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Not all my fault": Genetics, stigma, and personal responsibility for women with eating disorders

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Abstract

Medical researchers and clinicians increasingly understand and present eating disorders (anorexia and bulimia nervosa) as biologically-based psychiatric disorders, with genetic risk factors established by high heritability estimates in twin studies. But there has been no research on interpretation of genetic involvement by people with eating disorders, who may hold other views. Their interpretations are particularly important given the frequent presumption that biogenetic framing will reduce stigma, and recent findings that it exacerbates stigma for other mental illnesses. To identify implications of genetic framing in eating disorders, I conducted semi-structured interviews with 50 US women with a history of eating disorders (half recovered, half in treatment; interviewed 2008–9 in the USA). Interviews introduced the topic of genetics, but not stigma per se. Analysis followed the general principles of grounded theory to identify perceived implications of genetic involvement; those relevant to stigma are reported here. Most anticipated that genetic reframing would help reduce stigma from personal responsibility (i.e., blame and guilt for eating disorder as ongoing choice). A third articulated ways it could add stigma, including novel forms of stigma related to genetic essentialist effacing of social factors. Despite welcoming reductions in blame and guilt, half also worried genetic framing could hamper recovery, by encouraging fatalistic self-fulfilling prophecies and genetic excuses. This study is the first to elicit perceptions of genetic involvement by those with eating disorders, and contributes to an emerging literature on perceptions of psychiatric genetics by people with mental illness.

Keywords

USA; stigma; eating disorders; genetics; women; anorexia nervosa; bulimia nervosa; mental illness

Medical researchers and clinicians increasingly understand and present eating disorders as biologically-based psychiatric disorders, frequently with the presumption that biological explanations will help reduce stigma (e.g., Herpertz-Dahlmann et al., 2011). Genetic risk factors are part of this multifactorial biological model, and supported by the high heritability of eating disorders as estimated in twin studies (48–76% for anorexia nervosa (AN); 50–83% for bulimia nervosa (BN); Striegel-Moore & Bulik, 2007). As with many other psychiatric disorders, the heritability identified by twin studies is still largely "missing" because no candidate genes have been correlated with eating disorder phenotypes (Pinheiro

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et al., 2010; also see Manolio et al., 2009). Nevertheless, biogenetic models of AN/BN have been disseminated via major media (e.g., Tyre, 2005), medical websites (e.g., Mayo, 2012), advocacy groups (e.g., EDC, 2008), and other sources. Against this backdrop, I consider in this paper how genetic reframing is interpreted by people with AN/BN.

There is evidence that genetic framing is helpful for countering stigma in eating disorders, despite findings to the contrary for other mental illnesses, such as schizophrenia. Recent studies show mental illness stigma is usually exacerbated by biogenetic framing (see reviews by Angermeyer et al., 2011; Read et al., 2006). Yet studies of AN/BN suggest stigma-alleviation with genetic etiology (Crisafulli et al., 2008; 2010; Wingfield et al., 2011), likely because eating disorder stigma centers on personal responsibility, rather than dangerousness or unpredictability (which genetic framing appears to exacerbate; Angermeyer et al., 2011).

In this article, I consider the impact of genetics on stigma according to people with a history of AN/BN. Stigma studies more often assess public attitudes than those of mental health consumers or recovered people (Schulze & Angermeyer, 2003). However, the labeled person's perceptions are important in the stigma process (e.g., Link et al., 1989). I examine whether people with AN/BN expect genetic causal attribution to exacerbate or alleviate stigma and self-stigma, and how it may do so. This study is the first to examine implications of genetics for stigma from the perspective of people with AN/BN, and contributes to an emerging literature on implications of genetics for people with mental illness generally (Laegsgaard et al., 2010; Meiser et al., 2005; Rusch et al., 2010). It also calls attention to unique features of eating disorders and how they shape interpretation of genetics.

Genetics and Stigma for Different Psychiatric Diagnoses

Genetic causal attribution tends to exacerbate stigma of mental illness (e.g., Angermeyer et al., 2011; Read et al., 2006). The optimistic predictions of attribution theory--in which the presence of a genetic causal factor increases compassion for individuals (Weiner, 1986; Phelan, 2005)--are not usually borne out. Indeed, a recent review contends that "biogenetic causal beliefs and diagnostic labeling by the public are positively related to prejudice, fear and desire for distance," for schizophrenia and other serious mental illnesses (Read et al., 2006: 303). Moreover, genetic causal attribution also makes mental illness seem more serious and persistent (e.g., Phelan, 2005; Bennett et al., 2008; Dietrich et al., 2006), and therefore more like an essential, defining aspect of the person ("genetic essentialism," Lippman, 1992; Nelkin & Lindee, 1995; Phelan, 2005).

However, the impact of genetics on stigma ought to vary by diagnosis, not least because stigma is different across mental illnesses. Schnittker (2008) found genetic attributions to correlate with perceived dangerousness in schizophrenia, but enhance social acceptance in depression (also see Goldstein & Rosselli, 2003; Cook & Wang, 2011; Dietrich et al. 2006). People with depression or eating disorders are similarly stereotyped as more "competent" (and "warm") than those with schizophrenia (Sadler et al., 2012), suggesting greater responsibility. As with depression, describing AN in terms of genetics elicited less blame or responsibility (Crisafulli, 2008; 2010; Wingfield, 2011), as well as less perceived triviality, weakness, and selfishness (Crisafulli, 2010), and self-destructiveness (Wingfield, 2011).

In general, interpretations of genetics in one disease context ought not be presumed to transfer to another (Sankar et al. 2006; Shostak et al., 2011; also see Timmermans & Haas, 2008). Eating disorders have specific features that may affect interpretations of genetics and stigma; in addition to the perception of those with eating disorders as competent, intentional actors, there is also a striking gender disparity (9:1 women to men; APA 2000). Gender stereotypes may encourage interpretation of AN/BN behaviors and their stigmatization as

vain, trivial, and voluntary. Despite their high mortality rate, eating disorders have been likened to "contested illnesses" (Giles, 2006: 466), which disproportionately involve women (see Barker, 2010). While emphasizing biological factors may help validate the existence of a condition (Barker, 2011; Fausto-Sterling, 1992; Zavestoski et al., 2004) and help women get their needs met, it can also serve an ideological function against women, by making social disparity appear natural and immutable (e.g., Nelkin & Lindee, 1995; Fausto-Sterling, 1992). The feminist cultural model of eating disorders (e.g., Bordo, 1993; Malson & Burns, 2009; Orbach, 1986) is thus critical of medical models for locating the problem in the individual body, rather than cultural contexts that direct many women toward destructive bodily practices. Cultural actors may be aware of gender stereotypes, gendered causal factors, and/or feminist perspectives as they interpret genetics in the specific context of eating disorders.

Although there are no genetic tests for treatment or diagnosis, some people with eating disorders and their families are likely to have encountered information about genetic risk factors and genetic research through the media, patient-oriented literature, and advocacy groups. For example, genetic research and theories on eating disorders have been disseminated by major media (e.g., a *Newsweek* cover story sub-titled with the phrase, "anorexia is probably hard-wired"; Tyre, 2005: 50) and popular medical websites (e.g., "There may be genes that make certain people more vulnerable to developing eating disorders"; Mayo, 2012). Biomedically-oriented advocates have also strategically publicized genetic research to "fight stigma with science," (EDC, 2008) and to argue for better insurance coverage (Bernstein, 2007). Some individuals with family history of AN/BN may have developed their own theories about genetic transmission, like people with other disorders (Walter et al., 2004). Even those without family history may interpret clinicians' questions about it to indicate genetic etiology, particularly given widespread public endorsement of other psychiatric conditions as "genetic or inherited problems" (Pescosolido et al., 2010). In summary, people with AN/BN may have heard of or thought about the idea that genes play a role, and are likely to interpret its potential meaning and consequences specifically for eating disorders.

Two Kinds of Stigma in Eating Disorders

AN and BN are characterized by bingeing, purging, and/or food restriction, with serious health consequences and high mortality rates compared to other psychiatric disorders (Arcelus et al., 2011; Sullivan, 1995). Their classification as psychiatric disorders (APA, 2000) confers the stigma of mental illness. Yet they are also trivialized as behavioral choices, which is theorized as stigma in studies of AN (e.g. Crisafulli, 2010). The interpretation of eating disorders as voluntary, chosen behavior is hereafter referred to as "volitional stigma." "Volitional" stigma provides an interesting contrast to stigma as usually studied. Rather than stigma from being mentally ill and set apart from "normals," volitional stigma involves being judged by normal behavioral standards. Stigma in eating disorders can thus be very broadly divided into two types: (1) stigma from being perceived to have a mental illness; and (2) stigma from AN/BN being interpreted as an ongoing voluntary behavioral choice rather than as a mental illness. After describing each broad type of stigma, I examine how genetic framing might affect each.

In the first type of stigma, the label of mental illness "marks" a person as different and "spoils" his identity (Goffman, 1963) by linking her to negative stereotypes, resulting in status loss and discrimination. Perception of the mental illness as severe would exacerbate such stigma by emphasizing difference from "normal" people (Phelan 2005). For eating disorders, being perceived to exhibit psychopathology is indeed stigmatizing (Rich, 2006).

This broad type of stigma resembles that of other mental illnesses, in which individuals are perceived to be unstable, dangerous, and fundamentally different from others.

By contrast, the second kind of stigma involves the "trivialization" of eating disorders as behavioral choices rather than serious mental illnesses. People with eating disorders are often perceived as *choosing* to behave as they do, because they are morally bad (vain, conformist, greedy for attention), and/or because eating disorders must not be so bad (Crisp et al., 2000; Crisp et al., 2005; Mond et al., 2006; Holliday et al., 2005; Stewart et al., 2006; Crisafulli et al., 2008; Roehrig & McLean, 2010). This "volitional" stigma is measured by endorsement of survey items describing people with AN/BN as "acting this way for attention," or "able to pull themselves together if they wanted to," etc. (Stewart et al., 2006: 322; Crisp et al., 2005; Roehrig & McLean, 2010). While personal responsibility is a component of stigma in other conditions (e.g., Corrigan et al., 2002), survey respondents hold people responsible for eating disorders more than for schizophrenia, depression, panic attacks, or dementia (Crisp et al., 2000). Greater volitional stigma in AN/BN may be due to a number of reasons: people are usually held responsible—or even given credit--for similar food-related behaviors (Roehrig et al., 2010); some with AN intentionally pursue and defend it as an identity (e.g., Giles, 2006; Rich, 2006); agency is implied in understandings of AN/BN as protest--"hunger strike" (Orbach, 1986)—or capitulation to women's oppression (Malson & Burns, 2009); and psychiatric definitions themselves imply volition (e.g., "refusal" to gain weight; APA, 2000).

Genetics and Two Kinds of Eating Disorder Stigma

What impact might genetic causal attribution have on these two broad forms of stigma? Genetic etiology tends to make mental illnesses seem more medical (Shostak et al., 2008), more serious (Phelan, 2005), and necessitating hospitalization and medication (Phelan et al., 2006). Therefore, the idea of genetic influence could exacerbate the first form of stigma (from being perceived as mentally ill), and alleviate the second form (from being perceived as behaving badly, rather than being mentally ill).

For the first form of stigma, biological attribution would locate the problem in the individual body. As with schizophrenia, genetics would be expected to exacerbate stigma by making the problem seem to be an essential and permanent aspect of the person (Phelan, 2005); rather than *having* a problem, the person *is* a problem. This "genetic essentialism" (Phelan, 2005; Lippman, 1992; Nelkin & Lindee, 1995) would make those with AN/BN fundamentally and permanently different from others. In the context of eating disorders, genetic essentialism would also draw attention away from social factors as outlined in the feminist/cultural model (e.g., Bordo, 1993), which has been theorized as a form of stigma because it sets women who struggle with disordered eating apart from the continuum of normal women as mentally ill (Way, 1995).

Regarding the second, volitional form of stigma, genes would help to alleviate stigma. The eating disorder may become something one *is* or *has,* but it may no longer seem like something one *does.* If genetic involvement makes mental illness appear more serious (Phelan, 2005) and disease-like (Shostak et al., 2008), volition is less plausible because people don't "choose" to get serious diseases. Second, genetic involvement is often counterposed to free will (Levitt & Manson, 2007; Parens, 2004), and following causal attribution theory, would relieve responsibility for causing the disorder (Phelan, 2005; Weiner, 1986). By either logic, perception of AN/BN as "genetic" would serve to alleviate volitional stigma. Researchers and advocates hope genetic factors will make people take eating disorders more seriously (Bulik, 2004; Stewart et al., 2006; Holliday et al., 2005;

O'Hara & Smith, 2007; EDC, 2008), and there is evidence to support such a strategy (Crisafulli et al., 2008; 2010; Wingfield, 2011).

Genetics and stigma reception

One way to examine the potential impact of genetics on mental illness stigma is to interview people with the diagnosis, whose evaluations draw upon past experiences and observations in their social contexts. Expectations about stigma – its content and likelihood--are highly important in shaping the impact of stigma. Stigma may be internalized (Corrigan & Watson, 2006; Livingston & Boyd, 2010), or resisted--by deflecting it from oneself (e.g., "I'm not like that") or challenging its validity (e.g., "we are not like that") (Thoits, 2011)--demonstrating the importance of recipients' interpretations. Moreover, stigma need not be enacted by others to be important; "felt" stigma—shame about a condition and fear of stigma-enactment (Jacoby, 1994)—also affects labeled persons, including whether and how one interacts with others (Link et al., 1989).

Although stigma-recipients' perceptions are central to stigmatizing process, their interpretations of genetic framing are rarely considered. Diagnosed people can provide more proximal information about the likely impact of genetic framing than the general public, whose views may not be communicated to or internalized by "targets." There are few data of this kind, and findings are mixed and of uncertain relevance to eating disorders. In a sample of adults with diverse psychiatric diagnoses, endorsement of genetic involvement was associated with increased guilt but not perceived responsibility (Rusch et al., 2010). For a sample of adolescents with diverse psychiatric diagnoses, endorsing biogenetic explanations correlated with greater self-stigma (but so did endorsing social, familial, and trauma-based explanations; Moses, 2010). However, two qualitative studies found that people with bipolar disorder (Meiser et al., 2005) or with depression (Laegsgaard et al., 2010) expected genetic involvement to relieve stigma.

Genetic reframing may or may not be deemed helpful for countering stigma by women with eating disorders. It may increase stigma for respondents who resist the idea that they have a mental illness (first form of stigma, above), reduce it for those who resist its classification as voluntary behavior (second form), or have other effects. How do women with eating disorders conceive of the impact of genetics on stigma? Do they expect genes to exacerbate or reduce stigma, and if so, how?

METHODS

Recruitment and Sample

This dataset consists of semi-structured interviews with 50 women who were in treatment for, or recovered from, either AN or BN (see Table 1). I recruited those in treatment (inpatients, outpatients, and participants in a BN treatment study) through a hospital-based clinic at a large US university. I recruited recovered women via mass email to the same university's faculty, staff, and students. Recovery status was defined as three years without significant restricting, bingeing, or purging (self-reported based on five questions). I excluded those with current AN/BN who were not in treatment, to reduce the risk of harm from fatalistic interpretations of genetics. While fatalism was possible for any respondent, it seemed less likely for recovered people, and mitigated for those receiving treatment. Moreover, interview questions and debriefing discouraged deterministic interpretations. Four clinicians approved the guide. A safety plan provided professional counseling in case of serious distress during the interview (there was none), and all respondents received information about local and national eating disorder treatment resources. In recruitment and consent materials, I described the study topic as "your ideas and opinions about eating

disorders and what causes them," as genetics was not the sole topic. The study was approved by the university IRB.

Most respondents were white, with at least some college education (see Table 2). This sample is generally similar to *clinical* populations of women with eating disorders, which tend to be white and educated (Striegel-Moore & Bulik, 2007: 182). However, those who meet criteria for eating disorders frequently do not seek treatment (Hudson et al., 2007; Hoek & Van Hoeken, 2003), and demographics of the general population of people with eating disorders are difficult to characterize because of low prevalence and under-reporting by respondents (Hoek & Van Hoeken, 2003). However, among young adults, white women are more likely to have experienced AN or BN than black women (Striegel-Moore et al., 2003). Respondents in the present study ranged from 18 to 64 years of age. Some had experienced little or no treatment; others multiple hospitalizations. Those currently in treatment had more extensive treatment histories than those who had recovered (p=.017, Fisher's exact test, not shown).

Data Collection and Analysis

The interview guide elicited the most salient responses to genetic involvement in AN/BN, whether these included impacts on stigma or not. I began by asking about experiences and understandings of eating disorders, to provide context and enable genetics to come up naturally. About halfway through the interview, I introduced the idea of genetic involvement. I began with general reactions to the idea ("Some say genes could play a role in eating disorders…"), and targeted progressively more specific topics (e.g., whether and how genes could plausibly influence AN/BN, whether and how genetic framing had good or bad implications). Some questions were asked in the context of two hypothetical scenarios. The first presented "a media campaign to publicize the idea that genetics play some role in [AN/BN]," and asked what good or bad effects this would have, whether for people with eating disorders or others. The second scenario described a hypothetical test for genetic susceptibility to AN or BN, to elicit reflection about whether they would prefer to find out (post-diagnosis) whether they had a genetic predisposition or not, and what consequences this might have. To guard against deterministic interpretations, I described it as follows: "Because both genes and environment play a role, it's not likely that a genetic test could predict whether a person would develop an eating disorder. There is no test like that. But, for a moment let's say you could get a genetic test to find out if your genes made you more likely to develop an eating disorder." At interview I clarified this was not a *predictive* genetic test, but one given after developing an eating disorder.

I conducted all interviews, which were 1½–2 hours long. Interviews were digitally recorded (with respondents' permission), professionally transcribed, and de-identified. All interviews took place in private locations, usually one of two offices on the medical campus (for recovered people, outpatients, and treatment study participants), or private clinic locations (for inpatients and day program patients). Participants were paid $40 cash.

Although I did not ask directly about stigma, questions about past experiences and preferred understandings of eating disorders (e.g., as a "mental illness," as a "choice") prompted discussion of stigma-relevant topics. Questions about genetics did as well, particularly probing about good or bad implications of genetic framing or predisposition. I did not use the word "stigma" unless respondents did first. (At the end of the interview, I also asked some respondents a leading question about volitional stigma but omit results here.)

I analyzed transcripts in several stages. The first was extensive note-taking while reviewing transcripts for accuracy and removing identifiers. After entering transcripts into a qualitative software program (N6), I began coding, that is, I identified and tagged material relevant to

research questions, first with "open" coding, then more "focused" coding, and ultimately taxonomy-creation (Lofland et al. 2005, Strauss & Corbin 1990). While coding I also created theoretical memos to record observations, hypotheses, and ideas for future coding.

Link and Phelan (2001) observe that stigma is defined in different ways by different investigators in working on different disorders and coming from diverse disciplines. I chose to use a broad definition of stigma centered on the negative evaluation of people with AN/BN (not their relatives), including negative *self*-evaluation. This general definition of individual stigma as devaluation is consistent with classic approaches (e.g., Goffman, 1963), includes both felt and enacted stigma (Jacoby 1994), and is flexible enough to accommodate what matters most to respondents in their moral experience and social worlds (Yang et al. 2007; Shostak et al. 2011). I coded as "stigma" any and all allusions to negative beliefs, images, stereotypes, social evaluations, and discriminatory treatment of people with eating disorders.

For the present analysis, coding focused on discussions of stigma solely in relation to genetics. When coding text indicative of stigma reduction or exacerbation via genes, I included only discussions clearly related to individual stigma. I therefore excluded the following themes unless respondents clearly linked them to stigma: feelings of "relief" if genetics played a role (because genetics might relieve cognitive uncertainty as well as moral responsibility); concerns about genetic fatalism (because fears about recurrence or persistence of behavior indicate severity but not necessarily stigma); guilt about passing "bad genes" to children (because sorrow and concern about prognosis and prevention need not be stigmatizing). Respondents who offered ways that genetics could both reduce *and* exacerbate stigma were coded in both categories; coding was at the level of the text rather than the respondent. To categorize subthemes, I grouped answers according to similarity and labeled them using concepts from stigma literature, when applicable.

RESULTS

The clear majority of respondents (45/50 or 90%) anticipated genetic causal attribution would reduce stigma of AN/BN (see Table 3). About a third (17/50 or 34%) anticipated it would exacerbate stigma; 12 mentioned both. After presenting subthemes that illustrate expectations about stigma reduction and exacerbation, I place in context the most frequent sub-theme--volitional stigma (mentioned by 44/50 or 88%)—by characterizing the value of volition to respondents, despite its stigmatizing potential. Theme frequency did not differ by treatment status (recovered vs. in treatment); treatment experience (ever in highly structured treatment program vs. never); primary diagnosis (AN vs. BN); age (30+ vs. younger); or education (BA+ vs. less) (Fisher's exact test, data not shown). Quotations are attributed using pseudonyms, recovery status (R=recovered, T=in treatment) and primary diagnosis (AN or BN).

How genetics may alleviate stigma

Almost all who expected a positive impact from genetics upon evaluation of people with eating disorders mentioned themes I coded as volitional stigma (44/45), that is, critical ascriptions of fault, blame, and responsibility for causing or not stopping AN/BN. This clear majority expected genetic involvement to increase compassion and reduce blame by making the eating disorder seem less like a choice. As respondents put it, rather than being "their fault," a "personal failing," and something to be ashamed of, genetics would provide evidence that there was "an objective reason," "something real," "something that does exist," to explain their behaviors. Jackie (T-BN) explained, "Having people realize that you really can't help it… would do a lot for the stigma." If genetics played a role, respondents expected less stigmatizing treatment from others. Liana (T-AN) expected her parents would

"be a little more accepting to think, '… this wasn't her choice. This was just part of her body.'" Respondents imagined ways of using genetic reframing to actively resist volitional stigma and self-stigma. For example, "it might be a little bit of relief. Because … if there was a genetic factor, I might not be as like… "What were you thinking? Why did you do that?" … just criticizing yourself for like leading yourself down that path" (Barbara R-AN). Genetics also strengthened resistance to stigma from others. For example, Isabelle (T-BN) could say to a critical friend who saw BN as a matter of willpower, "Hey. Here's this research…I've got, like, science to back me up here."

Respondents expected genetic reframing to assist in alleviation of several other forms of stigma as well. Many of these were negative moral ascriptions and logically dependent on the idea of voluntary choice. With genetic causal attribution, eating disorders would no longer be seen as a "lazy," "bad," or "stupid" method of losing weight. Genetics would challenge stereotypes of people with eating disorders as "shallow," "vain," "conceited," "selfish," or "indulgent." Those with AN/BN would not seem as "weird," "crazy," "bad," or "freaky" if genetics were involved, often because such qualities were ascribed only to those who chose such behaviors. For example, Irene (R-BN) expected that with a genetic explanation, people would be "[f]eeling less stigmatized. Feeling less of a failure. Like 'I've chosen to do this messed up thing. And, now I'm a freak.' I think it would be helpful to them."

How genetics may exacerbate stigma

A third of respondents (17/50) anticipated more negative evaluations of themselves as a result of genetic framing, with three broad sub-themes (see Table 3). First, several raised concerns I grouped together as "genetic essentialism," a broad category centered on reduction of the person or her condition with her genes. "[I]t would almost put like a stigma on my genes… I wouldn't want to have, like, a bad gene" (Yolanda T-BN). Zinnia (T-BN) anticipated others would think, "because it's a genetic problem that there's something wrong with them." Nell expected others might speculate as follows:

> [I]f you have this genetic pre-disposition for anorexia, what else is wrong with you?… [M]aybe the gene for anorexia is also close to the gene for mental illness or schizophrenia or something that people are really scared of… [W]hat if it's related to the gene that makes you abuse your kids or something like that? Or makes you do something else in some other horrible way… that makes people scared." (Nell R-AN)

Having a genetic problem could exacerbate stigma if it implied other, scarier genetic psychiatric problems.

Respondents were also concerned about the reduction of the eating disorder itself to genetics, which I included as a form of stigma from genetic essentialism if respondents anticipated negative evaluation as a result. For Irene (R-BN), if BN were "essentially" genetic, it would supplant her personal narrative centered on childhood abuse, which would make her feel worse about disclosing it to others.

> *[Interviewer: Is there anything appealing about having that genetic idea to tell people?]* Irene: Make me feel less of a stigma?… No. I think in fact if anything it would make me feel less good about telling them about my bulimia. Because, I think it's a very graphic, dramatic way to explain the awfulness of my childhood. "It resulted in this." But, if that is genetic, it takes away from the drama of explaining everything that happened to me. (Irene, R-BN)

Reframing the disorder as "genetic" would overshadow childhood abuse and dismantle a dramatic understanding of the disorder that enabled Irene to feel better about herself. A few

others expected that genetic involvement would lead to a medically reductionist, oversimplified understanding of eating disorders as easily curable with an injection or pill. They anticipated that this erroneous presumption would exacerbate blame because ongoing struggle against AN/BN would be interpreted as refusal to be cured.

> I think some would be outraged. "…– I work really hard to be – to get to where I am. Or to recover. And how dare you say that this is something that 'Shoot. All you got to do is give her a shot and she's fine.' " I think that would be really frustrating for some people. (Sydney, T-BN)

Genetic medical reductionism could downplay individual effort in treatment and recovery, leading others to overlook or minimize the challenges, hard work, and achievements of those with AN/BN. For these respondents, genetic causal attribution would leave out important aspects of the treatment and recovery process that were important for others to understand.

Wendy (T-BN) made the clearest and fullest statement connecting stigma to reductive genetic essentialism of eating disorders. She said she was "against" the idea of a genetic explanation because it seemed to confer more blame and make the disorder a permanent and essential part of her (boding ill for recovery).

> … it makes it sound like it's my fault that I have it…Well maybe not so much my fault. But just like that's who I am. And I don't like that. Because I don't see my eating disorder as who I am. I see it as something that's, like, invaded my life and that I want to get rid of.

Wendy attributed her eating disorder to problematic interactions with her parents. Therefore, locating the eating disorder in her body implicitly increased her own role, albeit non-volitional. Reacting to the idea of a media campaign featuring genetics, she went on to say,

> I feel like that would lead to people judging you… Like it being an inherent part of you. And just people labeling you as, "Oh. That person is a bulimic person." … it would just kind of in the back of people's heads teach them to make snap judgments about bulimia. And just assume that there's an easy cure. … Like it's like a shot. You get a shot. And you're cured. Or just something like that. But bulimia you don't cure it like that. It's thought patterns. And it takes a long time to cure. You have to re-train yourself how to think. And you also have to re-train your behavior how to eat. And it takes time.

Describing BN in terms of genetics was thus an oversimplification that led to labeling, judgment, and blame.

Second, a few raised concerns generally related to discrimination from school, work, or insurance on the basis of genetic test results. As Alyce put it (R-AN), "there may be one or two genes that make such a strong contribution it would be obvious if you get your child genetic testing. It could be part of everyone's school. But, I don't think we have the protections for that. People with genes for different conditions could really be in bad shape then." Ingrid (T-AN) even imagined future "genetic engineering" to get the "perfect baby" with "perfect mental health," implying genetic discrimination and devaluation of people with AN/BN. Genes would thus determine one's value to society or an organization, leading to bad treatment from others.

Third and last, several respondents anticipated stigma resulting from genetic causal attribution in a different way: that invoking a genetic explanation for behavior would lead others to think them irresponsible. Amy (T-AN) expected her family to react by saying, "Okay, you're still making excuses." Likewise, Mary (T-AN) implied that others would challenge claims about genetics, and say, "'Oh, yeah. Blame it on something else.' Rather

than the person taking responsibility for it." Such reactions suggest that claiming genetic influence would be interpreted simplistically, and therefore rejected not only as implausible but irresponsible.

Reasons to retain volition, despite stigma

Because 90% of respondents anticipated that genetic causal attribution would reduce stigma, compared to just a third who expected an increase, one might conclude genetic framing of AN/BN is good for people with AN/BN. Indeed, respondents did not want to be stigmatized, and cited benefits from alleviation of volitional stigma (self-forgiveness, treatment-seeking, social support, hope for a cure). But the picture is more complicated, because alleviation of volitional stigma via genetics—mentioned by 88% of respondents—entailed unwanted implications for future health and behavior if responsibility were entirely removed. Here I focus on the finding that, while discussing genetics, about half of respondents (26/50, 52%) articulated a desire to preserve some measure of volition—even volitional stigma--to help them recover from eating disorders. Far more respondents likely shared this view, given widespread concerns about genetic fatalism or hopelessness generally, and majority endorsement that AN/BN had elements of "choice." (Indeed, only four respondents strongly rejected the idea of AN/BN as "choice," and never raised concerns about either genetic loss of agency or genetic fatalism; all four were receiving treatment in the inpatient unit or day program.)

The 26 respondents raised concerns about the potential for a self-fulfilling prophecy; if genes played a role, people with eating disorders might believe themselves to be helpless and give up. For example, Eva (T-BN) said people "might not work as hard at recovery as they could." Recovery was understood to require hard work and effort, and there was a danger that genetics could not only discourage people with AN/BN. Furthermore, respondents said it could also provide an "excuse" and a way to rationalize the behaviors; "if this is a person that's going to choose to have anorexia anyway… they would just say, 'Okay. I've got the [genetic] link. So, why not just go for it all the way?'"(Amy T-AN). Fran, who developed and recovered from BN decades earlier without treatment, reflected:

> I think it [genetics] would have been an enabler for me. I wouldn't have stopped the behavior. Because, I would have thought I couldn't. I would have seen that as the reason I couldn't. That I didn't have control over it." (Fran, R-BN)

Delia (T-AN) theorized that her exposure to biogenetic explanations actually did prevent recovery:

> Well, that kind of made it easier for me to continue doing it. Because, then it took the blame off me. And, say "Oh. I can't control this as much. Because, it's biological. It's in my genes. So, therefore I can't control it."

Others spoke about genetics providing a "green light," an "excuse to do it," a way of "staying in denial," a "cop out," and "a crutch," which would appeal to but perhaps ultimately harm some individuals.

DISCUSSION

I found respondents far more likely to expect genetics to reduce stigma than exacerbate it, primarily because they expected genetic explanations to reduce stigma from perceived volition and responsibility for behavior. However, half anticipated that less perceived agency could be harmful for people with eating disorders. A third identified additional stigma from genetic framing, reflecting some unique characteristics of eating disorders. Taken together, these findings are more congruent with the "second form" of stigma (AN/BN assessed as voluntary behavior) than the "first form" (as mental illness). All respondents

had personally experienced an eating disorder, giving them firsthand knowledge of the types of stigma likely to be experienced, internalized, and/or resisted, and how these might be affected by genetic framing.

This study relied on respondents to spontaneously volunteer their stigma-relevant thoughts as they contemplated the idea of genetic involvement. To capture the most salient anticipated consequences of genetic re-framing, I did not explicitly ask respondents about stigma, much less restrict them to a set of response options. In addition to maximizing salience, my approach enabled respondents to use their own words, follow their associations, and explain their logic in response to probes. A disadvantage was less standardization across interviews, such that findings reflect diverse interpretations of genetic influence, and some may apply to imagined others rather than respondents themselves. Moreover, my asking about genetics likely prompted any initially skeptical respondents to consider the idea more seriously as the interview continued; answers were produced in a specific interview context. Last, my sample excluded women with current *un*treated AN/BN, and men, who may have different perceptions of stigma and genetics.

The general finding that genetics would more likely reduce than exacerbate stigma for AN/BN is congruent with prior research on students (Crisafulli 2008, 2010; Wingfield 2011), and with advocates' expectations (e.g., EDC, 2008), as well as qualitative studies of people with bipolar disorder (Meiser et al. 2005) and depression (Laegsgaard et al. 2010). Findings are also consistent with Schnittker's (2008) argument that the impact of genes on stigma depends on the disorder; although I examined only one disorder, its characteristic "volitional" stigma was central to the interpretation of genetics by respondents. For disorders perceived as irresponsible behavior rather than serious and dangerous mental illness, conveying genetic etiology might reframe the problem just enough to make it less volitional. The potential "costs" of claiming genetic etiology may thus be less for eating disorders than schizophrenia and other diagnoses, at least with regard to stigma. Future research could compare the impact of genetic etiology on stigma in different diagnoses, perhaps comparing diagnoses stigmatized as volitional (eating disorders), dangerous (schizophrenia), or both (conduct disorder, addiction to illegal substances).

Results uncover novel aspects of stigma from "genetic essentialism," reflecting the eating disorders context. Phelan (2005) operationalized genetic essentialism for mental illness stigma generally as differentness, seriousness, persistence, and the expectation that blood relatives would also have the problem. Differentness was theorized as a direct indicator of stigma; the other three as moderators affecting the degree of stigma (from social rejection or reproductive restriction). Only about a third of my respondents discussed exacerbation of stigma via genetic attribution, and few of these linked differentness, seriousness, or persistence, to stigma (e.g., Wendy). Genes connoted differentness and seriousness, but this seemed to mitigate rather than exacerbate stigma, by making AN/BN something outside normal everyday experience, and therefore not evaluable by customary standards as choice behavior. While genes did connote persistence and familial inheritance, these elicited concerns about prognosis rather than stigma. Only one indirectly raised the issue of reproductive restriction as stigmatization (i.e., Ingrid on genetic engineering). Nevertheless, Phelan's (2005: 319) definition of genetic essentialism—"the closely related ideas of genes as the basis for human identity and of genetic reductionism and determinism"—was general enough to usefully encompass several forms of stigma raised by these respondents. Genetic-essentialist interpretations exacerbated AN/BN stigma by disrupting self-understandings and stigma-resistance strategies; genes distracted from causal narratives centered on abuse or other environmental causes, and trivialized the personal effort involved in recovery. With regard to the latter, the presumption of a simple genetic cure implied a tradeoff: less

responsibility for causing AN/BN, but more responsibility for solving it quickly and medically (see Brickman et al., 1982).

Based on these findings, I recommend that genetic essentialism continue to be examined in relation to mental illness stigma, but (1) that it not be equated with stigma; (2) that it be considered to have multiple dimensions, perhaps the four suggested by Phelan's (2006) operationalization--differentness, seriousness, persistence, and expectations about blood relatives having the same problem--plus two suggested by these data--oversimplification of the illness experience and of the individual's role in recovery; and (3) that these dimensions be separately correlated with other measures of stigma to determine whether and how they are stigmatizing for different disorders.

Despite near-consensus that genes implied less personal responsibility, respondents anticipated skepticism and even backlash, if taken as a claim that genes were exclusively or mostly to blame. This reductive interpretation limited the plausibility and utility of genetic reframing, and some even anticipated a new form of stigma from such reframing itself: genetic "excuses" for AN/BN reflect irresponsibility. The anticipated harms largely flowed from reductionist and deterministic theories of genetics. Notably, respondents' own genetic theories were usually more complex, with ample roles for environment and personal volition interacting contingently over time (data not presented). Nevertheless, they presumed others to have simple theories, a presumption that has empirical support (Dar-Nimrod & Heine, 2011, but see Condit et al., 2006). Stigma and other potential harms from genetic framing are likely to depend on exactly how genetic involvement is presented and interpreted, including environmental and volitional inputs (see Peay & Austin, 2011: 87ff for a helpful analogy: a mental illness "jar" filled with diverse risk factors). Further research is needed to assess impact on stigma of non-simplistic presentations; findings thus far are inconclusive (e.g., Phelan, 2005; Crisafulli, 2010). It remains to be seen whether the perceived benefits of genetic reframing will persist within a more complex genetic model where environment matters and genes are not "for" a mental illness (Kendler, 2005).

At least half of respondents balanced reductions in volitional stigma against other interests, suggesting a wish to preserve some volition. While a clear majority did not want to be blamed or held solely responsible for their eating disorders, at least half were concerned that genetic attribution would weaken self-perceived agency. Treatment for eating disorders frequently depends on some presumption of agency, and not just for "mild" cases; hospitalized inpatients are rewarded with greater privileges if they eat according to program rules. Respondents interpreted their behavior as somewhere between volitional and non-volitional, and held themselves both responsible and not responsible for behavior. In this liminal space, genetics helped make the case that the eating disorder was "not all my fault" (skeptical backlash about irresponsible "genetic excuses" notwithstanding).

If holding people responsible for their eating disorder behavior is essential to the recovery process, some volitional stigma could theoretically promote positive behavioral change. It has been argued that stigma is a public health tool in the case of smoking cessation; if tobacco use were de-normalized and shamed, people would smoke less (Bayer, 2008). Proponents of the feminist/cultural model might agree to some extent, as stigmatizing widespread "normal" behaviors such as extreme dieting could help prevent eating disorders. Respondents appeared to hold a somewhat volitional model of eating disorders themselves, like other samples of people with eating disorders (Higbed & Fox, 2010; Rich, 2006). Yet the idea of AN/BN as volitional was also stigmatizing. For people with eating disorders, the perception of volition is complex, changes over time, and presents ethical challenges, and so should be approached with caution and sensitivity.

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References

Angermeyer MC, Holzinger A, Carta MG, Schomerus G. Biogenetic explanations and public acceptance of mental illness: systematic review of population studies. Br J Psychiatry. 2011; 199:367–372. [PubMed: 22045945]

APA (American Psychiatric Association). Diagnostic and statistical manual of mental disorders: DSM-IV-TR. Washington, DC: American Psychiatric Association; 2000.

Arcelus J, Mitchell AJ, Wales J, Nielsen S. Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. Arch Gen Psychiatry. 2011; 68(7):724–731. [PubMed: 21727255]

Barker, KK. The social construction of illness: Medicalization and contested illness. In: Bird, CE.; Conrad, P.; Fremont, AM.; Timmermans, S., editors. Handbook of medical sociology. Nashville: Vanderbilt University Press; 2010.

Barker KK. Listening to Lyrica: contested illnesses and pharmaceutical determinism. Soc Sci Med. 2011; 73:833–842. [PubMed: 21840638]

Bayer R. Stigma and the ethics of public health: not can we but should we. Soc Sci Med. 2008; 67(3): 463–472. [PubMed: 18502551]

Bennett L, Thirlaway K, Murray AJ. The stigmatising implications of presenting schizophrenia as a genetic disease. J Genet Couns. 2008; 17(6):550–559. [PubMed: 18773286]

Bernstein E. Families fight insurers over eating disorders. The Wall Street Journal. 2007 Jan 2.

Bordo, S. Unbearable weight: feminism, Western culture, and the body. Berkeley: University of California Press; 1993.

Brickman P, Rabinowitz VC, Karuza J, Coates D, Ellen C, Kidder L. Models of Helping and Coping. American Psychologist. 1982; 37(4):368–384.

Bulik, CM. Role of Genetics in Anorexia Nervosa, Bulimia Nervosa, and Binge Eating Disorder. In: Brewerton, TD., editor. Clinical handbook of eating disorders: an integrated approach. New York: Marcel Dekker; 2004. p. 165-182.

Condit, C.; Parrott, R.; Harris, T. Laypeople and Behavioral Genetics. In: Parens, E.; Chapman, AR.; Press, N., editors. Wrestling with behavioral genetics: science, ethics, and public conversation. Baltimore: Johns Hopkins University Press; 2006. p. 286-308.

Cook TM, Wang J. Causation beliefs and stigma against depression: Results from a population-based study. Journal of Affective Disorders. 2011; 133(1–2):86–92. [PubMed: 21489636]

Corrigan PW, Larson JE, Watson AC, Boyle M, Barr L. Solutions to discrimination in work and housing identified by people with mental illness. J Nerv Ment Dis. 2006; 194(9):716–718. [PubMed: 16971826]

Corrigan PW, Watson AC. At issue: Stop the stigma: call mental illness a brain disease. Schizophr Bull. 2004; 30(3):477–479. [PubMed: 15631240]

Crisafulli MA, Thompson-Brenner H, Franko DL, Eddy KT, Herzog DB. Stigmatizing of anorexia nervosa: Characteristics and response to intervention. Journal of Social and Clinical Psychology. 2010; 29(7):756–770.

Crisafulli MA, Von Holle A, Bulik CM. Attitudes towards anorexia nervosa: the impact of framing on blame and stigma. Int J Eat Disord. 2008; 41(4):333–339. [PubMed: 18186057]

Crisp AH. Stigmatization of and Discrimination Against People with Eating Disorders Including a Report of Two Nationwide Surveys. European Eating Disorders Review. 2005; 13:147–152.

Crisp AH, Gelder MG, Rix S, Meltzer HI, Rowlands OJ. Stigmatisation of people with mental illnesses. British Journal of Psychiatry. 2000; 177:4–7. [PubMed: 10945080]

Dar-Nimrod I, Heine SJ. Genetic essentialism: on the deceptive determinism of DNA. Psychol Bull. 2011; 137(5):800–818. [PubMed: 21142350]

Dietrich S, Matschinger H, Angermeyer MC. The relationship between biogenetic causal explanations and social distance toward people with mental disorders: results from a population survey in Germany. Int J Soc Psychiatry. 2006; 52(2):166–174. [PubMed: 16615248]

EDC (Eating Disorders Coalition for Research, Policy, and Action). Congressional Briefing. 2008. http://www.eatingdisorderscoalition.org/congbriefings/2008/CongBriefFeb2008.htm

Fausto-Sterling, A. Myths of gender: biological theories about women and men. New York, NY: BasicBooks; 1992.

Giles D. Constructing Identities in Cyberspace: The Case of Eating Disorders. British Journal of Social Psychology. 2006; 45(3):463–477. [PubMed: 16984715]

Goffman, E. Stigma: Notes on the management of spoiled identity. New York: Simon & Schuster, Inc; 1963.

Goldstein B, Rosselli F. Etiological paradigms of depression: The relationship between perceived causes, empowerment, treatment preferences, and stigma. Journal of Mental Health. 2003; 12(6): 551–563.

Herpertz-Dahlmann B, Seitz J, Konrad K. Aetiology of anorexia nervosa: from a “psychosomatic family model” to a neuropsychiatric disorder? Eur Arch Psychiatry Clin Neurosci. 2011; 261(Suppl 2):S177–181. [PubMed: 21866370]

Higbed L, Fox JR. Illness perceptions in anorexia nervosa: a qualitative investigation. Br J Clin Psychol. 2010; 49(Pt 3):307–325. [PubMed: 19580703]

Hoek HW, van Hoeken D. Review of the prevalence and incidence of eating disorders. Int J Eat Disord. 2003; 34:383–396. [PubMed: 14566926]

Holliday J, Wall E, Treasure J, Weinman J. Perceptions of illness in individuals with anorexia nervosa: a comparison with lay men and women. Int J Eat Disord. 2005; 37(1):50–56. [PubMed: 15690466]

Hudson JI, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychiatry. 2007; 61:348–358. [PubMed: 16815322]

Kendler KS. “A gene for…”: the nature of gene action in psychiatric disorders. Am J Psychiatry. 2005; 162(7):1243–1252. [PubMed: 15994704]

Jacoby A. Felt versus enacted stigma: a concept revisited. Evidence from a study of people with epilepsy in remission. Soc Sci Med. 1994; 38:269–274. [PubMed: 8140453]

Laegsgaard MM, Stamp AS, Hall EO, Mors O. The perceived and predicted implications of psychiatric genetic knowledge among persons with multiple cases of depression in the family. Acta Psychiatrica Scandinavica. 2010; 122(6):470–480. [PubMed: 20346073]

Levitt M, Manson N. My genes made me do it? The implications of behavioural genetics for responsibility and blame. Health Care Anal. 2007; 15(1):33–40. [PubMed: 17373130]

Link BG, Phelan JC. Conceptualizing stigma. Annual Review of Sociology. 2001; 27:363–385.

Link BG, Struening E, Dohrenwend BP, Cullen FT, Shrout PE. A Modified Labeling Theory Approach to Mental Disorders: An Empirical Assessment. American Sociological Review. 1989; 54(3):400–423.

Lippman A. Led (astray) by genetic maps: the cartography of the human genome and health care. Soc Sci Med. 1992; 35(12):1469–1476. [PubMed: 1485194]

Livingston JD, Boyd JE. Correlates and consequences of internalized stigma for people living with mental illness: a systematic review and meta-analysis. Soc Sci Med. 2010; 71(12):2150–2161. [PubMed: 21051128]

Lofland, J.; Snow, DA.; Anderson, L.; Lofland, LH. Analyzing Social Settings: A Guide to Qualitative Observation and Analysis. Wadsworth Publishing Company; 2005.

Malson, H.; Burns, M. Critical feminist approaches to eating dis/orders. New York, NY: Routledge; 2009.

Manolio TA, Collins FS, Cox NJ, Goldstein DB, Hindorff LA, Hunter DJ, et al. Finding the missing heritability of complex diseases. Nature. 2009; 461(7265):747–753. [PubMed: 19812666]

Mayo Clinic staff. Eating Disorders: Causes. 2012. Retrieved March 24, 2012, from http://www.mayoclinic.com/health/eating-disorders/DS00294/DSECTION=causes

Meiser B, Mitchell PB, McGirr H, Van Herten M, Schofield PR. Implications of genetic risk information in families with a high density of bipolar disorder: an exploratory study. Soc Sci Med. 2005; 60(1):109–118. [PubMed: 15482871]

Mond JM, Robertson-Smith G, Vetere A. Stigma and eating disorders: Is there evidence of negative attitudes towards anorexia nervosa among women in the community? Journal of Mental Health. 2006; 15(5):519–532.

Moses T. Adolescent Mental Health Consumers' Self-stigma: Associations with Parents' and Adolescents' Illness Perceptions and Parental Stigma. Journal of Community Psychology. 2010; 38(6):781–798.

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

O'Hara SK, Smith KC. Presentation of eating disorders in the news media: What are the implications for patient diagnosis and treatment? Patient Educ Couns. 2007; 68(1):43–51. [PubMed: 17521841]

Orbach, S. Hunger strike: the anorectic's struggle as a metaphor for our age. New York: Norton; 1986.

Parens E. Genetic differences and human identities. On why talking about behavioral genetics is important and difficult. Hastings Cent Rep. 2004; 34(1):S4–35. [PubMed: 15098406]

Peay, H.; Austin, J. How to talk with families about genetics and psychiatric illness. New York & London: W. W. Norton & Company; 2011.

Pescosolido BA, Martin JK, Long JS, Medina TR, Phelan JC, Link BG. "A disease like any other"? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. Am J Psychiatry. 2010; 167:1321–1330. [PubMed: 20843872]

Phelan JC. Geneticization of deviant behavior and consequences for stigma: the case of mental illness. J Health Soc Behav. 2005; 46(4):307–322. [PubMed: 16433278]

Phelan JC, Yang LH, Cruz-Rojas R. Effects of attributing serious mental illnesses to genetic causes on orientations to treatment. Psychiatr Serv. 2006; 57(3):382–387. [PubMed: 16524997]

Pinheiro AP, Bulik CM, Thornton LM, Sullivan PF, Root TL, Bloss CS, et al. Association study of 182 candidate genes in anorexia nervosa. Am J Med Genet B Neuropsychiatr Genet. 2010; 153B(5):1070–1080. [PubMed: 20468064]

Read J, Haslam N, Sayce L, Davies E. Prejudice and schizophrenia: a review of the 'mental illness is an illness like any other' approach. Acta Psychiatr Scand. 2006; 114(5):303–318. [PubMed: 17022790]

Rich E. Anorexic dis(connection): managing anorexia as an illness and an identity. Sociology of Health and Illness. 2006; 28(3):284–305. [PubMed: 16573717]

Roehrig JP, McLean CP. A comparison of stigma toward eating disorders versus depression. Int J Eat Disord. 2010; 43(7):671–674. [PubMed: 19816860]

Rusch N, Todd AR, Bodenhausen GV, Corrigan PW. Biogenetic models of psychopathology, implicit guilt, and mental illness stigma. Psychiatry Research. 2010; 179:328–332. [PubMed: 20493559]

Sadler MS, Meagor EL, Kaye KE. Stereotypes of mental disorders differ in competence and warmth. Soc Sci Med. 2012; 74:915–922. [PubMed: 22321391]

Sankar P, Cho MK, Wolpe PR, Schairer C. What is in a cause? Exploring the relationship between genetic cause and felt stigma. Genet Med. 2006; 8:33–42. [PubMed: 16418597]

Schnittker J. An uncertain revolution: why the rise of a genetic model of mental illness has not increased tolerance. Soc Sci Med. 2008; 67(9):1370–1381. [PubMed: 18703264]

Schulze B, Angermeyer MC. Subjective experiences of stigma. A focus group study of schizophrenic patients, their relatives and mental health professionals. Soc Sci Med. 2003; 56(2):299–312. [PubMed: 12473315]

Shostak S, Conrad P, Horwitz AV. Sequencing and Its Consequences: Path Dependence and the Relationships Between Genetics and Medicalization. American Journal of Sociology. 2008; 114(Supplement):S287–S316.

Shostak S, Zarhin D, Ottman R. What's at stake? Genetic information from the perspective of people with epilepsy and their family members. Soc Sci Med. 2011; 73:645–654. [PubMed: 21831495]

Stewart MC, Keel PK, Schiavo RS. Stigmatization of anorexia nervosa. Int J Eat Disord. 2006; 39(4): 320–325. [PubMed: 16523470]

Strauss, A.; Corbin, J. Basics of qualitative research: Grounded theory procedures and techniques. Newbury Park, CA: Sage Publications, Inc; 1990.

Striegel-Moore RH, Bulik CM. Risk factors for eating disorders. Am Psychol. 2007; 62(3):181–198. [PubMed: 17469897]

Striegel-Moore RH, Dohm FA, Kraemer HC, Taylor CB, Daniels S, Crawford PB, et al. Eating disorders in white and black women. Am J Psychiatry. 2003; 160:1326–1331. [PubMed: 12832249]

Sullivan PF. Mortality in anorexia nervosa. American Journal of Psychiatry. 1995; 152(7):1073–1074. [PubMed: 7793446]

Thoits PA. Resisting the Stigma of Mental Illness. Social Psychology Quarterly. 2011; 74(6):6–28.

Timmermans S, Haas S. Towards a sociology of disease. Sociol Health Illn. 2008; 30:659–676. [PubMed: 18564975]

Tyre P. Fighting Anorexia: No One to Blame. Newsweek. 2005; 146(23):50–59.

Walter FM, Emery J, Braithwaite D, Marteau TM. Lay understanding of familial risk of common chronic diseases: a systematic review and synthesis of qualitative research. Ann Fam Med. 2004; 2:583–594. [PubMed: 15576545]

Way, K. Never too rich… Or too thin: The role of stigma in the social construction of anorexia nervosa. In: Maurer, D.; Sobal, J., editors. Eating agendas: food and nutrition as social problems. New York: Aldine de Gruyter; 1995. p. 91-113.

Weiner, B. An attributional theory of motivation and emotion. New York: Springer-Verlag; 1986.

Wingfield N, Kelly N, Serdar K, Shivy VA, Mazzeo SE. College students' perceptions of individuals with anorexia and bulimia nervosa. Int J Eat Disord. 2011; 44(4):369–375. [PubMed: 21472755]

Yang LH, Kleinman A, Link BG, Phelan JC, Lee S, Good B. Culture and stigma: adding moral experience to stigma theory. Soc Sci Med. 2007; 64(7):1524–1535. [PubMed: 17188411]

Zavestoski S, Brown P, McCormick S, Mayer B, D'Ottavi M, Lucove JC. Patient activism and the struggle for diagnosis: Gulf War illnesses and other medically unexplained physical symptoms in the US. Soc Sci Med. 2004; 58:161–175. [PubMed: 14572929]

- Presents semi-structured interviews with 50 women who have a history of eating disorders (recovered and in treatment).
- Most expected genetic causal attribution to reduce stigma, mainly by alleviating personal responsibility for the disorder (volitional stigma).
- A third suggested ways that genetic causal attribution could increase stigma, usually by oversimplifying eating disorders.
- At least half of respondents wanted to retain some degree of personal responsibility, despite its stigmatizing potential.
- This study is the first to examine how people with eating disorders interpret genetic causal attribution.

Table 1

Respondents' Diagnosis, Recovery and Treatment Status (N=50)

	Primarily AN[a]	Primarily BN[a]	Total
Recovered	14 (56%)	11 (44%)	25 (100%)
In treatment	13 (52%)	12 (48%)	25 (100%)
Inpatient	*9*	*0*	*9*
Day program	*2*	*2*	*4*
Outpatient	*2*	*0*	*2*
Treatment study	*0*	*10*	*10*
Total	27 (54%)	23 (46%)	50 (100%)

[a]AN= Anorexia nervosa, BN=bulimia nervosa

Table 2

Demographic Characteristics of Sample. Frequency (%) or Mean (S.D.) (range) (N=50)

Treatment experience	
Little or no treatment	10 (20%)
Outpatient treatment	15 (30%)
One structured program[a]	11 (22%)
Two or more structured prog.	14 (28%)
Age: mean (S.D.) (range)	32.7 (12.8) (18–64)
Race/ancestry/ethnicity	
White/European descent	42 (84%)
Black/African descent	4 (8%)
Asian descent	2 (4%)
Hispanic/Latino	2 (4%)
Education	
H.S. diploma or less	2 (4%)
Some college/Associate's	19 (38%)
Bachelor's degree	8 (16%)
Some grad./Master's	18 (36%)
PhD or MD	3 (6%)

[a]"Structured program" refers to psychiatric hospitalization, residential program, or structured day program.

Table 3

Anticipated impact of genetic explanations on stigma. Frequency (%) (N=50)[a]

Alleviation of stigma	45 (90%)
Volitional stigma: Eating disorders are a person's choice or fault, can be controlled with willpower, people do this to themselves, they are guilty and responsible	
Miscellaneous other negative stereotypes: Vain, indulgent, weird, crazy, stupid, silly, insecure, morally weak, trying to be cool	
Exacerbation of stigma	17 (34%)
Genetic essentialism: Genes are the essence of the person or disorder: bad genes define the person; genes define and oversimplify the disorder and its treatment	
Genetic discrimination: DNA test results as basis for discrimination	
Irresponsible to use genetic "excuses": Genetic framing is a cop-out	

[a]Does not add to 100% because 12 respondents articulated both.