibling, particularly regarding intimate forms of contact involving dating, marriage, and having children.

The genetics or genomic revolution, embodied in the Human Genome Project (HGP), is a scientific undertaking of unprecedented proportions. Francis Collins, director of the HGP, judges it “the single most important project in biology and the biomedical sciences” (Collins et al. 1998:682). Its champions have high expectations for the benefits of the HGP, predicting that it will “revolutionize the practice of medicine in the twenty-first century by providing the tools to determine the hereditary component of virtually all diseases” (Collins and Mansoura 2001:221).

Because the HGP represents a purposeful social action on a grand scale, we may expect its unanticipated social consequences (Merton 1936) to be significant as well. One subtle but potentially powerful consequence is “geneticization,” that is, the ascendancy of genetics as a basis for understanding human beings and human behavior (Lippman 1991). Just as the Darwinian and Freudian revolutions changed the way we view ourselves as human beings, the genetics revolution is likely to influence the way we understand the causes of human behavior and the power of the social environment to shape it (Krimsky 1991), the way we view what human groups and individuals have in common and what divides us, and perhaps even what the essence of a person is (Nelkin and Lindee 1995). One social phenomenon for which geneticiza-
tion has clear implications is stigma. This article empirically assesses the impact that geneticization may have on the stigma associated with deviant behaviors.

GENETICIZATION OF DEVIANCE

Conrad and Schneider (1992) argued that the increasing power and prestige of medicine in the twentieth century led to the medicalization of deviance, resulting in the redefinition of many forms of stigmatized behavior—such as mental illness, alcoholism, and criminality—from moral to medical problems. There can be little doubt that a parallel process of geneticization is under way.2 The HGP’s prestige and influence are attested to not only by huge monetary investments in genomic research but also by official governmental endorsement and widespread positive publicity about the HGP and genomic research in the mass media (Conrad 2001). Genetic definitions and interpretations are also making their way into wider cultural arenas, including policy debates, fictional works, and advertisements (Kitcher 1996; Nelkin and Lindee 1995). Genetic bases of many forms of deviance such as mental disorders, criminal behavior, homosexuality, and obesity are actively investigated, and positive findings are reported prominently in the press (Conrad 2001; Nelkin and Lindee 1995). In addition, to the extent that stigmatized behaviors have already been medicalized, they will in turn be geneticized by claims such as this: “except for some cases of trauma ... virtually every human illness has a hereditary component” (Collins and McKusick 2001:540).

GENETICIZATION AND STIGMA

According to Link and colleagues (Link and Phelan 2001; Link et al. 2004), stigma occurs when (1) a sufficiently powerful group of people identify a human characteristic as being socially relevant and warranting special labeling or categorization; (2) dominant cultural beliefs link labeled individuals to undesirable characteristics or stereotypes; (3) labeled persons are placed in distinct categories separating “us” from “them”; (4) people have negative emotional reactions to labeled persons; and (5) labeled persons experience status loss and discrimination that lead to unequal outcomes.

Although stigma is regularly described by scholars as being historically specific, it is just as regularly studied in static social circumstances. The genetics revolution gives sociologists the opportunity to directly study the impact of a major cultural and social change on existing states of stigma. Very different visions have been offered as to what this impact might be. On the one hand are hopes that genetic interpretations will reduce stigma by eliminating blame. On the other are fears that entirely new labels and forms of stigma may develop (e.g., “carrier” or “high risk”) and that genetic information will provide new mechanisms of discrimination (Chadwick 2000; Phelan 2002; Phelan, Cruz Rojas, and Reiff 2002; Wachbroit 2001).

To understand how the face of stigma may change in an increasingly geneticized society, concepts about stigma need to be integrated with theory and concepts relating to causal attributions. Two sets of ideas are particularly relevant. Attribution theory (Corrigan 2000; Weiner 1986, 1995) leads to optimistic predictions about the impact of geneticization on stigma. Another set of ideas relating to “genetic essentialism” (Lippman 1992; Nelkin and Lindee 1995) lead to pessimistic predictions.

Attribution Theory

The destigmatizing potential of geneticization is prominent in the arguments of activist groups such as the National Alliance for the Mentally Ill, as well as some gay rights activists (Conrad 1997; Johnson 1989; Whisman 1996). These arguments are based on the principle that genetic and other biological explanations of stigmatized behaviors reduce blame. For example, consider the following passage from the web site of one prominent activist organization: “A ‘mental illness’ is not caused by bad parenting and is not a character weakness . . . These illnesses are due to biochemical disturbances in the brain . . . The shame and fear once associated with cancer has largely been dispelled by accurate information and understanding. The same will happen for brain diseases—mental illnesses—once the facts are known” (National Alliance for the Mentally Ill Oregon 1997).

Attribution theory (Weiner 1986, 1995) and empirical tests of the theory support these arguments. The theory states that the attributions people make about the cause of an outcome
influence emotions, expectancies, and behavior toward the individual affected by the outcome. One important application of the theory has been to stigmatized behaviors (Corrigan 2000; Weiner, Perry, and Magnussen 1988). According to the theory and empirical studies of stigmatized behaviors, attribution of low causal responsibility for a stigmatized characteristic (e.g., brain dysfunction due to accidental injury rather than illicit drug abuse) is associated with less blame and more positive emotions, that is, pity rather than anger, which in turn lead to an inclination to help the person and a disinclination to punish (Corrigan et al. 2000; Reisenzein 1986; Rush 1998; Weiner 1988). In addition, research shows that the causes of physical illnesses are seen as less controllable than the causes of mental or behavioral characteristics (Weiner et al. 1988) and that mentally ill persons are viewed as less blameworthy and amoral when subjects perceived mental illnesses to be biological rather than psychosocial in origin (Furnham and Rees 1988; Mehta and Farina 1997). Taken together, these findings suggest that genetic attributions should reduce the perceived causal responsibility and the negative emotions and behaviors associated with a stigmatized characteristic. Only one study I am aware of examined genetic explanations of a stigmatized characteristic in relation to attribution theory variables: Phelan and colleagues (2002) found that people who ascribed an individual's schizophrenia to genetic factors were less likely to think the person did something to cause the problem. A few studies have examined the association between genetic attributions and outcomes that are outside the explicit scope of attribution theory but that may be influenced by attribution processes. Martin, Pescosolido, and Tuch (2000) found that people who attribute a mental illness or substance use problem to genetic factors desire less social distance from a person with that characteristic. Similarly, experimental research suggests that genetic causal attributions are associated with less prejudice against homosexuals (Piskur and Degelman 1992). However, Teachman and colleagues (2003) found no impact of genetic causal attributions on bias against overweight people.

**Genetic Essentialism**

Several authors have argued that "genetic essentialist" thinking is on the rise (Lippman 1992; Nelkin and Lindee 1995). In a genetic essentialist view, genes form the basis of our human and individual identities (i.e., "we are our genes") and are strongly deterministic of behavior, so that if one has genes associated with some behavior, that behavior will definitely occur and is fixed and unchangeable" (Alper and Beckwith 1993:511). According to James Watson, "our fate is in our genes" (quoted in Jaroff 1989).

A genetic essentialist viewpoint suggests that genetic characteristics are irrevocably, or at least very firmly, attached to an individual and, by extension, to those with whom the person shares genes. Consideration of positively valued characteristics such as beauty or intelligence make it clear that genetic essentialism is not inherently stigmatizing. However, when applied to negatively valued qualities, genetic essentialism should exacerbate stigma via its influence on several perceptions: (1) that the person is fundamentally different from others, (2) that the problem is persistent and serious, and (3) that the problem is likely to occur in other family members. These perceptions in turn should increase behavioral orientations of social distance and reproductive restrictions.

**Differentness.** If a stigmatized characteristic is perceived to be genetic and genes are viewed as the basis of personal identity, the stigmatized person may be seen as more fundamentally different ("them") from others ("us").

**Persistence.** Once a person has been "marked" with a stigmatizing label, the label is difficult to shed (Jones et al. 1984). Genetic essentialism should exacerbate this problem. If the stigmatized behavior is "in your genes" and your genes are seen as the essence of you as a person, then optimism for full and permanent "recovery" from the deviant status may be further reduced.

**Risk to family members and associative stigma.** Evidence suggests that family members of stigmatized individuals suffer "associative" (Mehta and Farina 1988) or "courtesy" (Goffman 1963) stigma (Mehta and Farina 1988; Phelan, Bromet, and Link 1998; Wahl and Harman 1989; Weyland 1983). Goffman described courtesy stigma as resulting from a person's social connectedness to a stigmatized individual; geneticization allows social connections to be fortified by biological ones. If we believe that genes determine behavior and genes are shared, then geneticization should increase...
the expectation that the stigmatized behavior will emerge in heretofore "normal" biological relatives, particularly younger relatives who are seen as still being in the risk period for onset of the behavior.

**Seriousness.** Not only should an enduring, "contagious" problem (as discussed above) be seen as more serious than one that is transient and self-contained, genetic essentialism may heighten perceived seriousness in its own right: A problem that affects one's essence (one's genes) must be a serious problem.

In contrast to attribution theory, little empirical data bear on genetic essentialism (but see Condit and Williams 1997). However, consistent with these ideas, Phelan and colleagues (2002) found that people who ascribed an individual's schizophrenia to genetic factors believed the problem was less likely to improve and that family members were more likely to develop a similar problem.

Of the foregoing beliefs related to genetic essentialism, only perceived differentness is a direct measure of stigma. However, I expect perceptions of seriousness, persistence, and transmissibility to affect the degree to which deviant individuals and their families are stigmatized via social rejection and reproductive restriction.

**Social distance.** Social rejection is a core component of stigma (Goffman 1963; Jones et al. 1984; Link and Phelan 2001). If genetic essentialism is operative, a genetic attribution should increase social distance for several reasons. First, if the person is viewed as more different, social distance should increase. Second, if the stigmatized behavior is seen as a core and permanent part of the person, then any contact has the potential to become a long-term commitment to social relations with a deviant person, and contact may therefore be avoided. Third, because of the idea that deviance-determining genes are shared by biological relatives, intimate social distance (e.g., unwillingness to date or marry) involves an added dimension of "genetic contamination" and should be particularly heightened by geneticization. Because genetic essentialism is predicted to increase associative stigmatization of family members, genetic attributions should increase social distance both from the stigmatized individual and, even more strongly, from his or her biological relatives.

**Reproductive restriction.** Because genetic essentialism should heighten fear that a stigmatized characteristic will be passed to the next generation, it should increase endorsement of reproductive restriction when those characteristics are understood to be genetically influenced, as occurred during the eugenics movement of the late nineteenth and early twentieth centuries (Haller 1963; Kevles 1985).

**The Two Theories**

Attribution theory predicts that geneticization will reduce stigma, while genetic essentialism predicts that stigma will be exacerbated. However, different outcomes are implied by the two theories. Attribution theory predicts stigma reduction via reduced blame, anger, and punishment and increased sympathy and helping. Genetic essentialism predicts stigma magnification via increased perceptions of differentness, and—indirectly through increased perceptions of seriousness, persistence, and risk to family members—via increased social distance and reproductive restriction. It is possible that both theories are correct and operate simultaneously. It is also possible that geneticization will have little effect on stigma. This would be the case if perceptions of the stigmatized behavior itself (e.g., fear of violence by people with mental illness or aversion to sexually deviant acts) are the primary determinant of stigmatizing responses rather than beliefs about its causes.

**RESEARCH STRATEGY**

Mental illness was chosen as the focus of this initial study of geneticization and stigma for several reasons. Mental illness is highly stigmatized and has long been examined by students of deviance and stigma. There is clear evidence for the importance of genetic factors in several forms of mental illness (Kendler and Diehl 1993; Members of the NIMH Genetics Workshop 1997), and interest in their genetic determinants is intense (Conrad 2001). Also, there is keen interest among advocacy groups in the impact of biological explanations on stigma associated with mental illness. To enhance generalizability of the findings, I examine two distinct disorders that are recognizable to a large proportion of the American public (Link et al. 1999): schizophrenia and major depression.

A direct examination of the impact of the genetics revolution on stigma would involve
tracking changes over time in beliefs about the causes of mental illness and in stigmatizing attitudes and beliefs. In the absence of such data, my colleagues and I conducted a vignette experiment in which a hypothetical person was described who had been hospitalized for schizophrenia or major depression, and respondents were randomly assigned to hear different versions of the cause of the problem, from strongly genetic to not genetic. Respondents were then queried about stigma-relevant beliefs, attitudes, and behavioral orientations. Thus we ask: If a stigmatized characteristic is attributed to genetic factors (as would occur increasingly with increased geneticization), how are people's reactions affected? The vignette experiment was embedded in a large, nationally representative telephone survey, combining the strong internal validity represented by the experimental design with a level of external validity not often obtained in experimental studies in social psychology.

DATA AND METHODS

Sample

The target population comprised persons age 18 and older living in households with telephones in the continental United States. The sampling frame was derived from a list-assisted random-digit-dialed telephone frame. A respondent was randomly selected from among all adults in the household. Telephone interviews were conducted with 1,241 respondents between June 2002 and March 2003. Interviews were conducted in English, Spanish, Mandarin, and Cantonese and averaged 20 minutes. The sample was stratified to oversample Puerto Ricans, Chinese Americans, and people with a family history of psychiatric hospitalization. Using estimation procedures of the Council of American Survey Research Organizations (1982), the response rate was 62 percent.

Comparison with the Census. To evaluate sample selection bias, I compared the weighted analysis sample with 2000 Census data for gender, educational attainment, household income, and age (see Table 1). Correspondence with the Census is good in terms of age, but the sample overrepresents women and people with higher socioeconomic status (SES). To assess the possibility that sampling biases affected the findings, results will be examined for their generality across gender, SES, age, and ethnicity.

Weighting. Results are weighted to account for poststratification adjustment to national counts by race/ethnicity and differential selection probabilities based on race/ethnicity and family history of psychiatric hospitalization. To calculate statistical tests, I used Sudaan (Research Triangle Institute 2001), which estimates standard errors for complex survey designs.

The Vignettes

All 1,241 respondents participated in one of two vignette experiments; 641 were randomly assigned to the experiment analyzed in this article. In the vignette descriptions, the following information was randomly varied: whether the vignette described a person with schizophrenia (N = 307), major depressive disorder (N = 301), or ruptured disk (N = 33); the vignette subject's gender and SES; and cause of the illness. The vignette subject's race/ethnicity was matched to the respondent's, and appropriate names were selected for each ethnic group. Below is one version of the vignette; ital-

| Table 1. Comparison of Selected Sample Characteristics of Weighted Analysis Sample (N = 426) with 2000 Census Data for Individuals Age 18 or Older |
|---------------------------------|--|--|
| **Weighted Analysis Sample** | **Census** |
| Female (%) | 63 | 51 |
| High school education or greater among those 25 or older (%) | 90 | 80 |
| Median household income (in dollars) | 48,000 | 41,994 |
| **Age (%)** | | |
| 18 to 44 | 51 | 53 |
| 45 to 64 | 35 | 30 |
| 65 and older | 14 | 17 |

* The Census reports educational attainment for individuals who are 25 or older.
icized text indicates characteristics that are varied, and alternative versions of the variables follow this example.

Imagine a person named Anne. She is a single, 25-year-old white woman. Since graduating from high school, Anne has been steadily employed and makes a decent living. Usually, Anne gets along well with her family and coworkers. She enjoys reading and going out with friends. About a year ago, Anne started thinking that people around her were spying on her and trying to hurt her. She became convinced that people could hear what she was thinking. She also heard voices when no one else was around. Sometimes she even thought people on TV were sending messages especially to her. After living this way for about six months, Anne was admitted to a psychiatric hospital and was told that she had an illness called "schizophrenia." She was treated in the hospital for two weeks and was then released.

About a year ago, Anne started having severe pain in her lower back and left leg. On some days, just going about her daily business seemed like agony, and she had to miss work several times. Taking aspirin or Tylenol wouldn't make the pain go completely away. After having these problems off and on for about six months, Anne was admitted to a hospital and was told that she had a "ruptured disk" in her back. She had to have surgery.

**Alternate causal statements.** Causal statements were varied in one of two additional ways: (1) "partly due to genetic or hereditary factors. In other words, her genetic makeup played a role in contributing to the problem, but other factors were also involved," or (2) "not due to genetic or hereditary factors. Her problem was definitely not genetic."

**Sibling.** After some questions about the vignette subject, information was introduced about a sibling: "Here is some more information about Anne. Anne has a sister who is 20 years old, five years younger than Anne. Her sister has never shown any signs of having a problem like Anne has." The sibling is always the same gender as the vignette subject.

**Independent Variable**

Three versions of the causal attribution statement were presented. However, the three levels were not monotonically related to most outcome variables, precluding treating causal attribution as a continuous measure. Mean levels of the outcome variables were more similar for respondents who heard the genetic and partly genetic versions than for those who heard the nongenetic version. Given these facts, I dichotomized genetic attribution (genetic or partly genetic = 1; not genetic = 0).

**Dependent Variables**

**Attribution theory.** These constructs were measured with single items: (1) "Anne is to blame for her condition" (blame); (2) "When completely worthless and even had thoughts about killing herself. After having these problems off and on for about six months, Anne was admitted to a psychiatric hospital and was told that she had an illness called "major depressive disorder." She was treated in the hospital for two weeks and was then released.

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you think of Anne, you feel anger toward her” (anger); (3) “When you think of Anne, you feel sympathy toward her” (sympathy); (4) “Anne should be given government assistance for health care for her problem if she needs it” (help); or (5) “If Anne did something violent because of her problem, she should be dealt with by the police and courts just like any other person would be” (punishment). Response options were “strongly agree,” “somewhat agree,” “somewhat disagree,” and “strongly disagree.” This response scale is used for all dependent measures except where indicated otherwise.

_Genetic essentialism._ Perceived differentness was measured with the item, “I believe there is something about Anne that makes her fundamentally different from most people.” Perceived persistence was measured with the item, “In your opinion, Anne will probably continue to have problems like the ones I described for her whole life.” Perceived risk to family members was measured with two individual items: (1) “In your opinion, how likely is it that Anne’s sister will develop the same kind of problems that Anne has?” and (2) “In your opinion, how likely is it that a child of Anne would develop a problem like she has?” (“very likely,” “somewhat likely,” “not very likely,” and “not likely at all”). Perceived seriousness was measured with the item, “How serious would you consider her problem to be?” (“very serious,” “somewhat serious,” “not very serious,” and “not serious at all”).

I measure social distance from the vignette subject with a five-item scale (Cronbach’s alpha = .89). Three items involve what I term “intimate” social distance: “How would you feel about having Anne date/marry/have a baby with one of your children?” (“very positive,” “somewhat positive,” “somewhat negative,” and “very negative”). Two involve “casual” social distance: (1) “How willing would you be to make friends with Anne?” and (2) “How willing would you be to have Anne start working closely with you on a job?” (“definitely willing,” “probably willing,” “probably unwilling,” and “definitely unwilling”). Social distance from the sibling (alpha = .89) uses the same items, but asks about the sibling, for example, “Anne’s sister.” Subscales were also computed as follows: Intimate social distance from the vignette subject, alpha = .94; casual social distance from the vignette subject, alpha = .69; intimate social distance from the sibling, alpha = .93; and casual social distance from the sibling, alpha = .75.

Respondents were randomly assigned to answer social distance questions either about the vignette subject or the sibling, and the two forms of social distance are analyzed separately. Thus, analyses of social distance are based on roughly half the number of respondents as the other outcome measures.

_Reproductive restriction_ is measured with a two-item scale (alpha = .67): (1) “Anne should not get married—that is, she should stay single,” and (2) “Anne should not have any children of her own—that is, she should remain childless.”

_Covariates_

Because the independent variable was randomly assigned, confounding is not an issue. However, I controlled for several demographic and attitudinal variables (1) to increase the precision of the estimate of the effects of genetic attribution and (2) to assess the generality of the effects of genetic attribution across these variables. These items were asked before the presentation of the vignette.

_Sociodemographic factors_ include gender (1 = female; 0 = male); age; education (eighth grade or less = 1; some high school = 2; high school graduate or GED = 3; trade or technical school = 4; some college = 5; college graduate = 6; postgraduate work/advanced degree = 7); and _household income_ (under $20,000 = 1; $20,000 to $39,999 = 2; $40,000 to $59,999 = 3; $60,000 to $79,999 = 4; $80,000 or more = 5). For the present analyses, _ethnicity/race_ was measured with dummy variables for Asian, Hispanic, black, and “other,” with white as the reference category.

_Attitudinal factors_ included _political conservatism_ (very liberal = 1; somewhat liberal = 2; moderate = 3; somewhat conservative = 4; very conservative = 5). Prior belief that genetic factors are important for mental illness (importance of genetics) was measured with the item, “How important do you think a person’s genetic makeup is in influencing whether or not a person will develop a serious mental illness?” (“very important,” “somewhat important,” “not very important,” or “not important at all”). _Eugenic concerns_ were measured with a scale (alpha = .60) comprising three items: (1) “If a couple has a 1-in-4 chance of having a child with a serious genetic defect, they should not give birth to any children of their own”; (2) “Every person...
should be required to have a genetic screening test before getting married”; and (3) “In choosing a marriage partner, it’s important to know whether there is history of mental illness in the family.”

Manipulation Checks

At the end of the interview, recall was assessed by asking respondents whether the genetics expert said Anne’s problems were “strongly,” “partly,” or “not due to genetic or hereditary factors” and agreement by asking, “Do you accept what the expert said—that is, do you agree that what the expert said about the cause of the problem could be correct?” Because I am interested in the effects of believing a mental illness is genetically influenced, I limit the results to respondents who recalled the expert’s statement correctly and who “strongly” or “somewhat” agreed with the statement. Of the 608 respondents assigned to the schizophrenia or major depression vignettes, 182 were eliminated on the basis of recall and agreement, leaving a final sample of 426 for the main analyses (212 for schizophrenia; 214 for depression).8

RESULTS

Are Mental Illnesses Stigmatized?

Before turning to the main findings concerning geneticization and stigma, I address a more basic question: Is there evidence in the present data for negative attitudes toward mental illness? To address this question, I compared social distance for vignettes describing the two mental illnesses and a presumably nonstigmatized problem, ruptured disk. The reason for this comparison is that, unless expressed attitudes are extremely negative, the use of a comparison point may be the only way of judging whether stigma is present. For example, if 60 percent of people express willingness to be friends with a person who has been hospitalized for schizophrenia, it is difficult to say whether this indicates social rejection unless we know how many would be willing to be friends with a similar person without schizophrenia. Table 2 shows that significantly less social distance was desired from the vignette subject with a ruptured disk than from either of the vignette subjects with a mental illness.

Geneticization and Stigma

My main goal is to evaluate the effect of geneticization on the stigma associated with mental illnesses, and the remaining analyses are based on the schizophrenia and major depression vignettes only. Because of the use of multiple dependent measures and consequent concerns about Type I error, I first conducted multivariate tests to evaluate whether genetic attribution significantly affects the attribution theory outcomes as a set and the genetic essentialism outcomes as a set. I then regressed each individual outcome measure on genetic attribution, controlling for vignette disorder (schizophrenia vs. depression); respondent gender; age; education; family income; race/ethnicity; political conservatism; perceived importance of genetics; and eugenic concerns. To evaluate the generality of the effects of genetic attribution, I assessed its interaction with each of the covariates. Interaction terms were retained only if the interaction terms as a set significantly increased R-squared ($p < .05$) and the coefficient for the individual interaction term was significant ($p < .05$). Table 3 reports unstandardized coefficients for genetic attribution after all control variables and significant interaction terms were entered.

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<tr>
<th>TABLE 2. Mean Scores (and standard deviations) on Social Distance from Vignette Subjects with Schizophrenia, Major Depression, and Ruptured Disk</th>
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<td><strong>Schizophrenia (N = 57)</strong></td>
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<td>Social distance</td>
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*** $p < .001$

Notes: p-values indicate significance of difference between schizophrenia and ruptured disk and between major depression and ruptured disk, based on regressing social distance on dummy variables for schizophrenia and major depression. Standard deviations are in parentheses. Only vignettes with a nongenetic causal attribution are included in this analysis.
TABLE 3. Regressions of Stigma-Related Outcomes on Genetic Attribution, Vignette Disorder, Sociodemographic and Attitudinal Factors, and Interactions of Genetic Attribution with All Other Independent Variables

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<td>(.093)</td>
<td>(.068)</td>
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* p < .05; ** p < .01; *** p < .001

Notes: Educational attainment, family income, age, gender, ethnicity (dummy variables for black, Asian American, Hispanic, and "other"), political conservatism, eugenic concerns, and belief in the importance of genetic factors in mental illness are controlled. Results for interactions are reported in notes to the text. Differences in sample size are due to missing data on dependent variables. Sample sizes for social distance outcomes are substantially smaller because respondents were randomly assigned to answer social distance questions about the vignette subject or the sibling. Unstandardized coefficients and standard errors are shown only for genetic attribution. Standard errors are in parentheses.

Both sets of hypotheses predict that geneticization influences cognitive and emotional factors that in turn affect behavior. Therefore, for all behavioral orientation outcomes (i.e., help, punishment, social distance, and reproductive restriction) that were significantly influenced by genetic attribution, Table 4 presents a series of multiple regression models in which the mediating role of theoretically relevant cognitive and emotional factors is assessed by adding them to the Table 3 equations.

Attribution theory. Attribution theory predicts that blame, anger, and punishment should be decreased by genetic attribution and that sympathy and help should be increased. A multivariate test indicated that genetic attribution was not significantly related to these dependent variables when considered as a set. Considered individually, panel A of Table 3 shows that genetic attribution significantly affected only one dependent measure: It decreased punishment \( p < .05 \), as predicted by attribution theory.9 Not surprisingly, given these findings, panel A of Table 4 shows that the impact of genetic attribution on punishment was not significantly mediated by the attribution model attitudes and emotions.

Genetic essentialism. Based on ideas about genetic essentialism, I predicted that genetic attribution would increase the perceived differentness of the individual, persistence and seriousness of the problem, and risk that the person’s child and sibling would develop a similar problem. I also hypothesized that, because of its impact on these beliefs, genetic attribution would increase social distance from the vignette subject, social distance from the sibling, and reproductive restriction. The multivariate test showed that genetic attribution was significantly associated with these outcomes as a set (Wald F-statistic; \( p < .001 \)).

Beliefs. Panel B of Table 3 shows that genetic attribution affected all these beliefs in the predicted direction and that all but one association (perceived differentness) was statistically significant. Perceived seriousness \( p < .01 \), perceived persistence \( p < .01 \), perceived risk to sibling \( p < .05 \), and perceived risk to child \( p < .001 \) were all increased.

Behavioral orientations. I have argued that,
to the extent that genetic essentialist views prevail, genetic attributions should increase the desire for social distance and reproductive restriction. Panel C of Table 3 shows that genetic attribution had no significant impact on social distance from the vignette subject or on reproductive restriction. However, it did increase social distance from the sibling ($p < .05$). Genetic essentialism also suggests that, because of the fear of genetic contamination, genetic attributions should affect intimate social distance more strongly than casual social distance. Therefore, I repeated the analyses with these two subscales. Genetic attribution had a small and nonsignificant effect on both forms of social distance from the vignette subject. For the sibling, the positive impact of genetic attribution was larger for intimate social distance ($B = .264, \text{s.e.} = .086, p < .05$) than for casual social distance, which did not attain statistical significance ($B = .151, \text{s.e.} = .082, p = .066$).

A genetic essentialist view predicts that the significant increase in intimate social distance from the sibling should at least in part be explained by beliefs that the problem is more serious and persistent, the person is more different, and the sibling and child are at greater risk of developing a similar problem. Panel B of Table 4 shows that this was so. When these beliefs were added to the model, the coefficient for genetic attribution was reduced by 20 percent and lost statistical significance.

**DISCUSSION**

This article addresses one potential social ramification of the genetics revolution—the impact that geneticization of deviant behavior may have on stigma. I used attribution theory and ideas about genetic essentialism to derive contrasting predictions about the consequences of geneticization for stigma. Attribution theory suggests that when the cause of a stigmatized behavior is viewed as being out of the individual’s control (as is the case with genetic causes) the individual is seen as blameless and thus elicits more positive emotions and behavior. On the pessimistic side is the idea of genetic essentialism, according to which genetic factors are increasingly viewed as the basis of an individual’s identity and as strongly deterministic of behavior. Genetic essentialism implies that geneticized deviance should be viewed as more serious, less likely to change, and more likely to appear in biological relatives and should make the stigmatized person seem more fundamentally different from others. In turn, these perceptions should worsen stigma outcomes of social rejection and reproductive restriction.

I tested these predictions using the example of mental illness and a vignette experiment embedded in a large national survey. I experimentally manipulated whether schizophrenia or major depression was attributed to genetic factors and measured a broad array of outcomes related to the two sets of hypotheses.

Support was weak for hypotheses based on attribution theory. Genetic attribution was not
significantly related to the hypothesized outcomes (blame, sympathy, anger, helping, and punishing) as a set. Considered individually, only one prediction was supported: When mental illness was attributed to genetic causes, respondents were more likely to endorse leniency in how the police and courts should deal with the person if he or she did something violent as a result of his or her problem. It is important to note that the relatively weak support for these predictions does not negate the validity of attribution theory per se. The findings do, however, question the optimistic implications of the theory that genetic explanations for deviant behaviors will reduce stigma.

Support was stronger for predictions based on genetic essentialism. Genetic attribution was significantly related to the hypothesized outcomes as a set ($p < .001$). Except for perceived differentness, each of the beliefs implied by genetic essentialism was increased by genetic attribution, most at the .01 or .001 probability level. Thus, genetic causes seem to imply to people a greater seriousness, tenacity, and pervasiveness or "spread" of the deviance. Predictions regarding behavioral orientation outcomes were only partially supported. Genetic attribution had no impact on social distance from the person with mental illness or on reproductive restrictiveness. Genetic attribution did, however, increase social distance from the mentally ill person's sibling, particularly with regard to intimate social distance, and this effect was partly mediated by genetic essentialist beliefs.

For the most part, the effects of genetic attribution were consistent across demographic groups. The only notable exception was for Asian Americans versus European Americans, where genetic attribution had a more pronounced effect on Asian Americans' reactions, substantially reducing blame and anger and increasing perceived risk to the sibling. This suggests Asian American attitudes may be more strongly influenced by the genetics revolution. However, the net effect with regard to stigma is not clear, because reduced blame and anger are destigmatizing outcomes, whereas an increased perception of risk to the sibling suggests an increase in associative stigma.

Two inherent limitations of the vignette experiment should be noted. First, vignettes describe specific scenarios. We presented two relatively severe mental illnesses that led to hospitalization because we wanted to observe the effects of genetic attribution on cases that would carry a significant "baseline" level of stigma. We described first episodes because we wanted to allow respondents to imagine what course the illness would take. These choices necessarily limit our ability to generalize. In particular, the effects of genetic attribution might differ for less severe forms of mental illness or illnesses that did not result in hospitalization, but stigma is also less of a concern in these cases. Second, respondents are confronted with a hypothetical situation, and only behavioral intentions, not actual behaviors, are measured. It is possible that real-life reactions to real people would differ from those observed in the vignettes. Nevertheless, it is reasonable to suppose that people's stated levels of social distance would have some bearing on their actual behavior. A third potential limitation shared by virtually all social research but perhaps especially noteworthy in this case concerns generalizability across time. The observed impact of genetic attribution on attitudes reflects notions the public currently hold about the meaning and implications of genetic causation. It is possible that, as the genetics revolution moves forward, a more complex and accurate understanding of genetics will be adopted by the public, and that in 10 years the same experiment would produce significantly different results.

An unexpected finding was that the three levels of genetic causation (genetic, partly genetic, and not genetic) were generally not monotonically related to the outcome measures. This suggests a more complicated relationship between genetic attribution and attitudes than I expected or than I can explain. A partly genetic explanation does not simply work like a weaker dose of a purely genetic explanation. This finding, combined with the fact that the partly genetic explanation was the one most likely to be accepted by respondents, suggests that the public's ideas about gene-environment interactions should be studied.

Because of the experimental design employed, the observed associations between genetic attribution and outcome variables cannot be attributed to confounding factors, and we can be confident that these associations reflect the causal impact of genetic attribution. Moreover, because the experiment was embedded in a nationally representative survey and because the findings, for the most part, held within demographic subgroups of the sample, we can conclude that the findings are reasonably representative of the adult U.S. population. Given the findings and
the strengths and limitations of the study, the following conclusions are warranted.

CONCLUSIONS

Genetic attribution had its strongest and most consistent effects on a set of related beliefs about the seriousness of the mental illness and the degree to which it persists over time and transfers to biological relatives, beliefs that are consistent with the idea of genetic essentialism but that do not directly indicate stigma. Here I consider two questions: (1) Are these public beliefs consistent with common scientific beliefs? and (2) What is their bearing on stigma?

To what extent do perceptions that genetic causes increase severity, persistence, and transmissibility reflect common scientific beliefs about genetic influence, and to what extent do they reflect exaggerated beliefs indicative of "genetic essentialism?" Alper and Beckwith (1993) have convincingly argued that there is no necessary association between the degree of genetic influence on a behavior and the behavior's malleability or persistence. Regarding transmissibility, it is clear that genes transmit traits to offspring. However, environmentally determined traits such as wealth and choice of religion are also powerfully transmitted to offspring. Are genetically borne traits more transmissible than other traits, in general? This question is currently unanswerable. However, for the mental illnesses investigated in this article, particularly schizophrenia, twin and adoption studies indicate that more of the familial aggregation of the characteristic is due to genetic than to nongenetic familial factors (Kendler and Diehl 1993). This suggests that respondents' reactions to the causal statements in the vignettes in part reflect perceptions that are congruent with current psychiatric research findings. However, studies of people undergoing genetic testing and counseling have found that individuals tend to overestimate the actual risk of developing a disease based on genetic profiles or family histories (Lerman et al. 1995; Evans et al. 1993). This suggests that public beliefs about the transmissibility of genetic characteristics are likely exaggerated by genetic essentialist thinking.

Finally, regardless of congruence with scientific thinking, it is important to consider the consequences of beliefs that genetic causes of a mental illness increase its seriousness, persistence, and transmissibility. As I have noted, they are not in themselves indicators of stigma. However, they have the potential to influence stigma indirectly by magnifying the perceived seriousness of the negatively labeled behavior and the firmness with which the label is attached to the person and his or her family. Is there evidence that these beliefs affect stigma? For the person with mental illness, the answer is no (a point to which I return below). For the person's sibling, however, these beliefs demonstrate their relevance to stigma by explaining a significant portion of the effect of genetic attribution on social distance.

Let us consider, finally, the overall impact of genetic attribution on direct indicators of stigma. Here we find significant but more modest effects and a situation that is somewhere between the worst fears of the pessimists and the best hopes of the optimists. One stigma outcome—the tendency to punish—was significantly reduced by a genetic causal attribution, and one—social distance from the sibling—was significantly increased. As noted earlier, the processes described by attribution theory and genetic essentialism are not mutually exclusive, and these findings support the idea that the effects of geneticization on stigma are complex, ameliorating stigma along some dimensions while exacerbating it along others. An important aspect of this complexity has to do with family relationship. In these results, the aspect of stigma that was reduced—punishment—affected the person with the illness, whereas the aspect of stigma that was increased—social distance—affected the sibling. If two important consequences of geneticization are decreased blame and increased fear of genetic contamination, then geneticization may be most beneficial to parents and most harmful to younger relatives, such as siblings, children, and cousins of the ill person. Geneticization may absolve parents of causal responsibility, a benefit not likely to be shared by siblings or children who would not be held responsible for the illness under any causal explanation. Issues of genetic contamination are most salient for younger relatives, individuals who will be seeking mates and who may be seen as squarely in the risk period for developing the illness themselves. These issues are less problematic for parents.

For the person who actually bears the deviant status, issues of causal responsibility and genetic contamination may both be relevant. However, in this case, the effects of causal explanation may be largely overwhelmed by negative
responses to the illness itself. Consistent with this possibility, social distance from the person with mental illness was nearly impervious to the manipulation of cause. However, social distance was strongly influenced by the type of disorder described in the vignette (schizophrenia, major depression, or ruptured disk). In addition, factors such as fear (reflecting perceptions of the behavioral manifestations of mental illness itself, not its causes) strongly influenced social distance and restriction (results not shown).

In conclusion, three findings are particularly noteworthy. First, the most prominent expectation about the consequences of geneticization is that it will reduce stigma, and public education campaigns and direct-to-consumer advertising currently promote the idea that mental illnesses are biologically based. The present findings do not confirm these positive expectations, and enthusiasm for this idea and practices based upon it should be reevaluated. Second, genetic attribution had strong effects on beliefs about persistence and transmissibility. Further research should explore the implications of these beliefs for a broader set of outcomes, including orientations to mental health treatment, genetic testing, and selective abortion, and for other stigmatized statuses. Research should also be specifically designed to assess the accuracy of these beliefs. Finally, in thinking about the impact of geneticization on stigma, it is essential to consider the whole family. The present findings indicate that the most harmful effects of geneticization are not for the individual with a deviant status but for family members who may never in their lives display the deviancy in question but who may nevertheless become tainted and rejected via a genetic connection to their stigmatized relative.

NOTES

1. From a labeling theory perspective, the term “deviant” is not derogatory. Nevertheless, in common usage, the term has negative connotations. For this reason, I will usually employ the term “stigmatized” rather than “deviant.”

2. Medicalization and geneticization are related but independent phenomena. Both involve movement away from moral definitions of stigmatized behavior, but genetic and medical definitions are not identical: Discovering genetic bases of sexual orientation would not imply that homosexuality is a medical condition (Conrad 2000), and to define a behavior in medical terms does not imply that it is influenced by genetics. There is, however, a large overlap between medicalization and geneticization, especially with the strong focus of the HGP on illness. Moreover, many consequences of geneticization are likely to mirror those of medicalization, for example, the conditional legitimation of the stigmatized behavior, dislocation of responsibility, and the domination of expert control (Conrad and Schneider 1992).

3. The theory deals with three dimensions of causal attribution: controllability, stability, and locus. However, controllability is the dimension hypothesized to affect evaluative reactions to others, and this is the dimension that has been considered particularly relevant to the problem of stigma by both researchers and activists.

4. I restrict the meaning of the term “geneticization” to an increased tendency to define human characteristics in genetic terms and use “genetic essentialism” to describe the closely related ideas of genes as the basis for human identity and of genetic reductionism and determinism.

5. This idea is also consistent with attribution theory (Weiner 1995). Controllability of the onset of a characteristic is often perceived to vary inversely with its stability (Jones et al. 1984; Weiner et al. 1988; Fisher and Farina 1979; Furnham and Rees 1988).

6. Race/ethnicity is not included in this comparison because data were weighted to the Census in terms of race/ethnicity.

7. This condition was included only to ascertain the presence of stigmatizing reactions toward the mental illnesses relative to a non-stigmatized physical condition (see Table 2). Genetic cause was not varied.

8. Because this selection modifies the random assignment of the independent variable and could introduce confounding factors, I did several checks. The only control variable that predicted retention in the final sample was younger age (p < .001). In the reduced sample, none of the control variables was significantly associated with the independent variable (genetic attribution), reducing concern about confounding factors. Finally, I repeated the analyses including all respondents regardless of recall or agreement status. The impact of genetic attribution was atten-
uated in these analyses, but the pattern of findings was similar to that reported here.

9. There were three significant interactions. A nongenetic attribution strongly elevated both blame and anger only among Asian Americans (p < .05 for both). Genetic attribution increased anger somewhat toward subjects with depression while decreasing it somewhat toward those with schizophrenia (p < .05).

10. For the multivariate test, the analysis pooled respondents who answered social distance questions about the vignette subject and those who answered about the sibling.

11. Genetic attribution significantly interacted with type of disorder and race/ethnicity in its effect on perceived risk to sibling. Whereas genetic attribution strongly increased perception of risk for both disorders, the elevation was greater for schizophrenia (p < .05 for the interaction). Genetic attribution increased perceived risk to the sibling among whites, but this elevation was even more pronounced among Asian Americans (p < .01). Surprisingly, for Hispanics, perceived risk to the sibling was higher when the problem was not attributed to genetic factors (p < .01).

REFERENCES


Jo C. Phelan is associate professor of sociomedical sciences at Columbia University. Her broad research focus is on social inequalities, particularly on social psychological aspects of those inequalities. Her current research interests include public attitudes and beliefs about mental illness, in particular stigma and the potential impact of the genetics revolution on stigma.