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The Blinding Power of Genetics

Manufacturing and Privatizing Stories of Eating Disorders

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In narrative therapy, the therapist engages with the client's own understanding of his or her problems and helps the individual to open and develop new possibilities through re-storying. This undertaking is guided by the ethic of being respectful of the client as a subject and is therefore opposed to therapeutic practices that objectify clients or reinforce stories that individualize their problems and pathologize them. Currently in the area of eating disorders, there is a biological narrative emerging that has the potential of eclipsing understandings developed over the last two or three decades. This new narrative features genetic research made possible by the Human Genome Project,¹ and there are good reasons to be concerned about how the findings from this research are being placed in the overall conversation about eating disorders. What is at stake is which narrative about eating disorders will dominate among mainstream clinicians and researchers. This has implications for how those with eating disorders are characterized as well as where responsibility for preventing eating disorders and treating them lies: Are they individual pathologies or understandable responses to a disturbing culture, and is preventing and treating them a private or social responsibility? In this chapter, the emerging genetic story about eating disorders and its implications will be tracked and questioned.

Genetic findings are the trump card of biological explanations because they are seen as establishing causal relationships between genes and their manifestations in characteristics or conditions. Such causal connections are integral parts of scientific explanations, and explanations that have been legitimated as scientific tend to disqualify competing or parallel accounts. We might expect, then, that as genetic explanations proliferate from the Human Genome Project, they will dominate the space previously occupied by sociocultural and other kinds of accounts.

For example, in a very recently published *Handbook of Eating Disorders and Obesity* (Thompson, 2004), the first section is devoted to “Etiology, Risk, and Prevention,” and the first chapter is “Genetic and Biological Risk Factors.” “Sociocultural Aspects of Eating Disorders” make their appearance in the last section of the book, a section that is marginalized both by its location in the book and by its title, “Special Topics.” The lead paragraph of the book privileges biology and minimizes sociocultural understandings of eating disorders:

Historically, sociocultural and family theories of etiology have dominated the scientific literature on eating disorders. There was certain sound logic to the belief that the pervasive emphasis on thinness as a symbol of beauty and control somehow “caused” eating disorders or that certain family interaction patterns were more likely than others to bring food and eating-related issues to the fore as a center of familial conflict. These explanations had considerable face validity—they seemed like common sense. However, they were not rigorously tested as true prospective risk factors. For decades, biological researchers have been working in the background of the scientific community of eating disorders. A small but dedicated group of researchers has continued to forge ahead with the notion that biology plays a substantial causal role in the etiology of anorexia nervosa (AN) and bulimia nervosa (BN). In this chapter, I address how research on genetic epidemiology and genetics of eating disorders is forcing us to refocus our understanding of the balance of the contributions of genetic and environmental factors to the etiology of anorexia and bulimia nervosa. (Bulik, 2004, p. 3)

While the passing away of sociocultural explanations is proclaimed and the nobility of biological research as “real” science is reinforced in the preceding paragraph, no specific, replicated genetic results have yet been found. This is particularly objectionable and underlines the dominating effects of genetic discourse in explanations of human behavior, because there are many significant findings in the area of sociocultural research that are simply submerged by statements such as those in the passage above.

Researchers like Anne Becker have shown that sociocultural characteristics can create increased vulnerability to eating disorders. A study conducted by Becker and colleagues in the Nadroga province of Fiji between 1995 and 1998 shows how its young women have become very vulnerable to Western

women's preoccupations with weight and shape, due to Nadroga's transition from a traditional agrarian economy to an industrial economy that has imported Western cultural values (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002). In Fiji, traditionally, a rounded body shape had been expected and valued in women, and up until 1995, dieting was rare. In 1995, television was introduced to Fiji for the first time, with a choice of two channels showing American, Australian, and British dramas, comedies, and advertising. By 1998, after 3 years of television, eating practices and weight-related attitudes had substantially changed among young women.

Sixty-nine percent of the 63 young women interviewed reported dieting to lose weight, and 74% reported feeling too big or too fat. Three times as many girls as previously had scored in the high-risk range for the development of eating disorders. While there had been no reports of self-induced vomiting in 1995, 12% of the young women reported this behavior in 1998. In their interviews, the young women said, for example, they did not want to be "fat" like their mothers; they felt fatter when watching television shows; and they wanted to have the lifestyles that went along with the body shapes they saw on the television shows they watched.

There is a plethora of other sociocultural research demonstrating, for example, increased body image dissatisfaction for women after viewing fashion magazines (Turner, Hamilton, Jacobs, Angood, & Dwyer, 1997); increased anger and negative mood after viewing fashion magazines (Pinhas, Toner, Ali, Garfinkel, & Stuckless, 1999); and increased eating-disorder symptomatology with increased viewing of fashion magazines—and the reverse, decreased eating-disorder symptomatology with decreased viewing of fashion magazines (Vaughan & Fouts, 2003); and there is evidence for a cumulative effect (over 2 years) of appearance-related TV commercials on body dissatisfaction and drive for thinness (Hargreaves & Tiggemann, 2003). Despite this research, the genetic model is successively moving the mainstream professional community in eating disorders from a biopsychosocial model that at least acknowledged a significant role for sociocultural factors (albeit with a rather superficial understanding of them) to a model that privileges the biological over the sociocultural and redescribes the psychological in terms of the biological.²

This process can be seen to be occurring in the area of eating disorders, where two versions of a *gene story* about eating disorders are present: a strong version and a weaker one. In its strong version, the genetic model is claimed to supercede the sociocultural, as in this quote from Cynthia Bulik, president of the Academy for Eating Disorders and the only endowed professor of eating disorders in the United States: "Socio-cultural factors are only important in that they might elicit an expression of someone's pre-existing genetic predisposition" (DeAngelis, 2002, p. 2); or "Historically, eating

disorders have been considered to be primarily of socio-cultural origin. Over the past decade, this perspective has been resoundingly rebuffed by a systematic series of enlightening family, twin, and molecular genetic studies” (Bulik, Jordan, & Jordan, 2004, p. 1). In its weaker version, the genetic model claims only that “genes may play a role in the underlying vulnerability to developing an eating disorder” (Kaplan & Woodside, 2003, p. 1). Researchers in the area can be seen to vacillate between the two stories, using the weak version in more formal contexts and the strong version in less formal ones, but backing themselves up with the same research in either case.

This vacillation may reflect a change that has occurred in how genes are conceived scientifically, a change that has not yet been fully integrated into popular consciousness. As the possibility of mapping the human genome became more real, the assumption among geneticists was that human beings would be found to have more genes than any other species (the estimates ranged from around 90,000 to 150,000) because of the greater complexity of the human organism. This would be consistent with the view of the gene as a powerful determiner of traits and with the strong version of the gene story of eating disorders, the version with the most punch, which purports to say something really significant.

As it turns out, we have about 25,000 genes: fewer than the mouse and about the same number as the roundworm. Clearly, the complexity of our species cannot be explained by the sheer number of genes we have, nor can it be explained by some qualitative difference in our genetic material compared with those of other organisms, since all DNA is made up of the same chemical and physical components.³ Genes, in fact, do not have the determinative power they were once credited with, and our characteristics are created through many-layered processes. As the psychologist David Moore states,

Biologists no longer question the following two facts: (1) a bit of chromosome can do no more than provide information about the order of amino acids in a chain; and (2) traits are constructed in cascades of steps—many involving nongenetic factors—that lie between amino acid sequencing and final trait production. Given these facts, one conclusion is inescapable: genetic factors cannot themselves cause traits, even traits widely thought to be “genetic,” such as hair color, eye color, or body type. . . . Just as no single domino in a series can be called *the* cause—or even the most important cause—of an outcome, neither can a genetic factor alone be *the* cause—or even the most important cause—of the development of a trait. Instead, genetic and nongenetic factors determine traits’ appearances *collaboratively*. (Moore, 2001, p. 76)

This view of the role of genes is much more consistent with the weak version of the gene story at best, and possibly not with any currently existing version of a gene story about eating disorders.

Let's consider the following summary of the gene story about eating disorders (paraphrasing the point of view of various researchers) to look at its claims more closely:

We researchers noticed that there are more people with eating disorders in some extended families than in others. This could be because of the influence of family members on one another (learning), or it could be because of genes. Our comparison of identical twins (who share 100% of their genes) with non-identical twins (who don't share more of their genes than any two siblings who are not twins) shows that more identical twins have eating disorders than do nonidentical twins. The relationship is strongest for anorexia nervosa but also strong enough to warrant believing there is a significant genetic contribution for bulimia nervosa. We estimate heritability to be in the range of 80% for both conditions (Bulik, 2004; Bulik, Sullivan, Wade, & Kendler, 2000).

We also looked for other psychiatric disorders in individuals with eating disorders and in their families. We looked for things like depression, substance abuse, obsessive-compulsive disorders and some personality traits like perfectionism and impulsiveness, and we found that there were more of these than is average. These shared characteristics made us think that probably some people have genes that make them more vulnerable to developing eating disorders than are people who don't have these genes. That would explain why only some people develop eating disorders when all of us share the same socio-cultural environment. People with these genes might react to shared and non-shared environments in ways that people without them would not (Klump, Wonderlich, Lehoux, Lilienfeld, & Bulik, 2002; Lilienfeld et al., 1998).

So, out of 237 people and their family members who had eating disorders, we picked those who had specific characteristics—that is, we identified a phenotype. For anorexia nervosa, we picked the people who had been ill for at least 3 years and had always restricted their food intake, but had never binged or purged. For bulimia nervosa, to identify a phenotype, we picked the people who purged by vomiting only (Kaplan & Woodside, 2003; Price Foundation Collaborative Group, 2001).

We scanned samples of the DNA of each of the people in these groups to find out whether there were locations of linkage on particular chromosomes for each of the groups. We did find linkage on chromosome 1 for anorexia and on chromosome 10 for bulimia. It will take time to find the specific genes, because there are 300 genes just within the area of linkage on chromosome 1, but we already have some ideas about what kind of genes to look for. We asked geneticists and computer scientists to look for any genes having to do

with appetite, eating, weight, and reward systems. That means looking at genes that control serotonin, dopamine, and opioids. We found some statistically significant connections, but we have not been able to replicate our findings. In the future, we will also be looking for genes that are linked to personality traits like perfectionism, impulsivity, and obsessiveness (Bergen & Kaplan, 2004, Grice et al., 2002; Kaye et al., 2004).

There are many reasons—scientific, logical, and political—we should be skeptical about this narrative.

Scientific Issues: Heritability, Linkage, and Culture Mirrored in Nature

Heritability

Heritability is not the same as *inheritability*, but most readers unfamiliar with the limited field of behavior genetics will not be aware of this (Moore, 2001, p. 45), and researchers in the area of genes and eating disorders do not explain it. When researchers make heritability estimates, they are using a concept that originated with plant and animal breeding, which has a very specific meaning in that context (Joseph, 2004, p. 142). In plant and animal breeding, environment can be controlled so significantly that variation in the characteristics of organisms raised in a given environment can reasonably be understood as being genetic in origin. Since it is unethical to control human's environments to this extent, we can never have the same degree of certainty about the variation of human characteristics. Furthermore, heritability estimates are reliable only in relation to the population from which they originated: They are not applicable to organisms from populations that did not share the original environment, and they do not describe the importance of genetic factors as they relate to a particular individual (Joseph, 2004, p. 139; Moore, 2001, p. 44). Using the concept of heritability in contexts outside of plant and animal breeding, without contending with the implications of its use out of context and without explaining its specific meaning, will result in readers misunderstanding it.

Above, for instance, in the summary of the genetic narrative from current research, it would be reasonable for the general reader to assume that genetic factors contribute 80% of what causes eating disorders. Bulik states that it should actually be read as saying that approximately 50% to 85% of the variance in liability for eating disorders is due to genetic effects (Bulik, 2004, p. 6). What she doesn't say is that although this means that we may have some reason to believe that among the people who were studied, the variation in eating

disorders is due to genetic effects, it does not mean that in any individual case, we can say that the person has an eating disorder because of some genetic factor or factors. Heritability is not the same as inheritability.

Linkage

A similar problem occurs with the idea of linkage. When genetics researchers report that they found linkage on chromosome 1, it sounds as though they found a definite connection between the genes in that area and anorexia nervosa. What the term *linkage* means within genetic discourse is that it is likely that when there is a certain characteristic (anorexia of the restricting type), there will also be another characteristic (a certain gene or genes). The linkage proves nothing about the nature of the relationship between these two things; it indicates only that in the population tested, they occurred together more often than chance would predict. To use the word *linkage* without explaining its technical meaning is to play on the general reader's understanding of linkage as indicating more than a statistical correlation. We would, for instance, say that smoking is linked with lung cancer, meaning that there is a causal relation between the two. Since most laypersons would still subscribe to the view that genes cause individual characteristics, it is likely that they would see "linkage" between a gene and a characteristic as a causal mechanism.

Culture Mirrored in Nature

Notice also in the summary story that there is a close connection between the genes that are being looked for and our psychological and psychiatric classification systems (e.g., personality disorders, mood and anxiety disorders, personality traits). Since this classification system is not even culturally universal, it seems a kind of hubris to assume that it will be mirrored on a molecular scale in nature. Yet researchers are proceeding as though these traits are good candidates with which to search for genes. All of these aspects of the gene story are legitimated by conclusions that are presented as authoritative, objective, and unequivocal and, being scientific, are treated as inherently real and independent of human construction or meaning making.

Logical Issues: The Notion of Susceptibility Genes

In its weak version, the gene story states that any gene found to be related to anorexia or bulimia will at best be of small-to-modest effect and will most likely be one of several contributory genes, which together will increase

a person's *susceptibility* to developing an eating disorder, *given certain environmental conditions* (Bulik, 2004; Grice et al., 2002; Kaplan & Woodside, 2003). This is, of course, a much weaker claim than that of finding a gene for anorexia. But what sort of claim is it?

Janet Treasure (2003, p. 1) gives phenylketonuria (PKU) as an explanatory example of a hereditary condition in which the risks associated with having the condition become manifest in a certain environment. Those born with PKU have mutations on both alleles of a particular gene. Practically, this means that they lack a liver enzyme that is required to digest an amino acid commonly found in protein-containing foods, including breast milk. When babies who are born without this enzyme ingest such foods, they sustain brain damage. Brain damage is avoidable if the baby is diagnosed with PKU immediately after birth and the feeding "environment" is manipulated to eliminate certain protein-containing foods, that is, to eliminate the risk represented by having PKU. A person with PKU could be identified as such on the basis of the genetic mutation that constitutes PKU: Environment plays a role only in whether the risks of having PKU are made manifest or not.

Treasure (2003) also gives as an example "risk of diabetes in people of Asian and Native American ancestry, which becomes manifest in an environment when food is abundant" (p. 1). This is a more complicated example than PKU, because although about 10 loci in the human genome have now been found that seem to confer susceptibility to type 1 diabetes, we do not yet know all of the factors that contribute. We know something about how to keep people with diabetes healthy, but we don't know exactly what causes it and are postulating a combination of many genetic factors and some nongenetic factors.

Anorexia and bulimia are unlike either of these examples. We know they are unlike PKU in that there is no mutation such that if you have the mutation, you also have anorexia or bulimia and should therefore concentrate on avoiding the risks associated with them. In type 1 diabetes, the body's immune system mounts an immunological assault on its own insulin and the pancreatic cells that manufacture it. While the "mechanism" of how this happens is not yet understood, at least it makes some sense to think of there being a mechanism involved here, one that could be the result of a critical mass of genetic and nongenetic physical factors. On the other hand, to "qualify" as having either anorexia or bulimia, a person must have certain *attitudes*, for instance, attitudes reflecting body image disparagement and an extreme valuing of thinness. So, with anorexia and bulimia, we are bringing in the idea that certain attitudes must be present for the terms *anorexia* or *bulimia* to be correctly applied to any individual. Attitudes are not part of the diagnosis of PKU or diabetes. Attitudes are not in the same logical

category as the factors in the gene story that are supposed to explain them. What sense does it make to think of these attitudes as somehow caused by a biological mechanism in concert with a particular physical environment? In other words, if we did happen to find a group of genetic factors that contribute to a person not eating, or vomiting after eating, would we have found genetic factors contributing to anorexia and bulimia—or to a syndrome that could be described and explained without requiring reference to attitudes? (For someone to have diabetes, for example, does not require that he or she have any particular attitudes or beliefs.) And what difference does it make when the environment that supposedly “triggers” the manifestation of the “illness” is a familial or cultural environment, not a physical one? The more we look at the ways that PKU and diabetes are different from anorexia and bulimia, the more it seems that the former are extremely limited sources of analogy for the latter.

Susceptibility to “Disease” or Normal Variation in a Problematic Context?

Suppose we think of a related genetic vulnerability that all of us have: For example, we will become unwell or die without appropriate types and quantities of nourishment. At any given time, there may be a lack of appropriate or sufficient food because of the natural, cultural, or political environment in which a person lives. A naturally occurring drought could result in there being little or no food available. A culturally significant social system, for example, a caste system, could limit access to quantity and variety of food for a segment of a population. A political situation, for example, a war, could slow down production and trade, with the result that there would be much less food for almost everyone. With human beings, of course, it is possible, and in fact likely, that all three types of environments could be operating at the same time. Drought conditions that make resources scarce increase the chances of war, and during war, those lower on the social hierarchy have more limited access to the few resources that are available. Some of the less privileged people have genetic dispositions that allow them to withstand conditions of deprivation for a longer period of time than others. Shall we privilege the role of genes when we tell the stories of their lives and deaths by saying that those who fell soonest were those whose genes left them most vulnerable?

The situation could perhaps be better likened to finding some genes that are not “for” any “disease,” but are simply part of the ordinary variation that occurs among members of any species. That certain environments might turn these genes into vulnerability factors for a disease says more about the

limits we should consider setting on the environments we create for ourselves to live in than it says about some genetically based disease entity. Perhaps we will soon hear researchers talking about the genes that are responsible for our requiring oxygen as though they are susceptibility genes for illness in an environment of poor air quality (Jasper, 2003).

While offering a newborn baby breast milk is usually in the baby's best interest and might arguably be described as a natural choice, it happens not to be a good choice for a PKU baby. This is a situation in which it makes sense to speak of susceptibility genes. The "environment" we provide for girls to grow into women—a cultural and political environment that inequitably values and devalues persons and *specifically generates intolerance toward women's bodies*—is not good for most girls and women. It doesn't make sense to speak of susceptibility genes here. Women's bodies tend to get fatter with puberty (Smolak & Levine, 1996, pp. 216–217), while men's bodies become leaner and more muscular with puberty (Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999, pp. 30–31). Fat is an object of disgust and is associated with many characteristics our Western culture disapproves of (MacInnis, 1993, pp. 70–71). It is in this environment that the vast majority of North American women have come to dislike their bodies (Maine, 2000, pp. 1–3) and in which a far greater number of females than males develop eating disorders⁴ (Thompson et al., 1999, p. 30), a fact that is almost never mentioned in the scientific genes and eating-disorders literature.⁵ Surely this is a paradigm example of a situation in which it makes most sense to speak of normal genetic variation in a problematic context, not of genetically based susceptibility.

Political Issues

Within dominant scientific discourse, cultural pressures are understood to be the same for all women, so they aren't seen as explanatory for why all women exposed to these pressures do not develop anorexia or bulimia. This picture discounts the many ways in which culture and its variants differentially affect women (Bordo, 1993, pp. 61–62), as well as the ways different families magnify, reinforce, or offer resistance to cultural pressures and the differences in how individual women relate to cultural ideas in the process of making meaning of their own lives. In its tendency to look for simple, linear, causal explanations, the reductionist scientific view also renders invisible the continuum of eating troubles and weight and shape preoccupation commonly experienced among women in Western and Westernized cultures (Brown, 1993, pp. 53–68). Nor does the gene story account for the predominance of eating problems among women, for the fact that among

males, gay males are at higher risk for developing eating disorders, or for the fact that eating disorders are historically situated such that genetic changes in the population could not account for them.

Perhaps what makes the scientific gene story so attractive and worth manufacturing is that it avoids confrontation with the values of Western culture and all the economic and political interests involved, while appearing to be objectively founded on scientific “facts.” There are, after all, serious financial interests in the many businesses that cater to weight loss and other “improvements” to appearance. The recently renewed attacks on fatness in response to the so-called epidemic of obesity also feed these business interests. As long as anorexia and bulimia are marginalized as individual pathologies, the connection between our culture’s serious phobia with fat and the generation of widespread, troubled eating patterns among females need not put a damper on the anti-obesity business. The problem of eating disorders becomes an individual one, with a private-enterprise solution. At most, the cultural pressures on women are seen as triggering something that already existed within an individual prior to her contact with the culture. It is this “thing” within the individual that is targeted for correction, effectively neutralizing any collective sense of responsibility for prevention of eating problems.

Since the late 1970s, there has been a rise of right-wing governments and economic policies in Britain, the United States, and Canada. Such policies favor individualizing social problems and cheaper methods of dealing with them. The massively widespread use of pharmaceuticals, like “antidepressants,” for a wide range of “illnesses” and the preferential funding of studies that look for biological causes are consistent with socially and economically conservative government. In this context, with regard to eating disorders, there is no apparent need to critically assess a pathogenic culture and less need to fund unwieldy and expensive prevention programs. The dream, no doubt, is to find pharmaceutical interventions.

There are ethical and practical concerns related to the few available ideas about how the genetic research will actually have an impact on prevention and treatment programs. Identifying the relevant genes will supposedly make possible early identification of those vulnerable to eating disorders. One researcher in the area of genes and eating disorders, Janet Treasure (2003), explains to the general public,

[It may be] possible for people to adjust their environment so that unhelpful interactions between their genetic propensities and their ecological context are minimized, e.g., someone with high trait anxiety may choose to ensure that they have high levels of safety, a close support network, and low risk goals; others who are somewhat rigid will flourish best in an environment where the rules are predictable and unchanging. (p. 2)

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So, one of the effects of the genetic endeavor may be the project of distinguishing people who are susceptible to developing eating disorders from those who aren't, such that the former can be targeted for preventive treatment, whether that be pharmaceutical⁶ or behavioral. An effect of this would be that girls (or boys) who are identified as carrying the susceptibility gene(s) could be seen as categorically different from those who are not. Girls (or boys) in this category could be treated differently from others in ways that could become discriminatory and disadvantageous.

Imagine explaining to a young woman in the "susceptible" or "vulnerable" category that unlike the other girls who are encouraged to try to "be the best they can be" and are told they can "be anything they want," she should choose low-risk goals and stay in environments that are predictable, "safe," and highly structured by rules. With Treasure's modest proposal in mind, it is interesting to return for a moment to the Fiji study. It seems that the young women of Fiji, whose eating-disordered attitudes have increased with exposure to Western culture, are simultaneously interested in a less highly structured "lifestyle" in which the young people are more "free" than in their traditional culture. Here are some things they and their parents have observed in relation to these issues:

The rules that have been made by the village, they are not following it, [because] they are copying Western culture.

They look good on the television, how they act and also how their body looks like when they . . . do some jobs, they are free to move around and do their jobs.

I try to look at them change the way, my way, of dressing and also the ways of looking fit and look to lose weight.

I like *Shortland Street* [an Australian drama] because of the many young adults involved with it. . . . I want to be like that, I want to imitate them—the way they live, the type of food they eat . . . it gives me ideas of how to solve problems when being in this world. (Becker et al., 2002, p. 513)

Treasure doesn't give us any suggestions for how we might persuade young women in Fiji or in North America to be happy with "lifestyles," identity projects, and body shapes that are unlike the ones that they are culturally induced to pursue. Clearly, the cultural forces that seem so neatly sidestepped by focusing on genes cannot be sidestepped after all.

It is not out of the realm of possibility to think that insurance companies might refuse to pay for treatment for, say, a young woman of 15 who was identified at the age of 6 as susceptible to developing an eating disorder

because of her genetic makeup, but refused to take any of the preventive measures approved by and paid for by the insurance company. What supports and therapies will then be available to her?

It is obvious that much of the excitement about discovering genes for susceptibility to eating disorders is motivated by economics, just as it is in numerous other areas of medicine and psychiatry. The search for genes is being funded at unprecedented levels,⁷ and scientists are increasingly affiliated with for-profit biotechnology firms rather than research institutes belonging to universities. The paradigm of genes as units that can cause characteristics and illnesses seems to thrive despite all the evidence there is to support the view that emphasizes the partnership of environment, both at micro- and macrolevels, in all developmental processes. As long as the role of genes is overstated, the tendency will be to act in terms of the genetics, because a “genetic approach” appears more likely to be effective, less expensive, apolitical, and objectively based.

Implications for Therapeutic Practice

A risk that comes with bringing genetic factors into therapeutic conversations about anorexia and bulimia parallels the risk of bringing them into theoretical discussions. Clients and therapists may allow the idea of genetic factors to replace or dominate other understandings about eating problems. Imagine scenarios that are something like the following.

The DeSousa-Jameson Family

Elena⁸ is a 13-year-old girl who is an inpatient in a children’s eating-disorders unit for the second time. Her parents Jack and Catalina are very frightened about her low weight and the fact that it does not seem to be getting any easier for her to eat. They have heard that of all psychiatric disorders, eating disorders have the highest mortality rate and are very difficult to treat. Elena recently read an Internet article in which experts reported that anorexia almost certainly has a genetic cause. She shows the article to her parents and says they should listen to her when she says the hospital treatment isn’t helping and is a waste of time. She sounds urgent when she tells them to take her home and promises that if they do, she will eat more than she did before, but she also says that if they make her stay in the hospital, she will make sure to lose any weight that she gains as soon as she does get home. Catalina, looking panicky, tells Elena that she may be able to come home very soon, and Jack mentions

that a sister of his had an eating disorder. Catalina and Jack are wondering if the reason that Elena is not getting better may be that her illness is genetically based. They ask if there might be a genetic cure on the horizon.

Elena is terrified of eating and of gaining weight. Catalina and Jack are frightened by Elena's behavior and by how scared she is to eat, but they are also angry that she is making threats to lose weight if they don't do as she asks. They want to believe Elena's promise that she will eat more if they take her home, and they are seduced by the idea that taking her home could make all the uncharacteristic conflict among them disappear. This powerful mix of emotions and ideas creates a cloud of confusion in Catalina and Jack.

The genetic story is a welcome distraction from the emotion, conflict, and confusion that is characteristic in families trying to resist anorexia or bulimia. It dissolves confusions by appearing to reduce a multilayered, complex problem to a one-dimensional, material one, and it generates hope of a solution that would bypass interpersonal conflict. This hope is misplaced, since it rests on a misunderstanding of the nature of genes and of the possible role genetics plays in the development of anorexia and bulimia.

The risk is that clients and practitioners will be "blinded by science," that is, that the genetic explanation will first of all be misunderstood in terms of "classic" genetics and/or that it will be taken as "the real story," which will eclipse other important story lines, reducing the struggles a girl experiences to the material/genetic and implying that salvation resides in a manipulation of her material being. The latter, of course, echoes a practice that she is already convinced is necessary.

Whether or not there is a meaningful way in which we can say that certain genes represent risk factors for anorexia or bulimia, Catalina and Jack will need to find a way to help Elena face her fear of eating and gaining weight, because it is not a sustainable practice to avoid eating. Fear of eating is not like some fears, for example, the fear of spiders or of flying, because while a person could live a long and healthy life without ever making friends with spiders or flying in an airplane, it is not possible to avoid food (and weight gain) indefinitely. Fear of eating is also unlike the fear of, for example, staying in a burning building, where the fear of fire drives us to escape, ensuring our survival. Escaping eating rather ensures our demise. So, Elena's parents are in the difficult position of asking her to do something she is genuinely terrified of doing but which they know is necessary. This will involve discussing ways that they can support Elena in having regular adequate meals, at the same time understanding and caring that this is intensely difficult for her. She may be able to manage increased eating and weight only in stages, and this may take a long time. It could also include Jack and Catalina insisting on a longer hospital stay. Avoiding these aspects of supporting Elena colludes with her avoidance of food and weight gain.

Equally important is having Elena, Catalina, and Jack explore the ways in which resisting eating is important to Elena and the ways in which Catalina and Jack may be inadvertently supporting anorexia. This could involve many avenues of exploration. In what ways have Catalina and Jack themselves been affected by our culture's methods of generating body insecurity? Are there ways that they resist these cultural pressures? How might their body regulation and eating practices fuel or help resist anorexia? Can they find their way to helping Elena feel secure in a body of greater substance? Is Elena taking very little food as a way of punishing herself? If so, what aspects of her environment contribute to her sense that she should be punished?

Many other areas could be explored with Elena. Is Elena spending more and more time on her schoolwork, but not feeling satisfied with the results of her efforts? Might this be happening in the context of increased expectations from the teachers in her high school? Is talk of the difficulties of getting into a good university charging up her worries? Are worry and self-doubt making trouble for Elena as she sets about making new friends in her high school? Is Elena finding that she is not ready to bring her developing sexuality into relationships in the ways that her peers do seem ready for? Or is Elena ready, but finds herself stymied by her parents' worries? Is not eating or not gaining weight a way of resisting increased expectations or registering a vote against these expectations altogether? Would dominant stories about her be fractured if she needed help, wanted to shirk responsibility sometimes, or didn't feel confident? Would it provide an alternate identity in the face of her rejecting the ones that are offered at school or within her family? Would Elena prefer delaying declaring an identity, or does she need support in articulating or presenting one? The challenging and multifaceted work of developing an identity in a culture that generates personal and body insecurity while simultaneously demanding confidence (even of adolescents) cannot be avoided by reducing some adolescents' struggles in this area to stories about genes.

Paula

Paula⁹ is a 30-year-old woman who has a history of many hospitalizations related to anorexia, some following near-death emergencies. She endured forced treatments several times, and medical complications have resulted in sustained damage to some of her internal organs. Nevertheless, she has now been doing well for over 2 years, is in a relationship, and has become pregnant by choice. An ultrasound reveals that the fetus is female. Paula recently read an article stating that the genes related to serotonin regulation and the genetic underpinnings of personality traits like perfectionism and novelty seeking are considered to be susceptibility genes for eating disorders. She is terrified at the thought that her daughter could go through

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experiences similar to those she has had. As she is in her first trimester, she is considering an abortion.

Paula has begun to think of her personality and brain chemistry as immutably flawed or defective and that these defective aspects of herself could be passed on to her daughter. The genetic story line thus encourages Paula to compare her personal characteristics with those of people who have not struggled with serious eating problems. She may be thinking that it would be better to have a baby with characteristics different from her own or that she is already a bad mother. In this way, the genetic story about eating disorders creates the idea that some psychological and chemical characteristics are defective, mirroring the culture-based judgment that certain body types are defective. Rather than encouraging an appreciation of natural and cultural variation that could encourage resistance to pressures to regulate our bodies, the gene story supports the rigid kind of thinking that gives eating problems an easier entry into our lives. In a narrative therapeutic conversation, Paula could be supported to deconstruct the gene story line so that the idea of genes as determining characteristics would be unraveled. She could also be encouraged to articulate the ways in which her personal characteristics have helped her ultimately to resist anorexia, thus challenging the idea of these characteristics as flaws or defects. Finally, Paula could be supported to recognize that she has insider knowledge of anorexia and that this knowledge provides a basis for strategies that would help protect her daughter in the future, giving her daughter an advantage rather than a liability.

The best impact that increasing knowledge about genetics could have for those with eating problems is to improve the specificity of medications that can play a role in supporting recovery from eating problems like anorexia. Currently SSRIs (selective serotonin reuptake inhibitors; predominantly fluoxetine [Prozac] for adolescents) and atypical antipsychotics (usually risperidone or olanzapine) are used to help ameliorate depression, anxiousness, and/or obsessiveness to a sufficient extent that the person is able to make use of other therapeutic supports. No research supports the view that any of these medications is sufficient when used without other therapies, nor does any research support the view that medications are always necessary; but research and clinical practice do support the view that medications can be helpful adjuncts to other therapies in many cases. Currently, there are undesirable effects (called "side effects") accompanying all of these medications, and sometimes a considerable amount of time-consuming and unpleasant experimentation is required before a useful medication is found for a particular individual. If genetic research were to make it possible to refine our understanding of, for example, the biological contributions to depression and to develop medications that target different variations of these, it is possible

that more effective medications with fewer unwanted effects could be produced. These could play an important though time-limited role in the process of helping women to resist troubled eating.

A narrative approach is open and curious about the multiple experiences and meanings in women's lives that may support and assist in the resistance of troubled eating. It allows for many possibilities, and many forms of expression by individuals in their cultural contexts, which fits with the empirical fact that most women and girls are preoccupied with weight and that most associate being thin with status, self-esteem, power, and a sense of control. It also fits with findings that an individual woman might be dieting and preoccupied with weight at one time, anorexic at another, bulimic later on, and then, later yet, not particularly preoccupied with weight nor restrictive of food intake. Most important, those working from the narrative perspective are committed to respecting the agency of the client and working collaboratively to understand problems and develop solutions.

Conclusion

In the gene story, a pathogenic culture is recast as neutral or made invisible, and the nature-given, gender-irrelevant genes of a small percentage of the population are seen as causing vulnerability to a highly gendered disorder. This makes individuals responsible for fixing their culturally induced problems and leaves the economic and political interests that benefit from promoting a range of perfectionistic and obsessional body-regulating practices free to continue to do so without criticism. As these practices become more normative,¹⁰ it becomes more difficult for people to question their effects and to see their costs. Manufacturing medical model, privatized accounts of eating disorders masquerading as objective scientific discovery depoliticizes and decontextualizes their development in cultures and in individuals.

The larger modernist quest to make the gene story part of mastery over our biology promises control over disease, aging, disability, and practically anything else we don't like, including what we call mental illnesses. It seduces us into the project of creating the perfect human being, who is seemingly very beautiful and never dies. This project may be conveyed as one that will benefit all of us, but it has historically required that some be sacrificed for the benefit of others, and access to its purported benefits has always been political. Even if genes are included in our understanding of eating disorders in a way that makes good theoretical sense, it will also be necessary to use this understanding in a way that makes good political and ethical sense.

Notes

1. A *genome* is an organism's complete set of DNA. When the Human Genome Project, undertaken by the U.S. Department of Energy in the 1980s, succeeded in drafting the human genome, it meant that geneticists had identified the approximate number of genes in human DNA (at the time, it was thought to be about 30,000, but more recent estimates are closer to 25,000, a number shared with the roundworm). It also meant that the particular side-by-side arrangement of bases along the human DNA strand could be described or sequenced (U.S. Department of Energy Genome Programs: <http://www.ornl.gov/hgmis>, p. 1).

2. Personality "traits" like perfectionism are now being described as "phenotypes," with the obvious implication that there are matching genotypes.

3. According to the U.S. Department of Energy Web site on the Human Genome Project, "The genetic key to human complexity lies . . . in how gene parts are used to build different products [proteins] through alternative splicing" and in the "thousands of chemical modifications made to proteins and the repertoire of regulatory mechanisms controlling these processes" (<http://www.ornl.gov/hgmis/publicat/primer/>, p. 3).

4. Although boys and men are increasingly subject to similar prescriptions to have a body that is "worked on" or otherwise made to fit an image that increases their social value, resulting in their being increasingly concerned with body image (Pope, Phillips, & Olivardia, 2000, p. 27), the current requirement for males in terms of body shape is to increase muscularity, which is consistent with the direction their bodies developmentally tend to take. This explains why the increase in concern with body image among males has not resulted in a corresponding increase in eating disorders among them.

5. The only exception I know of is Howard Steiger's (2004) article "Eating Disorders and the Serotonin Connection: State, Trait, and Developmental Effects" in the *Journal of Psychiatric Neuroscience*, in which he comments in passing that dieting is associated with more pronounced alterations in postsynaptic 5HT (serotonin) activity in women than in men (p. 23).

6. An entire issue of the journal *Current Drug Targets* was devoted to the subject of eating disorders in 2003 (see Tozzi & Bulik, 2003).

7. The value of the current National Institute of Mental Health (NIMH) grant funding the search for genes related to eating disorders is \$10 million.

8. The "DeSousa-Jamesons" are a fictional family.

9. J. Hubert Lacey raised an example like this during a panel discussion on genetics and eating disorders at the 2005 International Eating Disorders Conference in Montreal, Canada.

10. In the October 2004 issue of *Harper's* magazine, it was reported that the town council of a city in northern Tuscany voted unanimously on a resolution by Mayor Sandro Donati to subsidize council members' expenses for cosmetic surgery, because the resulting improvement in council members' appearances and self-esteem could cash out in terms of "positive economic repercussions for the community" (Donati, 2004, p. 16).

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