

“SOME SORT OF LARGER FORCE AT WORK”:
MEANINGS AND IMPLICATIONS OF GENETICS
FOR WOMEN WITH EATING DISORDERS

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ABSTRACT

MICHELE M. EASTER: “Some sort of larger force at work”: Meanings and implications of genetics for women with eating disorders.

(Under the direction of Peggy A. Thoits and Andrew J. Perrin)

Recent research supports the idea that genes play a role in the development of many complex psychiatric and behavioral disorders, including eating disorders. Although no genes have been linked to anorexia or bulimia nervosa, heritability estimates from twin studies have been described in scientific and popular literature. Genetic causation may compete with other causal narratives that implicate individual choice, family problems, and cultural norms. In this dissertation, I treat genetic ideas as newly available “cultural tools” for individuals to use as they manage identities, organize action, and conceptualize their own behavior and condition. Through semi-structured interviews with fifty women who have a history of anorexia or bulimia nervosa, I describe their understandings of genetic causality for this complex disorder and its implications. Half the sample was currently receiving treatment (recruited from a hospital-based clinic); the other half had recovered (self-reported and recruited through a mass email).

Chapters 3 and 4 illuminate respondents’ understandings of eating disorders and their causes before the idea of genetic influence is brought up as a topic of discussion. In Chapter 3, I present the complex, dynamic causal models put forth by respondents and their perceptions of their own ambiguous, ambivalent agency in carrying out eating disorder behavior. Respondents spoke of eating disorder causation in ways that were not reducible to biological, “environmental” (e.g., social, cultural, familial factors), nor “individual” (e.g.,

psychological, agentic) factors and frequently involved elements of all three in interaction with each other over time. I illustrate this dynamic interactive causation by highlighting three complex causal factors frequently cited by respondents: valorization of thinness, coping responses, and repetition over time. The language respondents used to describe eating disorder behavior suggested that they held complex notions of agency; the disorder and its constitutive behaviors were not simply “chosen” but there was nevertheless some role for agency or quasi-agency.

In Chapter 4, I focus on respondents’ understandings of eating disorders and responses to specific terms describing eating disorders. There was consensus that eating disorders were problems, but disagreement about whether they were psychological problems, mental illnesses, brain diseases, physical illnesses, or choices. As respondents discussed whether and why a given term was appropriate (or not) for eating disorders, they revealed not only how they thought about eating disorders but also how they defined the terms presented. I summarized their reactions to all five terms with an index of medicalized term endorsement and found that respondents who were currently in treatment and those who had received more extensive treatment endorsed more medicalized terms.

In Chapter 5, I examined respondents’ initial reactions to the idea that “some say there are genetic causes” followed by their more considered speculations about how genes *could* play a role. Most respondents had already mentioned genetics in relation to eating disorders before I brought it up. There were negative and positive initial statements and reactions about genetics. Some respondents found the idea implausible and characterized genetic explanations as simplistic and deterministic, with an inadequate role for environmental causes. Others thought it made sense and expected that it would remove

blame and stigma from people with eating disorders and offer hope for new genetically-based treatments. People who had earlier endorsed medicalized views of eating disorders were more likely to hold “positive” initial views about genetics, as were people with more or current experience of treatment. Respondents’ genetic theories allowed a great deal of room for agency and environmental influence. Most respondents found genes specifically “for” AN or BN less plausible than genes “for” something more general, such as personality type or addictive tendencies. I identified four ways that respondents combined genetic and non-genetic influence and rank-ordered theories according to how much conceptual “room” they allowed for non-genetic forces to shape an outcome, arguing that genes “for” AN or BN offered the least room and genes “for” body type the most.

Chapter 6 addresses the perceived implications of genetic causal ideas for people with eating disorders. In response to hypothetical scenarios involving genetics, most respondents perceived genetics to medicalize eating disorders by increasing their resemblance to other less contested diseases, by making treatment by healthcare professionals seem more necessary, or by raising expectations for biologically based treatments. In general, respondents interpreted genetic influence to imply less personal responsibility for the eating disorder, which in turn had a number of implications for their agency and future action vis-à-vis eating disorders.

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LIST OF ABBREVIATIONS

AN = Anorexia nervosa

BN = Bulimia nervosa

R-AN = Recovered from AN

R-BN = Recovered from BN

T-AN = In treatment for AN

T-BN = In treatment for BN

CHAPTER 1

GENETICS AND THE MEDICALIZATION OF A CONTESTED ILLNESS

Research tells us that anorexia nervosa is a brain disease with severe metabolic effects on the entire body. While the symptoms are behavioral, this illness has a biological core, with genetic components, changes in brain activity, and neural pathways currently under study. (Letter from Thomas R. Insel, Director of the National Institute of Mental Health, to National Eating Disorders Association, October 5, 2006) (Insel 2006).

As one who has battled the eating disorder bullshit, I don't like all this 'explaining away by genetics, etc.' stuff at all. I'd be willing to bet the vast majorities of [cases of eating disorders] are birthed by our cultures [sic] hatred of all women weighing over 100 pounds. The genesis lies here. The blame goes here. (Blog comment posted by "Kim" in response to science reporting about genetics and anorexia nervosa, March 16, 2006) (Kim 2006).

The purpose of this study is to find out whether and how women diagnosed with eating disorders incorporate genetic information into their self-understandings and their understanding of the causes, controllability, and treatment of the disorder. Eating disorders, such as anorexia nervosa and bulimia nervosa, are characterized by extreme attention to body image, weight control, and eating behavior. Psychiatric researchers argue that these conditions are caused by genetic factors in combination with environmental factors such as family dynamics and culture, rather than by genes or environment alone. Clinicians expect that genetic information will become more important in the understanding and treatment of psychiatric disorders generally. However, there is little knowledge of how people diagnosed with psychiatric disorders understand and use the idea of a genetic basis for their disorders.

As genetics are increasingly connected to a wide range of diseases and disorders, people's conceptions of them are likely to change. The process of coming to see more and

more diseases, disorders, and even character traits as based in genetics has been termed “geneticization” (Lippman 1991a). Geneticization resembles the process of medicalization, familiar to sociologists, in which a condition or behavior comes to be defined and treated as a medical rather than a moral or other kind of problem. Geneticization is likely to further the arguably incomplete medicalization of eating disorders because it places biology, rather than psychology or culture, at the earliest phase of the disorder. It is not known whether people with eating disorders are aware of the discourse about genetic factors nor what it means to them. Because academicians, clinicians, and others identify positive and negative aspects of geneticization, it is important to find out whether people diagnosed at some point with AN or BN share their hopes and fears, and/or introduce new ones. Thus, my study is concerned with how people “living under the description” (Martin 2007) of an eating disorder use and resist medical, biological and genetic explanations in making sense of their disorder and themselves. Such explanations can be seen as materials in the cultural repertoire, or “tools” in the “cultural toolkit” (Swidler 2003), for actors to use or not. This project diverges from other studies of medicalization because rather than observing a *society’s* shift toward a medicalized or geneticized conception of a category of behavior, I focus on how *individuals* use – or do not use – medical or genetic cultural materials. This focus on individual agents reflects the idea that culture is negotiated and recreated by individuals rather than monolithic.

This study contributes to our understanding of the meaning and consequences of genetic discourses for explaining a psychiatric disorder. Eating disorders, though already officially medicalized, have been further defined in medical terms by emphasis on their biological, and especially genetic, causes. This study demonstrates how people with eating disorders incorporate genetics when they talk about themselves and their disorder. This

broad research question comprises several sub-questions: Will respondents mention genetics at all, before I ask them about it? Will their understandings of genetic influence be simple or complex? Will they contest or accept the idea of genetic causality, or will it be ambiguous? How will they reconcile it with other possible causes and risk factors? Do they use genetics to manage their identities as people diagnosed with a psychiatric disorder? Through semi-structured interviews that include broad questions about the causes, controllability, treatment, and the nature of eating disorders, along with more specific questions about genetic and other causal factors, my study will shed light on the uses and meanings of genetics for people with a disorder that is undergoing greater medicalization and geneticization.

1. MEDICALIZATION OF EATING DISORDERS

In this study, I argue that genetic explanations are increasing the ongoing medicalization of eating disorders. “Medicalization” is the process in which a condition or behavior comes to be defined as a medical disorder (Conrad and Schneider 1992, Conrad 1992, 2007). Medicalization depends on how information is interpreted and used by social and political actors (Conrad and Schneider 1992). Conditions that at one time were not seen as medical – addiction, menopause, senile dementia, and eating disorders – are now frequently treated as medical disorders. Some conditions have been demedicalized over time, such as homosexuality and masturbation, but Conrad asserts that medicalization has generally increased over time.

Medicalization is a social and political process in which medical professionals, consumers, pharmaceutical companies, and others promote medical definitions for particular conditions. Many studies of medicalization concern the transformation of *normal* healthy human conditions, such as menopause or baldness, into treatable disorders. Other studies

address the transformation of *negative or deviant behavior* from “badness to sickness” (Conrad and Schneider 1992), from moral failure and deviance to illness. From this perspective, eating disorders fall into the negative and deviant category because they involve behaviors that depart from contemporary conceptions of healthy and normal because they are unusual (purging), extreme (restricting food to the point of emaciation) and entail negative physical effects including starvation, disability, and death. Anorexia nervosa (AN) has the highest mortality rate of any mental illness, with 5.6% of people with AN dying each decade from starvation, cardiac arrest, or suicide (Sullivan 1995, Herzog and Eddy 2007). Bulimia nervosa (BN) has a similar mortality rate (Crow et al. 2009), involving serious cardiac, dental, and gastrointestinal consequences (Herzog and Eddy 2007) and, as in the case of Terri Schiavo, ultimately coma (NEDA 2005). The prevalence of AN, BN, and binge-eating disorder (BED) for adult women in the U.S. and western Europe is 0.9%, 1.5%, and 3.5% respectively (Hudson et al. 2007). A greater percentage of the population has eating disorders that do not fit into these three categories or are below the diagnostic threshold for them (Hoek and van Hoeken 2003, Berkman et al. 2006).

Eating disorders have been medicalized in their classification in several editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM) (e.g., DSM-IV, APA 2000a) and medical treatment, but I argue that the current geneticization of these disorders provides cultural materials that further the conception of them as biologically based. I am concerned with eating disorders as diagnosable psychiatric disorders, rather than with the continuum of disordered eating that extends into the “normal,” such as calorie-counting, crash dieting, eating too much, and feeling fat when one is at a healthy weight.

Eating disorders have not always been seen as psychiatric disorders.¹ Joan Jacobs Brumberg describes the shift of people with AN “from sainthood to patienthood” in the U.S. over the 19th century (2000: 43). At the beginning of this shift, “fasting girls” were treated as miraculous and spiritual because they seemed to survive without food, suggesting the young woman’s divine rather than earthly nature. By the end of century the spiritual explanation was discredited and replaced with a diagnosis of physical disease (Brumberg 2000, also see Silverman 1997 for a description of AN as a treatable disorder as early as 1689). AN thus went from goodness to sickness, though arguably there was an interim period of “badness” as some people with AN and their families were accused of defrauding those who paid to view the miraculous fasting girls. AN was temporarily labeled “hysterical aepsia,” but “aepsia” assumed an inability to digest food and “hysteria” assumed involvement of the uterus, and so this term was replaced in 1873 by “anorexia nervosa” to describe a lack of appetite related to the central nervous system, rather than an inability to digest (Brumberg 2000). (“Anorexia” is also a miscategorization, except in advanced stages, because most people with AN do not lack appetite but rigidly control it.) Since the 1930s, psychiatrists began to use psychoanalytic and psychotherapeutic approaches more often, sometimes focusing on sexual repression or family dynamics (Brumberg 2000: 217-228), particularly the girl’s relationship with her mother (Vanderven and Vanderven 2003). In the last several decades, psychological

¹Assessing the changing perception of eating disorders over time presumes eating disorders to be objective medical entities, with changes in perception due to different cultural lenses rather than changes in eating disorders themselves. Such a perspective assumes a universal, biological essence beneath various socio-cultural constructions. Yet some have argued that eating disorders may be “culture-bound” syndromes restricted to or produced by Western modernity (see Swartz 1985, Banks 1992, Keel and Klump 2003) or defined too narrowly using Western criteria (Lee 2001). However, it is probably fair to interpret eating disorders over the last few centuries in the West as one phenomenon, as long as cases fulfill contemporary diagnostic criteria, even though the experience of AN over that time has changed to include behaviors such as excessive exercise (see discussions in Gremillion 2002 (footnote 3), Gooldin 2003, Fabrega and Miller 1995 for further information).

research on eating disorders has focused on the contributions of family and culture, including exposure to images of very thin women (Striegel-Moore and Bulik 2007).

Eating disorders have been successfully medicalized by most criteria for this process.

Conrad describes these as follows:

The key to medicalization is the definitional issue. Medicalization consists of defining a problem in medical terms, using medical language to describe a problem, adopting a medical framework to understand a problem, **or** using a medical intervention to ‘treat’ it. This is a sociocultural process that may or may not involve the medical profession, lead to medical social control or medical treatment, or be the result of intentional expansion by the medical profession. Medicalization occurs when a medical frame or definition has been applied to understand or manage a problem....” (Conrad 1992: 211, emphasis mine).

Conrad focuses on how a problem is defined rather than who defines it (medical professionals) and with what purpose (professional power) and consequences (social control). His definition thereby expands on previous definitions (Strong 1979, Davis 2006) and theoretically opens the study of medicalization to include studies of non-medical professionals and how they use medical terminology.² I rely on his expansive definition in this paper and do not presume that medicalization is necessarily tied to professions, power, or social control. By both the narrow and expansive definitions, eating disorders have been medicalized: they are officially under medical jurisdiction, there are medical interventions, and medical terminology is used to describe them.

The inclusion of eating disorders in the Diagnostic and Statistical Manual of Mental Disorders (DSM), the official classification of psychiatric disorders by the American Psychiatric Association, exemplifies both medical terminology and medical jurisdiction over eating disorders. “Eating disorders” were introduced into the third edition of the DSM in

² The definition was expanded to recognize that patients can drive the medicalization of their conditions, as with fibromyalgia (Barker 2002, 2008), and that some conditions become quasi-medical even if not treated by medical professionals, such as alcohol codependency and even child abuse (Conrad 1992, Pfohl 1977, Van Wormer 1989).

1980 (Kashubeck-West and Mintz 2001, APA 1980), replacing the category “feeding disturbance” from the second edition (APA 1968, see APA 1980: 383). “Eating disorders” comprised anorexia nervosa, “bulimia,” pica, and rumination disorder of infancy; in 1987, a revised version of the DSM used the current term “bulimia nervosa” (APA 1987). The diagnostic criteria for anorexia nervosa (AN) continue to be revised, but as of 2000 the DSM-IV Text Revision (DSM-IV TR) listed them as follows. Criteria A through D all must be present for a diagnosis:

- A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
- B. Intense fear of gaining weight or becoming fat, even though underweight.
- C. Disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
- D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles... (APA 2000a: 589)

Currently there are two types of anorexia nervosa as defined by the DSM-IV-TR: the “restricting type,” in which there is excessive food restriction and no binge-eating or purging, and the “binge-eating/purging type.”

For bulimia nervosa (BN), the DSM-IV-TR describes binge-eating followed by “compensatory” actions, such as vomiting, and use of laxatives, enemas, or diuretics (in the “purging type” of bulimia nervosa), or fasting and excessive exercise (the “nonpurging type”). These are the criteria for BN:

- A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
 - (1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances
 - (2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

- B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.
- C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.
- D. Self-evaluation is unduly influenced by body shape and weight.
- E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa. (APA 2000a: 594).

More than half of people diagnosed with eating disorders do not fall into either BN or AN, but in a residual category “Eating Disorder Not Otherwise Specified” (EDNOS) (Fairburn et al. 2007). This includes people who meet all but one criterion for AN or BN, and those who binge-eat without purging, a behavior that is under study and may become its own category of “binge-eating disorder” (BED) in the next revision of the DSM (APA 2000a: 785-787).

Inclusion in the DSM alone is sufficient evidence for medicalization according to Conrad’s definition above. But eating disorders also have physical consequences and some are treated medically through hospital-based inpatient or partial hospitalization programs. Compared to other psychiatric diagnoses such as schizophrenia, biology and medicine are unambiguously involved because AN produces starvation, which visibly affects the body (Estroff 2007, personal communication). Starvation also affects the mind. Regardless of what led to the starvation, even those who do not advocate biological origins for the disorder incorporate biology to explain later stages of the disorder, as with the concept of a physical “addiction to starvation” (Brumberg 2000: 32-33, Bordo 1997: 102). The initial goal of a hospital-based treatment is medical stabilization (Bowers et al. 2004: 353) because starvation can lead to life threatening complications that are not solved by eating alone. Hospital programs include medical personnel, laboratory testing, and medical interventions, such as a nasogastric tube if the patient is forcibly fed. The first goal is to restore weight: “In an

inpatient or partial hospitalization setting, food is the patient's primary medicine. Sometimes it is the only medicine" (Bowers et al. 2004: 354). This "nutritional rehabilitation" involves close monitoring of caloric intake and body weight by a registered dietician. The treatment team also includes psychiatrists, who may prescribe psychiatric medications for some patients, and psychologists, social workers, occupational therapists, or recreational therapists. As the patient gains weight, she or he engages in the emotional and cognitive work required to change eating behaviors through cognitive behavioral therapy (CBT) and other approaches (Bowers et al. 2004).

1.1 Contestation despite medicalization

Despite their institutionalization in the DSM and frequent treatment by doctors in hospital-based clinics, eating disorders, like most other psychiatric disorders, also seem incompletely medicalized compared to diseases such as cancer. Giles suggests that eating disorders are "contested illnesses" much like chronic fatigue syndrome (2006: 466). The marginal status of eating disorders is evident in at least four ways: inadequate insurance reimbursement, fluctuations in their definition, the kinds of methods used to treat them, and skepticism by the lay public about their medical status and particularly their biological origins.

Eating disorders are included in the current DSM, but insurers do not consistently reimburse for them and when they do it is often inadequate (Kalisvaart and Hergenroeder 2007). The epigraph from Thomas R. Insel, Director of NIMH at the beginning of this chapter illustrates this aspect of contested medicalization, as well as the role of consumers as "engines" of medicalization (Conrad 2005). The quoted letter was posted on the website of the advocacy group, National Eating Disorders Association (NEDA), presumably as a

resource for families to use in their fight to obtain reimbursement from insurance companies. The letter was so important that it was featured in the title of the link to the “Parent, Family and Friends Network” with the title “Ask an Expert, Voices of Hope, *Dr. Insel NIMH letter*” (emphasis mine). A second link to the letter itself summarizes its significance: “A Letter from Thomas H. Insel, MD NIMH director states *anorexia is a ‘brain disease’*” (NEDA 2002, emphasis mine). Insel composed the letter in response to a request from the advocacy group for “a statement that underscores that eating disorders are brain diseases and that appropriate treatment can work” (Insel 2006). One blogger put it this way: “Note to insurers: All the recent research on eating disorders shows a strong genetic and biological component to the disease. The suffering of those with anorexia is real and based in biology. Pay up--or have the suffering and deaths of children on your corporate conscience” (Brown 2006).

A second reason for the marginal status of eating disorders is that the DSM criteria and labels are not stable and mutually exclusive. A recent review by psychiatric researchers summed up the situation: “The eating disorder diagnoses remain best construed as open and falsifiable diagnostic constructs in need of further scientific study... rather than discrete disease entities that have been discovered in nature in their true form” (Wonderlich et al. 2007: 167). As mentioned above, the DSM-IV-TR lists three types of eating disorders: AN, BN, and EDNOS. The DSM criteria and categories for eating disorders have been revised several times since 1980 and are expected to be revised again for the DSM-V. Clinicians and scientists note a lack of discrete division between anorexia nervosa and bulimia nervosa because people alternate between diagnoses; indeed having AN is a risk factor for developing BN (Herzog and Eddy 2007, Tozzi et al. 2005). In addition, the residual category “Eating Disorder – Not Otherwise Specified” (EDNOS) accounts for at least half of all eating

disorder diagnoses (Herzog and Eddy 2007, citing Fairburn and Bohn 2005). This lack of a “specific diagnosis” contributes to denial of insurance coverage (Striegel-Moore and Wonderlich 2007). Some researchers advocate that this residual category be differentiated into binge-eating disorder, purging disorder, and folded into expanded definitions of AN and BN. The fluctuation of DSM categories, the large residual category, and the crossover of individuals from one category to another may make eating disorder diagnoses seem less “real” and, therefore, less medical than other disorders.

Third, eating disorders do not currently have a standard pharmaceutical treatment. Biologically based treatments, primarily re-feeding, are part of the treatment for eating disorders if a person has significant weight loss, cardiac arrest, or electrolyte imbalance; “re-feeding” lasts many weeks in the hospital, may (rarely) involve the use of feeding tubes, and is monitored by medical doctors. However, the psychological features that caused these physical problems are not usually treated biologically. For AN, “no drug as yet exists that effectively treats the core features of the disorder: body image disturbance, obsessionality and perfectionism, and extreme anticipatory anxiety” (Kaplan and Noble 2007: 138). For BN, fluoxetine (Prozac) has been approved by the FDA, but pharmacotherapy is not the primary method for treating any eating disorder (LaVia 2007, Berkman et al. 2006). Cognitive behavioral therapy is considered to be the most effective treatment for BN, and is used for treating all eating disorders in many hospital units. Individual and family therapy are also used along with occupational therapy, relaxation, and other modalities (Bowers et al. 2004). These approaches are not incompatible with biological causation, but they do not connote biological treatment the way a pharmaceutical intervention would. By contrast, a “brain disease” suggests structural problems, biological pathways and biologically-based

treatment such as pharmaceuticals or surgery. I argue that the current lack of biologically-based treatment contributes to the perception that eating disorders are less biomedical than other disorders.³ Similarly, although medical monitoring and giving food – the “primary medicine” (Bowers et al. 2004) – are clearly biological, to some extent these address the consequences of the eating disorder rather than the eating disorder itself. Clinicians and researchers see appetite and weight “dysregulation” as key components of AN, such that the disorder has core biological components. However, some biological components are clearly the effects of starvation and malnutrition, and can be distinguished from the eating disorder itself: altered brain function, abnormal blood pressure, heart rate, electrolyte imbalance, dehydration (APA 2000b: 5-6). From this perspective, food and monitoring are part of medical stabilization, which is a prerequisite to treating the eating disorder through CBT and other psychotherapies for a patient who is severely underweight.

Fourth, there is evidence that the public, including academicians, does not always view eating disorders as medical disorders or as brain diseases. As Conrad notes,

in most cases of medicalization of deviance, public acceptance ‘lags behind’ professional and bureaucratic support. The public remains more skeptical about medical designations than do professionals, especially in the cases of alcoholism, opiate addiction, and homosexuality. This skepticism provides a reservoir of potential support for future challenges to medical deviance designations. (1992: 271).

This “reservoir” of alternative views is evident in survey data: A British survey found that over a third of the general public thought people with eating disorders had “only themselves to blame” (34.5%) or were “able to pull themselves together” (38.1%). Respondents blamed people with eating disorders for their conditions less than those with alcohol and drug

³ Viewed from the perspective of biologically-oriented researchers, this would be a side-effect of the “trap” of face validity (Bulik 2005: 335, 2006): the strong face validity of social and familial causation discourages research into biological causation, thereby preventing the development of biologically-based treatments, and in turn reinforcing the sense that eating disorders are not biologically caused.

addictions, but more than those with panic attacks, depression, schizophrenia, and dementia (Crisp et al. 2000, also see Holliday et al. 2005b, Stewart et al. 2006). If a condition is volitional, it is less like a disease and suggests a non-medical view of the disorder. In addition, it is common for clinicians and advocates for eating disorders to lament the widespread perception that eating disorders are volitional, reflecting vanity or character flaws, rather than a disease for which the person is not responsible (e.g., Holliday et al. 2005b, Keel and Klump 2003, Bulik 2004, Stewart et al. 2006). The consequences of eating disorders may be physical, but those consequences are seen as the result of a volitional choice. I include in the “reservoir” of skepticism non-medical academic explanations of eating disorders that also oppose the medical interpretation of eating disorders and use alternative terminology. For example, a feminist sociologist writing about eating disorders consciously resists medical terminology:

I avoid using the term eating disorder because it categorizes the problems as individual pathologies, which deflects attention away from the social inequalities underlying them (Brown 1985). However by using the term problem I do not wish to imply blame. In fact, throughout, I argue that the eating strategies that women develop begin as logical solutions to problems, not problems themselves. (Thompson 1992, footnote 1, citing Brown 1985)

Hepworth (1999) writes, “My aim is to challenge the dominant conceptualization of AN as a psychopathology... [that is] separable from the social practices through which it became defined” (pp. 3-4).

In this proposal, I assume that eating disorders have been medicalized, following Conrad’s definition, and I use the term “disorder” in accordance with the dominant medicalized understanding of AN, BN, and EDNOS. When I describe eating disorders as having been medicalized, I am not doubting that they are harmful, serious, “real” disorders but describing their current conceptualization (see Brown 1995, Conrad 1992: 211-213). My

purpose is to understand how people are thinking about genetic explanations of their conditions. The term “disorder” is appropriate for this project because I focus on women who have been formally labeled as having an eating disorder by DSM criteria. I recognize that I am using a term that some scholars and people “living under the description” of AN or BN may not accept. Because of this, my interview guide avoids terminology that assumes a medical understanding.

1.2 Re-conceptualizing medicalization

The case of eating disorders clarifies the underlying logic of the medicalization thesis, specifically the idea that even after a diagnosis has been institutionalized it can be further medicalized. Certainly analysts recognize “degrees” of medicalization (Conrad 1980) and acknowledge a continuous rather than dichotomous process, but usually these continua are applied to conditions that have not yet been institutionalized. Yet Conrad notes that genetic and neuroscientific discoveries not only “reaffirm” the medical status of a mental disorder, but can render it “more highly medicalized” (Conrad and Schneider 1992: 281). This idea has received little study. In studies of medicalization, the classification of a disorder as medical and its treatment by medical professionals comes at the end of the story, rather than being a point of departure as it is in my study.

In order to make sense of eating disorders as undergoing further medicalization, I propose that medicalization can be conceptualized as having two converging dimensions: the pathological and the biological. The pathological dimension ranges from healthy and normal to sick, while the biological dimension ranges from phenomena that are not clearly biological to those that are (see Table 1.1). A psychiatric condition may be considered “sick” even without a biological cause or treatment. Because psychiatric diagnoses are not equivalent to

“brain disease,” the two dimensions can be usefully disentangled to describe the location of eating disorders along a continuum of the medicalization process. The cells of Table 1.1 are separated by dashed rather than solid lines to convey that the dimensions are continuous rather than categorical.

I believe Table 1.1 (below) is a clarification of, rather than a departure from, the thesis of medicalization as advanced by Conrad. Clearly medicalization occurs when a phenomenon is pathologized into a treatable disorder by the medical profession, and this is the explicit focus of those who study medicalization. However, there is less clarity about whether it must also be perceived as biological, even though the word “medical” is often taken to mean physical or biological. As Conrad states, “Medicalization doesn’t require specific claims about cause, although the assumption is often biological; certain medicalized problems like child abuse make no biological claims whatsoever” (2000: 329). For example, child abuse and its effects were medicalized by virtue of using medical-sounding words like “syndrome” (“battered child syndrome”) and identification of the syndrome by radiologists and pediatricians (Pfohl 1977). However, battered children were not necessarily medicated, treated by psychiatrists, nor thought to have acquired the syndrome through biological origins; medicalization need not be biologization.

Table 1.1. Medicalization Status as a Convergence of Two Dimensions: Pathology and Biology. (Lighter cells denote greater medicalization than darker cells.)

<i>Pathological status</i>	<i>Biological status</i>	
	Biological	Not clearly biological
<i>Sick</i>	Biological AND pathological (e.g., Cancer, diabetes, “brain disease”)	Psychiatric diagnosis (e.g., Social Anxiety Disorder listed in the DSM)
	Lay formulations: (e.g., Fibromyalgia, Chronic Fatigue Syndrome as physical rather than psychological conditions)	Lay formulations: (e.g., alcohol codependency by family members of alcoholics is seen as a type of behavioral disorder)
<i>Undesirable state, negatively deviant, in some cases the person’s fault</i>	Undesirable physical states (e.g., obesity, baldness, short stature, erectile dysfunction may be treated medically)	Character flaws, problem behaviors (e.g., drug use, frequent sadness may be medicated)
<i>Normal, healthy</i>	Genetic predisposition for anything “undesirable” or “sick” ⁴ (e.g., genetic predisposition to ovarian cancer makes someone a “pre-patient”)	Personality traits (e.g., shyness may come to be treated as Social Anxiety Disorder)
	Normal physical states or conditions (e.g., menopause, childbirth can be treated medically)	
<i>Superior Health or Ability</i>	Enhanced physical abilities	Enhanced cognitive abilities (e.g., intelligence, memory),

In Table 1.1, medicalization occurs with movement from darker to lighter cells. Conditions listed in a cell may have moved there recently or may be on their way to a new cell. Movement upward reflects increased pathologization and arrival in the top row requires official classification as a disorder or disease. The top row includes clearly biological conditions in the left cell (e.g., infectious disease, cancer, heart disease) and conditions that are not clearly biological in the right cell (psychiatric disorders). As psychiatric disorders come to be seen as biologically caused, they move to the left (e.g., autism, schizophrenia, bipolar disorder). Although psychiatric disorders in the top right cell have been officially

⁴ Only appears on the biological side because genetic predisposition is biological by definition.

classified as such by the DSM, this classification does not mean that symptoms, origins, or treatment are necessarily biologically based; “Whatever its original cause, [a disorder] must currently be considered a manifestation of a behavioral, psychological, or biological dysfunction in the individual.” (APA 2000a: xxxi)⁵ For some readers, inclusion in the DSM may be tantamount to biologization because of the biological orientation of the psychiatric profession (Luhmann 2000, Horwitz 2002). However, in Conrad’s work, medicalization is a matter of degree, and even a DSM category can have its medical status heightened, “furthered” or “reaffirmed” by the discovery of a genetic origin.⁶ To move leftward, a condition would need to be described or treated as “biological” in some way – perhaps because it is located in a bodily organ (e.g., “brain disease”), or is thought to have a biological cause (e.g., neurotransmitter abnormality), or is treated with medication (e.g., Prozac). The division between biological and “not clearly biological” is not absolute, just as the division between physical and mental is not (APA 2000a: xxx). Biological conditions can have social causes, for example. Horizontal and vertical movement may take place over centuries, as when the dominant, widely-shared meaning of a condition changes, or in a single clinic visit, as when a person comes to redefine her symptoms in terms of misfiring neurotransmitters. This table is meant to illustrate that medicalization is a continuous process

⁵ Horwitz offers this observation however: “Although diagnostic psychiatry is officially agnostic about the variety of factors that lead people to develop mental diseases, the medicalized system of classification it uses emphasizes underlying organic pathologies... Although many mental health professionals continue to locate the origins of psychological problems in disturbed childhood relationships with parents, the study of the biological foundations of discrete mental disorders has gained unquestioned primacy in the profession of psychiatry.” (2002: 3) Horwitz bases this on the greater attention to biology in the psychiatric profession as a whole, not the DSM per se.

⁶“Perhaps new genetic associations with more marginal psychiatric disorders like obsessive compulsive disorder or attention deficit-hyperactivity disorder could provide evidence for claims of further medicalization, although it would depend on how the evidence was used” (Conrad 2000: 326), “With the advent of the Human Genome Project... it is very likely that more genetic grounds of mental disorders will be uncovered... This, of course, very likely will lead to a *reaffirmation* of the medicalization of madness” (Conrad and Schneider 1992: 281, emphasis added.)

with two dimensions. When a condition moves up or to the left, it is more medicalized than before, and when it fits into the top left cell its medicalization is complete, within the scope of this project.

Case studies of medicalization fit into this conceptual table. The example of homosexuality illustrates the two dimensions. Homosexuality once occupied the top right cell because it was classified as a disorder by the DSM, but was not viewed as biological in origin. When it was removed from the DSM, it moved downward into the other categories along the right column: unofficially “sick,” flawed, or healthy depending on the observer. With recent studies suggesting a genetic origin for homosexuality (Hamer 1993), it becomes biologized and moves leftward, again depending on the extent to which a genetic origin is embraced. It is important to note that a genetic causal attribution does not move a condition up the table along the pathologizing dimension: a genetic explanation biologizes but does not necessarily pathologize homosexuality because blue eye color is also thought of as genetic without being a disorder (Conrad 2000). Case studies of women’s health illustrate how healthy, normal conditions – childbirth and menopause – moved upward along the left column over time as they were increasingly treated by medical professionals using medical procedures or prescription medication, thus coming to resemble disorders more than normal life events (Riessman 1983). PMS moved into the top row when it was classified as a psychiatric condition in the DSM-IV (Figert 1996, though in the DSM-IV-TR it is listed as awaiting further study, APA 2000a: 771-773); its origins in the menstrual cycle are clearly biological so it could occupy or approach the top left cell. In the case of Attention Deficit and Hyperactivity Disorder, behavior that was once seen as deviant moved upward to become a psychiatric diagnosis (Conrad 2006). Enhancement, in which normal human

qualities are improved or altered, is reflected in the bottom row, labeled “super-healthy” or “better than well” (Conrad and Potter 2004, Kramer 1993, Elliott 2003). By the very existence of a category of “better than well,” characteristics that once seemed normal and healthy – balding, shortness, shyness (Conrad 2007) – are medicalized because they are no longer the least pathological state. Other examples are provided in the cells of the table, and the case of eating disorders will be described momentarily. Although cases can be fit into the cells of Table 1.1, the point of the table is to illustrate movement across a continuum and only the top right cell has specific criteria for entry.

My formulation overlaps with and differs from two other sociological models of medicalization:⁷ Adele Clarke et al.’s (2003) recently introduced concept “biomedicalization” and Phil Brown’s (1995) tabular presentation of the “naming and framing” of medical disorders. Clarke and colleagues focus on transformations in U.S. medicine rather than how behaviors and conditions are defined. Their addition of the prefix “bio-” to “medicalization” is not related to my emphasis on biology as a dimension of medicalization: “We signal with the “bio” in biomedicalization the transformations of both the human and nonhuman made possible by such technoscientific innovations as molecular biology, biotechnologies, genomization, transplant medicine, and new medical technologies. That is, medicalization is intensifying, but in new and complex, usually technoscientifically enmeshed ways” (p. 162). “Bio” for Clarke connotes contemporary biotechnology and “technoscience,” and while this includes geneticization (p. 169), Clarke’s focus is on technical interventions such as predictive genetic testing, gene-based interventions such as

⁷ It also overlaps with and differs from numerous models that address individual understandings of disease, such as the distinction between clinician-defined disease and patient-defined illness (Kleinman 1988). Because I am concerned here with perceptions at the societal level rather than the individual level, I do not discuss them here. But such models are certainly relevant to how individuals use medicalized and geneticized cultural materials, which is discussed later.

pharmacogenomics, and even gene therapy. Because these are changes in medical practice rather than redefinitions of problems, they are only distantly relevant to this project.

Brown (1995) captures the varieties of social construction in medicine along two dimensions which define a four-fold table: “biomedical definition applied or not” to a condition, and “condition generally accepted or not accepted as a biomedical entity” (40). He does not focus on psychiatric conditions and how they may seem less or more medicalized depending on a biological etiology or treatment. He does acknowledge that some conditions have a biomedical label despite not being “generally accepted” as medical conditions, for example, chronic fatigue syndrome, late luteal phase dysphoric disorder (LLPDD or PMS), and chronic pain syndrome. However, these biomedical labels were not official medical labels and would perhaps be better labeled “bio-medicalizing.” My conception of medicalization differs from Brown’s because I focus explicitly on biological rather than “biomedical” concepts, on official rather than “generally accepted” labels, and view the process of medicalization as more complex than does Brown.

1.3 Heightened medicalization of eating disorders

The place of eating disorders in the table has changed over time and, I suggest, is in the process of changing now. Table 1.1 provides a framework for understanding these changes. When extreme abstinence from food was seen as evidence of a holy nature, eating disorders might have been in the bottom right cell as superior health because the saint’s abstinence from food was a form of positive deviance. When eating disorders were seen as negative and deviant they moved upward in the right column, and when viewed as disorders they reached the top row: close to the left cell if attributed to a problem with digestion or reproductive organs (Brumberg 2000), closer to the right cell if attributed to family dynamics

or patriarchy.⁸ Currently, by virtue of classification in the DSM, they are in the top row, if we assume that medical classification reflects a dominant understanding. A dominant understanding is not the only understanding. In some current contexts – the modeling industry, the dance profession – self-starvation and purging may be statistically normal behaviors, and among those few who aspire to get AN, so-called “wannarexics” (Bauman 2007), these behaviors may even seem “better than well.” However, eating disorders, like other psychiatric disorders, are arguably “not clearly biological” for the four reasons noted above: they are commonly seen as volitional rather than as having a biological origin, the most effective treatment is CBT, the diagnostic categories shift, and insurance reimbursement is less than adequate. Like AN and BN, other psychiatric disorders appear to be moving in a biological direction as well (Horwitz and Wakefield 2007, Luhrmann 2000).

For eating disorders and other psychiatric diagnoses (top right cell), discovery of biological causation, the location of the disorder in the physical brain, or the use of biologically based treatments would strengthen the claim to biological status, and thus a more complete medical status (Mizrachi 2002). There is recent evidence of a push to move eating disorders to the left, more biological side. The letter from Insel in the epigraph illustrates the importance of biology in making the case for a medical definition of psychiatric disorders. Rather than AN having *effects* on the biological brain and body as a result of starvation, it is a “brain disease” with a “biological core” which suggest biological *causes*. The inclusion of genetics places biology at the very earliest possible point of a causal chain. Insel was urged to issue this statement by a leading advocacy group (NEDA, described earlier). The idea of

⁸ This table would need additional dimensions to accommodate these three concepts as well: that eating disorders are a normal response to a pathological society (Hepworth 1999, Thompson 1994), that “problems in living” are wrongly described as mental illnesses (Szasz 1960), and contestation over environmentally-defined conditions such as “black lung” (Brown 1995).

mental illness as a “brain disease” is most famously disseminated by the National Alliance on Mental Illness (NAMI), an advocacy organization, which asserts that “[j]ust as diabetes is a disorder of the pancreas, mental illnesses are medical conditions that often result in a diminished capacity for coping with the ordinary demands of life” (NAMI 2008). According to a leading geneticist of eating disorders, sociocultural explanations for eating disorders divert attention from potential biological explanations, with bad effects in her view: “The face validity of these [sociocultural] explanations has inhibited our progress due to a burden of plausibility. The sheer convenient believability of sociocultural explanations has influenced research directions and hindered recognition of the seriousness of eating disorders” (Bulik 2004: 165, Bulik 2005). This suggests a rationale for promoting biological explanations; in contexts where sociocultural reasons are not taken seriously, those who want eating disorders taken seriously would do better to frame them as biologically based. Unlike many critics of pharmaceutical companies, some researchers and advocates for people with eating disorders regret that the lack of a standard pharmaceutical treatment prevents companies from making the case to the public that eating disorders are real illnesses through their advertising campaigns: “[a]t this time AN is unable to realize such ancillary benefits from the emergence of effective psychotherapies” (Stewart et al. 2006, also see Conrad 2005 on the “drivers” of medicalization).

The perception of a genetic cause for eating disorders would also push eating disorders to the left of Table 1.1, thereby helping to medicalize them. Geneticization, a process similar to medicalization, in which a condition comes to be described and understood in terms of genetics, is the focus of this study.

2. GENETICS AND THE COMPLEX CAUSES OF EATING DISORDERS

To prepare for discussion of the geneticization of eating disorders, why it matters and what it might mean to people with eating disorders, I review expert accounts of the genetics of eating disorders, those elements that are relevant to this project, and what people with eating disorders might know about them. The genetic explanation for eating disorders is part of a complex causal model that includes culture, gender, family, personality, and other factors. This complexity makes the study of eating disorders particularly interesting because people may grapple with how to reconcile multiple, potentially conflicting causal explanations and how to understand their implications.

Including genetics in complex causal models helps scientists explain why people who share other risk factors do not always go on to develop eating disorders. These causal models do not presume that a genetic explanation is the only explanation but also include environmental triggers, risk factors, and protective factors. Those scientists who promote the idea of a genetic cause do not tend to think of eating disorders as Mendelian “genetic disorders” like hemophilia or Huntington’s, but disorders with complex genetics, like diabetes or alcoholism. Genetic influence has been established through twin studies, though scientists have not identified which genes are important, which aspects of eating disorders might be heritable, and how various eating disorders are related to each other. Indeed, genetic findings may alter current disease classifications (Faraone 2002).

Based on twin studies, in which identical or “monozygotic” (MZ) twins are compared to fraternal or “dizygotic” (DZ) twins of the same sex, researchers have concluded that there is evidence in support of a genetic component to eating disorders (Bulik 2004). Classic twin research design compares MZ and DZ twins to assess whether the former are more

commonly concordant (that is, whether they have the same phenotypes or outcomes) than the latter. Such twin studies theoretically hold environment constant because twins are raised in the same household. Monozygotic twins share 100% of their genetic material;⁹ heterozygous twins share on average 50% of their genetic material. This ratio of 100/50 or 2/1 provides the basis for the estimate of “heritability,” or the genetic contribution to the variance observed. This method has produced estimates of heritability ranging from 48-76% for AN, 50-83% for BN and 41% for binge-eating disorder (Striegel-Moore and Bulik 2007). It is important to note that these heritability estimates are based on Western samples only. Western rates of eating disorders are higher than non-Western rates, suggesting that sociocultural factors matter a great deal. If a twin study included both Western and non-Western twins, or twin pairs in which one was raised in the West and the other not, it is likely that the environmental contribution to the variance would be higher because there would be greater variability on that key independent sociocultural variable. Some researchers argue that AN is “more genetic” than BN because some diagnosed with AN have had no exposure to Western culture (Keel and Klump 2003).¹⁰

⁹However, recent studies show that even monozygotic twins may differ genetically because of “copy-number variation,” a type of “structural variation” in which the number of copies of DNA segments varies (Bruder et al. 2008). The existence of copy number variation among MZ twins suggests that when their phenotypes are discordant, it cannot be attributed only to the environment but may also be genetic.

¹⁰ Some scholars criticize the assumptions of twin studies and cast doubt on their claims. These criticisms come not only from social scientists, who are arguably motivated to resist biological explanations (e.g., Horwitz et al. 2003a), but also from geneticists who use different methods (e.g., Wahlsten 2007). The validity of this project does not depend on the validity of twin studies and therefore I do not evaluate them here. In a recent example, sociologist Allan Horwitz and colleagues cast doubt on one of the essential assumptions of twin studies: the “equal environment assumption” that MZ twins do not have more similar environments than DZ twins in ways that matter for the outcome studied. If this assumption were violated, it would suggest that the greater similarity of monozygotic twins could be due not only to genetic but also to environmental similarity. In their study of depression and alcohol abuse using a twin sample that was more diverse than other twin samples (the National Longitudinal Study of Adolescent Health, a.k.a. Add Health) they found that key social environmental variables – spending more time together, having friends in common – were more similar for identical twins than for fraternal twins and that they explained some or all of the variance in outcomes. Horwitz suggests that estimates of genetic heritability take “credit” for social environmental causes. (Horwitz et al. 2003a, and their critical exchange with Freese and Powell 2003, Horwitz et al. 2003b) Some geneticists also criticize the assumption

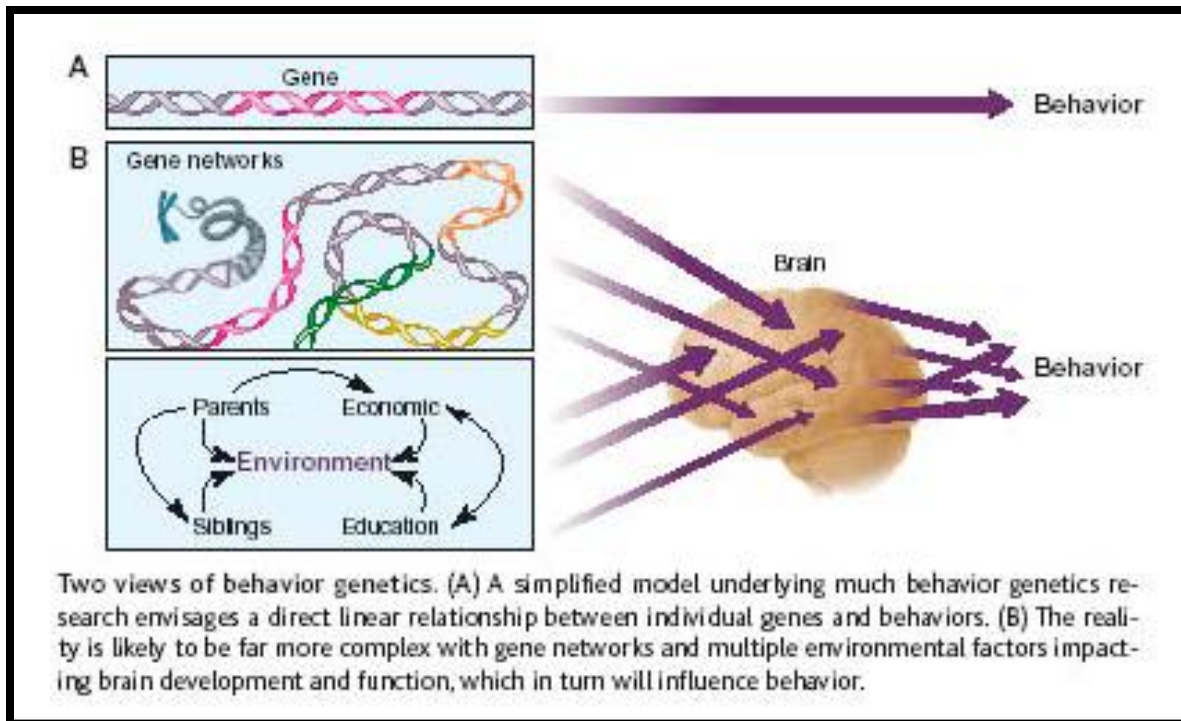
Most chronic diseases and psychiatric disorders are thought to have complex genetic contributions, as opposed to Mendelian or no genetic contributions. Chronic diseases such as obesity, diabetes, and heart disease are thought to have complex genetic explanations (what I refer to as “complex genetics”). Most cancers are thought to have complex genetics, despite some subtypes of breast, ovarian, and colon cancers being linked to specific individual genetic variants. Behavioral and psychiatric disorders such as alcoholism, autism, schizophrenia, depression, bipolar disorder, eating disorders and many others are also thought to have complex genetics. Complex genetic explanations involve multiple genes, interactions between genes, possible gene-environment interactions, and environmental factors that confer protections or risks for a disorder. In the early 2000s many geneticists came to the conclusion that despite evidence that genetics played a role in many disorders, the inability to find “the gene for” those disorders suggested that single genes were unlikely to account for the observable outcomes or “phenotypes” they studied. Many scientists became skeptical that new genetic “main effects” would be discovered, because findings would have been replicated by now if single genes were indeed causal factors (e.g. Hamer 2002). In the last two years, however, the hope of finding single genes has been rekindled because new study designs, large-sample Genome-Wide Association Studies (GWAS) and then “re-sequencing” or NextGen technologies enables the testing of tens of thousands of genes and identification of unexpected genes, rather than being limited to “candidate” genes

that variation can be partitioned into genetic and environmental components at all because if genes and environment interact then the genetic and environmental contributions are not discrete and cannot simply be added together. In addition, critics argue that several additional assumptions are unwarranted: that there is no selective placement of step-siblings (in adoption studies), negligible gene-environment correlation, and the equal environments assumption criticized by Horwitz (Wahlsten 2007, Perrin and Lee 2007). For counter-arguments see Freese and Powell 2003, Loehlin 1992, Rowe 1994: 38-40. Some researchers argue that the equal environments assumption for eating disorders is warranted because eating-disorder-related variables are not more likely to be shared by MZ twins than DZ twins (e.g., Klump et al. (2000), see discussion in Bulik 2004: 170ff).

identified earlier (WTCCC 2007, Frayling 2007, ten Bosch and Grody 2008). In one sense, these large studies represent a paradigm shift in genetic research because of their immense scale, but they also are a return to an older conceptualization of genes directly linkable to disorders. In the following I discuss a previous paradigm shift, in which the search for single genes gave way to a search for complex explanations involving environmental variables.

Complex genetic models for behavioral and psychiatric phenomena involve the brain as a mediator of the effects of multiple genes and environmental factors. Below is a figure comparing the “old” and “new” models for behavioral genetics (Hamer 2002, featured by Schaffner 2006). This figure illustrates a recent paradigm shift in behavioral genetics. The “old” genetics expected to find a single gene for the phenotype under study (Model A in Figure 1.1 next page). This model is also referred to as the “gene for” model or the one-gene-one-disorder, or OGOD model. The “new” genetics for behavioral disorders theorizes many intermediate and interacting variables, all of which act through the brain at some point (Model B in Figure 1.1). The new paradigm is characterized by gene-environment interaction, expectation that large numbers of genes will be involved, and the greater role of neuroscience.

Figure 1.1. Two Views of Behavior Genetics (Reproduced from Hamer 2002: 71)



Although Model B is more complex than the “old” Model A, it leaves out much complexity too. Model B would be an oversimplification even for behavioral geneticists if only because behavior – such as self-starvation or alcohol consumption -- also acts back upon the brain (though a behavior such as alcohol consumption might be reconceptualized as part of the “environment” in genetics). In Figure 1.1, this would be represented by an arrow from behavior back to the brain. In AN, the behavior of self-starvation would directly affect brain function by depriving the body and brain of adequate nutrition. AN might also influence the person’s social environment because it is likely to change activities, friends, and even her exposure to the “thin ideal” as she may seek so-called “thinspiration” from media images of very thin women. Behavior thus changes environment as well. Some researchers would also hypothesize an arrow from genes to the body and then to behavior or brain, if genes that impair the metabolism of alcohol or food affect behavior directly. Social scientists would

likely conceptualize causation differently, paying more attention to the environment and less attention to the brain. The Model B diagram is thus a theoretical model of the initial development of a disorder rather than how it is maintained or altered over time.

For this dissertation, the important parts of the “new” model of behavioral genetics are those that diverge from the “gene for” model.

1. Combinations of multiple genes or “gene networks” are theorized, rather than a single gene. In eating disorders, some candidate genes are thought to be related to perfectionism, anxiety, and perseverance.
2. Diverse pathways may be involved. Different genes affect the brain in different ways, such that a single outcome can be the result of multiple pathways (termed “equifinality”). Thus many different gene and environment combinations can lead to a single diagnosis such as AN or BN.
3. The brain mediates the effects of gene or environment via neurochemicals such as serotonin.
4. The new model includes the environment, however vaguely or incompletely described. In AN, environmental risk factors include participation in gymnastics, ballet or any other activities that reward weight loss; exposure to the “thin ideal” of Western modernity via media images of celebrities and models; and the experience of teasing about weight and shape, among others.

In addition, a few elements that are not pictured in the diagram are important for this project because they may affect respondent self-understandings and understandings of the disorder:

5. The focus on the brain as intermediary gives rise to the concept of the “intermediate phenotype” or “endophenotype” in behavioral genetics, which is a biological or

behavioral characteristic along the theoretical causal pathway between genotype and phenotype, specifically on the causal arrow between brain and behavior (Gottesman and Gould 2003). For example, people with AN might have cognitive characteristics (e.g., trouble with “set shifting”, Holliday et al. 2005a) or personality tendencies (e.g., perfectionism, Bachner-Melman 2007) that are less common in the general population. Their biological relatives may have the same characteristic, without having the disorder. Endophenotypes are detectable only using specialized instruments and techniques such as experimental testing, personality questionnaires, and functional magnetic resonance imaging (fMRI) of the brain. These characteristics are not easily apparent phenotypes (*pheno* connotes the visibility of the outcome) but more difficult-to-detect *endophenotypes* (*endo* connotes internal). In order to be true intermediate phenotypes, they must not be a consequence of the disorder.

6. Because genotypes and endophenotypes are shared by people with and without the disorder, there is a continuum between normal and abnormal rather than a categorical difference. This aspect of psychiatric genetics overlaps with the genetics of personality and behavior and deconstructs dichotomous disease classifications and categories. Psychiatrists looking ahead to the DSM-V and future versions expect that genetic research will lead to the recategorization of diagnoses (Faraone 2002).

Given these complexities, there are several ways that genes may play a role in eating disorders. Striegel-Moore and Bulik (2007) cite two possibilities other than the possibility that research may ultimately uncover a main effect of the kind portrayed by Model A in Figure 1.1 (perhaps through Genome Wide Association Studies). The first possibility is that

genes might interact with the environment (a “gene-environment interaction model”), in which a genetic susceptibility to an eating disorder makes a difference because the person is exposed to an environmental risk. For example:

[A]n individual with Genotype A might experiment with her first extreme diet, find the experience aversive and uncomfortable, and reject the behavior on the basis of it not being at all reinforcing. In contrast, an individual with Genotype B might experience that first episode of severe caloric restriction to be highly reinforcing by reducing her innate dysphoria and anxiety, providing her with a sense of control over her own body weight and resulting in her receiving positive social attention for weight-loss attempts. That individual might then adopt the behavior in a persistent manner because of its multiple reinforcing effects, which would then set the stage for the development of anorexia nervosa. (Striegel-Moore and Bulik 2007: 188)

In this example, the extreme diet is the environmental trigger, possibly brought on by involvement in an activity in a cultural context that values weight loss. People with low genetic susceptibility would not develop AN even if they went on an extreme diet. The gene-environment interaction model has frequently been described with the memorable slogan, “Genes load the gun, environment pulls the trigger” (e.g., Bulik 2007 presentation).

A second alternative model is that the current diagnostic constructs are merely a “grab bag of symptoms” (Striegel-Moore and Bulik 2007: 188), only some of which can be directly explained by genes. There may be genetic main effects for a few symptoms, but these can be discovered only if the definition of the phenotype is purified and refined:

Genetic main effects could primarily account for the core symptoms we see in eating disorders that have persisted throughout history— such as maintenance of low body weight, binge eating, and self-induced vomiting—but the pervasive culture of the times may have “filled in the gaps” by providing an explanatory context and padding the definition of syndromes with environmentally mediated and contextually plausible symptoms. For example, the maintenance of low body weight seen in anorexia nervosa has been observed for centuries, and indeed, the core phenotype of persistent low body weight appears to have genetic underpinnings . . . , yet the psychological “fillers” that have been added to create the syndrome of anorexia nervosa have changed over time. (Striegel-Moore and Bulik 2007: 188-9).

An example of a culturally specific “filler” is the Western emphasis on fat phobia, which is not as common among Asian patients who fit the other criteria for AN (see Lee 2001). The quest to refine the phenotype is interesting from a social constructionist angle; it recognizes the constructedness of current diagnostic categories but still seeks an unconstructed genetic core. Refinements are likely to change the DSM categories and therefore will eventually influence the conceptions of non-geneticists and lay people (Faraone 2002).

3. MEDICALIZATION AND GENETICIZATION ON THE GROUND: STUDYING PEOPLE WITH EATING DISORDERS

The introduction of genetics as a partial explanation of eating disorders – whether a simple or complex model – constitutes a “geneticization” of eating disorders, and, as I have argued, heightens their medicalization.¹¹ There are many definitions of geneticization (see Hedgecoe 2001). I define geneticization by adapting the expansive definition of medicalization from Conrad above:

Geneticization consists of defining a condition in genetic terms, using genetic language to describe it, adopting a genetic framework to understand it, or using a genetic intervention to ‘treat’ it... Geneticization occurs when a genetic frame or definition has been applied to understand or manage a problem.... (Adapted from Conrad 1992: 211).

In this section I explain how my study diverges from other studies of medicalization and geneticization, and blends with sociological approaches.

My study will address the understudied concept of “heightened” medicalization through interviews with women diagnosed with AN or BN and contribute to an emerging

¹¹It is important to note that geneticization is not equivalent to medicalization. The causal role of genetics for blue eyes, extraversion, high intelligence, or homosexuality does not imply pathology. For this project, I see geneticization as a type of medicalization because it increases the resemblance of eating disorders to biomedical diseases. At the same time, I recognize that genetics can be used to *de*-medicalize a trait: the genetics of homosexuality are also used as evidence for its naturalness and normality as part of human variation (Conrad 1997: 147, 2000).

empirical literature about genetics and the lay person. Because studies of geneticization and medicalization are concerned with changes in the dominant view of a category over historical time, they tend to be concerned with official classifications, professional turf wars, and media representations rather than the perspectives of people with a condition. My study takes seriously the idea that medicalization is a continuous and negotiated process even after an institutional definition has been accepted at the professional and societal levels. People with a diagnosis provide a window into this process and how it affects those who have the most to gain or to lose.

By studying people with eating disorders and whether and how they use genetic ideas, my approach departs from traditional sociological approaches to both medicalization and geneticization. Early studies of medicalization by Wootton (1959), Zola (1983), Szasz (1960) and Freidson (1970) focused on official medical jurisdiction. Conrad's redefinition of medicalization, which I have adapted for geneticization, allows what might be called personal or individual medicalization because it does not insist on the involvement of the medical profession. People may use biological and pathological terminology to describe their experiences even in the absence of an official medical label, and they may reject such terminology even when there is a medical label. Early treatments of geneticization also focused on the societal level (see Hedgecoe 1998).¹²

At least one critic has argued that the expansion of medicalization to include individual and non-professional definitions has rendered the theory a "complete muddle" (Davis 2006: 51). Davis argues that medical professionals must be central to the definition of medicalization because "outside the sphere of medicine, we have no way to determine what

¹² According to Hedgecoe, scholars of both medicalization and geneticization have tended to begin with polemical assumptions about the dangers of each. My empirical study strives to avoid this assumption as well, as discussed in the section on hopes and fears about geneticization.

constitutes a "medical" term or framework" (54). He contends that Conrad's theory claims that the medical profession is not necessary for the process, but smuggles it back into the theory. For example, he notes that by Conrad's own definition, "demedicalization" must involve organized opposition to the medical profession, which presupposes the medical profession's involvement (55). Davis also objects to Conrad's value-neutrality about medicalization, preferring to reserve the term for a critical appraisal of illegitimate encroachment by the medical profession. In addition, Davis is concerned that studying individual-level medicalization distracts from the study of societal categories:

If medicalization literally means "to make medical," then including individual-level diagnostic interactions in the physician's office would seem to make sense. Strictly speaking, that pain in my abdomen is made medical when the doctor diagnoses it as appendicitis... In the original meaning, new definitions shifted problem categories (not individual instances of those categories) to medical jurisdiction. Now "medical terms," "medical language," and "medical framework" are no longer limited to those defined and used by the medical profession. Any group or individual's use of such terms/frameworks represents an instance of medicalization. (53-54).

For Davis, studying individual usage diverts from societal level usage.

There are also strong arguments for studying medicalization by focusing on non-medical non-professionals. Three other criticisms of medicalization literature would be resolved by such an approach. According to Charles Rosenberg, there is a "tendency to conceptualize medicalization as a reified, monolithic, and inexorable *thing*—a point-of-view that obscures the complex, multidimensional, and inconsistent nature of the way in which medical concepts and practices have laid claim to larger realms of social action and authority" (Rosenberg 2006: 408 fn.1, emphasis in original). Listening to diagnosed people enables the complexity and inconsistency of medical definitions to emerge, as they draw on multiple systems of meaning to create understandings. They may incorporate or oppose or recreate medical understandings, and they may not be consistent from time to time. A

second critique of medicalization literature, as well as Foucaultian approaches to similar phenomena, is that it assumes a passive patient who simply receives medical definitions from more powerful others (Williams and Calnan 1996, also Conrad 1992, Clarke 2003, Moreira 2006, Lupton 1997). This tendency has been counteracted by more recent studies that cast patients as agents of medicalization for difficult-to-detect conditions (such as multiple chemical sensitivity and fibromyalgia) (Barker 2002, 2008, Kroll-Smith and Floyd 1997) and as active managers of their own problems, using medicalized understandings and approaches if the treatments seem safe and avoiding them if they don't (Hislop and Arber 2003). My study will contribute to this area by examining how people with eating disorders use medicalized definitions, if they do. A third criticism is that studies of medicalization presume bad effects on patients and society. By tacitly assuming that medicalization is equivalent to *overmedicalization*, and that its identification is tantamount to social critique, its critics presume too much (Rose 2007a, Bury 2006). Studying lay people with a disorder lets us examine the complex meanings of medicalization and geneticization for people who experience their effects.

My conceptualization of medicalization in Table 1.1 avoids problems identified by these critics as well as by Davis. It gives the medical profession an important, though not exclusive role (top right cell of Table 1.1); it allows for complexity, inconsistency, and individual agency by positing a continuous rather than a dichotomous process, and defines "medical" by specifying two conceptual dimensions, biology and pathology. I agree with Davis's criticism that the theory of medicalization is muddled to some degree, and I believe my framework clarifies and extends Conrad's theory while taking into account some of Davis's objections.

My approach to medicalization and geneticization is compatible with symbolic interactionism, social constructionism, and the “toolkit” strand of the sociology of culture. These approaches emphasize the role of individuals in creating meaning and a sense of reality, rather than the imposition of meanings by an abstract “actor” such as society or culture. Genetic and medical terminology and concepts can be thought of as tools in a cultural repertoire for individuals to use (Swidler 2003). The medical profession’s adoption of genetic and medical terms for eating disorders grants the terms greater cultural power, but individuals may or may not use them (Schudson 1989). My focus on one cultural area – biomedical and genetic explanations of behavior – resembles cultural sociologist Ann Swidler’s (2003) focus on the discourse of love in American culture.

I expect that people will use genetic and medical cultural tools to accomplish goals, such as making sense of their condition, exculpating themselves, or providing accounts of their behavior to others (including an interviewer). According to Scott and Lyman (1968), an account is provided when there is a discrepancy between expectation and action. People with eating disorders are potentially confronted with many such discrepancies: how to account for having a mental illness, how to explain why they engage in behaviors that are seen as undesirable (bingeing, purging) or extreme (restricting), why they cannot control these behaviors (or do not wish to), how recovery happens, and why some people have eating disorders and others do not (also see Boltanski and Thévenot 1999). Because I am asking questions about causality and personal experience, I expect respondents will frequently respond in narrative form. Narratives can be seen as “accounts which contain transformation (change over time), some kind of ‘action’ and characters, all of which are brought together within an overall ‘plot’” (Lawler 2002). In interviews, people are not simply reporting facts

but constructing their stories and identities and actively making meaning using existing cultural materials. If genetic ideas are cultural tools, then, are people using them, and if so, how, and in what combination with other ideas?

The idea of individual-level medicalization or geneticization has much in common with other social science research into how patients make sense of their conditions or behaviors as medical problems, including mental illnesses and eating disorders. Although many of these literatures do not link their work to sociological thought about medicalization (see Conrad 1997), they are nevertheless relevant to this project and ought not be overlooked. For example, studies of labeling (Phelan and Link 1999, Link and Phelan 1999) address whether and how individuals accept their medical labels and what the consequences are. Studies of illness narratives describe individuals' experiences of illness and medicine that diverge from biomedical understandings of disease and some address the extent to which diagnosed people incorporate official medical versions of their experience (e.g., Bury 2006, Kleinman 1988, see Rich 2006: 287 for a list of others). Studies of explanatory models (Kleinman 1980), illness accounts (Estroff et al. 1991), illness identities (Barker 2002), health identities (Hislop and Arber 2003), illness perception or representation (Moss-Morris 2002), and lay understandings of medicine, science and technology (e.g. Prior 2007) also address such issues.

4. LAY GENETICIZATION OF EATING DISORDERS?

By the definition of geneticization above, eating disorders are being geneticized by professionals, the media, and to some extent by lay people. Although there are no genetically-based treatments for eating disorders, genetic terminology, frames, and definitions are used to describe and understand eating disorders. A genetic contribution to

eating disorders is accepted in academic journals and textbooks. According to O'Hara and Smith (2007), many clinicians include it in their discussions with patients.

Genetic ideas about eating disorders are available to lay people from many sources. One study of U.S. newspapers found that few articles about eating disorders mentioned a genetic or biological cause between October 13, 2004 and October 13, 2005 (two out of a subsample of 90 articles in 7 U.S. newspapers, O'Hara and Smith 2007). However in December of 2005, *Newsweek* ran a cover story entitled "Fighting Anorexia: No one to Blame" (Tyre 2005) focusing on the role of genetics in AN among children. In addition, the National Eating Disorders Association (NEDA) has tried to "spread the message about the powerful influence of genetics on both our physical and mental shape" during the annual eating disorders awareness week (NEDA 2008). Recent survey results show that even some members of the general public without special knowledge of eating disorders have heard of genetic causes. According to a poll commissioned by NEDA, 30% of the members of a consumer panel (n=1500) thought of genetics as one of the "primary causes" from a list of possible causes of eating disorders, though much higher percentages cited dieting (66%) and the media (64%) as primary causes (GMI and NEDA 2005).

To assess the ease with which contemporary respondents might have come into contact with such information, I conducted simple Google internet searches. Of the top ten results of a Google search using the search string "anorexia," four mentioned genetics as a cause. Of the top ten results using the more focused search string "anorexia cause", nine mentioned either genetics (8) or "inherent biological factors" (1). Of the top ten results for the search string "bulimia cause", five included information about genetics; for "binge

eating disorder' cause", six included genetics.¹³ People who seek internet information about the causes of eating disorders are likely to encounter the idea of a genetic cause, though it may be overshadowed by information about sociocultural and psychological causes. Anecdotal evidence from a hospital-based eating disorders clinic also suggests that most adult eating disorder patients have heard of genetic causation before entering treatment (LaVia 2008, personal communication).

Although there is reason to think that people diagnosed with AN or BN may have heard general information about genetics in connection with eating disorders, there are little data about whether they accept it and how they understand it. Previous studies of how people with eating disorders interpret their condition, its causes, and treatment have not focused on genetic or biological understandings. In qualitative and quantitative studies of perceptions of causality thus far, people with eating disorders have tended neither to mention nor to endorse genetic risk factors, both before one might expect people to have heard of genetic research (Button and Warren 2001, Nevenon and Broberg 2000, Tozzi et al. 2003) and after (Holliday et al. 2005b, Mond et al. 2004, Quiles-Marcos et al. 2007, Stewart et al. 2006). Patients also did not tend to mention biological causes at all. Those asked to identify the causes of their eating disorders cited instead weight-related problems and interpersonal problems (Nevenon and Broberg 2000), loss of control and relationship problems (Button and Warren 2001), family dysfunction, weight loss or dieting, and stress (Tozzi et al. 2003). People given forced-choice questionnaires also tended to endorse psychological and social causal factors more than biological factors such as genetics (Quiles-Marcos et al. 2007), as is true for other psychiatric conditions (Read et al. 2006). Like patients, recovered people also

¹³Google searches for AN and BN conducted by the author on November 11, 2007, for BED December 27, 2007.

did not tend to credit pharmaceutical interventions, but therapy was important for many (though more cited a supportive partner relationship) (Tozzi et al. 2003). My respondents may mention genetics more than past respondents because of recent publicity and because I will ask them about risk factors in addition to causes, which are more likely to elicit thoughts about genetic predisposition (French et al. 2005).

I expect that genetic explanations will be incorporated differently by different respondents. Just as medical definitions are not uniformly accepted by all individuals or groups, people with eating disorders may differ in the ways they incorporate or reject genetic explanations. The existence of a cultural tool does not compel a person to use it. Individuals may reject, revise, misunderstand and ignore cultural tools that are available to them (Schudson 1989, Griswold 1994, Gamson 1992). In the sections that follow I explore the variety of ways people may use genetic cultural tools.

5. HOPES AND FEARS ABOUT GENETICIZATION

In this section I address the hopes and fears about medicalization and geneticization presented by clinicians, academics, and other commentators. I have described the contested medicalization of eating disorders and how genetic discoveries may help to medicalize them further. I explained my focus on lay understandings of medical and genetic accounts of eating disorders, and speculated on the extent of geneticization of eating disorders among prospective respondents and how respondents' understandings might vary. The hopes and fears of clinicians and others suggest some of the potential diversity of meanings and implications of genetics for respondents.

The process of medicalization is thought to have both good and bad aspects. Conrad and Schneider list the "brighter side" of medicalization: "the creation of humanitarian and

non-punitive sanctions; the extension of the sick role to some deviants; a reduction of individual responsibility, blame, and possibly stigma for deviance; an optimistic therapeutic ideology; care and treatment rendered by a prestigious medical profession; and the availability of a more flexible and often more efficient means of social control” (than criminalization) (Conrad and Schneider 1992: 248). They also describe a “darker side” of medicalization: the individualization of social problems, the depoliticization of deviance (i.e., that power is involved in defining and punishing deviance), the dislocation of responsibility, the assumption of medicine’s moral neutrality, control of public debate by experts rather than lay people, and greater social control by medical professionals (Conrad and Schneider 1992: 248-251, also see Wootton 1959, Zola 1983, Riessman 1983, Illich 1976, Szasz 1960, Freidson 1970). Like medicalization, geneticization has both bright and dark sides in commentators’ views. Many of these resemble bright and dark sides of medicalization, reflecting the ability of geneticization to heighten medicalization (see Table 1.2 for examples relevant to eating disorders). Doctors, scientists, and patient advocates hope that scientific discoveries about the genetic role will not only contribute to improvements in the understanding and treatment of disease, but also remove blame from the individual. For example, “We predict that... identifying genes involved in behavioral disorders will do much to improve public perception and tolerance of behavioral disorders” (McGuffin, Riley and Plomin 2001).

The “dark side” of geneticization includes genetic versions of medicalization’s drawbacks (listed earlier), as well as other concerns specific to genetics. The individualization of social problems and the depoliticization of deviant behavior become particularly troubling if the social arrangements that produce differences in behavior across

race are ignored in favor of genetic explanations of that behavior (Duster 2003, Gould 1996, Herrnstein and Murray 1994). Genetic testing expands medicalization by monitoring and even treating those who are not sick but only genetically at risk (Conrad 2000, 2005, Kenen 2007, Konrad 2003). Expectations for gene therapy medicalize those whose heretofore normal attributes might one day be genetically improved or enhanced (Conrad 2004, 2007). The logic of disease prevention through prenatal genetic testing and pregnancy termination could be a “backdoor to eugenics” (Duster 2003). The dislocation of responsibility associated with medicalization can be extended even farther with genetic explanations of personality and behavior, leading to the question “Are we free to choose to behave as we do or is it caused by our genes?” (Parens 2004: S4). And if control of public debate by experts rather than lay people was of concern under medicalization, it is even more so for genetics because the influence of genes not only seems harder to understand than other biological explanations, it is still in the process of being discovered.

Going beyond medicalization, genetics introduce some unique problems. Genetic origins may make a problem seem permanent, uncontrollable or unchangeable and lead to fatalism (Alper and Beckwith 1993), though there is evidence to the contrary (e.g., Novas and Rose 2000). There are many other potential concerns, including essentialism, reductionism, genetic discrimination and privacy, but I do not describe all of them here (see Press 2006, Pilnick 1999, Kerr 2004, Carson and Rothstein 1999, Conrad and Gabe 1999, Rose 1995, Lippman 1991, Nelkin and Lindee 1995, Conrad and Schneider 1992, Riessman 1983, Davis 2006, Henderson 2008, Duster 2003). Many concerns about geneticization currently do not apply to eating disorders because there are no identified genes and hence no predictive genetic test that exposes people to risks from genetic disclosure including

discrimination by insurance companies based on test results. Concerns that are relevant to the study of eating disorders appear in Table 1.2 and are explored in greater depth below.

Negative features such as reductionism, essentialism, and determinism once were assumed and even incorporated into early definitions of geneticization (e.g., “Geneticization refers to an ongoing process by which differences between individuals are **reduced** to their DNA codes...” (Lippman 1991a: 19, emphasis mine). For this “critical” group of writers, identifying the geneticization of a phenomenon constituted “an activist’s rallying cry, a way of raising public and political consciousness about the possible drawbacks of genetic technologies and information” (Hedgecoe 2001: 307). My definition of geneticization (see above section on geneticization of eating disorders) does not assume positive or negative consequences. Many recent studies are also neutral about the effects of genetics, describing new identities and “biosocialities” related to identified genes (Atkinson et al. 2007, Gibbon and Novas 2008, Glasner et al. 2007, Schaffer et al. 2008), theorizing a cultural shift toward “somaticization” of personality (Rose 2007a, 2007b, Vrecko 2006), and strategizing about how best to integrate genetics into clinical practice (e.g., Richards 1993, Marteau 2004, Senior 1999, Parrott et al. 2004, Smith 2007). My definition and approach follow Hedgecoe and others by avoiding these assumptions, though my study is partly motivated by concerns about negative consequences.

My study examines how people with eating disorders actually use genetic concepts and whether they think about some of the hopes and fears that have been articulated by academicians. While they may not have the same understanding or concerns as these experts, the hopes and fears expressed in academic literature suggest some hypotheses for what they might bring up and a sense for what might be at stake.

It is important to note that most of the literature about hopes and fears addresses only simple genetic explanations. Ideas about genetic explanations ought to differ depending on whether genetic causation is understood in the older simpler way or in the newer more complex way (Figure 1.1 above). Although genetic explanations for eating disorders are complex, they may be oversimplified by those unfamiliar with the science, particularly if messages about genetic influence do not also emphasize that environment matters. Thus, Table 1.2 (next page) outlines hopes and fears that may apply to simple and complex understandings of a complex genetic causal model for eating disorders. One goal of this study is to learn how complex explanations are understood and used by lay people. Table 1.2 does not exhaust the possible meanings of genetic explanations for people with eating disorders. After I describe each cell of Table 1.2, I suggest additional details and variations that I expected to emerge in interviews.

Table 1.2. Genetic Explanations for a Complex Psychiatric Disorder: Hopes and Fears about Simple and Complex Explanations

	<i>Simple genetic explanation for complex disorder</i>	<i>Complex genetic explanation for complex disorder</i>
<i>Hopes</i>	<ul style="list-style-type: none"> • Remove blame for individual and family • Remove stigma for individual and family • Improve family’s ability to cope with the eating disorder of a family member • Draw attention to an understudied disorder by increasing perception of severity • Make insurance reimbursement more likely 	<ul style="list-style-type: none"> • Provide some or all of the benefits from a simple genetic explanation (see cell to the left) • Promote a balanced and accurate understanding of what can and cannot be controlled by the person • Encourage greater awareness of environmental causes and how to avoid them • Harmonize with common sense understandings of disease causation
<i>Fears</i>	<ul style="list-style-type: none"> • Exacerbate stigma • Add new kinds of stigma • Increase determinism and fatalism about prevention, treatment and recovery • Decrease plausibility of and commitment to non-biological treatments. • Promote biological reductionism • Decrease attention to non-genetic causes (e.g., sexism, trauma) • Provoke a reaction against any biological or genetic explanations 	<ul style="list-style-type: none"> • Provide some or all of the harms from a simple genetic explanation (see cell to the left) • Increase confusion through the proliferation of causes, risk factors, protective factors and environmental triggers • Increase distance between lay and expert understanding

5. *Hopes for simple genetic explanations*

The benefits of a simple explanation for eating disorders are listed in the top left cell of Table 1.2: Removal of blame and stigma, improving coping, and underscoring the idea that the disorder is serious and worthy of insurance reimbursement. People seeking treatment may be aware of these potential advantages and use genetic ideas accordingly.

Less blame and stigma for individuals. The main source of stigma for people with eating disorders is that they are held personally responsible for their disorder. Unlike people with schizophrenia, they are not stigmatized for being dangerous (Crisp et al. 2000, Mond et

al. 2006, Holliday et al. 2005b, Corrigan et al. 2002), though in one study survey respondents say they “pose a greater danger to others” than people with asthma or those with no known disorder (Stewart et al. 2006). Many advocates for eating disordered people and the medicalization of eating disorders expect that more focus on genetics as opposed to social and cultural factors and personality will reduce the blame assigned to the sufferer and her family (O’Hara and Smith 2007, Tyre 2005, Duffy and McElhinny 2007). According to attribution theory, a genetic or other biological explanation removes blame from the person and should reduce stigma. Some studies show that disorders attributed to a biological cause are perceived to have a less controllable onset and to elicit greater pity and less anger from others compared to disorders that are not (Weiner et al. 1988, Corrigan 2000, Meiser et al. 2005). A study of nursing students found that those told of a genetic cause were less likely to hold the eating disorder patient responsible (Crisafulli et al. 2008), though medical professionals in training may be more likely to accept the biological and genetic causal explanations than the general public, which tends not to (Read and Harré 2001). For some, the idea that one’s disorder is genetic might make one better able to take action because one does not have to accept all of the blame for it (Herman 1993). Genetic explanations for body size rather than one’s eating behaviors might be appealing for people with eating disorders, as well (e.g., the “embrace your genes” campaign, NEDA 2008). People with eating disorders who perceive themselves to be overweight may find a genetic explanation for their body size is helpful for destigmatizing their weight (Cordell and Ronai 1999, Martin 2002). These strategies would work best in a context of wide public awareness and acceptance of a biological rather than volitional model of causation. For people who are afraid of being

stigmatized as vain (as with AN or BN) or as having no willpower (BED), an eating disorder diagnosis may remove barriers to seeking treatment (Stewart et al. 2006).

Less blame and stigma for families. The idea that eating disorders are genetic also relieves the self-blame of families, enabling them to make positive changes that help the affected person recover. Parents who have a child with a mental illness tend to blame themselves, “What did I do [or not do] to have caused this?” (Austin and Honer 2007). Anecdotal evidence supports the idea that families of people with eating disorders respond positively to the idea of a genetic influence: it reduces anxiety and guilt for parents who felt they must have done something wrong, allowing families to conclude “If I didn’t cause it then I can help it go away” (Flanagan 2008, personal communication). A woman who recovered from AN expresses it this way: “Perhaps the impulse to blame, in fact, diverted essential energy and attention from the real business of recovery. Mothers who subscribed to *Vogue* might play a hand in some cases of anorexia, but according to Strober they no more ‘caused’ the problem than chilly temperatures caused pneumonia. Nor did anorexics ‘choose’ this infuriating and dangerous behavior – any more than they could snap out of their genetics on command” (Liu 2007: 23).

Disorder taken more seriously. Several eating disorders researchers hope that a genetic cause will prompt people to take the disorders more seriously (Bulik 2004, Stewart et al. 2006, Holliday et al. 2005b, Duffy and McElhinny 2007). Stigma in eating disorders works differently than for other psychiatric disorders such as schizophrenia: a perception of greater severity¹⁴ is expected to *reduce* stigma rather than exacerbate it (Mond et al. 2006). Eating disorders may be seen as volitional, and not potentially fatal conditions (Bulik 2004).

¹⁴ Severity was measured with questions about how distressing it would be to have the condition, whether it is seen as a passing phase, whether the respondent ought to be able to ‘get over it,’ and ‘how severe’ the condition is (Mond et al. 2006:523).

For example, in a survey of female college students, about 30% said that they had at least occasionally felt it “might not be too bad” to have AN and 37% at least somewhat admired the ability of someone with AN to lose weight (Mond et al. 2006). Surveys on other psychiatric disorders show that a biological or genetic cause makes them seem more serious or severe (Phelan 2005, Phelan et al. 2002, Phelan et al. 2006, Kuppin and Carpiano 2006). To the extent that eating disorders are contested as illnesses (Giles 2006), severity can validate the existence and importance of the disorder. The widespread perception of severity could encourage more research attention and lead to improved treatment options and prevention. Finally, a simple genetic explanation for an eating disorder might also convince insurance companies to fully cover treatment for eating disorders because the disorder would be seen as “biologically based,” a criterion for coverage by insurance companies in several states (Kaiser Family Foundation 2004).

5.2 Fears about simple genetic explanations

Some of the potential concerns about genetic explanations for eating disorders are listed in the bottom left cell of Table 1.2. Some are negative aspects or implications of the hopes described above.

Increased stigma. Genetic causal attribution could lead to increased stigma, making people reluctant to embrace it. The optimistic predictions of attribution theory are not borne out in studies of stigma for people with schizophrenia; indeed a recent review contends that “biogenetic causal beliefs and diagnostic labelling by the public are positively related to prejudice, fear and desire for distance” (Read et al. 2006: 303). As noted before, in a study of the geneticization of deviant behavior, Phelan (2005) found that when deviant behavior is attributed to a genetic cause, it seems more serious, life-long and chronic (also see Phelan et

al. 2002, Hagger and Orbell 2003, Bennett et al. 2008). Although the perception of severity may assist the goal of medicalization and increased attention, as some hope, the fact that it is internal to a person may also add stigma. Phelan presented vignettes about people with mental illnesses and experimentally varied them so some gave genetic and others non-genetic causal attributions for a condition. This study aimed to compare attribution theory, which would predict reduced stigma by absolving the sufferer of responsibility and blame, with genetic essentialism theory, which would predict increased stigma by making the disorder seem more severe and persistent and making the person seem categorically different from other people. Phelan found more support for the theory of genetic essentialism than attribution theory. Read and Harré describe similar findings in their (non-experimental) survey: endorsement of a biological or genetic cause was correlated with negative attitudes toward mental illnesses (2001). Attribution of behavior or mental illness to a genetic cause suggests an external locus of control even though genetics are internal to a person (Weiner 1985, Maher and Kroska 2002). An inherited disorder also implicates biologically related family members and produces a desire for greater social distance, particularly for younger relatives of those affected (Phelan 2005).¹⁵ Although in the case of eating disorders, the perception of severity may actually reduce rather than exacerbate stigma, these pessimistic findings ought to be taken into consideration.

Hard to reconcile with non-biological treatment. Although the perception of severity may draw positive attention toward eating disorders, it may lessen confidence in the treatments commonly available for eating disorders. People in treatment may struggle with

¹⁵ Other concerns arise for disorders that are linked to specific genes, if these genes are also linked to more stigmatized disorders. Genotypes may give rise to multiple outcomes, known as “multifinality” and “pleiotropy.” This is the case in smoking behavior; genes identified for it are also implicated in addictive behavior and psychiatric disorders thereby conferring additional stigma (Caron et al. 2005, Shields et al. 2004).

how to reconcile the idea of a genetic cause with the psychotherapeutic treatments they are receiving. If they feel therapy is helping, a genetic cause may seem implausible or threatening. Survey respondents who attributed mental illnesses (schizophrenia or depression) to genetics found biological and hospital-based interventions more plausible than other treatments (Phelan et al. 2006 reporting on the 1996 GSS and the “Genes, Disease, and Stigma” vignette experiment, Kuppin and Carpiano 2006). Most troubling, in Phelan et al.’s vignette experiment, respondents who were told that a mental illness had a genetic origin were more pessimistic that mental health professionals could help (2006). Given that most treatments for eating disorders are non-biological therapies – cognitive-behavioral, family – a “gene for” eating disorders renders these implausible. It would be harder to justify the effort and expense of engaging in such therapies if the “real” cause were genetic and neurochemical. Pharmaceutical treatments targeted to a gene-related biological pathway would be most logical (Striegel-Moore and Bulik 2007). Some may even hold out for gene therapy. But clinicians rely heavily on non-biological methods of treating eating disorders. My study will uncover whether respondents think about these issues and whether it might affect their commitment to particular treatments. There is a danger that the primarily non-pharmaceutical treatments for eating disorders will be perceived as ineffective by virtue of a genetic attribution for a disorder. The worst case scenario is that people with eating disorders, their families and insurers forego non-biomedical treatments that might have helped.

Fatalism about prognosis. A related concern among critics is the idea that a genetic cause may lead to determinism and fatalism about long-term prognosis (Alper and Beckwith 1993). Phelan found that survey respondents (non-patients) who were given a partially or completely genetic explanation for “Anne’s” mental illness were more likely to endorse this

statement: “In your opinion, Anne will probably continue to have problems like the ones I described for her whole life” (Phelan 2005: 313). Mehta and Farina (1997) found that a biological cause made a disorder seem more fundamental, real, and immutable (see also Corrigan and Watson 2004). Similar findings apply to physical illnesses (Senior et al. 1999, French et al. 2005). This is a problem because if a complexly caused disease is seen as inevitable it will discourage action that might have helped, including the biological and hospital-based treatments that seem most plausible. A woman recovering from AN wrote that “powerlessness is the most dangerous thing an anorectic [sic] can hear. It grants license, exoneration” (Hornbacher 2006: 131). However, people with lifelong genetic conditions and those with a high probability of becoming sick do not necessarily become fatalistic about treatment, as they may be able to manage the condition even if they cannot cure it (e.g., Novas and Rose 2000, Skinner et al. 2003, Kenen 2007) or to maintain uncertainty about what the future holds (Whitmarsh et al. 2008). In addition, knowing about a genetic predisposition may only change perceptions of *how* to control a disorder, not whether control is possible (Marteau et al. 2004). On the other hand, AN is not necessarily experienced as a problem that needs controlling or managing: it may feel like a part of the self rather than an unwanted “condition” or “disorder” (Rich 2006, Hornbacher 2006). If a genetic cause connotes permanence it may discourage people from working on it.

Reductionism of self, behavior and will. The perception that genes are the source of behaviors and the self challenges everyday ideas about human identity and agency. In the case of eating disorders, a simple genetic model implies that a gene directly causes the behaviors of bingeing, purging, and restricting. If people with eating disorders are not familiar with the complex model of genetic causation, they may wonder how a gene can

cause a behavior and what their own role in the behavior would be. They may doubt their ability to go against genetic “will.” Even if genes are seen as the source of personality traits that in turn produce behaviors, it is still challenging. Genetic explanations for behavior may suggest a genetic “essentialism” that “reduces the self to a molecular entity, equating human beings, in all their complexity, with their genes” (Nelkin and Lindee 1995: 2) or “neurogenetic determinism” (Rose 1995). If eating disorders are often experienced as part of the self, and even “a crucial part of self” (Surgenor 2003: 711, Rich 2006, Hornbacher 2006), and if eating disorders are genetic, perhaps the self will be seen as genetic too. As Kendler notes, a physical condition is something that you *have* (“I have allergies”) whereas a personality trait is something that you *are* (“I am introverted”). Psychiatric conditions are not clearly one or the other: “Am I schizophrenic or do I have schizophrenia?” (Kendler 2006, Parens 2004). The reductionism of a genetic explanation for behavior, personality and self may be disturbing and contribute to fatalism.

Loss of individual responsibility. A genetic causal attribution could also affect one’s sense of responsibility. Although the relief from blame and responsibility is clearly desirable for reducing stigma, there are potential downsides. As one woman recovering from both AN and BN wrote of her recovery, “It is not a sudden leap from sick to well. It is a slow, strange meander from sick to mostly well. The misconception that eating disorders are a medical disease in the traditional sense is not helpful here. There is no ‘cure.’ A pill will not fix it, though it may help. Ditto therapy, ditto food, ditto endless support from family and friends. *You fix it yourself*” (Hornbacher 2006: 284, emphasis mine). Kendler notes that after publishing about the genetics of alcoholism he received a letter that asked, “How dare you give my Aunt Diana yet another excuse to say that drinking is not her fault!” (Kendler 2006,

also see Caron et al. 2005 on nicotine addiction). In court cases, defense attorneys offer genetic explanations to change the jury and judge's sense of the defendant's culpability (Farahany 2006). If genes seem to produce behavior directly, or indirectly when mediated by personality, how does one understand behavior aimed at getting better? Is that genetic too, or does a non-genetic "part" of the self exert control over the genetic "part"? Knowing about one's own genetic risk for genetic disorder appears to impose new perceived responsibilities (Hallowell 1999, Novas and Rose 2000, Arribas-Ayllon et al. 2008); there is evidence that this logic may be extended to genes for behavior as well, such that people are held responsible for controlling their own personality tendencies (Condit et al. 2006). Might people wonder whether the very ability to gain such control is also "genetic"? These questions are philosophical and recall discussions of free will; they are also folk theories of the self and agency. People who have rejected a medical (biological and pathological) explanation of their eating behavior may accept a genetic explanation of their personality and behaviors.

Less attention to cultural causes. Simple biological and genetic explanations could additionally divert attention from important environmental factors, including enabling and constraining conditions. Cultural explanations of anorexia and other eating disorders usually focus on thinness as the ideal body type for women (Striegel-Moore and Bulik 2007). Those who explain eating disorders using a biomedical model often recognize that socio-cultural factors are important to explain why eating disorders are more common in Western countries, among higher economic classes, among women, and at certain historical periods (Brumberg 2000, Striegel-Moore and Bulik 2007). Eating disorders have been called "culture-bound syndromes" (Keel and Klump, Swartz 1985, Banks 1992), meaning that they appear only in

some cultures and not others, and anthropological studies demonstrate the influence of Western cultural influences and media on eating disorder symptoms in Belize (Anderson-Fye 2003) and Curaçao (Katzman et al. 2004). The greater prevalence of AN and BN among women compared to men, and the increased incidence of eating disorders in recent decades also provide evidence for culturally based explanations (Striegel-Moore and Bulik 2007). Genetic explanations of eating disorders could conceivably threaten cultural and political explanations by shifting attention away from them (Lippman 1992, Duster 2003). The second quotation from the beginning of this paper illustrates the potential of genes to exculpate culture and power: "...I don't like all this 'explaining away by genetics, etc.' stuff at all. I'd be willing to bet the vast majorities of Eds [sic: cases of eating disorders] are birthed by our cultures hatred of all women weighing over 100 pounds. The genesis lies here. The blame goes here" (Kim 2006). Environmental risk factors, ranging from exposure to images of very thin women in the media to participation in cheerleading or dancing, would be overlooked in a simple "gene for" model. This might inhibit individual and collective action to change social and cultural contributors to the disorder.

Depoliticization of eating disorders. Feminists connect the thin ideal and other cultural messages about women's bodies to capitalism and patriarchy to explain eating disorders. "Anorexia is an attempt to resolve at the level of the individual body the irreconcilability of individuality and femininity in a bourgeois patriarchal culture" (MacSween 1993: 252). According to one woman who recovered from BN, whose essay was included in an edited collection of "voices from the next feminist generation" (Findlen 1995), "gaining weight and getting my head out of the toilet was the most political act I ever committed" (Chernik 1995: 81). "Diet culture" disempowers women: "we keep on

shrinking, starving away our wildness, our power, our truth” (Chernik: 83). Feminists and others would likely resist genetic explanations as a way to obfuscate the true cause, disempower feminist critics, and turn a political problem into a personal affliction (Orbach 1986, Wolf 2002, Bordo 1993, Brown 1985, Thompson 1994, Chernik 2001). Genetic explanations would likely be seen as a new way in which biology is used to justify gender inequality (Lorber 2004 [1993], Fuchs-Epstein 1988, Fausto Sterling 1992). If a labeled disorder is in fact a reasonable response to a problem of living (Thompson 1994, Karp 1996, Szasz 1960), the emphasis ought to be on the problem of living rather than the individual’s genes or biology. As Karp put it in his study of depression, “Medicine nearly always interprets illness as a reflection of individual physical pathology and rarely as *a normal response to pathological social structures...* [hyping] medication as the cure for depression [would be] both scientifically arrogant and politically retrograde.” (Karp 1996: 80, italics in the original). If Westerners, particularly Western women, endure “pathological social structures” without recognizing them nor realizing that change is possible, genetic explanations would serve to naturalize such structures, divert attention from alternatives and inhibit social movements to change them (Eagleton 1991, Berger and Luckmann 1967).

Family problems blamed on individual biology. Simple genetic explanations could also divert attention from the family (though not its genes) and prevent a full understanding of an individual’s illness. Genetic explanations for mental illness tend to remove blame from parents (Phelan 2005, Austin and Honer 2007), which is clearly positive in ways discussed earlier. However, in an internet chat with the author of the Newsweek cover story that focused on the genetics of AN (Tyre 2005), a participant recovering from AN expressed concern that genetic explanations could absolve the family:

I know that these articles on anorexia are focusing on biological predispositions to it. ... It bothers me because it seems that parents could read this and feel relieved [sic] of any responsibility and not examine their [sic] own behaviors. ... [S]houldn't parents not just focus on "fixing" the child and seeing the child as the problem but also to examine that maybe the child is an indication of a larger family problem? ... I'm just afraid [sic] these articles will foster misunderstanding and further the stigma that the child is somehow "defective" all on his/her own" (Wocester 2005).

A simplistic model of genetic causation could discourage parents and family members from examining their own behaviors and participating in family therapy. (Parents might blame themselves for passing along the genes, however.)

Rejection of any explanation involving genetics. For those who are already convinced of and committed to social and cultural causal models, a simple genetic model runs the risk of provoking a reaction against models that include biology or genetics in any way. The oversimplification of the "gene for eating disorders" model would prevent respondents from recognizing that genetic causal models for eating disorders also include environmental causes. Genes could thus be interpreted to mean "NOT environment," taking on their meaning from what they lack or ignore.¹⁶ The simple inclusion of genes in a causal model could, if oversimplified, prompt a backlash against genetic explanations generally as implausible and reductionistic. For those who hope that genetic research will lead to life-saving discoveries, such a backlash would be a negative consequence of the oversimplification of genetic causality.

5.3 Hopes and fears about complex explanations

The hopes and fears about simple genetic explanations may or may not apply to complex genetic explanations. Many of the fears about medical, genetic and biological

¹⁶ This idea is illustrated by Swidler's (2003) observation of how giving flowers to secretaries during Secretaries Week can be semiotically recoded to diverge from the meaning desired by florists: that the employer values the secretary. "A national secretaries union, publicizing a "Bread, Not Roses" campaign, might shift the code, pressing bosses to enter labor negotiations during National Secretaries Week and making flowers without a raise a sign of contempt." (164).

explanations of behavior center around reductionism: reifying complex human behavior to a syndrome or disorder, ignoring causes other than individual-level biological ones, casting doubt on individual agency and responsibility. These concerns may not be such a problem if genetic factors are only part of a “multifactorial” explanation.

One possibility is that a complex, partly genetic explanation would produce effects that are “in between” those of a fully genetic or non-genetic explanation. The same advantages or disadvantages might hold for complex explanations, but to a lesser extent than for simple explanations: somewhat less alleviation of blame, sense of severity, or fatalism, for example. However, Phelan (2005) found that when survey respondents were presented with a partly genetic explanation, as opposed to a fully genetic or non-genetic explanation of a vignette character’s condition, it did not produce “part” of the effect of a fully genetic explanation. That is, a partly genetic influence was not always in between the non-genetic and the fully genetic (but she does not report which way it tended to go). As Phelan put it, “A partly genetic explanation does not simply work like a weaker dose of a purely genetic explanation. This finding, combined with the fact that the partly genetic explanation was the one most likely to be accepted by respondents, suggests that the public’s ideas about gene-environment interactions should be studied” (Phelan 2005: 317).

Some argue that a multifactorial model might actually increase the perceived importance of environmental factors. Shostak argues “that the promise of the study of gene-environment interaction is in its direction of scientific, biomedical, and public health attention simultaneously inward, towards the gene/genome and the interior of the body, *and outward, towards particular practices, places and the exposures they contain and enable*” (Shostak 2003: 2328, emphasis mine). The idea that genes only matter in certain

environments could draw more attention to those environments, in part because the effect of those environments are newly “visible at the molecular level” (p. 2338). Alternatively, gene-environment interaction models may enhance the importance of both genes and environment. The interaction need not be theorized in a way that prioritizes genes; models can have environmental factors as independent variables and genotypes as moderators of the relationship with phenotype (e.g., Figure 1 in Caspi and Moffitt 2006).

Some hope that if a complex genetic explanation is the most accurate model for disorder causation, it will prevent frustration and promote the best outcome for people with eating disorders. Action would be taken only in the areas where it is most effective, responsibility would be apportioned only where justified. A person with an eating disorder could not only be absolved of blame, more knowledgeable about risk factors, and better positioned to avoid relapse. With new genetic knowledge may come new obligations and responsibilities (Novas and Rose 2000). Genetic counselors could help explain the meaning of genetic influence to affected people (though such counseling is not usually available) (Austin and Honer 2007).

It is also possible that in the future, consumers will be helped by information about their genetic risk profile. There is no identified “gene for” eating disorders, nor (most likely) single genes for the eating disorder phenotypes as currently constructed, but profiles based on multiple genes may be developed. Such genetic profiling is already available for some complex conditions through direct-to-consumer marketing, complete with interpretations of one’s level of risk (Harmon 2007, Gollust et al. 2003). It is not likely that physicians will offer predictive testing for psychiatric conditions because it will have little power to predict outcomes (Hamer 2006, Feldman 2006). If multiple genes are identified as causal factors for

mental illnesses, they are more likely to be useful in understanding the etiology of diagnosed cases and identifying the most appropriate treatment. For example, if there are two theoretical pathways to an outcome, one involving a genetically-based problem with dopamine and the other with serotonin, a genetic test could identify which is relevant for a specific patient and point to the relevant pharmaceuticals (Hamer 2006). Yager envisions that, in the future, predictive genetic tests could warn those at risk away from particular high risk environments, such as gymnastics teams (Yager 2004). Although some environmental causes may not be any easier to change than genetics (Parens 2004), others could be ameliorated through individual or collective action. There are practical benefits to the complex causal model, if it is an accurate portrayal, though some of the benefits are contingent on identifying and replicating associations with actual genes.

Complex genetic explanations harmonize with common sense understandings of the importance of both nature and nurture. A nationally representative survey taken in the U.S. in 1997 found that when asked about diverse conditions and traits, a plurality of people said that most of them were “somewhat” determined by genes, as opposed “not at all,” on one end of the spectrum, or “completely” and “mostly” on the other end. Eating disorders were not among those listed, but some that were – alcoholism, mental illness, neurotic behavior – may be similar enough to provide some guidance about what might hold in the case of eating disorders (reported in Singer et al. 1998). Similar results were found for a range of mental illnesses in the General Social Survey in 1996, suggesting that a multifactorial model makes sense to many people (Link et al. 1999). These survey findings are consonant with qualitative studies about genetic causation for a variety of health conditions (Condit et al. 2006, Condit 2004, Parrott et al. 2003, Lock et al. 2007). Lay people view causation as having both genetic

and environmental components, rather than seeing one or the other as the sole cause. However, people who can do this for cancer or heart disease may not do so for eating disorders, given that survey respondents do not typically endorse biological causal factors for eating disorders (Stewart et al. 2006, Holliday et al. 2005, Quiles et al. 2007). Some observers note that laypersons may acknowledge genes and environment but have trouble integrating them into a single model. Condit found that “participants cite examples of people who smoked and did not get cancer, or of people who have a family history of cancer but do not get sick, or who do get sick and do not smoke. No participants account for such variation by noting the interaction of genes and behavior or environmental factors. No one says, for example, ‘she smoked and didn’t get cancer, but that is probably because she did not have a genetic susceptibility.’” (Condit et al. 2009, also see French et al 2005). Yet it would not be surprising to hear someone say, “He smoked all his life and didn’t get cancer – I guess he has good genes” (Perrin 2007, personal communication). Recently an eating disorders survivor put it this way: “Here's what happens: I'm highly sensitive and I'm a perfectionist. If you take a person like me and if you flood me with fashion model [images], then culture plays a really strong role in triggering an eating disorder that might otherwise be latent” (Gura 2007). I think this is an accessible account and am curious to see if it is widely shared.

Despite evidence that a combination of both genetic and environmental influences makes sense to non-experts, there is nevertheless reason for concern about the increasing complexity of expert discourse. This is also true with regard to predictive testing for complexly caused conditions. As Condit writes, “[g]iven the difficulties involved in explaining the implications of a single genetic test, communicating the implications of having, for example, two risk-conferring alleles and two non-risk conferring alleles for

hypertension or skin cancer or osteoporosis will certainly be challenging” (Condit 2007: 819). In addition to genetic risks, there are also environmental triggers, risk factors, and protective factors. For psychiatric conditions, unlike skin cancer or osteoporosis, genetic susceptibility may be theorized in terms of cognitive and personality traits, adding further complexity. Information and even visual renderings of genetic influence on psychiatric disorders presented in scientific articles are daunting for nonspecialists, even those with a post-graduate education (e.g., Figure 2 in Gottesman and Gould 2003), and genetic discoveries are expected to change diagnostic definitions for mental and physical illnesses. Yet many articulate a need for the public to be well informed enough to provide a challenge to expert authority, to point out “when the emperor has no clothes” (see an overview in Kerr et al. 2007). Sociologists of medicalization have been concerned about the potential expansion of social control through medical intervention: interventions into certain behaviors are often justified in the name of health (e.g., Conrad 2007). But even when there is consensus that a condition is indeed medical, professional commitments and disciplinary interests may motivate causal accounts and ought to be tempered by public oversight, which requires understanding of what experts are saying.

6. OTHER POSSIBLE RESPONSES TO GENETIC EXPLANATIONS

The hopes and fears about simple and complex genetic explanations for a complex multifactorial disorder, outlined on Table 1.2 and described above, do not exhaust all possible reactions to the geneticization of eating disorders. They primarily reflect the thinking of academics and clinicians. There is reason to believe that respondents will add additional variation beyond that described in Table 1.2. Their characteristics and experiences, such as education and length of illness, may have a bearing on how they think

and talk about eating disorders. I suggest three additional points to consider: (1) people who do not think of eating disorders as psychiatric disorders or even as problems will likely think of genetics differently, (2) people may make sense of genetic causation in ways that are not adequately captured by the terms “simple” and “complex,” (3) people may see implications for the genetics of personality beyond those suggested by experts, particularly because eating disorders can feel agentic and empowering to the person experiencing them.

First, those who do not see eating disorders as medical problems may use genetic “cultural tools” differently than those who do. Table 1.2 reflected hopes and fears for the geneticization of a condition that is understood by experts to be a multifactorial psychiatric disorder. I focus now on anti-medical perspectives from people with AN and the lay public, not feminist anti-medical perspectives described earlier. Some people with AN do not see themselves as having a problem or medical disorder. As Fairburn and Harrison note (2003: 407), “...in anorexia nervosa there is a sustained and determined pursuit of weight loss and, to the extent that this pursuit is successful, this behaviour is not seen as a problem. Indeed, these patients tend to view their low weight as an accomplishment rather than an affliction...” (citing Vitousek et al. 1998). People with “typical” AN believe they are too fat, whereas those with “atypical” AN assent to a medical definition because they realize they are too thin and would like to control their symptoms (Yager 2004 citing Strober et al. 1999). Thus, those with “typical” AN may not see their eating behavior as a medical disorder or at least express confusion about it.

Studies show that people with eating disorders often resist medical understandings (Giles 2006, Fox et al. 2005), even when they are in treatment (Warin 2004, Rich 2006) or recovered (Shohet 2007, Button and Warren 2001, Weaver et al. 2005). In the internet-based

pro-anorexia or “pro-Ana” movement, participants embrace anorexia as a lifestyle that provides control, stability, and a sanctuary to cope with social, psychological, and cultural problems (Fox et al. 2005). According to another study of pro-Ana website users, the non-medical stance is not necessarily consistent: “even users themselves are unsure as to whether they are ‘celebrating’ their EDs, whether anorexia is a life-style choice, a medical condition, an illness, or a positive or a negative experience” (Giles 2006: 464). Even those who do try to preserve an anorexic identity are conflicted about it, feeling both empowered and potentially destroyed by it (Rich 2006). Unlike disease advocacy groups that strive to convince others of the medical status of disorders like fibromyalgia and environmental illness (Barker 2002, 2008, Kroll-Smith and Floyd 1997), pro-Ana groups seem to argue for the *non*-medical status of the behaviors. In Giles’ study of pro-Ana websites, contributors to the site discussed medical diagnoses without pathologizing them. For example, one contributor with the residual diagnosis EDNOS (Eating Disorder Not Otherwise Specified) lamented being “too much of a failure to earn a concrete diagnosis” (2006: 470). Moreover, it was common to distinguish between real and “wannabe” anas or “mias”. The very existence of “wannarexics” – teenagers who strive or claim to have anorexia (Bauman 2007) – underscores the prevalence of non-medical views. Others may reject the classification of their eating behavior as a disease or mental illness but nevertheless feel that it is a problem (Button and Warren 2001).

Genetic explanations for a behavior may carry a different meaning if that behavior is not pathologized.¹⁷ How would someone who somewhat embraces her eating disorder view

¹⁷Although my study links geneticization and medicalization, respondents may not link genetic explanations to medical statuses. As noted earlier, genetic origins can be used to normalize and naturalize behaviors, thereby de-medicalizing them as in the case of homosexuality (Conrad 1997: 147). Sankar and colleagues likewise found that genetic causal attributions had a variety of positive and negative consequences. For example,

information about genetic causal factors? She might resist a genetic cause if it seems to pathologize her behavior. Others might accept a genetic explanation for AN without perceiving pathology; genetic explanations for height and intelligence do not suggest pathology in and of themselves. Still other questions are raised: if anorexia is perceived as an achievement, how will genetics be incorporated? Whether a genetic origin medicalizes behaviors or not, it would demote their “achievement” to something programmed rather than chosen (Kendler 2006, Press 2006). This may particularly trouble those who view their disorder as a precious source of control (Bruch 1994), or a form of ascetic religious practice to control the body (Banks 1992) because the genetic body would be in control of the self and its behavior rather than the reverse. It seems possible also that such people might medicalize bingeing and purging but not food restricting, which seems more controlled, “pure,” moral, and of higher status to some people with eating disorders (Burns 2004, Giles 2006, Hornbacher 2006).

In a medical context, patients’ disagreement about whether they have a psychiatric disorder is typically deemed denial, a lack of “insight” and symptomatic of the disorder (David 1990).¹⁸ Estroff et al. state, “Despite the dominance and authority of biomedical paradigms..., the nature and meaning of psychiatric disorder remains contested, particularly by those so diagnosed.” (1991: 332, Read and Harré 2001). As a sociologist I am more

deafness due to genetic causes could provoke envy by those whose deafness was due to other causes, because genetically deaf people had deaf family members who were supportive and understanding of their condition (Sankar et al. 2006). In the writings of clinicians, scientists, and advocates for eating disorders, the concept of genetic origins supports a medicalized understanding and is largely assumed to benefit patients. Genetics may signify something quite different to respondents, as might medicalized understandings more generally.

¹⁸ “Lack of insight” is arguably quasi-medicalized through application of the term “anosognosia” (Estroff 2005, Albee 2004). Lack of “insight” denies a place for alternative views and contestation: “it contains within it an immediate value judgement: insight is either full/good or lacking/poor, with the implication that the latter is always inferior or undesirable” (White et al. 2000: 501).

interested in how people talk about the disorder and will treat alternative views of medicalization as contestation rather than as symptoms of illness.¹⁹

Second, interpretations of genetic influence may go beyond the simple or complex models outlined in Table 1.2. Respondents may understand genetics in other ways consistent with lay models identified in other studies (Henderson and Maguire 2000). For example, they may interpret heritability to mean the percentage of a person's behavior that is due to genes, and therefore how culpable the person is (Farahany 2006). They may interpret it as the percentage of cases due to genes, rather than the percentage of the variance explained by genes for a specific sample, leading to the conclusion that cases of eating disorders are either solely genetic or solely environmental (Goldstein 2006, Poyastro 2007). While researchers expect that “[s]ome proportion of individuals may have a highly genetic form of AN, some a highly environmental variant, and, in others, AN may result from interactions between genetic and environmental influences” (Bulik et al. 2007: 265), the above lay interpretation would be a misunderstanding. It is also possible that people will not find genetic factors salient or plausible based on their personal or family history (Shiloh 2006, Walter 2004, Cappella et al. 2005). Some may alternate between wholly genetic and wholly environmental explanations depending on the context of the conversation, and their tolerance for cognitive dissonance. Condit found that people grappling with gene-environment models sometimes alternate between the two, perhaps reflecting two unintegrated “separate discourse ‘tracks’ or neural networks” corresponding to gene discourse and environment discourse (Condit et al. 2009; 731).

¹⁹ However, I will not go so far as to conceptualize treatment as a form of social control or surveillance (Vogler 1993, Bell 2006) though certainly the “micro-politics” of therapy and involuntary commitment are important (Surgenor et al. 2003, Surgenor 2003). Although such an emphasis on social control and power is compatible with the theory of medicalization, I am more concerned with how the condition is defined by patients and how those definitions relate to their views of appropriate treatment.

Third, people with eating disorders might generalize geneticized understandings of personality and behavior in the context of a mental illness to normal personality and behavior. They may be aware of the continuum between “normal” dieting or body concerns and “abnormal” eating disorders (Haworth-Hoepfner 1999). If they link the genetics of personality to eating disorders, they may also be aware that others with that personality type do not necessarily have an eating disorder. My study could assess lay understanding of the following intriguing prediction by Conrad:

Recent discussion of ‘shadow syndromes’ (Ratey and Johnson 1997) contend that quirky behaviors may actually be mild mental illnesses that are tied to genes. Here illness and behaviors are seen on a continuum: one or two altered genes give you a little disorder; perhaps three or four create a serious personality problem; and seven give you a full-blown illness. If geneticists found actual evidence to support the ‘shadow syndromes’ conception, it is possible that the psychiatric net would widen and medicalization increase. (2000: 326).

This idea is related to endophenotypes (discussed earlier) and there is strong evidence for it in autism research (e.g., “broad autism phenotype,” in which parents of autistic children display autistic-like behavior, Losh and Piven 2007). Respondents might geneticize the personalities of others, especially family members who are genetically related to them. They may identify personality traits and medicalize them, perhaps to reduce the distance between themselves and those considered healthy or normal. Geneticists have investigated the heritability of the “big five” personality traits (OCEAN: Openness to Experience, Conscientiousness, Extraversion, Agreeableness, and Neuroticism) (e.g., Loehlin 1992), but how do lay people respond to the idea that genes are responsible for their personalities? It seems unlikely that many lay people have thought about this issue deeply. Individuals with eating disorders are more likely to have done so and are therefore an interesting population to study.

7. SUMMARY OF CHAPTER 1

The main questions that will be examined in this dissertation are as follows. Do respondents interpret genetic influences to suggest more medicalized conceptions of eating disorders? Do respondents whose thinking is already more medicalized find genetics more appealing and plausible than other respondents? What do respondents see as the positive and negative implications of the idea that genes play a role in eating disorders, and do these correspond with those identified by professionals and academics (summarized in Table 1.2 above)? Finally, how do respondents imagine genetic influence, and how do they combine it with other influences, if they do?

This study has both theoretical and practical applications. It contributes to medicalization theory by addressing the understudied idea of heightened medicalization and by defining its dimensions. If medicalization is a continuous process, it may well continue even after a diagnosis has been included in official medical classifications such as the DSM. An illness that is contested prior to inclusion in the DSM may continue to be contested afterwards. This appears to be the situation for many psychiatric disorders, judging from the lack of parity in insurance coverage for mental illnesses and widespread non-medical perspectives that persist despite campaigns to frame psychiatric disorders as “brain diseases.” In my proposed reconceptualization of medicalization theory, if a psychiatric disorder is described as biological it will heighten the medicalization of that disorder. My study will illuminate how people with AN or BN grapple with the idea of genetic and other biological and non-biological causes, in particular how these relate to medicalized and non-medicalized concepts of their disorder.

My focus on people diagnosed with a disorder and how they use medical and genetic concepts as cultural tools is also innovative for studies of medicalization. I draw on the assumptions of cultural sociology and symbolic interactionism to theorize medicalization at the individual level. Medicalization has tended to assume a top-down model of cultural transmission, in which professionals propagandize medical definitions and patients accept them. Recent thinking in cultural sociology and classic ideas from symbolic interactionism, emphasize the importance of individual agents in the construction of reality, including medical reality. My interviews with current patients and recovered people sheds light on how individuals use medical, biological and genetic concepts to account for their eating disorders and related behaviors.

My findings will contribute to a growing literature about how lay people conceptualize complex genetic causality. Not enough is known about how people interpret genetic influence when it is one of many factors. Some may ignore them or find them irrelevant, but they may nevertheless have a concept of what genetic influence means. For those who use genetic concepts (either with or without prompting from me), there are at least two genetic narratives they may draw upon, corresponding to the simple and complex models of genetic causality described earlier (Figure 1.1). One is the simple deterministic model: “I had the gene so I got the disorder.” Another is the more complex susceptibility model in which both genes and environment matter: “I was susceptible and then I was exposed to X so I got it.” Both models are simple compared to many sociological approaches, including life course and symbolic interaction, but their differences are crucial, as genes are often thought of in simple Mendelian terms. Of particular interest with eating disorders is the idea that media imagery and other sociocultural factors contribute to the disorder. How will

respondents make sense of these many potential factors? Will they reject genetic causality, sociocultural causality, or find ways to integrate the two?

This study also breaks new ground by examining perceptions of complex genetic causality for eating disorders, which has not been done before. Medical explanations of genetic influence for a multifactorial physical disorder are already complex compared to those for a genetic disorder. For a multifactorial psychiatric or behavioral disorder, the symptomatic behaviors are often assumed to be under the person's control, such that additional questions about free will or agency, individual responsibility and personality arise with such disorders. How do respondents think about the idea of a genetic source or influence for behavior that others, or even they themselves, perceive to be volitional? Respondents may use genetic cultural tools to manage their identities by lessening responsibility, blame and stigma. Those who do not accept a medical definition of their behavior may reject genetic accounts as pathologizing that behavior. The negative and positive implications of genetics as expressed by clinicians, academics, and others may or may not be relevant to diagnosed people, and this cannot be known without empirical research. My sample design, described in Chapter 2, includes variation in recovery status and diagnosis, which may also help to generate hypotheses about individuals' endorsement or rejection of medicalization and geneticization of AN and BN.

In addition to the theoretical applications already described, my findings will have practical applications. They may be useful to clinicians and others who communicate genetic information. In order to communicate effectively with patients, clinicians will find it useful to know how their patients understand and feel about genetic factors. As noted by Crisafulli and colleagues (2008), "prior to beginning any wide scale campaign highlighting the role of

genes in AN, it is important to consider potential unintended consequences of doing so and to present the message in a way that safeguards against them” (p. 338, also see Spriggs et al. 2008). Health messages are more successful if they can build on existing knowledge and address beliefs about illness (e.g., Parrott et al. 2004, Shiloh 2006, Leventhal et al. 1984). In the future, clinicians will be increasingly using genetic information in their practices, whether simply mentioning genetic risk factors or testing genes in order to target pharmaceuticals (APA 2008). Because discoveries about the genetics of eating disorders are likely to be covered in the media, even those providers who do not subscribe to a biomedical model will need to address genetics, if only to answer their patients’ questions. People with eating disorders, whether diagnosed or not, will likely hear about genetic risk factors through the media, internet and patient-oriented literature. With regard to practical applications, my goal is to identify concerns or understandings that could affect beliefs and actions related to treatment and recovery.

CHAPTER 2

SAMPLE AND METHODS

So little is known about how people with eating disorders think about genetic causality that exploratory work is needed first, and such work is best done using qualitative interview methods. Open-ended questions enable respondents to use their own words and categories rather than being restricted to a set of response options that may not apply. A flexible interview guide permits respondents to speak at length about relevant topics unanticipated by the interviewer. My research questions can thus be answered best using qualitative methods.

1. SAMPLE

I interviewed 25 women who were currently in treatment for AN or BN and 25 who had recovered (self-reported). Those undergoing treatment were recruited through one hospital-based eating disorders program, and those who had recovered were recruited via a university-based informational mass email system. The sample was designed to compare those who have recovered with those who have a current diagnosis, and those with different diagnostic histories. To maximize comparisons among respondents, I assessed severity by asking about current or past experience with inpatient, partial hospitalization, or residential treatment for their eating disorders.

1.1 Rationale for sample design

I divided the sample between currently diagnosed and recovered people because the experience of recovery could make a simple, deterministic model of genetic causality implausible. If there were a simple, deterministic “gene for eating disorders” how would recovery have been possible? Compared to people currently diagnosed with an eating disorder, I anticipated that recovered people would be more likely to articulate a complex model of genetic causality, in which environmental factors interact with genetic propensity and the disorder can be overcome. It was also possible that recovered people would simply conclude that they must not have had “the gene” at all.

Because severity could affect whether respondents think a genetic causal explanation applies to them, and therefore whether they grapple with complex or simple genetic causal accounts, I asked all respondents about hospitalization (inpatient or partial) for an eating disorder or attendance at a residential treatment center for eating disorders. This proxy for severity was more relevant to AN than BN; people with severe cases of BN may not receive insurance reimbursement for such programs because their bodyweight is not dangerously low. The subsample of currently diagnosed women was more likely to have experienced such programs compared to recovered women (72% and 28%, respectively; see Tables 2.2 and 2.3 in the last section of this chapter).

I sampled for two diagnoses, AN and BN, because different disorders may inspire different ideas about genetic causality. Stigma may be different for people with different disorders. To the casual observer, an emaciated person with AN is already “discredited” based on appearance, whereas a person of unremarkable weight who has BN is only “discreditable” (Goffman 1974). People with different symptomatic behaviors may also

think differently about stigma, as bingeing, purging, and laxatives may seem more deviant or embarrassing than restricting and exercising too much. (However, because bingeing and purging may be part of both disorders, a comparison of diagnoses is not necessarily a comparison of behaviors.) These differences in perception and experience of stigma may have a bearing on how people think and feel about genetic (and medical) explanations and how motivated they are to accept them.

AN and BN occasionally overlapped but were not difficult to distinguish for respondents in my sample. Because current or past diagnosis of AN does not exclude a past diagnosis of BN or vice versa (Tozzi et al. 2005), a neat comparison of the two is difficult to implement. Most (34 of 50) respondents in my sample only reported experiencing one, and those (16 of 50) who either self-reported both, or reported BN but also had a lowest lifetime body mass index (BMI¹) under 18.5, did not differentiate between them enough for me to ask the same questions for each disorder without the interview becoming tedious. A sampling method that insisted on diagnostic purity would have been flawed conceptually because such purity is not characteristic of eating disorders. To take one example, it would be difficult to find a recovered person who had “AN-only,” because she is likely to have been diagnosed with an EDNOS at some point during her recovery as there is no category for “AN in remission” (Bulik 2007).

The comparison of disorders should be seen as a comparison between people who saw their diagnostic histories as “primarily AN” or “primarily BN.” During telephone screening (described further below), I asked if they had ever received the other diagnosis, and

¹ BMI is a measure of body fat based on height and weight and has four categories (Underweight: BMI \leq 18.5; Normal weight: BMI =18.5-24.9; Overweight: BMI = 25-29.9; Obesity: BMI \geq 30). BMI is calculated based by multiplying lowest weight in pounds and dividing by squared height in inches ($703 * \text{weight} / \text{inches}^2$). (NHLBI [no date])

if so, whether one affected their lives more than the other. This simple, subjective assessment grouped people into categories of primarily AN and primarily BN, rather than exclusively one or the other. This screening question did not force a choice between disorders, nor did it restrict the interview to a single disorder. Although the question about whether one disorder has more affected their lives can be interpreted in several ways (e.g., which have they had the longest, which was the most difficult, which has had the most consequences), I left it to respondents to define for themselves.

Sampling for disorder overlapped to some extent with severity, operationalized as time spent in inpatient, residential, or partial hospitalization treatment for eating disorders. Insurance reimbursement and American Psychiatric Association practice guidelines are based in part on bodyweight, such that a patient with low weight is more likely to be given inpatient or partial hospitalization treatment (APAb 2000). Because a criterion for AN is low weight, people with AN are more likely to have received such treatment. Outpatients may have AN, BN, or EDNOS, though only those with AN and BN were eligible for my study.

1.2 Eligibility criteria

To be eligible, respondents with a current eating disorder diagnosis had to be female, 18 years of age or older, and in treatment for an eating disorder. In addition, program personnel verified whether they met criteria for either AN or BN² within the last 6 months and whether they were medically cleared for participation (the latter applies only to inpatients with AN). I focused female respondents for three reasons: males with eating

²To expand the pool of potentially eligible people, I relaxed criteria for both disorders by including the following types of respondents: women who met all criteria for AN except loss of menstruation and women who met all criteria for BN except that binge eating and vomiting or other compensatory behaviors occurred one time per week instead of two times per week. According to the DSM-IV-R, these subjects would have been classified as EDNOS, Eating Disorder – Not Otherwise Specified. Cynthia Bulik guided the choice about which criteria to relax.

disorders are rare enough that they would be difficult to recruit in sufficient numbers to represent them fairly without extending the period of data collection, their experience of stigma is likely to be compounded by the perception of their disorder as “female,” and genetic studies of eating disorders have focused on females. To be eligible, recovered respondents had to be female, 18 years of age or older, must have been diagnosed with AN or BN (or both) at some point in their lives, and must have had no active bingeing, purging or very low bodyweight for the last three years (Von Holle et al. 2008). Because some people can be considered recovered even with consistently low bodyweight or with an occasional lapse into purging, I described all cases of marginal eligibility to an eating disorders expert (Cynthia Bulik) and excluded some respondents.³

Patients in the hospital-based program are normally placed in one of the three “stepped” treatments – inpatient, partially hospitalized, and outpatient - depending on the assessment of the treatment team. In addition, the eating disorders program considers people involved in certain “treatment studies” to be under its care. Individuals who begin as inpatients can progress to partial hospitalization and then outpatient treatment according to the treatment team’s assessment of their needs. There are ten beds for hospitalized inpatients, and their length of stay is 30-45 days, depending on their weight gain and other progress and depending on the limits placed on treatment by their insurance programs. Up to 12 day program patients attend clinic programs Monday through Friday, from 8:15 am to 6:30 pm. Outpatients and people involved in treatment studies come to the clinic for specific appointments. In part because the inpatient and day program are more likely to admit women

³ Three women were never officially diagnosed but were told by a healthcare provider after the fact that they most likely had the disorder. One may never have been told by a healthcare provider. Three women had engaged in symptomatic behaviors or gone below a BMI of 18.5 for a short time during the last three years. Two women were currently at a BMI of 18.5 or slightly below but had been at that weight for decades. One woman was at a BMI under 18.5 because of other recent physical and psychiatric conditions.

with AN, I also recruited women with BN through a treatment study (See Table 2.1, next section). In addition, I recruited and interviewed five men with AN or BN and two women with binge eating disorder (BED, a diagnostic category currently under research investigation) for theoretical contrast purposes, but results from these seven interviews are not presented in this dissertation.

1.3 Recruiting respondents

Data collection was completed in April 2009. I recruited recovered respondents through mass email first (May 22, 2008) and then recruited respondents currently in treatment (early June 2008). Interviews with recovered respondents and those in treatment for AN were completed mid-September 2008; interviews with respondents with current BN were completed in March 2009. (Five men with AN or BN, and two women with BED were interviewed by April 2009.) Interviewing inpatients and partially hospitalized people posed logistical challenges and it was necessary to coordinate with clinic staff to arrange interviews that did not interfere with program activities.

I did not know patients' names unless they contacted me directly or through the clinic staff. I did not have access to patients' medical records or other information about their medical history. I attended three treatment team meetings in order to learn about the clinic, staff and procedures. During these meetings, staff people referred to patients by first name only and provided information about their physical and mental health and recent events or concerns. My purpose in attending the meetings was to learn about clinic life in general rather than to know about specific cases. I did not attend these meetings once I began interviewing patients. My notes on these early meetings did not include patient names so I am not aware if I heard about patients at a meeting whom I later interviewed as respondents.

When recruitment started, I announced my study to clinic staff prior to meetings to explain my study and facilitate recruitment. I did not attend treatment team meetings after having made the announcement in order to avoid violating patient and subject confidentiality by hearing information about them. Patients were informed that I would not attend treatment team meetings about them, nor have access to their medical records, nor discuss their answers with providers or others. I attended a meeting of clinical researchers nearly every week for more than a year to stay in contact with research and clinical staff, which greatly facilitated recruitment.

Respondents from the hospital-based clinic heard about the study at the clinic. These prospective respondents were involved in inpatient, outpatient, partial hospitalization programs or treatment studies for BN and BED. Prospective respondents saw a study flyer posted on a research bulletin board or heard about the study from clinic staff. The flyer included information about eligibility requirements and study participation and the name and contact information of the clinic research coordinator, and later, me (after the research coordinator position was vacated). Clinic personnel gave me names and contact information of eligible women interested in learning more. I then contacted the prospective respondent to screen for eligibility, answer questions, and, review the consent form and arrange for an interview. Interviews with inpatients and day program patients were scheduled with the assistance of the nursing staff and took place on site. I arranged interviews with all outpatients and treatment study participants. Consent forms were hand-delivered to the inpatient and day program units, and mailed or emailed to all others.

Recovered respondents learned about the study through the university-based informational mass email system. This mass email was sent to female students, faculty, and

staff who had not opted out of receiving informational emails (the system enabled filtering by gender). The subject line of the email read, “Have you recovered from an eating disorder?” and the body of the email read, “If so, I would like to hear your ideas about eating disorders and what causes them.” The email included information about eligibility requirements and study participation, as well as my contact information (email address, mail address and telephone number), and a URL (www.unc.edu/~mmeaster/recovered) for a copy of the consent form. Those who were interested contacted me by email, mail or telephone to provide their name and contact information. I contacted prospective respondents to answer questions, review the consent form and arrange for an interview. Respondents who had not been able to download the consent form from the website were mailed or emailed a copy.

There was a risk that respondents would become distressed because of the interview. Some harms were minimized by recruiting currently diagnosed respondents through a treatment program to be sure they had a professional with whom to talk about their feelings. I gave currently diagnosed and recovered respondents a list of resources before beginning the interview so all would have somewhere to turn if they needed support after the interview. Although some respondents cried, none wanted to terminate the interview nor expressed plans of self-harm. Indeed, many respondents seemed to enjoy the interview. Thankfully, the Safety Protocol describing my plans in case a respondent spontaneously reported plans or thoughts of self-harm was never implemented.

1.4 Conducting interviews

I have “outsider” status among eating disordered respondents, which had advantages and disadvantages. I have not had an eating disorder, so there may have been less rapport or trust than for an “insider.” Because the clinic permitted and facilitated recruitment,

respondents may have perceived me as an authority figure connected to the clinic, particularly if I were older than them. As a result, they may have avoided disclosing some attitudes and activities because for fear I would report them to staff (Rich 2007 reports a similar experience). I took care to tell respondents during the consent process that I would not share their information with clinic personnel, except if they had plans or thoughts of harming themselves or others. My outsider status may have caused me to overlook or misunderstand some meanings that are based on shared knowledge, experiences, and even vocabulary (Rich 2007, Gremillion 2003). This disadvantage was also an advantage because it caused me to notice and ask questions about taken-for-granted meanings, thereby transforming them into objects for analysis. In addition, I tried to give respondents the sense that they were experts helping me with my project. This would have been more difficult if I were an eating disorder patient or clinician. I feel that my outsider status as sociologist was preferable to that of a psychologist or social worker because patients would be more likely to tell me things that differed from clinical accounts. These personal characteristics, as well as my female gender, white racial identity, and participation in a doctoral program, probably affected how respondents talked to me, what they emphasized, and what they thought required explanation or not. Differences between interviewers and respondents can increase discomfort and uncertainty at first but they can also open spaces for new understandings as rapport is established (Reinharz and Chase 2003).

Interviews lasted 1½ -2 hours, were digitally recorded (with respondents' explicit permission) and professionally transcribed with identifiers removed. Interviews with outpatients and treatment study participants were held in two private offices on campus to guard the confidentiality of patient participation and the information provided. Scheduling

took treatment needs into account; interviews were not scheduled immediately before therapy in order to avoid tiring patients. For inpatients and day program patients, interviews took place in private locations within the clinic (e.g., activity room, kitchen) during clinic hours of operation. All inpatient interviews took place during 2-hour blocks of free, un-programmed time. Because there was no free 2-hour block of time in the day program during clinic hours, respondents in this program missed up to one hour of occupational or recreational therapy as a result of participation in my study. Recovered people were interviewed at a private location of their choosing, usually one of the two private offices on the University campus.

Participants were paid \$40 cash for their participation. Some interviews lasted up to two hours, resulting in a payrate of approximately \$20 per hour depending on how long the interview was and whether the respondent had to pay for parking. Most interviews were between one and two hours but a few lasted longer. After completing the interview, I gave respondents the EDE-Q (Eating Disorders Examination Questionnaire), a self-administered paper-and-pencil questionnaire assessing eating disorder symptoms.

1.5 Achieved Sample

In Table 2.1 (below), recovered respondents are grouped according to their primary diagnoses (self-reported at time of interview) and respondents in treatment according to their current diagnosis as understood by me at the time of recruitment (with one exception⁴) Several respondents reported experience with both AN and BN but interviews normally focused on one. (See Tables 2.2 and 2.3 in the last section of this chapter for more detailed information about respondents.) Some self-reports of BN were at odds with diagnostic criteria. A few said they had BN but had never binged and a few others reported BN in conjunction with very low bodyweights that could have met criteria for AN. I suspected that

⁴During the interview Claire said she had AN-BP rather than BN and so was reclassified as AN

several who claimed to have a history of both AN and BN may have used the word “bulimia” to mean bingeing and purging; if these behaviors accompany low bodyweight they would be classified as AN according to the DSM-IV. (Confusion about BN was apparent even among people in treatment, as when a visibly emaciated woman on the inpatient unit approached me to volunteer for my study, because she heard I was recruiting people with BN and thought she would qualify.) I did not attempt to “correct” these self-reports. A few recovered respondents had very high scores on their EDE-Q symptom questionnaire; perhaps clinicians would determine some to have a current eating disorder (such respondents may have been helped by information I provided to all respondents about local and national resources).

Table 2.1: Respondents’ Diagnosis, Recovery and Treatment Status (N=50)
Frequency (Percentage)

	Primarily AN	Primarily BN	Total
Recovered	14 (56)	11 (44)	25 (100)
In treatment	13 (52)	12 (48)	25 (100)
<i>Inpatient</i>	9	0	9
<i>Day program</i>	2	2	4
<i>Outpatient</i>	2	0	2
<i>Treatment study</i>	0	10	12
Total	27 (54)	23 (46)	50 (100)

AN= Anorexia nervosa, BN=bulimia nervosa

Of 38 who responded to the mass email recruiting recovered women, 24 were interviewed. Eight were ineligible either because they had never been diagnosed with AN or BN, or because they were not recovered. Two screened eligible but declined to schedule an interview. Four others were never screened; 1 declined before screening and 3 were lost to follow-up. Of the 24 who were interviewed, 9 did not meet every screening criterion but were included because in the judgment of an eating disorders specialist their self-report indicated recovery (described above and in footnote). I included one additional interview, which had been conducted as part of a course in qualitative methods, for a total of 25

interviews with recovered respondents. This respondent went through a second consent process, answered additional interview questions, completed the EDE-Q, and received the \$40 incentive payment.

The total number of potentially eligible people for recovered and currently diagnosed samples is unknown. The recruitment email for recovered women went to 12,717 women's email addresses (4933 employees, 7784 students) and may have been forwarded to others. For people currently in treatment or in a treatment study, there is no estimate of the number of potentially eligible respondents because the number of patients and their length of involvement in the program varies and the eating disorders program does not have these data.

1.6 Reflections on sampling approach

Sampling from a clinic population has advantages and disadvantages. There are many people with eating disorders who have not been diagnosed (Keski-Rahkonen et al. 2007), and many who engage in disordered eating but are below the threshold for a diagnosis. There is evidence that only a minority of people with eating disorders ever seek treatment (Stewart et al. 2006; Fairburn et al. 1996, Keski-Rahkonen et al. 2007). All of my respondents received a diagnostic label at some point and received some kind of treatment, making them different from the universe of people with eating disorders or disordered eating (with one exception, see earlier footnote in the section "Eligibility criteria"). In addition, because clinical populations are likely to include more severe cases than the general population with eating disorders, my sample of people currently in treatment is "biased" toward severity. Such respondents are more likely to have other psychiatric problems, such as depression and anxiety (with AN and BN), and substance abuse (with BN and the bingeing-purging subtype of AN) (Lilenfeld 2004), along with their eating disorders: in psychological parlance, there is

high “comorbidity” in clinic populations. People who have multiple disorders or illnesses are more likely to seek or find themselves in treatment than those with only one. Because eligibility for my study is based on current or past diagnosis, it is more likely to include people with other psychological problems. This over-representation is known as “Berkson’s bias” (Lilenfeld 2004: 184). I did not exclude people with comorbid conditions. An advantage of these biases is that my respondents were more likely to have thought about medical terms and genetic information. This is important to my study, as I am interested in precisely how these medical concepts are used (or not) by people with eating disorders.

Sampling through the program had fewer disadvantages than some alternative sampling strategies. A snowball sample, which starts with one person and uses her social network to locate others with eating disorders, also has disadvantages: respondents who are talking to each other about their eating disorders are likely to have similar ideas. Eating disorders are rare enough that using a sampling frame such as a phonebook would be inefficient, and the condition is sufficiently stigmatized that approaching potential respondents by telephone could feel threatening and jeopardize the openness needed for qualitative interviews. An alternative approach would be to advertise widely in the community or on the internet to reach a more diagnostically diverse population. This approach has disadvantages too: respondents may not have an eating disorder and there is little information about the greater population from which respondents self-selected. In addition, for all of these alternatives there would have been a greater risk of harm to respondents because they may not have been receiving treatment. If some respondents find it difficult or even psychologically “triggering” to talk about the interview topics, it is better that they have a therapist or other clinician readily available to help.

My goal is to generalize theoretically, rather than statistically, from my sample (Weiss 1994). My study will produce concepts that are theoretically generalizable, by which I mean concepts that are useful not only for understanding this sample but also the processes of medicalization and geneticization on an individual level (as opposed to a societal level) and from a lay perspective (as opposed to an expert perspective). I do not make assertions about the distribution of views of genetics within the wider American population, people with eating disorders, people who have received hospital-based care for their eating disorders, nor even people who received care at this particular clinic. My sample is not based on probability and I cannot generalize statistically to a population. Respondents will represent some of the variety of perspectives of clinic patients and those who are similar to them. As noted above, a clinic sample enabled me to focus on people who definitely have eating disorders, who are in the care of a therapist, and who are likely to have grappled with the idea of eating disorders as medical, i.e., as pathological, biological, or both (See Table 1.1, Chapter 1). In the hospital-based clinic from which I drew most respondents, the idea of genetic causal factors was not completely new to patients. According to the clinic director, all patients heard about the idea of a genetic causal factor for eating disorders from patient literature or clinic staff, and according to the clinic psychiatrist, most had already heard about the idea before arriving at the clinic.⁵ Although treatment did not involve individual genetic information, patients recruited from the clinic were more likely than the general population to

⁵ These statements ought to allay concerns about the potential for harm in asking people with eating disorders about genetics. Indeed, according to one of the clinic psychologists, “Patients are more agitated by the food cart arriving than they would be about these questions. I really don’t see anything in the interview that would be distressing, even taking into consideration the fact that our patients often can take offense at benign content. The only negative reaction I have ever gotten when discussing genetics is that some patients say that they think it is irrelevant to their personal experience of the disorder.” This quote and other statements were personal communications to me but are not cited in order to avoid unnecessarily revealing the identity of the clinic.

have heard about genetic causality from their therapists or the patient handbook.⁶ I consider this to be an advantage because people who are familiar with genetic and biological explanations are more likely to have thought about the issues in diverse ways than those who are not.

My clinic sample was designed to contain diagnostic sub-groups because their experiences may lead to different viewpoints about eating disorders, medicalization, and genetics. As mentioned earlier, the experience of AN and BN may be different with regard to stigma. In addition, I expected respondents to vary in their endorsement of a medical model. Although all respondents have been diagnosed and treated for an eating disorder at some time and therefore exposed to medicalized accounts and narratives, they may yet resist medicalized understandings, as noted earlier. Even eating disorders therapists resist some medical language by avoiding the term “patient,” as I observed at a treatment team meeting prior to beginning data collection.

I also expected respondents to vary in their views by severity of illness. Some recovered and currently diagnosed respondents were treated in an inpatient, partial hospitalization, or residential treatment program. Participation in these programs is an imperfect proxy for severity because it also reflects access to treatment. If genetic causal attributions connote greater severity, a respondent with a mild case may conclude that genetic influence was irrelevant for her. Those who are more severely affected may also feel that they have less control over the disorder, making a biological explanation more plausible.

⁶ For example, the informational “module” given to inpatients with AN lists “family/genetic factors” first in its list of causes and explains that “Some of the family factors are genetic and some are due to the environment. For example, a family history of an eating disorder, depression, or alcoholism may have a genetic contribution. On the other hand, other factors, such as being harmed by other people, physically, sexually or emotionally are environmental factors” (p. 3 of clinic handbook for patients with AN, not cited in order to avoid revealing clinic’s identity).

Because severity could affect whether a genetic causal explanation seems relevant, it could affect how the respondent grapples with complex or simple genetic causal accounts.

Only diagnosis (AN vs. BN), severity (hospitalization or residential treatment vs. outpatient), and recovery status (recovered vs. not) were part of the sampling design. Understandings about genetics and perceptions of geneticization could also vary by socioeconomic status and education; better educated people may be more likely to endorse a biological model of mental illness (Karasz 2005). Because the clinic accepted Medicaid there is a higher chance of variation in SES than found in a private clinic. My sample was not designed to contrast socio-economic status and other demographic variables; I anticipated there would be enough diversity in the clinic sample for systematic comparisons of these subgroups.

2. INTERVIEW GUIDE

Qualitative methods can reveal unanticipated findings and investigate meanings and understandings of interview themes more fully than quantitative methods, but often must sacrifice uniformity of question wording and ordering in order to do so (Weiss 1994). To illustrate why qualitative methods are appropriate for this project, I explain why I considered and abandoned the idea of using the revised Illness Perception Questionnaire (IPQ-R) for this project. This questionnaire is based on Leventhal's self-regulatory model (Leventhal et al. 1984) and includes standardized questions with a fixed set of response options to assess perceptions of illness and has been used to study perceptions of eating disorders. This questionnaire includes subsections that are relevant to this project: causality (including the item "hereditary – it runs in my family"), control over symptoms, and expectations about treatment (Moss-Morris 2002). However, this questionnaire was not created specifically for

eating disorders and contains a number of inappropriate items. For example, the causal subscale asks respondents their level of agreement on a 5-point Likert scale with 18 causes, many of which make sense for disorders such as cardiovascular disease or cancer, but not for eating disorders. Some items are nonsensical for eating disorders (smoking, pollution in the environment), and others are indicators or outcomes of eating disorders: “diet or eating habits”, “my own behavior” and “my emotional state” (IPQ-R). Not surprisingly, this causal subscale was found to have low internal consistency in a study of eating disorders perceptions (Stockford et al. 2007). To make the IPQ-R questionnaire more relevant to AN, Quiles (2007) added a subset of eight eating disorder specific causes, such as “media influence” and “need to be perfect.” However, even this improved version is still a standardized fixed response questionnaire and would not allow me to understand the meanings and consequences of these causes for respondents, much less discover unanticipated themes. Standardization of questions would threaten validity at this exploratory stage (see Schaeffer and Maynard 2003 for a discussion of standardization).

The order of questions asked in the interview guides was flexible, with one exception (see Appendix for interview guide). If a respondent spontaneously brought up genetics before I asked about it, I asked general, non-directive probes, rather than following up with questions about genetics that appear later in the guide. These later questions would have introduced concepts of genetic causality that might have affected their answers about other, non-genetic topics. A few respondents knew ahead of time that there would be questions about genetics. At least one inpatient reported having heard about the interview from other inpatients who had already participated, and one recovered person did as well. For most interviews, I asked questions in the order of the interview guide (see Appendix).

The guide was flexible in the wording of questions to adjust to what respondents told me. As Charmaz has written, “Questions must both explore the interviewer’s topic and fit the participant’s experience” (2003: 315). In interviews, I was sensitive to the respondent’s own view of her eating disorder and avoided imposing a different view through my vocabulary. Before the interviews, I was concerned that some respondents might object to the term “eating disorder”, but this did not happen. With all respondents, I tried to elicit their perspectives and avoid terminology that could influence their answers or reduce rapport.

My interviewing technique was not as flexible as some other qualitative approaches. If qualitative and quantitative methods are on a continuum, my approach was toward the structured, quantitative end. Because my analysis (described below) involved a direct comparison of respondents’ answers to specific questions, I was less inclined toward flexibility than some qualitative researchers. I wanted everyone to answer most of the questions, except for probes designed only to fill out an incomplete answer. In addition, because this project was primarily about the meanings of genetic explanations for behavior, I introduced these concepts in a later part of the interview even if they were not already part of the respondents’ consciousness.

The interview was in eight parts. The following description of the interview guide is based on the guide for currently diagnosed patients; the version for recovered people is in the past tense. (The interview guide submitted with the dissertation proposal was shortened to generate an average interview of about an hour and a half.)

(1) *Background questions.* To begin the interview, I asked respondents easy-to-answer questions about their age, education, etc. I confirmed with the respondent her diagnosis and asked if she had ever received any other eating disorder diagnosis. While these

were short-answer questions and ran the risk of “training” the respondent to give short answers to future open-ended questions, they were useful for establishing rapport at the start of the interview. In subsequent sections the wording of questions and the use of probes let the respondent know that longer answers were preferred.

(2) *Personal experiences with eating disorders, with attention to causality.* This section began with very general questions asking the respondent for her thoughts about what AN (or BN) is, and how it began for her.⁷ I was as non-directive as possible in eliciting this history in order to get at the respondent’s own narrative of illness.⁸ These general questions also helped the respondent start talking about her experience in general terms and provided jumping-off points for questions about causality. I asked respondents what they thought were causal factors, risk factors, or contributing factors, and why the eating disorder started *when* it did.⁹ I asked if she thought she had been at risk for an eating disorder compared to other people she knew; previous research on genetic causality suggests that this framing of

⁷ I found this to be a better opening than two alternative strategies, one that was too broad – “tell me about your eating disorder” – and the other that seemed at odds with most respondents’ experience – “how do you talk about your eating disorder to other people.” Many avoided talking about it with others, as might be expected with a psychiatric diagnosis. Because the reasons why a person might want to hide their disorder are relevant to the stigma and conceptualization of eating disorders, I worked this question in when it felt more comfortable to do so.

⁸ Narratives are “interpretive devices, through which people represent themselves, both to themselves and to others” (Lawler 2002: 242). Many of my research questions implicitly involve narratives because they are about causes and how they influence outcomes. Eliciting a narrative early in the interview, before any specific questions about cause, enabled me to minimize my influence on the narrative created. Despite such efforts, patients being interviewed are giving accounts of their own behavior for particular audiences (including the interviewer) and may be motivated to talk about some things and hide others. Their sense of what is a socially desirable and convincing account was likely influenced by their impressions of me.

⁹ These two questions are adapted from Arthur Kleinman’s questions for uncovering explanatory models (1980). Several of these questions assume that respondents would agree they have a problem, which may not be the case for people with AN. I do not claim that these questions necessarily uncover explanatory models because respondent answers are also a way of accounting for themselves in a particular interview situation (Scott and Lyman 1968, Estroff et al. 1991, Young 1982, Groleau et al. 2006), and/or “constitute an imaginative attempt to find a legitimate and meaningful place for [the disorder or disease] in their lives” (Lawton 2003, describing Williams 1984). I have adapted the original questions to make sense for eating disorders and to encourage respondents to think not only about cause but also risk factors, which are important in conceptualizing genetic causality and may not be elicited with standard questions about cause (French et al. 2005).

the question is more likely to elicit ideas about genetics than exclusively causal language (French et al. 2005). To get at causality in yet more ways, I asked whether the eating disorder could have been prevented, and whether there are things that make the eating disorder worse or reoccur. I did not introduce specific contributing factors (e.g., genetics, gender) until later in the interview. During this part of the interview, I elicited the broad outlines of the respondent's experience with treatment, the number of times she had been hospitalized (if any), and information for calculating the lowest BMI for respondents with a history of AN.

(3) Perceptions of eating disorders In this section, I elicited the respondent's perceptions of eating disorders as well as her sense of others' perceptions of eating disorders. Because the respondent may not have had a medicalized understanding of her condition, I asked how she felt about having had an eating disorder, and (later in the section) whether and how eating disorders had been a problem for her. A question on preferred terminology was included but yielded little because respondents were comfortable with the official classifications (e.g., "anorexia"). To get at self-presentation, I asked how she tended to explain it to others when she had to, and if she preferred not to, why. I inquired about others' reactions, including unwanted reactions, wrong ideas and stereotypes about eating disorders. I asked how she would ideally want people to understand eating disorders.

(4) Reactions to specific ideas about eating disorders Here I asked respondents to react to seven different ways of viewing eating disorders. The respondent was told that some options might seem obviously true or false and her honest reaction was requested. I asked, "How do you react to the idea of [AN/BN] as a..." and inserted the following terms one at a time: psychological problem, mental illness, brain disease, physical illness, choice, lifestyle,

and problem with our society or culture. I probed their reactions to mental illness, brain disease, and choice in particular, to find out what aspects of the eating disorder made these terms appropriate or not. I then inquired about whether there were other ways of thinking about eating disorders that seemed accurate and followed up about models they had mentioned that were not among the seven (such as addiction). I asked how they themselves preferred to think of eating disorders and whether their views had changed over time. Last, I asked those with a diagnosis of AN if they saw BN any differently, and vice versa.

(5) *Specific causes.* This set of questions focused on specific causally relevant areas that respondents may not have mentioned before: gender, biology, and genetics. I began by surfacing any remaining ideas about causality before introducing specific ideas, by asking if there were any other important causal factors, even if they were not personally relevant. I asked, “are some kinds of people more likely to develop eating disorders than others?” and, “are there some situations, settings, and environments that make people more likely to develop eating disorders?” Following this, I asked for more of their ideas about social and cultural causes, introduced already as one of the seven models of eating disorders, and asked specifically why more women and girls develop eating disorders compared to men and boys.

Before asking about genetics directly, I told the respondent, “Some say there are biological causes, where something in your body or brain could make you more likely to have [AN/BN],” and asked what they had heard. This question was designed to bring to the surface any thoughts about genetics before asking the same question again, this time specifying “genetic causes, where something about your genes could make you more likely to have [AN/BN]”. I asked for their reaction to this idea, probing for negative or positive emotional reactions, and how plausible or relevant it seemed. I explained that I had several

more questions about genetics and reassured them that I did not expect them to know correct answers but was interested in their best guesses about how genes might be involved in eating disorders. Would everyone with those genes develop [AN/BN]? Does everyone with the disorder have those genes? What makes more sense, genes for [AN/BN] specifically, or genes for something more general that influences it? What would the more general thing be? I also asked for their reactions to the idea that genes could affect one's temperament or personality, making [AN/BN] more likely. All questions about genetics as a causal factor were framed as hypothetical and under investigation in order to minimize geneticization of eating disorders by my research (Brunger & Cox 2000, as cited by Cox & Starzomski 2004).

(6) *Hypothetical scenario 1: Media campaign.* To elicit ideas about the implications of genetic conceptions, I asked respondents to imagine there were a media campaign to promote the idea that genetics play some role in the development of eating disorders. I said that this might involve posters saying "Genes matter for eating disorders" and asked what the reaction might be. I probed about possible good or bad effects for people with AN or BN.

(7) *Hypothetical scenario 2: Test for genetic predisposition.* In this section I focused on the complex model of genetic causality, in which genes predispose a person to eating disorders. This section came last, after I had already explored fully the respondent's ideas about genetic causation with minimal influence from me. This section enabled exploration of the meaning and implications of genetic susceptibility or genetic risk factors. In order to make the conversation less abstract, I asked respondents to imagine that there were a test to assess their genetic predisposition, and consider whether they would want it.¹⁰ In the

¹⁰ A flaw of this question is that it does not accommodate an important lay theory of genetic causality in eating disorders, namely that genetics may predispose them to a *broad range of disorders*, of which AN and BN are only one possibility. Asking them about a predictive test for AN or BN would suggest genes that are specifically linked to one of these disorders. Thus, while the question had been designed to get at complex

preamble I provided explicit information to discourage a deterministic view: “Because both genes and environment play a role, it is not likely that a genetic test could ever predict whether a person will develop an eating disorder. There is no test at this time. But for a moment let’s say you could get a genetic to find out if your genes made you more likely to develop [AN/BN]. Would you want to know?” They were then asked if they would prefer to find out they did or did not have a genetic predisposition. I then asked them to imagine that they found out they had been at a high genetic risk for an eating disorder and how this would change their and others’ views on eating disorders, including causes, treatment, recovery, and genetic family members, and responsibility.

(8) *Closing questions* To conclude the interview with a general question that was still relevant to interview themes, I asked, “If you were giving advice to someone with [AN/BN] about how to think about it, what advice would you give?” I followed by asking what kind of research they would be interested in learning about and what advice they might have for treatment providers. I concluded with a few questions to assess the interview questions and how they felt about the interview.

After every interview, I administered the EDE-Q questionnaire to assess eating disorder symptoms.¹¹ In sociology, the self-report of diagnosis by a healthcare provider would be sufficient to describe my sample, but to make my research potentially useful to psychologists and other clinicians, I provided objective information about eating disorder symptoms of both currently diagnosed and recovered people (Bulik 2008, personal communication). I chose the self-report paper version rather than the in-person interview

causality by focusing on the probabilistic nature of genetic influence, it inadvertently channeled genetic causality into a narrow “gene for” model, albeit probabilistic.

¹¹ This questionnaire was originally included as an appendix but removed because making it publically available could detract from its utility as a clinical instrument.

version (Cooper and Fairburn 1987) because stigmatized behavior tends to be more accurately reported when the mode of data collection distances the interviewer and respondent (Lyberg and Kasprzyk 2004 [1991]). The self-report also enabled me to avoid taking the role of clinician (Kleinman 1980). Because the questionnaire reflects a medicalized view of eating disorders that could influence respondent answers, I administered it after the interview was over. Several respondents ran out of time at the interview, completed it apart from me, and returned it to me later by mail. I informed respondents that they could skip any uncomfortable questions in the in-person interview and questionnaire. In a study comparing the EDE (Eating Disorder Examination) administered by a clinician with the self-administered EDE-Q questionnaire, the self-administered version had higher reports of binge eating and concerns about shape, though this may be because self-reporters had too expansive a definition of what constituted a binge and loss of control (Fairburn and Beglin 1994). A recent assessment of the EDE-Q found that it had satisfactory internal consistency (Peterson et al. 2007) The EDE-Q has 36 items and takes less than 15 minutes to assess frequency and severity of key eating disorder symptoms. (See Table 2.3 and discussion in the next section for more information.)

3. STRATEGIES FOR ANALYSIS

I analyzed transcripts and my own fieldnotes in several stages. The first stage of analysis was note-taking during review of transcripts and fieldnotes for accuracy and to remove identifiers. When transcripts and fieldnotes were entered into N6 (QSR 2002), I began coding, that is, I identified and tagged material relevant to my research questions. I also coded material that seemed important even if it did not relate to my original research questions. In addition, I coded simple information such as demographics, answers to yes/no

questions, the location of structured questions in the transcripts, and whether genetics were mentioned spontaneously before I asked about it. While coding I also created theoretical memos to record surprising observations, new hypotheses, and ideas for future coding.

At the second stage I compiled or disaggregated these codes into categories. I reviewed codes for consistency when an exact count was important, as for statistical testing (e.g., the index capturing endorsement of medicalized terms for describing eating disorders in Chapter 4). I collapsed some specific codes into broader thematic codes (e.g., coding to describe how respondents spoke about “mental illness” and “brain disease”, also in Chapter 4). I also approached analysis by considering one code, or a single section of the interview (e.g., all reactions to the term “psychological problem,” in Chapter 4, or all material after genetics were introduced, covered in Chapters 5 and 6), then categorizing common or conceptually important sub-themes without formally coding or counting them.

At the third stage, I examined whether selected codes and categories were correlated with other codes, categories, or respondent characteristics. For example, I present statistical analyses using Stata (StataCorp 2007) of whether people who are currently in treatment are more likely to endorse medicalized terms (Chapter 4), and whether people who endorse medicalized terms are more likely to have a positive view of genetics (Chapter 5).

4. DEMOGRAPHIC CHARACTERISTICS OF SAMPLE

Demographic characteristics of the achieved sample of 50 women are summarized in Table 2.2 below.

Table 2.2. Demographic Characteristics of Sample. Frequency (%) or Mean (standard deviation) (range) N=50

	All	Recovery status		Diagnosis	
	(n=50)	Recovered (n=25)	In treatment (n=25)	Anorexia nervosa	Bulimia nervosa
Age: mean (min, max)	32.7 (12.8) (18-64)	36.0 (12.6) (20-58)	29.4 (12.4) (18-64)	35.8 (14.4) (19-64)	29.1 (9.7) (18-52)
Race/ancestry/ethnicity					
White/European desc.	42 (84)	22 (88)	20 (80)	24 (89)	18 (78)
Black/African descent	4 (8)	2 (8)	2 (8)	1 (4)	3 (13)
Asian descent	2 (4)	1 (4)	1 (4)	2 (7)	0 (0)
Hispanic/Latino	2 (4)	0 (0)	2 (8)	0 (0)	2 (9)
Education					
H.S. dipl or less	2 (4)	0 (0)	2 (8)	2 (8)	0 (0)
Some college/Assoc.	19 (38)	8 (32)	11 (44)	10 (37)	9 (39)
Bachelor's degree	8 (16)	3 (12)	5 (20)	3 (11)	5 (22)
Some grad./Master's	18 (36)	13 (52)	5 (20)	12 (44)	6 (26)
PhD or MD	3 (6)	1 (4)	2 (8)	0 (0)	3 (13)
Marital					
Single	31 (62)	11 (44)	20 (80)	16 (59)	15 (65)
Married/partnered	15 (30)	12 (48)	3 (12)	8 (30)	7 (30)
Divorced/separated	2 (4)	2 (8)	0 (0)	1 (4)	1 (4)
Widowed	2 (4)	0 (0)	16 (8)	2 (7)	0 (0)
Children					
None	39 (78)	17 (68)	22 (88)	20 (74)	19 (83)
One or more	11 (22)	8 (32)	3 (12)	7 (26)	4 (17)
# Children (range)	(1-5)	(1-5)	(1-3)	(1-3)	(1-5)
Employment status					
Unemployed	4 (8)	0 (0)	4 (16)	2 (8)	2 (9)
Employed	23 (46)	16 (64)	7 (28)	10 (37)	13 (57)
Retired	2 (4)	0 (0)	2 (8)	2 (8)	0 (0)
Disabled	3 (6)	1 (4)	2 (8)	3 (11)	0 (0)
Primarily a student	18 (36)	8 (32)	10 (40)	10 (37)	8 (35)

Percentages may not add up to 100% due to rounding

The mean age of respondents was 33, with recovered people and those with AN tending to be older (36 for both groups) than those in treatment and those with BN (29 for both groups).

My sample was predominantly white (84%) and relatively well-educated (58% had a BA or above). Recovered respondents tended to have attained a higher level of education than those in treatment; the modal category for recovered respondents was “some graduate school

or master's degree" (52%) and for those in treatment it was "some college or associate degree" (44%). Over half of respondents were single, and those in treatment were disproportionately so (80%) compared to those who had recovered (44%). Most respondents had no children (78%), but more recovered people had one or more (32%) compared to those in treatment (12%). With regard to employment, a plurality of respondents were employed (46%), with more recovered respondents employed (64%) than those in treatment (28%). Other than age (noted above), demographic characteristics were not markedly different for those with AN compared to BN.

Table 2.3 illustrates respondent characteristics related to eating disorders, subdivided by recovery status. Respondents were fairly evenly distributed among four categories describing treatment history, ranging from little or no treatment to two or more highly structured programs (such as an inpatient stay or residential treatment facility). Recovered respondents had gone through less treatment than those currently receiving treatment; the distribution of responses for the two groups are nearly reversed. This inverse relationship was statistically significant ($p=.017$, Fisher's exact test) and it will therefore be difficult to distinguish and interpret the effects of either variable in bivariate analyses. Those with AN compared to BN were more similar with regard to treatment, though those with AN had more often been involved in two or more highly structured programs (41%) compared to those with BN (13%) (not shown, and I do not present other characteristics subdivided by diagnosis because the symptoms of those with AN and BN are already known to differ). Most respondents reported a body mass index (BMI) below 18.5 at some point in their lives (71%) and this did not differ by recovery status, though the mean lowest lifetime BMI for those currently in treatment was lower (14.10) than those who had recovered (15.38). As

would be expected, assessments of current symptoms using the EDE-Q instrument showed lower scores for recovered respondents than those in treatment (see legend of Table 2.3 for further information on the EDE-Q). This difference by recovery status was apparent in all four subscales (dietary restraint, eating concern, shape concern, and weight concern) and measures of self-reported behavior (vomiting, laxative use, diuretic use, “exercising hard,” and binge eating).

Table 2.3. Eating Disorder Severity and Symptoms for Women with AN or BN (n=50)
Categorical variables: frequency (%); Continuous variables: usually Mean (S.D.) (range)

	All (n=50)	Recovered (n=25)	In treatment (n=25)
Treatment experience*			
Little or no treatment	10 (20)	7 (28)	3 (12)
Outpatient treatment	15 (30)	11 (44)	4 (16)
One structured program	11 (22)	4 (16)	7 (28)
Two or more structured prog.	14 (28)	3 (12)	11 (44)
BMI<18.5 ever in lifetime**			
No	14 (29)	6 (25)	8 (32)
Yes	35 (71)	18 (75)	17 (68)
Mean lowest BMI (S.D.)	14.76 (2.39)	15.38 (1.81)	14.10 (2.79)
(range) (Yes answers only)	(8.92-18.29)	(11.37-18.29)	(8.92-18.29)
EDE-Q (last 28 days)***	2.86 (1.76) (0.43-5.86)	1.50 (1.18) (0.43-5.05)	4.23 (1.01) (1.61-5.86)
Dietary restraint subscale	2.41 (1.94) (0-6)	1.22 (1.27) (0-5)	3.60 (1.77) (0-6)
Eating concern subscale	2.12 (1.83) (0-5.75)	0.64 (0.85) (0-3.2)	3.61 (1.23) (1.2-5.75)
Shape concern subscale	3.51 (1.91) (0.25-6)	2.00 (1.43) (0.25-6)	5.02 (0.81) (3.25-6)
Weight concern subscale	3.42 (1.87) (0.25-6)	2.13 (1.54) (0.25-6)	4.71 (1.14) (2-6)
Vomiting			
No	37 (74)	25 (100)	12 (48)
Yes	13 (26)	0 (0)	13 (52)
Median # times	15	N/A	15
(range)****	(1-425)		(1-425)
Laxatives			
No	43 (86)	25 (100)	18 (72)
Yes	7 (14)	0 (0)	7 (28)
Mean # times (S.D.)	4.86 (3.58)	N/A	4.86 (3.58)
(range)	(0-10)		(0-10)

Diuretics			
No	48 (96)	24 (96)	24 (96)
Yes	2 (4)	1 (0)	1 (4)
Mean # times (S.D.)	8.00 (4.24)	5	11
(range)	(5-11)	N/A	N/A
Exercising hard			
No	33 (66)	20 (80)	13 (52)
Yes	17 (34)	5 (20)	12 (48)
Mean # times (S.D.)	10.41 (8.19)	10.00 (8.28)	10.58 (8.52)
(range)	(1-25)	(3-20)	(1-25)
Episodes of binge eating			
Mean score (S.D.)	1.22 (1.85)	0.36 (1.04)	2.08 (2.10)
(range)	(0-6)	(0-5)	(0-6)

AN=Anorexia nervosa, BN=Bulimia nervosa, S.D.=standard deviation

*Treatment experience variable based on interview coding, rather than a standard question with fixed response options. “Structured program” refers to psychiatric hospitalization for an eating disorder (not for symptoms like dehydration), or participation in a residential program, or structured day program. “Outpatient treatment” refers to any other kind of treatment. The “little or no treatment” category encompasses to people who were diagnosed only *after* recovering from the eating disorder, who mentioned dropping out of treatment after a short time, who mentioned counseling for another condition unrelated to the eating disorder, or who had just entered the BN treatment study as it was starting.

**BMI was calculated based by multiplying lowest weight in pounds and dividing by squared height in inches ($703 \times \text{weight} / \text{inches}^2$). Questions about height and lowest weight were asked of people with BN only if context suggested that there might have been two diagnoses or confusion about diagnosis (e.g., person who has AN reports having BN because of purging). N=49 because Carol was a child when she had AN and did not know her lowest BMI.

***EDE-Q= Eating Disorders Examination Questionnaire. The EDE-Q rating scheme is based on frequency and severity ratings ranging from 0 (feature is absent) to 6 (feature is constant/severe).¹² The four subscales are listed below the EDE-Q global score, which is the total divided by four. Dietary restraint subscale was based on the sum of questions 1, 2, 3, 4, and 5, divided by 5. Eating concern subscale was based on the sum of questions 6, 7, 9, 15, and 34 divided by 5. Shape concern subscale was based on the sum of questions 10, 11, 12, 13, 30, 33, 35, and 36 divided by 8. Weight concern subscale was based on the sum of questions 14, 29, 31, 32 divided by 4. For respondents with missing data for one or more questions on a subscale, I added values for the existing answers and divided by the number of existing answers.

****There was one outlier reporting 425 on this variable.

Looking ahead to bivariate analyses in Chapters 4 and 5, it is notable that recovered respondents had markedly different treatment experiences from those currently in treatment. This most likely reflects different recruitment strategies for the two groups: those in treatment were recruited through a hospital-based program and those who had recovered received a mass email through their affiliation with work or school. Ideally I would have

¹²The two sets of response options for frequency were (A) 0 – no days, 1 – 1-5 days, 2 – 6-12 days, 3 – 13-15 days, 4 – 16-22 days, 5 -- 23-27 days, 6 – every day, and (B) 0 – none of the times, 1 – a few of the times, 2 – less than half the times, 3 – half the times, 4- more than half the times, 5-most of the time, 6-every time.

recruited recovered respondents through the same clinic, but this was not possible. Therefore, hypothesized effects of the experience of recovery as an influence on genetic ideas cannot easily be assessed, if at all, because those who recovered also received less treatment. In addition, if those who received less treatment appear to differ in their interpretation of genetics, this will be difficult to interpret, because receiving less treatment could be a proxy either for having a less severe case or receiving less exposure to medicalized concepts. For these reasons, I will qualify all claims about the effects of recovery status or extensive treatment experience on respondents' views of genetics.

The next chapter examines the first question of the thesis, what do people with eating disorders believe to be the causes of AN and BN?

CHAPTER 3

COMPLEX CAUSATION AND AMBIGUOUS AGENCY: PERCEPTIONS OF HOW AND WHY EATING DISORDERS DEVELOP

How do people account for the development of their eating disorders? The interviews revealed that it is not a simple matter of causes leading directly to an eating disorder. Rather, respondents included complex combinations of contributors interacting over time, usually featuring some degree of individual control or agency. Both of these – complex causation and uncertain agency – make it difficult to think of eating disorders as exclusively medical phenomena.

I begin with a discussion of some challenges in coding causation for this project. I then illustrate complex causal accounting by presenting some of the most common ways people talked about the development of their eating disorders. Subsequently I present causal factors that may be particularly relevant to the meanings and implications of genetic theories for respondents. Because respondents discuss causation in a way that implicates their own agency, defined generally as the individual exerting control, making decisions, choosing, or taking action, I examine language that implies agency, a lack of agency, and something in between. Finally, I touch upon the importance of agency as respondents consider how they would ideally like others to view eating disorders. Throughout this project, I will use the word “cause”, “causal factor”, and “contributing factor” interchangeably and to encompass many different kinds of influence.

1. APPROACHING THE CATEGORIZATION OF CAUSES

The categorization of causes in this chapter requires some explanation and history. When beginning this research, I was interested in how broad categories of causes might be pitted against each other: biological forces vs. social forces and individual agency. If biological forces get center stage, do they push social forces to the wings, and change the politics of who ought to be held responsible? Do biological forces appear to remove individual agency? I developed three broad codes to describe the types of causes mentioned by respondents: “environmental”, which included anything that was clearly external to the individual; “biological”, which included anything about the material body or brain; and “individual”, which was created to capture diverse material that was neither external nor biological and was located in the individual. The latter code included statements about individual agency and personality, as well as thoughts, feelings, and beliefs cited as causal factors. When I began coding, I already knew I wanted to capture statements about environmental and biological causation. I created the “individual” code after reading transcripts.

I found, however, that when respondents spoke of causes they could not usually be categorized in this simple way. Frequently, a causal factor implied two or even all three categories.¹ For example, respondents often spoke about a felt imperative to control their weight as a causal factor. It was not uncommon to cite some aspect of the body as a contributing factor (coded as “biological”), and discuss dissatisfaction with it (coded as “individual”), and describe social contexts that promoted or intensified this dissatisfaction (coded as “environmental”). Similarly, coping with stress incorporated elements of

¹ This observation may be useful in thinking through the partitioning of variance in twin studies between genetic and environmental contributions.

“individual” and “environmental” factors. The code for “individual” included a wide diversity of phenomena, including psychological characteristics (e.g., low self-esteem, being depressed), desires or preferences (e.g., wanting to lose weight), interpretations (e.g., thinking that Dad left the home because the respondent was overweight), and agency. Many respondents thought of the eating disorder as a coping strategy or coping “mechanism” to deal with problems in their environment. Thus, the behaviors they adopted were influenced by the environment as well as some individual predilection or choice, and were combined into the concept of coping strategy, which cannot be reduced to either one. Coping strategies and other compound causes will be described in more detail in the next section.

Some causal factors seemed particularly likely to have a bearing on how respondents think and feel about genetics. All the material presented in this chapter appeared well before the idea of genetic causation or other specific causal factors were introduced in the interview. This early material is the best view I have of their pre-existing ideas, relatively free of influence from me. Some of the ideas appeared to be more fertile ground for genetics than others (a proposition that is tested in later chapters).

Discussions of causation were elicited by specific questions but also occurred spontaneously in other sections of the interview. All the material described in this chapter appeared not only before I brought up genetics, but also before I asked them to react to specific models of eating disorders (e.g., as mental illness) because these models frequently prompted respondents to think of new causes. (I refer to these questions about specific models as “reaction terms” and “reaction questions” as a convenient shorthand for the rest of the chapter.) The questions encompass themes that go well beyond some narrow definitions of cause. Specifically, I asked them how their eating disorder began; what causal factors, risk

factors, or other factors might have contributed to it; why it started when it did, why they might have developed the eating disorder as opposed to other people they knew, and whether it could have been prevented in some way. The purpose of this breadth was to bring to the surface any existing ideas about biological predisposition, as well as environmental factors.

2. COMMON, COMPLEX, AND COMPOUND CAUSES

Respondents' causal accounting included many different kinds of causal factors operating in conjunction with each other across time. Several said that their stories changed over time as they learned new things about themselves and about eating disorders. Sometimes they expressed uncertainty about whether something they cited was actually important. In the following I present three categories of causal factors presented by respondents: valorization of thinness, response to stressors or other problems, and factors that encourage the maintenance of the eating disorder. I organized commonly-cited causal factors into these three categories because the categories are general enough to encompass a great deal of variation. I do not report frequencies for causal factors and offer them primarily as background to illustrate the range and variety of causal accounting. These also help prepare the reader for subsequent discussion of individual agency and lack of agency in eating disorders.

2.1 Valorization of thinness

Most respondents described concerns about their weight or size as a contributing factor to their AN or BN. This was inseparable from their own valorization of thinness, and often closely tied to people and environments that valorized thinness. After a brief description of weight concern, I present in greater depth the most commonly mentioned contexts in which thin body size and shape were prized.

Almost all respondents reported a conscious desire to lose weight or avoid gaining weight. For most, this desire began as a result of being overweight or gaining weight themselves, and was identified as one of the causes of the eating disorder because it was the reason to begin dieting, purging and other eating disorder behavior. They reported being “pudgy”, “chunky” or “chubby” as a child and resolving to lose weight as they got older; some resolved to lose weight in order to avoid becoming as overweight as a parent. Several talked about gaining weight as an adolescent or adult as a result of puberty, pregnancy, birth control pills, injury (inability to exercise), or over-eating due to stress, which prompted them to take action in the form of restricting, exercising, and purging.

Some were not overweight but nevertheless came to desire weight loss. Some inadvertently lost weight and then adopted it as a goal. For example, Barbara lost weight because she began to play a new sport. When the season was over, she dieted and exercised to compensate for anticipated weight gain. Lynn lost weight because she had very little money for food during graduate school. Gillian began restricting because of a phobia about throwing up. As a result she lost weight and found that being smaller helped her avoid bullying. Willa consciously valued the extreme thinness of models and aspired toward that ideal, even though she was not overweight.

The concern about weight reflected a social valorization of thinness, in respondents’ estimation. Respondents spoke about monitoring and evaluation of weight by “society” generally, family members, and in athletic and performing arts contexts. Some linked it to gender, others did not.

For example, Ingrid describes the general valorization of thinness in “our society”, particularly among peers at her school. As someone who was “heavy” and “chubby” she developed a fear of food as an “enemy” because she associated it with abuse from her peers.

... you’re trying so hard to get away from the enemy that made you so fat. And, made you suffer as a result of that. And, by suffer I mean not just physiologically. But, emotionally. Socially. And, so forth. Fat is not fun. And, fat is not a sociable or likeable attribute to have. And, in our society anybody who is overweight is not considered worth even talking to. So, the thinner you got, the more – you would equate being thin with being popular. And, I was a hundred and seventy pounds when I was twelve years old. And, at four ten that’s a hefty size. And so, I went on this diet with a doctor who – I mean I went through emotional and verbal and physical abuse as a child from my peers. Classroom bullying. And, all these things. ... So, when I found something – a medium called dieting. Which allowed me in fact to feel better about myself. Suddenly the whole world opened to me. So, I started when I was about thirteen. Maybe fourteen. And, by the time I was in high school, I had reached the weight I wanted. I was extremely popular. My grades went up. And, gee. That’s the best thing since sliced bread. Why would I want to give it up? (Ingrid, T-AN²)

For Ingrid, “society,” specifically her peers, judged her negatively and excluded her because of her weight. By losing weight, her “whole world opened.” She equated dieting with feeling better about herself and being popular.

Most respondents described contexts that valorized thinness in ways that suggested that gender was important. This ranged from uncritical statements about trying to be attractive to men to critiques of beauty norms for women and broader critiques of patriarchy. Yolanda (T-BN) said that the main contributing factors for her eating disorder were “The fact that I was overweight for so long. And it felt so good to be skinny.” I probed about the good feeling she had about skinny, and she linked it to her attractiveness to men:

It was something that I had always been envious of and always wanted. And then I finally had it. And I was finally able to go to the store and pick out the clothes that I wanted. And think that it looked really good on me. And I was getting attention from boys. And beforehand I’d always have a crush on the boy. And like, I would just be

² “T-AN” stands for “In treatment for AN.” I will provide such brief descriptions when quoting or referring to respondents. “R” designates “recovered”. See List of Abbreviations at beginning of Chapter 1.

their friend. And I mean I was the same way. The boys that I had crushes on weren't overweight. So I was finally – I was just really, really happy.

She connected her happiness about being thin to positive attention from boys, and in this sense it relates to gender. Other respondents cited gendered beauty norms as causal factors, suggesting more critical distance than the excerpt from Yolanda. Rebecca (T-BN) spoke about comparing herself to media images and revising her sense of what was beautiful.

I do kind of blame the media a little bit. Because I feel like – I didn't have TV growing up. So until I was in high school I never really watched TV. And then I started going over to friends' houses. And we'd watch TV there. And I think it changed my perception of what beautiful was. Like, I'd always had, like, a lot of confidence in myself. And, like, thought I was a pretty girl and everything. And then like, I started putting on weight. And I started seeing like, these celebrities who were beautiful. And they're like, bone thin. And so I think my perception of like, what was beautiful changed. And I didn't feel like I matched – I matched what I thought of [as] beautiful anymore.

Rebecca's description suggests that she sees the celebrity version of beauty as one possible standard, rather than the only one. Some linked their eating disorder to gender inequality more directly. Alyce (R-BN) described puberty as a betrayal, because it meant she was a woman and therefore disempowered.

Why did it start when it started? It was totally puberty. I felt betrayed by my body. And, I felt it was a demotion. I was always taller than the boys. Smarter than the boys. Faster than the boys. Meaner than the boys. Better swimmer than the boys. Even those five years older I could lick 'em. And, it was totally disempowering. I'm still mad about it. Honestly. And, not many women will talk about it. I don't know. But, it just really sucked. All of a sudden I was like the back-up singer. Or, the secondary status of women just hit me like a log. And, I hated it. I hate it now. In retrospect I think I was conflicted. Because, part of you wants to conform. It's puberty. And, part of you doesn't. I was very conflicted.

Having a woman's body was a "demotion" from her previous status as a young competitive swimmer, whose low bodyfat was praised by her family and doctor. Alyce was one of 23 who explicitly tied the eating disorder to gender in a way that was more critical than others such as Yolanda above. Although puberty is important, Alyce does not present it as a

biological event but as the inauguration of an unwanted identity as a disempowered woman. By explicitly connecting gendered disempowerment to the eating disorder, Alyce may be more critical of medicalized ideas of and genetic explanations for eating disorders. (More about this more gender-conscious group of 23 will be presented later in this chapter, in the section on causal factors that are likely to inform a respondent's reception of genetic ideas.)

In addition to speaking about shared social norms, respondents spoke about specific people and contexts that valorized thinness. Family members created environments in which thinness was valued. Several respondents thought their parents had eating disorders or "eating issues" of some kind. Victoria described her mother's "competitiveness" with her about eating small amounts and being a small size:

... she's a lot smaller than I am. Like, I'm not just saying that. She really is. But she'll say things about how, like, she's so fat. And she like, can't fit into my clothes because I'm small. And she'll, like, send me like, clothes that are obviously too small for me. And that just makes me feel worse too. And she's like, "Oh. What's the matter? Didn't it fit? Were they too big?" I'm like, "No." They were, like, size zero like, pants she'll send me or something. I'm like, I obviously don't wear this size. It's like, stuff like that. I mean maybe she could. So it's just like, "Uh yeah." Or she'll just be, like, competitive about eating with me. Like when I just had surgery. Like, I couldn't eat a lot at certain times. Like, at night for the surgery because I had surgery in the morning. It was a bunch of different procedures that they did. And, like, she wouldn't eat if I couldn't eat. (Victoria, T-BN)

Victoria thought this kind of comparison and "competition" about body size and eating encouraged her eating disorder, even without explicit criticism. Willa also describes even more subtle encouragement from her mother about weight loss, against a backdrop of a self-described affluent area of the country.

And it's kind of understood I guess that – I mean you're supposed to be beautiful and thin and wealthy and all that kind of thing... My mom was very thin. And so she I mean never said "Oh my gosh. You really need to lose weight." Or anything like that. But anytime I made it clear that I wanted – was trying to do that. It was always encouraged. ... I mean to me moms eat salads. It was just kind of that was that's just

what you do. You grow up. And I honestly think I believed “Of course that’s what you do when you become a mom.” (Willa, R-AN)

Willa’s mother does not tell her to lose weight, but through example and encouragement about weight loss, the importance of thinness is clear; “it’s kind of understood.” (Clearly, Willa’s description of expectations for moms is related to gender, as are many of the following examples of specific contexts.)

Others reported comments from family members that were far from subtle. For example, Margaret and Tammy were told they were “chunky”, Emma’s mother declared, “Your thighs are starting to get big.” Fran’s father “harped on me...and offered me five dollars for every pound I lost.” Later, she married a man who monitored her weight in ways that were not only non-subtle but even abusive:

He liked me thin. That was what he was used to. He had no tolerance for any extra weight. So, by the time I was three months pregnant. And, I wasn’t huge. He wanted me to shower in the guest bathroom across the house so that he wouldn’t risk seeing me with a larger stomach from my pregnancy.... I was married to him for fourteen years. But, he wanted to know how much I weighed every day. Did not want me gaining any extra weight. Even made the statement that he’d divorce me if I got fat. (Fran, R-BN)

Weight control was also part of Yvette’s abuse as a child by her father. In addition she cites his attitudes toward her mother’s weight, some of which happened before she was born.

For me my biological father when I was little – he would tell me how fat I was. And he would tell me like basically how disgusted he was with me. And so when you’re a little kid, like, you don’t realize that your dad is crazy. And he did a lot of other – like, he abused me. And he’d have me and my older brother do stuff. ... it was like he wanted control over me and my brother. And like, my mom had said he always wanted her to be thin. He had always liked her being thin. And ... he had always prided when she – taking her out when she was really thin. And they wouldn’t do much when she got pregnant. He was, like, disgusted with her. And that’s why when she got pregnant with me he was more disgusted. Because he had to go through having a larger lady again. Even though she was pregnant. Like, I don’t know. So it was like, in my mind I was like, “Well if I want my dad to come back, I need to be thinner.” And so that was, like, my main cause. (Yvette, R-AN)

Yvette was thus affected not only by her father's abusive weight control but by his attempt to control her mother's weight. Several others also said they were affected by a family member's monitoring of another family member's weight, even when their own was not a topic of conversation.

Many respondents described specific contexts outside the family that promoted awareness of size, with consequences for gaining weight. Here I describe athletic contexts. Several shared Deena's experience of body-monitoring by coaches for a variety of sports, including gymnastics, swimming, field hockey, and cheerleading.

I was about ten or eleven. And, my period started. And then, that's when it really started to hit me. When my body started changing. And, like, beginning puberty. I really was very aware of my body changing and developing breasts. And, it was like, very troublesome to me. And, the gymnastics coach was very adamant about we shouldn't be too heavy. And, we can't be too big. We have to be very petite. And, he won't spot us. He can't lift us if we're too heavy. And so, that was very – like a fear of mine almost. Like, I was very afraid to gain weight or get big. And so, I was very restrictive of my food. (Deena, R-AN)

Carly's bodyfat was assessed every month as part of her involvement in swim team. This became more of a concern when she gained some weight due to going on birth control pills.

I was swimming all the time. And like, I had to be a certain weight. Because we were getting our fat tested every month. It was called "Getting Pete'd." Because the guy's name was Pete. And he would come in with the pinchers and stuff. And I mean I never thought about my weight. I was always so good about it. It was just easy for me to be skinny until I went on birth control. And I mean high school is when I guess your body is maturing and stuff. And you're gaining weight no matter what. But I just wasn't the weight I should be. And I don't know. I think I – the fat percentage tests and all that. Getting Pete'd. That had something to do with it. Because I was going up. But it wasn't anything like obese. It was, like, nineteen percent body fat. (Carly, R-BN)

Deena and Carly and others had their fat explicitly monitored by coaches. But even athletes who did not, reported disturbing effects of participation: their bodies had been using so many calories while in training that their weight went significantly upward at the end of a

season, which contributed to the eating disorder, by their account. On the other hand, for Jackie, injury and withdrawal from athletics prompted her to stop appreciating her “strong”, “solid girl” body that had always been so “helpful” in sports, and “I kind of had to start seeing my body differently. And then, I wasn’t happy with it.”

Like athletics, involvement in performing arts also heightened respondents’ consciousness of their bodies and rewarded vigilance. Sydney described being highly conscious of having to fit into costumes for theater.

And then I’ve always been into theater. Where you do a lot of productions. And you’re changing back stage in front of everybody. And you’re getting costume fittings. And you’re getting – so you’re very aware of your body size. And when you get cast in a show in October that’s not performing until January you have to stay the exact same size. And so that’s just – it’s something that’s in the back of your mind. (Sydney, T-BN)

Nell experienced something similar, even when she was only eight years old and would be expected to grow by the time she had to wear the costume.

And so but then my mom a couple of times put us on diets. We took ballet lessons for example. And they would measure us for these costumes in October. And we were supposed to be able to wear them in, like, May. When you’re like, eight. I mean it doesn’t make any sense. And one time my older sister had gained weight and was unable to fit into her costume. So my mom put us on this, like, crash diet when I was eight years old. And other times she would sort of help make us restrict or something like that. (Nell, R-AN)

Clearly the family is important here, along with the performing arts context; Nell’s experience highlights the multiple reinforcing components of cultural messages about thinness.

There is much more that could be said about the ways respondents connected eating disorders and environmental contexts, particularly as they relate to gender. I have reviewed a great diversity of experiences with weight in specific environmental contexts in order to convey an important causal factor. This causal factor, which I have titled “valorization of

thinness”, encompasses the meanings of weight as adopted by the person in interaction with a variety of social environments. For many respondents, this causal factor is inextricably linked to their material weight when they began the eating disorder, illustrating the difficulty in separating the physical body from its interpretation by the individual as influenced by a social context.

2.2 *Stressors and responses: Eating disorder as coping*

Another complex, compound contributing factor that respondents frequently mentioned was stress or other problems, with the eating disorder as a response. Most respondents cited stressful life events as contributing factors. Sometimes this was abuse or other bad treatment directly connected to bodily appearance as for Ingrid, Fran and Yvette above. But more often respondents described stressors unrelated to appearance or eating. Under stress, respondents said they turned to behaviors that developed into eating disorders. I will use the shorthand “stressor” to refer to a wide range of events, from abuse and trauma, to breaking up with a boyfriend, to moving. Like the “valorization of thinness” described above, the process of experiencing a stressor and responding to it over time cannot simply be categorized as an “environmental” factor. Respondents are zeroing in on their *responses* to the stressor, responses which eventually turned into an eating disorder. For this reason, I conceptualize the causal factor not simply as the external event but its combination with the individual response. Twenty-one (at least) respondents spoke of their eating disorder as a way of coping with stress or difficult emotions and yet more spoke about it more generally in terms of responding or reacting to a situation, without specifically talking about difficult emotions or stress.

Some described serious abuse or trauma as a child, long before the eating disorder started. This included sexual or other child abuse, domestic violence, abandonment, death or mental illness of a parent, and domestic violence. Respondents said these experiences led to negative states of mind such as low self-esteem, insecurity in relationships with others, or the sense of having a “hole” in the heart that needed filling. For some, these traumatic experiences became directly connected to the eating disorder, as for Reba, who said she avoided sexual attention from her father by becoming extremely thin. Often, they described the eating disorder as one way of coping with these negative experiences, and that alcohol or something else might have accomplished the same thing. Some spoke of “choosing” the eating disorder over another form of coping, an idea I return to later.

Many respondents also spoke of stressful events as precipitating factors that helped explain the timing of the eating disorder, rather than as background stressors from long ago. This included traumatic experiences like rape (Joelle, Gena), and, more commonly, less violent stressors such as moving, changing schools, and being away from home. A version of Eva’s experience was shared by several respondents:

I had moved from [southwestern state] when I was in fifth grade. So, I kind of started middle school without very many friends. I think it was a combination of, like, me developing faster. And, seeing that, like, a lot of the other girls were a lot thinner than I was. And then, not really having any close friends like I did in [southwestern state] before I moved. And so, I think I kind of like tied the two together. Like being thin and being happy. And, like, having friends and stuff. (Eva, T-BN)

Amy (T-AN) spoke of family problems: “The family was having problems. And, for me if I got upset, I refused to eat. It was just easier to deal with whatever I was feeling that way.”

Jackie was troubled by her sister’s health problems, and “used it [BN] as a coping mechanism. And then, all of a sudden I didn’t know any other way to cope.”

Respondents turned to eating disorder behavior to deal with the stress of daily life as well as specific stressors. Victoria explained, “when things are going poorly. And I’m like, anxious or stressed or angry or anything. It’s just kind of like what I go to. Either eating or not eating and throwing up.” Fiona said it “was a way for me to cope. That I had control of something in my life,” during a challenging semester at school. Zinnia described it as a “coping mechanism for stress” during graduate school; Emma as a “maladaptive coping mechanism,” Reba as an “unhealthy coping mechanism.”

Several said that the eating disorder behavior delivered a feeling that counteracted the bad feelings, which was connected to coping. Selena compared it to a “high” that enabled her to feel better: “it’s sort of like a drug. I mean whenever I have really bad stress it’s almost like a crutch. Like, I can – it gives me some sort of like, brain high. I don’t know what it is. But it helps me cope somehow.” Delia said the eating disorder “numbed me from pain”, and Karen also appreciated its “numbing effect.” Mireya felt “disconnected” and “safe” when she binged. Fran linked the bingeing to “suppressing” feelings, and the purging as “getting rid of those feelings.” Paula likewise thought of purging as a “metaphor.”

Many respondents thought of the eating disorder as one possible way of coping. They said they could have tried other things. According to Ingrid (T-AN), “anorexia is the way a person deals with stress in their life. Now, as long as we’re human, we’re all going to have stress. Some people deal with stress by chain smoking. Other people deal with stress by drinking. Other people deal with stress by anger. Arguing.” Yvette, whose father abused her and her brother, speculated that her brother handled his emotions with alcohol.

Why did respondents turn to an eating disorder rather than another way of coping or managing unwanted feelings? Mary thought her eating disorder was a better alternative than

expressing a “bad temper”: “So, it was kind of a way to control something and not hurt anybody.” For those who thought of it as an alternative to addiction, the eating disorder could seem preferable based on observations of alcoholic family members. Some said they had more access to food than they did to drugs, and that being a “good girl” prevented them from seeking these out. Nell spoke of having a “limited vocabulary” of ways to cope, due to a lack of access to people in “helping professions” rather than addictive substances:

I didn’t know about therapists. And I didn’t know about social workers or anything. I didn’t know that there were mentors and helping professions and all that jazz out there. So it was like, I didn’t have anybody. And I didn’t even know there was anybody that I could have possibly gone to. And so since there were – since I didn’t know anybody I had this limited array of possibilities of ways to cope with things. And so anorexia was in my limited vocabulary. That was a pretty good one. (Nell, R-AN)

Thus, for some the eating disorder was partly the product of a lack of other options or opportunities. It could also be the closest at hand, because of a family member or friend who modeled the behavior. Wendy thought she was “taught” or “trained” to have an eating disorder because her mother was “one of those compulsive dieters that is perpetually overweight and emotionally eats.” As she summarized, “I think that people turn to different things to deal with their emotions. Like a lot of people turn to alcohol. People turn to all sorts of things. And I think the reason I turned to food was because it was already engrained in me to turn to food.”

2.3 Repetition over time: maintaining and intensifying the eating disorder

In addition to the valorization of thinness and coping with problems, I identified a third general compound cause: repetition over time. This compound general cause encompasses aspects of the temporal dimension that contributed to eating disorders in and of themselves, according to respondents. They spoke about development of the eating disorder

over time, rather than a dichotomous state of either having an eating disorder or not. One might think about causal arrows, a list of causes on the left, with arrows leading from them to the eating disorder on the right. But respondents described intermediate steps entailing a series of imaginary arrows; recursive arrows reflecting the effects of eating disorder behavior on the person's body, sense of self, and activity in the world; and causes that matter at one time and not another (e.g., factors that intensify rather than incite the eating disorder). The beginning of the disorder is different from the middle; there is learning, development of routines, physical and other adaptation, and the meaning and role of the eating disorder in the person's life changes over time. In this section I describe selected aspects of the dynamic process, which are related generally to repetition over time: learning to purge, intensification and maintenance of the eating disorder, changing experiences of the disorder and the development of a "habit" that begins to feel like second nature. Other examples could have been chosen; these are meant to convey the temporal dimension involved in the discussion of causal factors.

For most respondents, the development of the eating disorder was gradual and took on new meanings over time, rather than arriving suddenly. To take one example, Jackie found herself gaining weight because an injury prevented her from working out at the level she had for her high school athletic teams. She "dabbled" in and then formed a "habit" of purging, which was under her control for a while.

I wasn't burning off nearly as much as when I was eating when I can exercise. And so, I started to gain weight. And, I couldn't exercise it off. Because, I was on crutches. And, one day I just thought to vomit. And, I'd heard of – I mean I had friends in middle school who dabbled in purging. As middle schoolers will dabble in things. And, I'd always thought it was ridiculous then. But, the first time I did it; it just seemed like such a natural and obvious thing to do. And, I mean the first time I did it, I hated it. Thought it was disgusting. Because, who really likes to throw up? But, it just sort of became a habit. And then, I don't even know when I started

noticing it. But, it just – I started doing it. Because, it made me feel better. Not just because I felt like I'd eaten too much. I wasn't always bingeing at that point. Sometimes I just threw up a normal meal... And, this just kind of became my secret thing.... And, I kind of loved it. Because, it was just my thing. And then, it would stop for a couple months all throughout high school. And then, come back for a few months. And then, it'd stop. But, I never really thought of it as an eating disorder. (Jackie, T-BN)

Then, Jackie went away from home for an internship and was alone for long periods of time, which she associated with an intensification of bingeing and purging.

...it kind of provided the perfect environment for bulimia to worsen. And then, I got in a really bad car accident. And, couldn't exercise anymore. And, just all of a sudden it went from something that I could turn to when I wanted to, to something that completely controlled my life. And, I mean I was restricting a lot. There would be days when I would only eat a cup of low sodium beef broth. And, that was it. And then, there would be days when I would eat a ton and a ton and a ton. And, throw up seven times. And, at that point I kind of realized what a problem it was. (Jackie)

Jackie cites different contributing factors at different times as the eating disorder developed, from weight gain, practicing purging till it became a habit, feeling it was a secret thing she “loved”, being in an isolated environment where it began to worsen and become a problem. This illustrates the sometimes complex, varied, and long-term temporal dimension of the development of eating disorders.

Respondents cited factors that helped maintain or intensify the eating disorder, which reflect the importance of development over time, rather than static cause and effect. I include external, environmental factors, such as being isolated and free of monitoring, as well as a hard-to-categorize causal factor: the force of habit. Some respondents felt that just incorporating it into their lives and having it become a habit gave it some momentum, because of physical or other adaptation.

Many spoke of social reinforcement for losing weight, in the form of compliments: “once I got started, I did get a lot of compliments. And, I didn't stop... Initially my mom was

just like, “Well, you look very well.” And she was glad that I was able to lose weight. And, we went shopping together. And, like, picked out clothes and things.” (Amy, T-AN).

Yvette’s account was similar:

And I had always gotten compliments about, like, being, like, pretty. But then I had never gotten like, “Oh. You look so thin. Oh. I’m so jealous. You look so good in this.” And then my friends were all pretty tiny. And so, like, it was just like it was nice to be able to share clothes. And so, like, it just – that helped me I want to say as much causal as much as perpetuated it. Like, that was definitely like an incentive. (Yvette, R-AN)

People may have begun restricting calories in order to lose weight or to cope with stress, but maintained it in part because of the positive feedback they got. Thus, the behavior affected the body, which affected how people treated them, which affected how they felt about themselves; these may be thought of as arrows leading from the eating disorder behavior to the social environment or the individual, and then back again to the eating disorder behavior. Those who lost weight accidentally also reported compliments as an important motivation to continue.

Several respondents noted that isolation, being away from family members who would have noticed and objected to weight loss, bingeing, or purging, made it easier to continue the behaviors. “[T]here was no one around who was really sort of watching me and watching those changes.” (Lynn, R-AN) Tammy described a similar experience:

When school ended. And I was kind of – my parents worked day shift. And I was there alone. And I had time to exercise and not eat or throw up or do whatever I wanted to do in private.... And I just kind of had the privacy to have more self-control over what I did without somebody finding out to the extent of what I was doing.

Although several respondents complained about monitoring of their behavior by self-appointed “food police”, many felt that if they had been confronted earlier or received some kind of intervention, they would not have developed the “habit”. A habit cannot be

developed unless it can be repeated for a time; conditions that enabled it to form were treated as a kind of causal factor.

The meanings and functions of the behaviors changed over time, as respondents grew to appreciate them or rely on them in new ways. Zinnia began purging as a way to compensate for stress-induced over-eating, then found that purging became a goal in itself.

And so to compensate for the eating it became – I turned to different ways of purging. But then it became sort of – there was a time when it wasn't even about the eating anymore. I sort of got a fulfillment in the purging itself. Sort of that was one of the only ways I could sort of feel anything. And that became the rush. As opposed to the actual eating in and of itself. (Zinnia, T-BN)

Lynn (R-AN) said that the place of her eating disorder in her life changed over time. She said she had started because she lost weight and connected this to being more successful dating men. But after she married, there were new reasons to continue:

[W]hen I got married, it continued. And, that really had to do with kind of me stepping out on my own. I married someone who my parents hadn't met before I brought him home. They weren't thrilled about that. And, I knew I'd taken some big steps. And, I just didn't have – inside me I didn't have confidence that those were the right steps.... So, I think that's what kind of kept it going. I know kind of where it started. And, the thing that kept it going was kind of a lack of confidence and a feeling that maybe I hadn't made good decisions. And, I had this new relationship that I wasn't really feeling that I was being particularly successful with.

Both of these examples demonstrate the changing meanings and functions of eating disorder behaviors over time.

Respondents adapted to AN and BN physically by developing a new sense of what was normal; eating disorders became second nature and seemed to have their own momentum.³ Several with BN described how easy vomiting became over time, to the point of being easier than swallowing and digesting normally.

³ Isabelle used this term later in the interview when I asked her to respond to different models of eating disorders. She compared it to addiction, and said "...you use it out of habit for so long that it really becomes

[I]t was so bad that my throat stopped knowing which way to go. From vomiting. That's pretty much the nutshell. I think I vomited – it got up to be about five times a day. I don't know if that's a lot or a little. But, it was to the point where I would stand there and think "Swallow." And, my throat wouldn't know how to swallow anymore. (Irene, R-BN).

... I had gotten to a point where I would literally – it was a mental thing. And so when I would see food and I would eat food I had gotten to the point where I would actually mentally make my – I would feel sick. And then I would throw up. It wasn't that I induced it. It would just – that was just the way it was. So that's why I'm saying it wasn't something that I could just – thought that I could just turn on and turn off. Before I was inducing the vomiting myself by either putting my toothbrush down to the back of my throat or a straw or my finger or whatever. But it had gotten [automatic] – that's when I realized I really had a problem. (Vanessa, R-BN)

Mireya talked about her body's adaptation to bingeing and purging:

I think basically it just got easier and easier, like, physically to throw up. And I think also there's something about a release or just a chemical addiction to food too that it kind of fed into it because the more I ate the more I knew I didn't have to, like, digest it. So it's just easy to purge. So I think physically it just got easier.... in eating, like, tons of like, ice cream or sweets like, there's this, I think for me, something that's set off by that that just completes – like, a complete disconnect from, like, anxiety or, like, issues that are going on. And the, like, amount of sugar probably that I process or amount of, like, junk food – like, there's something, like, chemical there that, like, for me makes me want to do it again the next day. (Mireya, T-BN)

She felt she developed a "chemical addiction to food", which contributed to a cycle of bingeing and purging.

Betty and Ingrid, who were in treatment for AN, also described a process of adapting to a new sense of what was an appropriate amount of food.

I just didn't eat enough. And, I think my stomach just got small. And, over the years it just became a way of life. I never missed a meal. I mean, I don't do anything like that. I've never tried to throw up or anything. I mean, I hate that. I just wouldn't have ever done that. But, I was real careful about what I ate. (Betty, T-AN)

second nature." The concept of second nature could be linked to the idea of habitus; "nature" can be learned and acquired. Genetics, which correspond to what might be called "first nature" are not the only way of explaining persistent bodily dispositions.

Betty explained that she adapted physically, and soon interpreted her condition as located in her stomach rather than as a psychiatric problem. Ingrid explicitly compared her AN behavior to “living and breathing” because it had come to seem so natural.

...[B]ecause you ignore the essential feeding mechanisms that most people would say if they're even slightly hungry “Wow. I'm hungry.” You just think “Wow. So, I'm hungry. So, big deal. We can go another day without it.” It becomes a natural life. Like living and breathing.

The eating disorder behavior comes to seem like a “natural life”, rather than a disruption or departure from it.

I have highlighted the temporal dimension in this section, because causal factors changed over time and the repetition itself exerted an influence. This dimension adds yet more complexity to the causal factors described earlier. There are many more examples that could have been chosen to convey respondents' perceptions of the dynamism and contingency involved in the development of an eating disorder.

To summarize, accounts of the development of eating disorders did not tend to draw on simple causal factors but complex, compound, factors interacting over time, with a role for the interpretations and actions of the person. The valorization of thinness, coping with stressors by means of an eating disorder, and repetition over time were impossible to capture simply as “biological” or “environmental.” In the next section I focus only on those causal factors relevant to genetic understandings, then turn to the role of agency in the development of eating disorders.

3. SELECTED CAUSAL FACTORS: COMPATIBILITY WITH GENETIC IDEAS

Above I have presented examples of causal factors that cannot be easily reduced into biological, environmental, or agentic components. In this project I am interested in how respondents incorporate genetic ideas into their causal accounts. Some causal factors may be

“fertile ground” for genetic ideas, others may be less. In this section I present results from a more focused and selective coding for causal factors according to their expected compatibility with genetic causal accounting. I draw from the same interview material as above. I will assess whether these factors are in fact related to reactions about genetics in Chapter 5. The purpose of this section is to describe how respondents speak about causation with regard to this select group of factors relevant to medicalization and geneticization.

First, any account of genetic causation would have some role for the individual body where the genes are located - the genes are located inside the physical person somewhere, not outside the person. By this logic, *a causal factor that is biological would be more compatible with genetic causation* than a causal factor that is not. If somebody talks about a chemical imbalance or a digestive problem that precedes the eating disorder, that would count as a biological factor. Bodily appearance does not count here because it is ubiquitous and difficult to see as an exclusively biological factor, even though it is an aspect of the material body. (Discussions of body size or type, having a slow metabolism, and weight gain with puberty or pregnancy might be understood as genetic, but are not clearly “biological” because respondents relate them to social understandings of weight and gender.) I also do not include physical characteristics that are developed as a result of eating disorder behavior, such as the idea that it became physically easier to binge and purge over time, nor digestive problems resulting from the eating disorder.

Second, an account of individual causation that invokes some kind of *enduring individual disposition would be more compatible with genetic causation* than one that does not. By “enduring” I mean a disposition that is not described as the temporary result of a specific situation or event but something that persists within the individual across multiple

situations. The word “disposition” is general enough to encompass both social and biological causation; Bourdieu used the word in connection with “habitus”⁴ and it is also compatible with biologically-based concepts. (To use “habitus” instead would presume social origins, thereby truncating my analysis by prejudging respondents’ perceptions of biological causation as fundamentally social. My analysis is agnostic about causation.) A person who says that her perfectionism, addictive personality, or tendency toward depression contributed to her eating disorder is citing an enduring disposition. By contrast, a person who never mentions a disposition might be less likely to embrace genetic causation, because she is not already connecting her disorder to some internal entity or force. This is important for how respondents might think about gene-environment interaction, as the effect of an environmental factor may matter more or less for certain kinds of people.

Third, if an environmental factor is strongly linked to abuse, trauma, or injustice, there could be resistance or rejection of genetic causal explanations on moral grounds.

Every respondent cited environmental causal factors. But some of these may be more at odds with genetic explanations than others for respondents. For example, if someone mentions child sexual abuse as a reason for the disorder, she may see genetic hypotheses as potentially offensive or simply inapplicable to her situation, depending on how she understands genetics. Similarly, even though many respondents spoke of gender in relation to their eating disorder, some sounded more critical of it as a social problem, rather than just accepting it as a fact of life. These respondents may also resist or reject genetic causal explanations because they draw attention away from important social factors. I now turn to each of the proposed factors

⁴ In *Logic of Practice*, sociologist Pierre Bourdieu defines the “habitus” as “systems of durable, transposable dispositions, structured structures predisposed to function as structuring structures, that is, as principles which generate and organize practices and representations that can be objectively adapted to their outcomes without presupposing a conscious aiming at ends or an express mastery of the operations necessary in order to attain them” (Bourdieu 1980:53).

in turn: biological characteristics, dispositions, morally charged environmental factors, and relatively greater gender consciousness.

3.1 Biological characteristics

About a third of respondents (n=18) mentioned biological characteristics that were connected to something psychological. Ten spoke of genetic contributions, two hinted at such origins by speaking of conditions running in the family, and five spoke about biology without mentioning genetics. Respondents mentioned biologically based addiction, depression, obsessive-compulsive behaviors, and low self-esteem. Some also suggested more direct biological contributions to AN and BN.

So you were asking about other factors. Genetics I think is. And then I have four sisters and three brothers. And I believe every one of us has some kind of an eating – maybe not a disorder. Two have been diagnosed with an eating disorder. Two other sisters. But everyone has some issue around food and weight. No one is overweight. One sister is still quite a bit under-weight. So I think genetics is a big link. And then when I have talked with cousins I have been informed that they have – that they are dealing with eating disorders. Anorexia and bulimia. So I think that's a really big factor in my family. (Reba, R-AN)

Few respondents (4) brought up some physical characteristic as a contributing factor. Alyce mentioned that she was “sensitive” and “reactive” to food, which went along with food allergies. Mary said that hormonal changes from pregnancy contributed. Two respondents said that their bodies' tendency to lose weight rapidly was a contributing factor: Amy said that having a high metabolism was “part of it” and Fiona, said that her body “just dropped more” weight even though she was eating the same restrictive diet as her identical twin (who also had an eating disorder).

The variety of biological and particularly genetic causes will be more carefully explored in Chapter 5, drawing on material from later in the interview.

3.2 Dispositions

Dispositions locate the problem in the individual. Even though the characteristic may have been acquired through interaction with the environment rather than inherent, it is a transposable disposition that the individual brings to new experiences, a “throughline” across different situations. Such a throughline means that when trying to understand behavior, one might see the individual as having a *pattern* of responding, as bringing something to the situation and being predisposed to interpret or react to the environment in a particular way. It is fertile ground for genetics because if a problem is already located internally, it is a shorter cognitive step to thinking of it as biological than if it were not. Dispositions are also cognitively closer to genetic explanations because they limit agency; an individual disposition may be theorized as a constraint upon choice (I couldn’t help it, that’s the way I am).

Thirty-two respondents clearly cited dispositions as causal factors. They frequently mentioned more than one, including mental illnesses, and attributed them to the environment, biological, and unspecified other sources. Sixteen mentioned perfectionism. Several spoke of “type A” personality, competitiveness, “obsessive personality” or obsessive-compulsive behaviors. Many spoke of generally feeling insecure and self-critical, or having low self-esteem, “negative self-concept”, and feelings of “not being enough”. It was also common for respondents to talk about insecurity in relationship to other people, such as “an “emotional hole” that needed filling, being a people-pleaser, not being able to “bounce back like normal people” from the disruption of a relationship.

Ten of these 32 also mentioned conditions officially classified as mental illnesses in the DSM: depression, anxiety, obsessive-compulsive disorder, phobia, bipolar disorder, and

addictions based on self-report (e.g., self-descriptions of “OCD” behavior might not mean a diagnosis of OCD). Discussing a mental illness was not incompatible with talking about personality traits. Karen spoke about both as she described taking on more and more responsibilities at work and how difficult this was because of her perfectionist nature:

I mean they just kept piling stuff on me. Because, I was a perfectionist. (She mimed patting papers on the table, as though to straighten them out and make them perfect.) And, I love to just do everything I could to make people happy. And, I got very stressed. Very stressed and weak. And, I didn't know what OCD was at the time. And, I was really – it really started coming in. Playing a role. I was getting – having a lot of rapid, just racing thoughts. And, kept thinking about things over. My desk had to be perfect. It was just getting really bad and out of hand. (Karen, T-AN)

Personality traits and tendencies toward mental illnesses co-existed in causal accounts.

Although mental illnesses are medicalized and personality traits are not⁵, respondents did not see them as incompatible.

People who mentioned dispositions also identified environmental causal factors. Having a certain personality did not preclude being influenced by one's environment, nor vice versa. Hannah spoke of her mother's dieting along with several dispositions as causal factors: being people-pleasing, perfectionistic, “type A”.

So, risk factors. Probably having a mother that dieted I guess would be one. And, having a self-perception of being chubby growing up. And, being very people pleasing. Wanting everyone to like me. That's a huge one I think. That's probably one of the top ones. I think I really just wanted – and, I was perfectionistic, too. Like I wanted to be the best at everything. I was already the best at violin. I was the best at tennis. I was getting straight A's. I wanted to be the best looking, too. So, definitely the type A. Wanting to be number one. (Hannah, R-AN)

Hannah, like most respondents, listed multiple factors and did not feel compelled to choose among them. Dispositions co-exist happily with environmental factors and this co-existence may transfer to ideas about gene-environment interaction.

⁵ Interestingly, some did use language that suggested the medicalization of personality traits: Joelle spoke of being a “recovering introvert”.

Indeed, environment was identified as a cause *of* the disposition by many respondents. Some located the origins of their disposition solely in their family environment, others in genetics, and some in both. Deena said she was profoundly affected by her father's absence from the household when she was young:

I always felt like I needed to impress my dad. Like, to get his approval. And, he was always – he's depressive. Depressive alcoholic. And so, I felt like I had done something to disappoint him. And so, I was always a perfectionist. Like top of my class. Best grades. I'd always bring home all my A's on my report card. And, always like, what could I do to get his attention? And, I think that affected my eating. Very much. Like that whole type A perfectionist kind of mindset. (Deena, R-AN)

Although Deena identifies her own perfectionism as a contributor to her AN, she attributes it environment by describing it as a response to a difficult situation rather than an inherent trait. Her attempts to get her father's attention and approval created a more general trait of perfectionism.

By contrast, Liana suggests that her perfectionism may be related to her family's genetics and cites a variety of traits and disorders that she sees as potentially related.

I think I had, like, a typical personality for somebody with anorexia nervosa. The type A perfectionism. Like, always on-the-go kind of thing. I have read in a lot of books that like a lot of disorders in my family. Just, like, genetics and stuff. Even if it's not, like, DNA genetic. I think it can also be, like, your personality. And, a lot of my family's – especially one side of my family is, like, really like goal-oriented. Like, really expects excellence kind of thing. I also have, like, alcoholics in my family. Panic disorders. And, stuff like that. So, I think that's a – kind of has impacted me like, just from the start. Like, I kind of was like more prone to have something. Some kind of disorder. And then, stress from my, like, sport, like, dance was a big impact. And, I think my personality as being a people pleaser. Like, I think that's a big impact. (Liana, T-AN)

Liana describes genetic origins for personality and leaves ambiguous whether some of her family's influence might also be classifiable as “environmental”; a family that “expects excellence” could have affected her by means of its expectations, rather than having genetics

underlie their expectations as well as her own behavior. (Concepts such as “DNA genetic” will be discussed in Chapter 5.)

Fifteen respondents did *not* cite an internal disposition as a causal factor. They cited many of the same factors that other respondents did, without theorizing a disposition. Rather than environment or genetics producing an enduring transposable disposition which in turn shaped actions and reactions to the environment, many of these respondents spoke about environment, actions, and reactions without an intervening disposition. Environment and experiences were certainly important, but they provided contexts, understandings, or lessons rather than instilling a disposition. (Those who did explicitly theorize dispositions also described the environment in these ways; the difference is in whether they talk about dispositions.) They may have become “obsessed” by losing weight or been depressed by a situation or a relationship, but these were temporary states of mind rather than enduring dispositions.

Some in this group grounded their eating disorder behavior in their own decisions and values, which is at odds with a medicalized understanding of eating disorders. I provide one example, though it is not representative of the group of 15.

R: I think my biggest thing was just that I have a history of being overweight. And I have, like, such a fear of ever being overweight again. And I place so much value on being skinny.

I: Can you talk about that? Your fear of being overweight and the value you place on being skinny.

R: I feel like my happiness has a lot to do with my weight. And I’m really into, like, fashion and everything. And I feel like everything looks good when you’re skinny. And I feel like if I were overweight again, like, I would be so depressed to have to go buy all new clothes. And I wouldn’t feel like anything looked good. And I would be – I wouldn’t be as outgoing. Because if I were, like, trying to introduce myself particularly to, like, a boy or something, I would, like, automatically be thinking that they thought I was fat. And so I just know I wouldn’t be happy if I was ever fat again. (Yolanda, T-BN)

Yolanda centers the eating disorder in her equation of thinness with happiness and later goes on to explicitly reject ideas about personality disposition: “I don’t have an eating disorder because I’m, like, concerned with being perfect or I’m concerned with controlling something. I just don’t want to be fat. I mean it’s, like, a lot more simple for me I guess.” She narrates her decisions as her own, rather than compelled by some problematic part of herself.

By not mentioning a disposition, these respondents arguably put the focus away from psychological characteristics within themselves and more onto specific situations or relationships, lessons learned from their environments, or simply deciding for themselves that weight loss was necessary to be happy. This did not mean that these respondents necessarily saw their eating disorders as free choices. While some, like Yolanda are arguably more agentic because they describe the eating disorder in terms of free decisions to maximize their happiness, many sounded very constrained or compelled by their environments. I do not propose that this group as a whole claims more agency than those who talk about dispositions.

To summarize, most respondents talked about some kind of disposition or bio-psychological factor or both. People who spontaneously brought up biological causal factors, including genetics, before I asked any questions that explicitly or implicitly suggest biological origins, seem more likely to endorse it in the later parts of the interview (an obvious proposition for those who mention genetics to begin with). It also seems possible that people who talk about dispositions as causal factors will connect them to genetics later in the interview, even if they had previously connected it to the environment. Because all respondents who talk about dispositions and biological factors also discuss environmental factors, I would expect them to endorse complex forms of genetic influence that include

environment, rather than solely genetic accounts. The factors I have discussed so far could be ordered as follows, from most likely to embrace genetics to least likely: biological factor > disposition that is not environmentally generated > environmentally generated disposition > no disposition.

3.3 Morally charged environmental forces: abuse, trauma, injustice.

Although environmental factors frequently co-exist with biological and dispositional elements in many narratives, here I characterize a subset of environmental factors that may discourage thinking in terms of biology. If a moral or social injustice has been done, respondents might reject a simple genetic explanation or any role at all for genetics. Some environmental conditions are described in ways that suggest moral injustice, abuse, trauma and the like. As above, here I examine only interview material prior to asking about specific terms for eating disorders (e.g., mental illness, etc.); respondents may bring up other traumas and injustices later. I speculate that if people are bringing up morally charged environmental causes, whether connected to gender inequality or traumatic events, they may be less likely to endorse simple or even complex genetic causation and more likely to identify negative moral implications of genetic causal explanations.

There is great diversity within this category, and it may be too broad narrow to capture environmental factors that may be crucial for genetic reasoning, if there are any. I included abuse by parents but usually not bullying or teasing by peers unless I interpreted the respondent to think of it as abuse. I included death of a parent or spouse but not break-ups with boyfriends. Many troubling and important family situations were excluded – parental divorce, difficult move to another country, abandonment by a parent, alcoholism, parents described as emotionally or otherwise dysfunctional. I did not include having a disability or

being injured. Seventeen people were classified as having abuse, trauma, or an unusually strong sense of injustice regarding gender in connection to eating disorders.

Eleven people described abuse of some kind. Of these, six reported physical or sexual abuse as a child (Amy, Gillian, Irene, Paula, Reba, Yvette). These and others also described witnessing domestic violence, sexual assault, and extreme peer bullying (e.g., Ingrid noting that “seven years worth of all this abuse really pretty much stifles if you ever had any self-esteem. It doesn’t exist anymore”).

Two described not abuse but trauma and grief from the death of a mother (Tammy) or husband (Natalie), which warranted coding because these respondents brought it up repeatedly during the interview.

Four spoke about gender in ways that seemed central enough to their self-understanding to constitute a morally charged environmental cause. Thirty-two (or more) respondents spoke about gender in connection to eating disorders, from feminist analyses to less reflective statements about wanting to be thin in order to be more attractive to the opposite sex. The four selected here stood out as well-developed statements in which gender inequality was central or articulated in moral terms. Very early in the interview, Alyce (R-BN) stated that “I feel a desire for justice... I want people not to feel ashamed like I did. I want people not to be targeted as being sick the way I was. I want it to be seen as more a rational reaction to the conflicting expectations put on people. And, unrealistic standards on women in particular.” Barbara (R-AN) said she was “really passionate”, “frustrated” and “angry” that “powerful, powerful women” are “sitting there doing caloric charts instead of like, going to a political debate or things like that. And I mean I do it too. But it gets me so

angry that so many of us do it.” Carol described her early experience of AN as a reaction to her depressed mother and restrictive gender roles.

I was eight to nine years old. And, growing up female was not looking very good to me. My mom suffered from untreated depression. And so, I grew up in a Polish Catholic family. And, the role of the female was pretty well established. That you had children. And, you raised your children. And, you took care of the house.... I was terrified of having to fit into the role. Because, it – I think as a child I thought “Oh, my God. This means I’m going to be like my mom. Which means I’m going to be depressed. Which means I’m not going to be leaving the house....” And so, I for some reason chose to stop eating as a way to kind of say “Help me. Get me out of here. Show me some other way.” It was a cry for help. I didn’t get that help. But, for some reason it was what I chose to do. (Carol, R-AN)

Carol portrayed herself as “choosing” AN as a “cry for help” within an oppressive context.

Margaret’s experience of therapy involved getting angry, which she connected to anger about gender inequality: “I stayed pissed off at the world for a while. I was becoming a feminist. I didn’t know it.” I asked her to elaborate on the connection to feminism and she said she developed that understanding after studying feminism formally in graduate school.

...to me it [feminism] seems to connect the dots. I don’t think I had this real clear understanding of what eating disorders were about until I started understanding more about gender inequality and women’s voices being silenced. And their feelings being kind of constrained to certain categories of feelings. (Margaret, R-AN)

Although they are all very different, these 17 respondents may have more reason to resist genetic explanations than the other respondents, all of whom also brought up environmental causal factors. The role of morally-charged environmental forces in genetic reasoning is examined in Chapters 5 and 6.

3.4 Gender consciousness

Some respondents displayed relatively more consciousness of gender than others as they spoke about eating disorders (n=23). I would expect this group to resist genetic explanations as a diversion from a social problem. These respondents included the four who

spoke about gender and were included in the “injustice” group along with 19 others. I defined gender consciousness expansively to include any statement that indicated any hint of critical distance from gendered norms of appearance or behavior. Again, I restricted my assessment to material appearing spontaneously before the first “reaction question”. I excluded gender-relevant statements about wanting to be attractive to men, being boy crazy, wanting to look nice, unless accompanied by implicit or explicit gender-relevant criticism of these themes.

Most respondents categorized here spoke about causal factors in ways that somehow questioned or criticized gendered norms of beauty and behavior, sometimes very subtly and vaguely. Frequently they articulated themes already described above in connection with the valorization of thinness, the first of the three compound causal factors. I also included gender-consciousness of a different sort, like that of Ingrid:

I think my feeling is that – and, I have talked to many, many other women young and old who deal with anorexia. I think that the root of the problem is lack of self-esteem. And, the desire to be perfect. Because – or, to have a perfect figure. Or, to not eat. Because, the eating – it’s a lack of self-love. And, the food is a way – if you eliminate the food, you learn to love your body better. Because, you don’t like your body for what it represents. It either represents emotional or physical unhappiness to you. Or, to me. Based on sociological and psychological factors. So, for example I don’t like my body because I don’t want to look like a woman. Because, I fear the sexual ramifications. And, I don’t want to be a woman in that way. I would rather be childlike. Nymph-like. Unisex as it were. And, not because I have a sexual preference one way or the other. But, because I don’t want the responsibilities that come along with being a woman. I fear sexual relationships. I just don’t really want any part of them. There’s too much involved. (Ingrid, T-AN)

Because respondents’ gender-related comments have been included in previous sections, I will not describe them further here. These respondents may be most resistant to genetic explanations of their eating disorders.

4. UNCERTAIN AGENCY IN EATING DISORDERS

In the above I described how respondents talk about causal factors, including those expected to be most relevant to their reaction to genetic explanations. As demonstrated by several examples above, they did not always see their behavior as the end result of causal factors but also as in some ways as the result of their own agency. They were actively pursuing weight loss, finding ways to cope with stressors, and learning and practicing ways of purging. It is not surprising that they would consider the role of their own agency because they are talking about their own behaviors, which are usually thought to be under personal control. Even though eating disorders are classified as a mental illness, people who have them may retain non-medicalized ideas about personal volition, rather than seeing themselves as acting on the basis of a mental illness. A comment from Thelma, currently in treatment for BN, illustrates this idea:

R: I just didn't necessarily, like, feel that in control of my eating. Like, it just was something that just felt like it happened TO me. Or it was something that was kind of out of my control. Which I mean is obviously a very, like, flawed logic.

I: Why?

R: I mean of course you're absolutely in control of your own actions. Like, that's simple and basic. (Thelma, T-BN)

In this section I examine how respondents talk about agency and their own responsibility for the disorder or related behavior. This sub-section complements the previous section on causal factors by focusing on agency, which I do not consider to be a causal factor but nevertheless important to the development of eating disorders and a repeatedly-discussed aspect of respondents' experiences. Agency will return in the next chapter, when respondents reason about what an eating disorder is, principally whether it is more like a choice or more like a disease (respondents' understandings of what eating disorders are is the topic of Chapter 4). It is also related to stigma and how outside observers view people with eating

disorders, because for many respondents genetics help make the case that an eating disorder is *not* a choice. Stigma in eating disorders is closely tied to the perception of outsiders that the behavior is a choice and the person could stop if she wanted to. I examine these areas in greater depth when I describe how respondents would ideally like others to see eating disorders (in the last section of this chapter), and in Chapter 6 when I discuss the perceived implications of genetic causality.

Before continuing, I address a few definitions of agency and language to talk about behavior. As noted at the beginning of this chapter, I am using a broad definition of agency in this project to refer to the active subject, who can decide, will, choose, control and direct her life or some part of it, rather than as the passive receiver or product of other forces. I use the word agency almost interchangeably with control, choice, and volition, to convey the capacity for voluntary action (Marshall 1998). In this project I have tended to use the word “behavior” rather than “action” to refer to what people with eating disorders do, that is, restricting, purging, and bingeing. I understand the word “action” would imply more agency than “behavior” and my use of “behavior” reflects both the medicalization of eating disorders and the worldview of respondents as articulated in the interviews. The politics of whether to use the word “behavior” or “action” are well captured by Gillian, who reflects on her past use of the agentic noun “plan” to describe her restricting.

I think that especially since a lot more people are aware of it in my family. And, the treatment team that I’ll be returning to at school. I think that now it’s very definitely not a plan. And, if I do anything plan related, it’s not called a plan anymore. It’s called restricting. Or, it’s called purging. Or, *it’s called a behavior*. It’s not called a plan anymore. (Gillian, T-AN, emphasis added)

Plans and action connote greater agency than behavior. If I were to use the word “action” instead of “behavior” I would seem to be promoting a choice-based model. I chose to use the

word “behavior” (and the term “eating disorder”) because it reflects how respondents speak about eating disorders, even though it seems to promote a medicalized model. (I have to choose words in order to refer to contested concepts, but my choices implicitly silence some of the contestation.)

Most respondents used language that both connoted personal agency and control *and* a lack of it when discussing their eating disorder. The level of responsibility, agency, control was implied indirectly through language used and addressed directly in discussions about that topic. Put very simply, their ways of talking about eating disorders – language used, models proposed or rejected – could be categorized by whether they implied more or less agency or were somewhere in between. I used three broad umbrella categories: agentic, quasi-agentic and non-agentic. These describe language, rather than distinct styles of thought held by respondents; I did not categorize respondents as using one as opposed to another because all respondents, without exception, used two or all three to describe their eating disorder. After describing the three kinds of language, I will describe their frequent co-occurrence and suggest reasons for it.

As above, I limited my description and analysis of this material to early sections of the interview, prior to questions about specific eating disorder terms. Because a question later in the interview asked respondents whether eating disorders can be described as “a choice,” language and views about agency mentioned before the “choice” question are less influenced by me.

4.1 Non-agentive language and concepts

At least 45 people (90%) spoke about their eating disorder in terms that suggested a lack of agency or control over their own behavior before I asked the reaction questions. I describe diverse overlapping themes below.

I interpreted a lack of agency when respondents described the eating disorder as a separate entity from them: a disease, an abusive boyfriend, a demon, a monster, a voice. Although they might be able to “fight” or “ignore” this entity, it was distinct and therefore not directly controllable the way one’s own behavior is normally thought to be. Indeed it had its own agency, and sometimes tried to convince the person to do things that were against her best interest.

Like, I went to eat with my parents. And, like, I knew what I had to do. And, I knew, like, everything. Like, I knew it. And, it was like – it’s almost like the eating disorder just wants to get in my head so bad that that’s what’s frustrating. . . . And, knowing everything that I had to do. But, still getting upset because the eating disorder was just getting stronger and stronger... they don’t know, like, all the secrets and all the stuff that the eating disorder can yell in your ear all the time. But, then having, like, the logical side of me be, like, “No. You know you want this to go good. You want to look back and say – know that you can do this. You’re with your parents.” And then, the eating disorder is like, “Well, just do what I say. And, you’ll feel more relaxed. And, you’ll have a better visit. Because, you’ll be relaxed.” And, all this stuff. And so, it’s really frustrating having to decide what to do sometimes. (Liana, T-AN)

Liana contrasted her “logical side” with the eating disorder voice. Clearly the voice is external to her in some way and she cannot control it. Similarly, a disease, demon, or monster are conceptually separate from the person and imply a separate agency. Several people shared Hannah’s (R-AN) view that it should be seen as a disease rather than a choice made by the person: “I would want people to understand that it is like a serious illness. And, like, that it’s not something that they chose for themselves.” Again, this was said before I presented them with models of eating disorders to react to.

This separate entity was sometimes more closely tied to the self than a monster, but still unwanted and difficult to control. They spoke of an “evil me” bent on destruction, something in me, an urge or drive, a compulsion or obsession. Joelle, recovered from BN, commented that at the time “it felt like an impulse. It felt like something I had to do as a compulsion.” Jackie compared it to “something inside of me” and elaborated as follows:

It’s just I know people need food. I know that. Biology tells you that. If you’re hungry, your stomach growls. But, there’s – I don’t want to sound like – not like a schizophrenic voice. But, there’s this other – there’s this half of me that’s going “You don’t need to eat that. You’re ugly. You don’t deserve to eat that.” Or, “If you eat that, you’ve got to throw it up.” And, it’s taken me a while to realize that it’s not me saying that. And, it’s a really uncomfortable thing. Because, I feel like I’m kind of re-meeting myself in recovery. (Jackie, T-BN)

Jackie identifies a “half of me” that is “not me”; separating the eating disorder voice from one’s true self is part of recovery for her. Mary said it was scary because “you’ve got this abusive thing inside you that basically wants to kill you” (in treatment for AN). Yolanda talked about feeling compelled to continue eating during binges:

it’s like tunnel vision sometimes kind of. Like, if I get hungry. And I start eating something. And then I keep eating. And I keep eating. I know in the back of my head somewhere, like, that I should just stop eating. And I’m full. I don’t really need to eat anymore. But it’s like a compulsion to just keep eating and eat whatever I can. And it’s just something – it’s not – habitual isn’t the word. It’s just kind of something that you feel like you have to do. So that’s why it’s not as simple as just stopping. (Yolanda, T-BN)

Control could be lost not only because of something inside or a compulsion but also because the body itself became unruly. As discussed earlier in connection with the development of a habit and a second nature, respondents described purging without agency (Irene, Vanessa). In a variety of ways, respondents reported that the eating disorder experience involved a lack of agency or a divided agency, depending on how the agent is defined (e.g., as part of the self

or not). (The question of how to define and locate the agent is a theme throughout this project and I discuss it explicitly later.)

I also coded a lack of agency when a respondent used language that suggested a lack of power over the disorder in more subtle ways. For example, Amy (T-AN) lamented that “it’s frustrating because for every relapse that I’ve had, they always end up worse than the one before.” Saying that she “always *ends up* worse” suggests a lack of control over the weight loss, with a passive verb tense indicating that it is not her choice. Similarly, some described recovery in terms that suggested a lack of agency. They “snapped out of it”, something “clicked”, and for unknown reasons it was no longer something they wanted to do.

Many respondents also spoke of an illusory sense of control conferred by eating disorders. They suggested that eating disorders could *feel* subjectively like they were under the individual’s control, but that this was not true. Margaret thought her AN and BN were under her control and part of a power struggle with her family. When she no longer lived with them and the behavior continued, she began to perceive a lack of control:

Like, it was a cycle I couldn’t break... And maybe it’s because the people I thought I was doing this in opposition to were not there anymore. So it was just me. Me and my eating disorder (laughing). I know. I laugh. Guess what? This is not about mom and dad. It’s about you and your issues with food and your body. (Margaret, recovered from AN/BN)

What seemed at first like a response to a situation, came to seem like her own “issues”.

Many spoke of the eating disorder going “out of control” at some point and some said this loss of control was a defining feature of an eating disorder; if it were in control then it would not be an eating disorder. (Arguably this puts the person with an eating disorder in a difficult position when working toward recovery.)

4.2 *Agentic language and concepts*⁶

On the other end of the spectrum, I also coded language and concepts that I interpreted to connote agency. At least 41 people (82%) used such language before I presented the “reaction terms”. Most used both agentic and non-agentic language (at least 36). As with the above material conveying a lack of agency, there was diversity within this code. Although all people coded here expressed ideas that sounded more agentic, it was rare for people to say they chose to have an eating disorder, and if they did there was often a sense that the choice was constrained or forced in some way (to be discussed in the section that follows this one).

Several described agency early in the process of developing the eating disorder. This agency could be seen as part of the early phase of an eating disorder, or as something distinct from the eating disorder (respondents saw it both ways). They usually saw the origins of the eating disorder as volitional goal-oriented behavior to control weight. Lynn, who recovered from AN, recalled:

initially when it started it seemed to me like a good deal. I was like, “Yeah. I’m paying attention to this. This is working. I’m being successful. I’m losing weight.” So, initially it seemed like a good thing. It was only when it really started to just take up a lot of space in my brain that it started to seem wrong.

Hannah had participated with her mother in Weight Watchers, an organization and activity that assumes the importance of controlling weight and the ability of individuals to do so.

Vanessa heard about vomiting as a weight loss strategy from her friends: “I saw that was working for them. And so I took a drive. And it started working for me. So it was just

⁶ My focus on agentic language should not be taken as a claim that respondents were currently pursuing eating disorders. Only very rarely did respondents talk about choosing to have AN or BN as a current conscious commitment at the time of the interview. Natalie was an exception, and some others acknowledged that a part of them wanted to have an eating disorder, suggesting a complex multiple agency.

something I continued to do.” “Taking a drive” sounds agentic, as does evaluating whether it helps toward the weight loss goal. When people describe the beginnings of the disorder, they are often agents striving toward a goal. (This was an important theme that I will return to when discussing their reactions to the idea of eating disorders as “choices” in Chapter 4.)

Some respondents spoke in terms that suggested that their eating disorder was a kind of accomplishment. Gillian illustrated this well:

I feel like – for a really long time there was almost a sense of not necessarily pride. But, more of a accomplishment. Like, I mean obviously there was accomplishment whenever you’ve lost another two pounds. Or, whenever you went another day without eating. Or, eating a certain number of calories. But, like, even in retrospect you’re almost amazed at the things you could and can do. And, like, no matter how logical or how much you know to the contrary, you almost think, like, “I’m the one person in the world who can live on twenty calories a day. Indeterminately. (Gillian, T-AN)

Gillian’s and others’ sense of accomplishment is tied to the valorization of thinness and self-control. When respondents spoke of it in terms of something that you can fail at, compete for, “make a career of”, or use as a spiritual practice, it sounded like achievement.

Respondents tended to think of AN as more of an admirable accomplishment than BN because of this self-control; Gillian above went on to contrast the two.

[W]ith the bulimia it’s definitely there’s not any sort of pride there... there’s no pride in telling people how many calories you’ve binged on. Or, even if you don’t tell anyone [about restricting]. You can have a sort of secret pride that “Oh, God. I just went a week without eating. That’s pretty impressive.” But, you can’t be like, “I just ate ten thousand calories. That’s pretty impressive.” It’s more of a “God, I want to die now” feeling. (Gillian, T-AN)

Though bingeing and purging were not aspired to the way restricting was, there were agentic features nevertheless. Some who purged spoke of it as “productive”, suggesting a willed action, despite feeling ashamed. Zinnia said that unlike diuretic teas and exercise, with vomiting “I can see the product” which provides “satisfaction” that something has been

achieved. For Thelma, purging “is something that’s productive. There’s an immediate kind of, like, goal that’s reached.” It could be talked about in terms that imply achievement; “I got really good at it” (Rebecca, T-BN).

Purging was also a skill that required practice for several respondents. Delia recounted that it was hard at first, but she persisted: “It took me a long time to be successful at it. Because, a lot of the time I had to put my finger – I had to force it. And, it was really hard. And, it hurt. And, it was painful. But, eventually I perfected throwing up.” As quoted earlier, for Jackie, “the first time I did it, I hated it. Thought it was disgusting.” But soon “it sort of became a habit.” Thelma described how it was difficult at first, but through trial and error found a way that “worked”:

Well I mean obviously there’s, like, a gag reflex. And your body says, like, “Don’t. What’s going on. I don’t like this. Like, make this stop.” But I just – I don’t know. I kept – just kept trying until eventually it worked. And like, whether, like, how it worked with different foods. And like, whether I – like if I drank a big glass of water before I ate or a big glass of water after I ate. Or like, drink a glass of water like, while I was eating. Like, how that like influenced – like, impacted it. (Thelma, T-BN)

Rebecca found that ice cream was easier to purge than other foods. For many of those who purged, it was something they consciously worked on until they were able to do it easily. Some learned about purging from friends, even receiving direct instruction. Selena asked a friend how she stayed so thin, “And she was like, ‘Well I just don’t eat. Or if I do, I get rid of it.’” In fifth grade Gena persistently asked her friend, “How do you stay so small?” and soon the friend agreed to show her.

And, I remember sitting in her bathroom. And, her literally standing over me and telling me how to make myself throw up. I was like, “I can’t do it. I tried. And, this feels bad.” And, I thought I was doing it. And, I would just be spitting. And, I was like, “Nothing’s happening.” ... And, she sat over me. And, she was like, “You’re not doing it right. You have to do it like this.” And, she went in the kitchen. Ate like some cookies. Like a bag of cookies or something. And, went back in the bathroom and showed me. She’s like, “This is how you do it.” And, I said “Okay.” So, I did the

same thing. And then, she was like, “Well, you got to do your fingers like this.” And then, the older I got; I kind of figured it out. (Gena, R-BN)

It was not easy for Gena to learn how to purge. Not only did a friend demonstrate bingeing and purging, but she had to practice in order to figure it out. Vanessa purged with a group of friends, even describing it as a pact that held them together as a group. After her friends “turned me on to this idea” she began to purge along with them.

I had some friends that could throw up at the drop of a hat. We used to kind of joke about it. But I mean it’s not funny now. But we used to joke about it because teenagers – I had a girlfriend that could swallow something. And then she could throw it back up almost instantly. It was amazing. Because she had done it for so long. (Vanessa, R-BN)

Vanessa’s group of friends incorporated purging into their friendship, and she described it as a “pact” they had.

Respondents described actively protecting the eating disorder from discovery and monitoring by treatment providers, family, or friends. Eating just enough to avoid the nasogastric (NG) tube, or to gain enough weight to be permitted to leave the hospital, can be seen as evidence of calculated agency (even though healthcare providers would likely interpret it as part of the disease). Amy (T-AN) described herself and fellow inpatients in terms that sounded agentic: “it’s easy for us to play games and get crafty and sneaky and try to play the system.” She recalled psychiatrists’ response to her refusal to eat during a prior hospitalization for AN: “they were like, ‘Well, we will transfer you to a psychiatric facility and do an NG tube.’ I’m like, ‘Well, okay. I’ll eat just long enough to get out of here so you don’t do that.’” Outside a hospital context, respondents report similar practices: “So at home it’s a lot of hiding things. And lying about where I’m going to be. And stuff like that. And I don’t like that. Because other than my eating disorder my mom and I have a great

relationship” (Yolanda, T-BN). Similarly, battles with parents who are “forcing you to eat” imply that the respondent has a will, which connotes agency.

Several spoke about agency as it related to recovery and treatment, namely that one could choose to recover or make choices as part of recovery. Often this was a choice to seek treatment, but several recovered people also described a self-initiated process of recovery without treatment, which sounded very agentic. Kathleen avoided doctors because she was afraid they would force her to gain weight too quickly, so she developed her own alternative program.

[W]hat I did essentially was just take little baby steps... I said “...the first thing I can do is just stop weighing myself. I’m not going to do that anymore.” And, that’s what I did. I just stopped. And, I did not weigh myself anymore ever. And, that kind of made it easier to do the next step. Which was – I think the next thing was “I’m not going to count any calories anymore. I’m going to forget how many calories are in everything. When I look at a menu. Or, I go to get some food. I’m not going to do that.” Which I did. And, it just kind of went from there. And so, it was a very gradual thing.
(Kathleen, R-AN)

Kathleen was one of several recovered people who reported recovering on their own. Carly, a college student, also described what she called a “tapering off” process that relied on the “self-motivation” she learned as a swimmer.

You’re kind of like on your own. Like, you’re in the water. Your head is in the water constantly. And you’re constantly talking to yourself. And you have no one there yelling. You can’t hear anyone when you’re swimming really. So you kind of have to push yourself to work hard in practice and to get up at 4:30 in the morning to make it to practice if you really want to do it. And I guess that helped me kind of eventually to be like that outside the pool. (Carly, R-BN)

Irene characterized herself as deciding to take a “leap of faith” that she could be happy even if she gained weight by stopping purging:

I decided to let myself eat whatever I wanted to. As much as I wanted to. As long as I didn’t vomit. So, that’s kind of how I got over my bulimia. Is to just – whatever you do, don’t vomit. Just eat whatever you want. And, take the risk. Take the leap of faith that you can be happy if you’re overweight. (Irene, R-BN)

People in treatment also evidenced agency by working hard to stop behaviors, like Liana who described a meal with her parents (presented above). Betty, in treatment for AN, described concrete skills taught as part of cognitive behavior therapy. These skills teach the person how to stop or challenge “automatic thoughts”, to weigh the pros and cons, to alternative thoughts of actions.

...they teach you how to think things. How to – some of the skills we use are pros and cons. What are the pros if I do this? What are the cons if I do? And what are the pros if I don't, and what are the cons if I don't? ...And then, we draw interruptions...between the situation and the thought. What could you have done to create a different thought? And then, if you can't catch it then or turn it around then, you might could do it after the behavior. (Betty, T-AN)

Whether in treatment or not, respondents spoke about behaviors in terms that suggested they were agents capable of control, or learning how to be so.

Even after recovering, the eating disorder could still be something for an agent to struggle against. Willa's commitment to recovery sounds agentic as she weighs its costs and benefits against AN:

Honestly I would be lying to you if I said I just didn't think it still looked really good. But I think I've just gotten to a point where I've said “You know what? That would never be worth it.” I guess. So I know what it takes to be there. And I was so miserable. And so it's just not worth it. And so that's how I look at it I guess.” (Willa, R-AN)

The ability to weigh the options and choose one suggests agency, and suggests that a recovered person could choose to develop an eating disorder.

4.3 Quasi-agentic language and concepts

Given that most respondents spoke in terms that suggested agency as well as its opposite, it is not surprising that most also talked in terms that were in between. Most of these examples may be described as forms of constrained agency; will or volition is involved

but it is impaired, lacks essential information or support, or is forced or compelled by environmental circumstances. They are acting, which implies agency, but what they are doing does not feel under control or chosen. Arguably all agency is constrained because nobody chooses the conditions she find herself in, or what material she has to work with. But what I have labeled “quasi-agency” always involves a reference to constraints or compulsion, whereas language categorized as “agentic” does not. The eating disorder might be a way to act out against something bad and achieve something better: a sense of control or power, feeling soothed or numb or euphoric, to express needs, to seek love or care or attention. The kinds of language presented here were difficult to categorize as either agentic or non-agentic because both were implied. The lines dividing language coded as quasi-agentic vs. agentic or non-agentic are were difficult to define precisely, but 46 respondents spoke in terms that I classified as quasi-agentic (92%). The ambiguity supports the overall argument that agency in eating disorders is profoundly complex and confusing for respondents (as well as analysts).

Many respondents spoke of their eating disorder as a way of coping with stress, emotions or other problems as described in the first section about responding to problems as a complex causal factor. My focus here is on coping as a type of constrained or compelled agency. Deena noted that “Eating disorders is just one way that... some people choose to cope. Because, they don’t know how else.” Although she describes it as “choosing”, a coping mechanism also suggests constrained choice because the person is forced to respond to a challenging situation using whatever methods are available. In addition, as Deena says the “choice” of an eating disorder as a coping mechanism is made without sufficient information; they may be agents but they have very limited options (or “skills” in the words

of several respondents). Many respondents saw their eating disorder as a flawed and harmful coping strategy. Rebecca said that “it did help me cope. It just hurt so much more than it helped. And it got to the point more recently where it no longer helped at all. It just hurt.” As she began to work on recovery she “realized that there were more healthy ways to deal with things.”

Helen describes her AN as a “disease” that began as a coping strategy, then went out of control. She thus implies that before the coping mechanism goes out of control, there must have been some individual control and agency involved.

It’s a very difficult disease to battle. I don’t think that it’s something you can will away. I don’t think it’s something that people choose to have. I really think it’s – it serves as a coping mechanism in some ways. And then, it really can wind up being out of control. Which is what the case is with me. Or, was with me. (Helen, T-AN)

Ingrid (quoted earlier) elaborated on the alternative ways of coping, including drinking, chain-smoking, arguing, and over-eating. To summarize, coping involves agency but it is always constrained, qualifying it as “quasi-agentic” in this project. For this reason it was not easily assimilated into the agentic nor the non-agentic codes. Coping is in the space where the individual meets the environment, responding to external situations, drawing on learned skills, acting to preserve well-being but in ways that are not satisfactory. Respondents frequently link coping to other themes presented below, such as gaining a sense of control via the eating disorder, “using” the eating disorder like an addictive substance, and “choosing” to express or gain something by means of the eating disorder.

Several respondents sounded quasi-agentic when explaining how they “chose” to have an eating disorder. As with coping, such statements did not seem clearly agentic because the choices were circumscribed by respondents: it could be one disorder or another, or it was a choice made under duress. Carol said she “chose” to stop eating as a “cry for

help”, Deena above talked about “choosing to cope”, Sydney “chose” BN rather than AN because in her family excessive restricting would not have been noticed.

My mom has disordered eating herself. And my dad kind of encourages that in her. Because he doesn't think it's disordered for a woman to just eat like a tiny bird and never have anything caloric and to – and so to them – like, that's probably another reason *I chose – or I didn't choose. But I became* bulimic rather than anorexic is had I become anorexic I'm not sure they would have (A) noticed or (B) done anything. Because that would be normal in my house... [T]o them they thought I was trying to diet and I was doing it in the wrong way. (Sydney, T-AN, emphasis added)

Sydney finds the word “choose” to be useful but in the end not adequate to capture the sense of what she is saying; “became bulimic” is less agentic. Her self-correction illustrates the uncertainty about agency in eating disorders.

Similarly, half or more said it was a way to achieve a sense of control or feeling more powerful (at least 24 before the reaction questions began). Although “control” and “power” imply agency, in context these respondents are describing a false sense of control and power in reaction to external events being out of control. Claire said her AN helped her feel more powerful, in contrast to the powerlessness of an overweight body.

I just always felt very powerless because of the way that I looked. Like, because of my extra [weight] in places. I just always felt very powerless. And then, when I figured out how to have power over it, I think something in my brain was just like, “Ah!” (*Claire made a pouncing gesture with both hands.*) Like, glommed on. (Claire, T-AN)

Reba thought of her AN as helping her to control abuse from her father: it had “a lot to do with my being in puberty. Trying to not be abused. And also it gave me something to feel like I could control. When I couldn't control my body changing. And developing breasts and hips and female features.” Helen said her eating was “the one thing I could control. It was the one thing I could be good at. And, practice perfection with.” Jackie reported that “whenever I would feel out of control of a situation, it was just something that I could do to

feel like I had control over it.” Similarly, Hannah responded to harassment from peers at school by trying to find control elsewhere:

I started to just feel like I wanted to control something. Because, I felt like I couldn’t control that part of my life. And so, I think that’s when I gradually just started. I didn’t even realize it. I just started, like, cutting down on my portions. And, like, eating half of a sandwich instead of the whole sandwich. ... dilut[ing] my milk in my cereal. (Hannah. R-AN)

Karen (T-AN) identified the control as a harmful illusion: “It’s just complete control. Where you think you’re in control. But, you’re honestly – and, actually you’re not in control at all.”

Gena described a “double edged sword”, which appears to confer control but does not serve the person.

[J]ust the whole idea of wanting to control something. Couldn’t control anything else. But, I mean it was a double edged sword. Because, no matter how hard I try, of course you’re never as small as you want to be. So, this constant pursuit of perfection. And, finding happiness in something that doesn’t really happen. (Gena, R-BN)

As is apparent from these examples, many respondents linked the eating disorder to a sense of control that was illusory.

In addition to coping and feeling in control, eating disorders were a way to achieve other desired ends: a way to feel more confident and positive about oneself; to receive love, care, acceptance, attention or admiration from others such as parents and peers; a way to fill an emptiness or emotional hole; among other things. These are goals an agent might have, and the eating disorder was presented as a way to achieve them, albeit self-destructive.

Controlling weight was linked to physical attractiveness, popularity, and romantic attention, as would be expected in contexts that valorize thinness. For many it was more than this.

Nell talked about it as a “desperate plea” for something, which she saw only in hindsight.

... I had felt “I’m hurting here. I need help here.” And nothing was getting it. So it might have been some sort of desperate plea for “I need to be recognized. And this is the only way I know how to do it.” Because I wasn’t going to, like, go make bad

grades or go get expelled from school. I wasn't going to do anything negative. But at the same time it was kind of like, "I really need help here." So there might have been some of that too. Even though at the time I wasn't thinking that. (Nell, R-AN)

AN was Nell's way of communicating a "plea", which might also have been conveyed by doing badly in school. At the time she "wasn't thinking that", suggesting a subconscious type of agency that is recognized only after the fact. Carol also talked about it as an "attempt to ask for help" and an "attempt at a solution to my pain." Irene described it as a "desperate attempt at something" in reaction to her abusive mother:

When I was thirteen or fourteen or whatever. And, saying to her "I want to drink those chemicals. I want to make myself vomit." I might have said "I'm so fat." Or, something. But, I said "I want to make myself vomit." And, she actually said to me "Go ahead. Do whatever you want. I don't care what you do." So, that may have been part of – I don't know if I partially did it to get back at her. Like, "I'm going to be really sick. Watch me be really sick." I don't know. (Irene, R-BN)

As mentioned earlier in reference to Fran and Yvette, being thin could also be a way of trying to stay connected to a parent who abandoned the family. Thinking of eating disorders as a way to achieve something implies agency, but respondents felt constrained and ill-served by it.

If an action directly harms or destroys the agent, it raises questions about whether the action was agentic. Does (or should) agency connote more than free and unconstrained action but also *positive and self-affirming* action? I have not categorized agency according to whether it led to positive or negative ends; given that all respondents saw their eating disorders as harmful (directly addressed in Chapter 4), this would have precluded the possibility of agentic language to describe eating disorder behavior. It is worth noting, however, that when respondents spoke about pursuing self-destruction, they frequently described themselves as constrained. Sometimes this agency was not conscious and was only seen or attributed in hindsight.

Several respondents spoke about “using” the eating disorder for many of the goals above. “Using” implies agency, but in context it also evokes constrained, or misdirected agency in these interviews. Claire (T-AN) asserted “I think in this disorder there’s a lot of fear and a lot of feelings of control over life through using food and exercise,” Zinnia (R-BN) spoke of using food to deal with stress, and Wendy spoke of using food to deal with emotions. Just as coping by means of an eating disorder suggests compelled action within constraints, “using” suggests a misdirected agency, which I also interpret as a type of constraint because the agent lacks the option to act directly on the problem and must substitute alternative and harmful action (e.g., eating instead of dealing with emotions for Wendy). Olga linked “using” directly to addiction:

And I really don’t think it’s a food thing. I mean looking back on it I think it’s a mental disorder. I don’t think it has anything to do with food. It’s just like, alcoholism probably doesn’t have anything to do with alcohol. It’s a tool for something. So I think the food is just a tool that you use. Just like later on when I was checking things. It’s just something that you do to make yourself feel like you’re in control of something. (Olga, R-AN)

Half of respondents discussed eating disorders in relation to addictions before the reaction questions, and although this alone may or may not warrant classification as quasi-agentic, it does suggest that “using” in eating disorders is related to “using” in addiction discourse. I interpret the language of “using” to be a way of underscoring the aspect of addiction that has to do with responsibility for behavior even though that behavior does not feel volitional to the “user.”

Several respondents (at least 11) used the word “habit” to refer to their eating disorder at some point before the reaction section of the interview. Earlier I discussed the development of habits as an example of dynamic developmental processes. Here I focus on habit as quasi-agentic. I coded it as in between agency and non-agency because habits are

not perfectly under individual control but commonly thought of as things that an individual can use willpower to break. Habits such as chewing fingernails, smoking, and over-eating are treated in everyday U.S. life as something that the individual is held responsible for. At the same time, there is the sense that they are not voluntary; through repetition over time they have acquired a solidity that other behaviors do not have. This dual nature makes them quasi-agentic for this project. Habits may also seem more like quasi-agency than agency if they are self-destructive, in addition to not being freely chosen, but this was not a criterion for coding quasi-agency in this project. Several respondents described their restricting, bingeing, and purging as “habits” that are hard to break. Fiona referred to “habit” in a way that evoked addiction (like “using”), listing it as one of the problems associated with having AN: “You become obsessed only with yourself and your habit.” For Gillian, “habit” connoted an ability to control it herself rather than needing help from others: “I think that for a long time I convinced myself as much as I convinced everyone else that it was just a habit. And, that I could deal with it. It wasn’t an issue. I’d get over it. It was fine.” Although Gillian no longer treated it as a habit, Thelma still thought of it that way: “If anything, I mostly think of it as like, maybe a bad habit. Like people bite their nails or do all sorts of things. And you just like, keep it to yourself. And as long as it’s not like, hurting anyone else. Then it can’t be that bad.” Fran stated that she overcame her BN by treating it as a habit. She had little access to treatment and based her efforts on a book given to her by a medical doctor.

And, now this was twenty-two years ago. And, the book said – it was about eating disorders. And, I think bulimia in general. And, it said that it was a habit. I thought “Okay.” I smoked some as a teenager. By the time I was twenty-one, I was smoking two packs a day... [Later] I quit smoking cold turkey that day from two packs a day. And, when I read that it was a habit, I said “Okay. I can stop.” (Fran, R-BN)

Taking into consideration all three types of language about agency, it was rare for respondents to describe their eating disorder behavior as freely chosen, despite the use of agentic language. Without exception, all who used agentic language also used either non-agentic language (n=36, 72%), or quasi-agentic language (n=37, 74%) to talk about eating disorder behavior. These numbers probably underestimate the actual co-occurrence because I have only counted language that appeared before the first reaction question. This constitutes strong evidence for the idea of “ambiguous agency” in eating disorders, as represented by respondents in these interviews.

4.4 Reasons for the co-existence of agentic and non-agentic language

There are several reasons why it makes sense that respondents used a combination of agentic, non-agentic, and quasi-agentic language. I will suggest three straightforward reasons and one that is more nuanced and interpretive.

The first of the three straightforward reasons is that respondents experienced both agentic and non-agentic action at different times. Many respondents described the capacity to choose or control their eating disorder behavior as something that shifted over time. Many spoke of the eating disorder behavior beginning with voluntary weight control goals then going out of control. Recovered people and others spoke of regaining the ability to control their behavior after treatment or as they recovered. It is possible that these beliefs about control at different stages of recovery were presented by treatment providers and simply repeated by respondents, but respondents adopted them as their own in the interviews.

Second, for many respondents, some aspects of the eating disorder were possible to control and others not. Some, like Liana whose self-observation during a dinner with parents was quoted above, said it was possible to choose not to listen to or obey the eating disorder

“voice” but they could not choose to stop hearing it. In a similar vein, Beth thought “it’s something that you never really get rid of unfortunately. It’s always there. You just have to learn to turn that voice off.” Several respondents said it was possible to control things that “trigger” the eating disorder, but once triggered there was little one could do. Claire talked about managing her level of stress, sleep, and exposure to “triggers”:

...it’s like I have triggers. And, I have, like, trigger foods. And, trigger thoughts. Usually what makes it worse is if I let myself get into a certain pattern of thinking. Or, if I don’t take care of myself. Like, if I don’t sleep enough or exercise enough in a given day. Then, I’m more vulnerable. (Claire, T-AN)

Similarly, Carly (R-BN) spoke about avoiding foods that would make her feel like she needed to purge: “I kind of don’t let myself go to the cafeteria as much. Or if I do, I limit myself to, like, a certain amount of food. And, like, healthy food. Because if I eat, like, greasy stuff or something, I’ll feel really tempted to throw it up.” Irene (R-BN) feels that she is overweight but feels cautious about exercising because “I’m afraid. I’m very afraid of becoming obsessed. And, I’m very afraid of feeling bad about myself. Criticizing myself and giving myself a hard time if I don’t stick to a regimen. So, I’m afraid to try to lose weight.” She is recovered in the sense that she controls whether she throws up, but does not feel she can exercise without becoming “obsessed.” Lynn (R-AN) makes sure she keeps her weight up:

I never think about doing that crazy stuff anymore. And, I try to keep my weight up. Sometimes I wonder if there’s something in that. If I start to drift down in my weight, I’m aware of that. And, I do what it takes to stop that. And, put the weight back on. So, sometimes I wonder if I do have some fear about being tempted to go back to it. But, I never do. (Lynn, R-AN)

Respondents are controlling things that might “tempt” them to restrict, binge, or purge. There is a clear implication that if they were tempted they would give in, so avoiding temptation enabled them to maintain control.

A third reason why both agentic and non-agentic concepts might apply is that the person views the behavior differently when they are “in” the disorder as opposed to when they are not, such as when they have recovered. Many respondents said that the eating disorder felt like a way to gain control, to feel more powerful, part of their will, an achievement. They carefully avoided detection by lying to others, bingeing or purging in secret, and concealing their weight loss behind baggy clothes. But with the benefit of hindsight (or alternatively of “insight,” as medical professionals might say), they came to see those agentic feelings and behaviors as driven by the disorder. By contrast, some respondents credited themselves with more agency in hindsight. By saying that the eating disorder was a way of trying to communicate something or get needs met, a goal they were unconscious of at the time of the eating disorder, they suggest that the eating disorder reflected a type of agency (though not a conscious one). What seems like a compulsion at one time can seem like agency at another.

In addition to these three reasons, some of the apparent contradiction is likely to be related to the difficulty of self-interpretation: what is agentic and what is not? People are influenced by others’ appraisals and internalize these views.⁷ Respondents are aware that other interpreters see eating behavior as controllable; if you want to eat you can, if you are purging you must want to, and over-eating is a matter of willpower. Respondents apply these standards to themselves, even when they subjectively feel the behavior is out of control (as the quote from Thelma at the beginning of the previous section illustrates). It was hard for them to understand their own agency, and even when they came to an understanding it was difficult to articulate in the interview. The category of “quasi-agentic language” may convey this most clearly, as respondents both had and did not have control, often simultaneously.

⁷ This classic social psychological concept is represented by Cooley’s (1902) theory of the “looking-glass self”.

Many of my respondents struggled with the problem of interpreting their own agency for themselves and others. This struggle is part of the experience of stigma in eating disorders, which I touch upon only briefly in this chapter and more expansively in future chapters, particularly when discussing the implications of genetic causal accounts.

5. COMPROMISED AGENCY: MANAGING IMPRESSIONS OF EATING DISORDERS

[I]t's hard for me to tell my parents "Something inside of me is telling me not to eat that." Because, they don't have that. So, it's hard for them to really understand and I mean believe me that there really is this thing that is keeping me – preventing me from eating food that I know I should be eating. (Jackie, T-BN)

Perceptions of causal factors and agency in eating disorders are closely tied to definitions of what eating disorders are. In Chapter 4, I focus on respondents' reactions to several terms used to describe eating disorders. In this section I describe how they would ideally like others to see eating disorders, a question I asked *before* the material to be discussed in Chapter 4. After eliciting respondents' theories about what caused them to develop an eating disorder⁸ and before asking them to react to specific models of eating disorders, I asked most respondents how they would *ideally* like *others* to understand eating disorders. Of the 45 who addressed this question, a clear majority brought up the compromised agency of people with eating disorders, frequently describing it in medical or psychological terms. They linked this compromised agency to the compassion they believe is warranted for people with eating disorders: it is not just a matter of choosing to behave otherwise.

⁸ Here I construct the sentence in a deliberately ambiguous manner, to encompass the preceding discussion of causal factors as well as the role of agency. There is both an agentic and a non-agentic sense of "develop"; one can develop cancer and one can develop a photograph or career.

Some focused on the emotions, issues, and other struggles involved in the eating disorder behaviors to convey that it is not simply a matter of choosing. They would like others to see AN and BN as the product of a compromised rather than free agency.

I: Well how would you ideally want people to understand anorexia?

R: I guess, like, everybody wants to group people together and put them in a nice little box. And, like, yeah, anorexia is you don't eat. But not everybody doesn't – chooses not to eat. Or it's not a choice. But I guess like, when you come down to it it's a choice whether or not you eat.

I: Can you say more about that?

R: Well, like, I can choose to pick this up or not... You can choose whether or not to eat it. Like, to pick it up and have food. But it's a mental thing. Like, we might be scared of it or something. So it's not just like a cut and dried choice. And so I wish people would understand that, like, the reasoning behind the not eating is not solely, like, just as simple as mechanically putting – picking up food and putting to your mouth and chewing it and eating it. Like, it's a lot – like, it's emotional and mental. And I just – if people could understand that, then I think it'd be like – I don't so much think anorexia should be accepted. But I think that the reasoning behind anorexia needs to be accepted to be able to help them... It didn't help me my [adoptive] dad saying “Well you can just eat.” Like, that doesn't help. (Yvette, R-AN)

Yvette's sentiments were shared by many respondents. It's not about food and simply choosing to eat; there are psychological reasons that others need to understand in order to have appropriate compassion. Emma pointed out that “no one would do that if they felt like they had another option” and that it should ideally be understood as a coping strategy (like others whose words were presented earlier).

That it's just a symptom of emotional turmoil or whatever you want to call it. But, it's a coping strategy that's not a healthy one. But, it's a coping strategy when one's having an emotionally hard time. And, it's complicated. So, it's not just a simple thing of eating too much and not – and then, throwing it up. I mean no one would do that if they felt like they had another option. But, at the time it's the coping strategy the person knows how to do. And so, I would want people to have some empathy about it. (Emma, R-BN)

Emma, Yvette and others would like social recognition that what seems like a simple choice is actually more complicated because of emotional and “mental” issues. Liana's description

of the eating disorder as something she fights against is another way of conveying the struggle that so few people seem to understand:

I wish that more people would understand how hard things are for me. And, maybe appreciate the hard work a little better. .. I mean, like, a lot of the stuff that is really hard for me is, like, a natural instinct to most people. Or, is really easy to most people. And so, they would be like, “Well, why would I feel so proud about you for trying such and such a food?” Well, I mean that’s, like, a really big deal to me. Because, that’s, like, really, really fighting against the eating disorder when I choose to try it. And, I think it would make me feel better if more people understood a little better. I mean I know nobody can really understand unless you’ve been through it. But, at least have some empathy. (Liana, T-AN)

Many respondents emphasized their compromised agency; they are struggling, fighting, coping the best they can.

Some wanted others ideally to see it in more explicitly medical terms, which also went along with more compassion. In the excerpts below, Melanie used the term “mental illness” and “disease” and Sarah used the word “illness.”

Well I would want them to think of it with empathy or with compassion anyway of that person who is in the middle of that. And not berate them or whatever. ... I know it’s in the DSM. I don’t know if it’s generally considered a mental illness or not. But I know it’s in there. But mental illness in our society general. I get very frustrated with people who just won’t think about it in terms of a disease that one might have or something. (Melanie, R-BN)

Yeah. I think that seeing it as an illness is really important that people don’t often view it as an illness. It’s thought of more as something that women are doing to themselves rather than suffering from. So I think that thinking about it more in terms of people are suffering from this rather than they’re causing it is one thing that’s important. (Sarah, R-BN)

The key for Sarah is that people with an eating disorder are not “causing it” or “doing it to themselves”. For many respondents, this is the reason they deserve compassion; if they were doing it on purpose then they might not. Rebecca (T-BN) preferred the terms “mental health disorder” and “addiction” and put it this way: “If it’s just a behavior, I feel like people, like, really judge you. I don’t know. I feel like they have a reason to judge you.” Several other

respondents used these words and phrases to describe it: “disease,” “complex disease”, “physical illness”, “mental health disorder”, “emotional and mental disorder”, “psychological disorder” and “addiction”. Several cited physical effects, including feeling “yucky”, having heart problems, and risk of death to underscore its serious medical status. A few compared it to physical illnesses: cancer, diabetes, and multiple sclerosis.

Yet some respondents found it difficult to fully believe in a disease model, even when they themselves advocated it. Later in the interview, Sarah expresses uncertainty about her statement quoted just above, and likens eating disorders to smoking with the idea that the person should be held responsible for stopping, just as patients with chronic obstructive pulmonary disease who continue to smoke ought to be held responsible for that as a choice. Claire notes that she would ideally like people to think of it in terms of a disease, but that it is hard for her because she blames herself.

R: Hmm. Just as a disease the way that, like, multiple sclerosis is a disease.

I: How come that’s a better way for you?

R: Because, it is. It’s a disease. It’s a disorder. It’s a – it’s not our fault. I’d like for people to understand that. Even, like, all the girls in the program. It’s like, it’s not – it’s not any of our faults. And, it’s really, like, empowering to think of it that way. Like, it’s not somebody’s fault that they have cancer usually.

I: Tell me how it’s more empowering you said.

R: Because, eating disorder patients – and, this is what I’ve been told a lot before. That eating disorder patients have a lot of self-flagellation and self-guilt and self-pity and self-harm and all of these things. And so, it helps so much to be, like, “This isn’t my fault.” And, I don’t believe that yet. And, I think that’s part of – that’s like, one of the core reasons that I’m struggling still. Is because I do believe that it’s my fault. Like, I believe that everything about my disorder and what’s been going on with me is my fault... And, if I didn’t believe that, then I think just in general I would have a healthier mind. (Claire, T-AN)

Claire states that defining it as a disease rather than as her fault would help her recover.

Paradoxically, she thinks that this removal of agency – it’s not my fault – would empower her and help her recover. (This theme of empowerment through the removal of agency will

be explored more fully in Chapter 5.) Claire's reference to the hospital-based program, and to Overeaters Anonymous in other parts of the interview, suggest that the disease concept may be learned during treatment. This is the case for Betty, an inpatient with AN. In her 60's, she may have been less exposed to the idea of eating disorders as mental illnesses and explicitly states that she is learning to see her behavior in medical terms during her inpatient stay.

I would want them to know that it is a disease. And, you can get over it. And, I've never thought of it as a disease. I just thought of it as kind of a condition or whatever. Something you chose for yourself that you could get over if you just would. But, I realize it takes a lot more than that. And, surprised me to find out how much I did not know about all of this. And, it's just been like being in school to me to be here and learning so much. (Betty, T-AN)

Elsewhere in the interview it is clear that Betty's changing view of her AN is not simply because of her presence in the inpatient unit but also the experiences that led her to need to be there. Yet such a re-thinking is clearly encouraged by program personnel. (In the statistical section at the end of the next chapter I examine relationships between treatment experience and medicalized discourse.)

Another theme in answer to the question about ideal understandings of eating disorders was the idea that others ought to take them seriously as negative and harmful problems, rather than frivolous or even somewhat positive behaviors. Because weight control is widely perceived to be good, it may be hard for observers to take eating disorders seriously according to Hannah. She was worried about a friend with AN and could not get others to take it seriously.

Like, everyone knows that she has anorexia. And, she is literally like, about to fall over dead. And, I've talked to the faculty about it. Like, I've talked to her about it. Trying to get her help. And, like, even the faculty don't do anything. And, like, I feel like that's ridiculous. Like, she is endangering her health. If she were an alcoholic, we would have no problem forcing her to get treatment. Why is it different for anorexia?

Because, it's socially acceptable to be thin. And, that's not acceptable to me. Like, I think that we need to take it just as seriously as we take drug problems. Because, it's doing just as much damage. (Hannah, R-AN)

Willa said she would ideally like people to see it as “very dangerous” and “that there's nothing fun about it”, and hopes that “our culture could get to a place where it's just understood that that [extreme thinness] is so unhealthy and doesn't produce any happiness.” The perception that there could be something desirable or acceptable about it undercuts the idea that it is a serious disorder, and makes it more plausible that the person can control her behaviors. In Gillian's and Reba's ideal scenarios, people would see that an eating disorder “is not shallow or attention-seeking”, and that it is “not a matter of vanity” (respectively).

A minority of respondents (n=5) presented an alternative perspective that distanced AN and BN from medicalization as serious psychological or mental disorders. I present them with greater detail than others to highlight conceptual differences among respondents.

Two grounded their answers in very similar language about eating disorders as a rational response to gender norms. This language resembles that of feminist analyses of eating disorders. Alyce (R-BN) wanted “people not to be targeted as being sick the way I was. I want it to be seen as more a rational reaction to the conflicting expectations put on people. And, unrealistic standards on women in particular.” Alyce is clearly locating the problem in society's expectations for women, for which AN is a “rational reaction.” Margaret (R-AN) also sees it as a “rational response” and a “response to powerlessness” of women confronted with “gender inequality”, and argues that “psychologizing” about it is like “blaming the women.” In response to my request to clarify what she meant by “psychologizing”, Margaret explained:

I think that psychologizing tends to treat the problem as if it were rooted in the individual nature of a human being. Of a single person. And that the problem is the

person. Either the way they think about the world. Or the way they feel about it. I think it tends to treat what I see as a social problem as more of a problem in, like, cognitive processing or something. That it's an irrational kind of behavior. That it's irrational because the person's not thinking properly or something. I think that it's a rational response to inequality. (Margaret, R-AN)

Her critique of the “psychologized”, medicalized perspective on eating disorders resembles Alyce's in its focus on cultural constructions of gender.

Similarly, Lynn focuses on AN as a response to a situation, though she does not link it to gender inequality. She began by stating a “big caveat” that there may “may be some biological basis for this for some people”, but went on to say that

I'd say to people it's a sign that this person's not comfortable in her own skin. Or, not comfortable in the situation she's in now. And so, to me it's kind of a symptom of a discomfort. And, that person who is an anorexic should take action about the things that are making her uncomfortable. (Lynn, R-AN)

Lynn draws a contrast between AN that has a biological basis and AN that is a response to a situation, and presents the solution as taking “action” to change the situation.

Two additional respondents ideally wanted others to understand that eating disorders are temporary and people with them should not be stigmatized permanently as having a serious mental illness. Vanessa's answer distanced eating disorders from being “crazy” which she understood to permanently affect sanity in every respect.

It is a problem, yes. Don't get me wrong. But just because a person is bulimic or anorexic I would not want them to equate that that person is mentally off necessarily in a whole sense of the picture. I mean yes. You are mentally off as far as your perception of self and what you're doing. But that doesn't mean you're completely crazy that you need to be put in a nut house. (Vanessa, R-BN)

She contrasted this permanent craziness with more temporary conditions - addiction and “a phase that you're going through” – which to her mind were more appropriate for eating disorders. Barbara's answer focused on her objections to other people monitoring her behavior, even after recovery:

And that's really bothersome. Because then they feel like they need to always kind of keep an eye on you. Like you're, like, a ticking time bomb. Some people will think that. And it's totally not the case. Like, years down the road you're completely able to be a fully functioning member of society that doesn't need to be watched twenty-four seven. (Barbara, R-AN)

It was possible to criticize gendered expectations for appearance in ways that were compatible with psychological and medical conceptions. Indeed, most respondents did so, but the following two respondents are unusual because they focused on it in answer to the question about how they would ideally like others to understand eating disorders. Kathleen (R-AN) said that to overcome eating disorders one has to "deal with the issues", but unlike other more psychologically-minded respondents, she linked these issues to gender: "It always seems to be a common theme that it's mostly girls. And, girls that have self-esteem issues. Which is sad. And, I don't like that for girls. And, I don't like the pressure of the world that we live in in America. Where everything is focused on that as a girl. It's all about how you look." Kathleen suggests that it is the pressure on girls about how they look that lowers their self-esteem. Her answer has elements of the more psychologically focused answers above, but by linking it to a cultural context she distances it from psychology alone. Rebecca also combined medicalized concepts with gender, describing BN as a "mental health disorder" but saying it stems from self-confidence problems related to unrealistic standards of beauty.

And like, what we view as beautiful today isn't necessarily healthy. And it's not real too. Like, I don't know. I, like, get angry at my boyfriend, like, for looking at pornography. Because I feel like, I think I can't live up to that standard. Like, they're plastic. They have liposuction. And how can women, like, live up to that? My mom's like, fifty-five or fifty-six. And, like, she has, like, a sagging face and she's wrinkled. But she just looks like a mom. Like, she looks like she should. And she, like, wants to get the face lifts and all these things. And it's just not natural. It puts, like, a lot of pressure on people to look perfect when it's not really the way we are. (Rebecca, T-BN)

In answering the question about how they would ideally like others to view eating disorders (n=45 answers to this question), the majority wanted it seen as something other than a simple choice, often as a mental health problem. A minority specifically criticized medicalized and “psychologized” perspectives, and the remainder gave answers that were not clearly relevant to agency or medicalization.

6. SUMMARY OF CHAPTER 3

The aim of this chapter was to describe how respondents understood eating disorders to develop before asking them to react to specific ways of thinking about eating disorders (such as “mental illness”) and before presenting them with the idea of a genetic causal factor. The chapter had two main goals: to illustrate (1) the complex, dynamic causal models put forth by respondents, and (2) their perceptions of their own ambiguous, ambivalent agency in carrying out eating disorder behavior.

Causal stories were complex and dynamic and involved compound, rather than simple, easily disaggregated causes, suggesting that any causal story incorporating genetic causes also will likely be complex rather than simple (their genetic causal models will be described in Chapter 5). To capture common themes in respondents’ descriptions of causality in eating disorders, I presented three frequently-cited causal categories: valorization of thinness, coping responses, and repetition over time. These were not reducible to biological, “environmental” (e.g., social, cultural, familial factors), nor “individual” (e.g., psychological, agentic) factors and frequently involved elements of all three. In addition to the three broad causal categories, I identified four, more specific causal elements that I hypothesized to be *compatible* with genetic ideas (biological factors, enduring individual dispositions such as personality) or *incompatible* (abuse, trauma, or injustice; gender inequality). Relationships

of these variables to respondents' initial reactions to the idea of a genetic causal factor will be examined statistically in Chapter 5.

As they described causal factors, respondents also directly and indirectly addressed the question of their own agency in the development and continuation of their eating disorders. In general, I (and they) found it difficult to simply summarize perceptions of their own agency. While respondents did speak in terms that connoted free agency, they always also spoke in terms that connoted either a lack of agency, or a constrained form of agency I labeled "quasi-agency." I offered four reasons why there might be this mix of agency, non-agency, and quasi-agency: that agency may be different at different phases of the eating disorder (e.g., starts as a choice but goes out of control); that some aspects of the eating disorder can be controlled whereas others cannot (e.g., choosing to avoid triggers that would make one lose control); that in hindsight what seems like agency really was not (e.g., the eating disorder had taken over, but at the time it felt like a choice); and that it may be difficult to self-interpret behavior as simply non-volitional when one is held responsible for it by others (i.e., others are telling people with eating disorders that it is a choice, which makes them wonder). The interpretation of agency had implications for stigma and guilt. Most respondents said they would ideally like others to see eating disorders as something neither chosen nor easily controllable: a struggle -- or even an illness -- for which compassion was deserved.

The next chapter goes into greater depth about what respondents think eating disorders are. I end with a quote from Karen, an inpatient, to convey the confusion she and others felt as they struggled to explain their experience.

It's a disease. It's an illness just like a physical – well, it is a physical illness also. To me it's mental and physical. Because, it's physically killing you. I would like them to

know that it's not something I asked for. It's not something I wanted. It just happened. It just kind of took over me. Because of my weakness. I don't know. The stress. OCD. My childhood. Of course the guy I was dating. It just everything kind of just played a part in it. But, I don't know if it was going to – it could have come regardless. I mean it just could have happened. I could have started – I told you I was already feeling uncomfortable with my body. My self. I felt at that time just wasn't happy with me. I didn't have a good relationship with any – my friendships weren't that good. They all had boyfriends at that time. And, I didn't. Until I met this guy. And, I just – I was thinking to myself “Well, maybe if I put on a little weight. What's wrong with me?” Even before it really took place. So, it could be something that just was going to happen. I don't know. (Karen, T-AN)

Karen says “I don't know” three times in this passage. She offers clearly medical language but is not confident that this explanation is sufficient, so she must explain how it took her over and why. She points to her own “weakness”, OCD, early childhood, the relationship that precipitated the disorder, but also suggests it could have developed without these factors being present. Other respondents also expressed a lack of certainty about causation. The sheer number and variety of causes mentioned was a testament to the complexity of and confusion about causation. As they struggle to explain why they cannot “just stop” the problem behaviors, some find medical concepts useful, as I have already illustrated. As I will show in later chapters, many will use or interpret genetics to be useful for making this case, though there will be dissenters. Now I turn to their models for what eating disorders are.

CHAPTER 4:

UNEVEN, UNCERTAIN MEDICALIZATION: PERCEPTIONS OF EATING DISORDERS

Based on Chapter 3, we know that these respondents conceive of causality and agency in complex ways. But what do they think eating disorders *are*? Are they diseases, choices, social problems, or something else? In this chapter, I discuss their reactions to several terms to describe eating disorders. First, I briefly discuss their views about eating disorders as a problem generally, and as a “problem with society or culture” specifically. Second, I examine their reactions to five concepts related to medicalization of eating disorders. Third, I present findings for an index of endorsement for medicalized terminology based on these five concepts. I begin by considering eating disorders as a problem and as a “problem with society or culture” because virtually all respondents endorsed these and they were not included in the endorsement index.

1. TERMINOLOGY FOR EATING DISORDERS

Before turning to the series of questions about specific terms, I touch briefly on language used by respondents to refer to their eating disorder. Respondents used the terms “eating disorder,” “anorexia” and “bulimia” to describe their eating issues, just as I did while recruiting and interviewing them. No one used less medicalized terms such as “ana” or “mia”, (from the pro-eating disorders lexicon). I asked many respondents if they used other terms besides the ones I was using, but they did not and seemed to find this question odd so I stopped asking it. (A less medicalized sample might have responded differently.) They

seldom used the suffix “nervosa” (14 out of 57 used this word at least once and of these only three used it more than once up to three times total). While using the phrase “eating disorder” certainly reflects its medicalized status, we cannot know exactly what its meaning is for each respondent. The series of reaction questions will be useful for systematically illuminating their individual definitions of eating disorders.

2. EATING DISORDER AS A PROBLEM?

In order to view a behavior as “medical” it must be seen as a problem; this is the ground on which medicalization or some other way of thinking about problems is based. In Chapter 1, I proposed that medicalization could be conceptually subdivided into “pathologization” and “biologization”, the first corresponding to the extent that the eating disorder is seen as a problem and the second corresponding to the extent that it is seen as biologically based. In my interviews I was careful to leave open the possibility that respondents did not pathologize it (see it as a problem) and avoided language to that effect (though by necessity I used the term “eating disorder” even though “problem” is implied by the word “disorder”).

To gauge whether eating disorders were seen as a problem, I asked all respondents first how they felt about having an eating disorder, and then asked most respondents directly if it was a problem in their lives. Without exception, all saw it as a problem.

The problems ranged from simple statements as mild as “I didn’t want to have it” (Kathleen) to long lists of bad consequences for relationships, finances, career plans, and physical health. Mary, in the inpatient unit for AN described the problems as follows:

I: How do you feel about having had anorexia?

R: It’s scary. And, something I don’t know if I’m ever going to get over. So, it’s depressing in that sense... Just that you’ve got this abusive thing inside you that

basically wants to kill you. And, it's just really controlling. It takes over everything. And, ruins relationships.

I: Well, what are some of the problems it has caused for you?

R: I've basically pushed my family away. And, I pushed my partner away. We're separated. And, when I went to the [residential treatment program]. Because of financial issues related to the eating disorder, we had to downsize from a house to a townhouse. And, when I got back and went to [the day program], Angie [my partner] basically told me she couldn't handle me living there. That she didn't want to have to monitor me. So, then I basically had nowhere to live. And, I had to go live with my parents.... It's affecting my daughter now, too. She's been having problems at school. And, we've actually got her seeing a play therapist.

Respondents were also concerned about potential physical problems caused by eating disorders, including problems with bones, heart, digestion, and teeth. Almost half of respondents (n=at least 22) spoke of death in connection to eating disorders at some point. Vanessa, who recovered from BN, had purged together with friends in high school without thinking of it as a problem.

And it didn't hit us until much later in high school what we were really doing to ourselves. By then for a lot of people it was a bit late. And we did lose one of our friends... we were all together when it – when she – she was throwing up. And she started bleeding. And she fell on the floor. And we had to call her mom in. And her esophagus had erupted or something. I don't remember the whole term. But she ended up dying...

Despite seeing eating disorders as a problem, many also saw good aspects. This would be expected in recovered respondents, who can look back and note that they have learned from the experience. Twenty-two people said that as a result of the eating disorder, they were able to help or educate others about eating disorders; still others felt it made them better able to empathize with people who had other kinds of problems. Some even felt fortunate to have gone through the experience.

I'm kind of glad it happened to me. Because, now I've been able to turn it around. And, I feel extremely – you want to say blessed or lucky or whatever. That I was able to turn it around and get healthy. I feel like I got kind of like a second chance. Because, like I said. I mean there's many nights when I didn't know if I was going to

wake up in the morning. So, I'm glad. Because, I'm able to turn around and help people. (Deena, R-AN)

Looking back at a resolved problem and finding good in it does not contradict the idea that it was a problem at the time. For some the compassion extended beyond eating disorders to people with other kinds of problems.

That [AN] and some other problems I've had with depression in some ways have made me a lot more sympathetic to other people. Even though, like, if I see somebody who's in a wheelchair. I've never been in a wheelchair. But it's like I can kind of – having dealt with some really difficult things I know what you're dealing with. And I can just cut people a break in my mind. And just be – I don't know. Just be more understanding and kinder and gentler I guess in my thoughts and actions to other people. So it's been a good thing. (Nell, R-AN)

Although some respondents identified good aspects, as I mentioned before, every one of them saw eating disorders as a problem. It may be possible to further differentiate between the perceived severity or importance of the problem in future analyses. For the present purpose of examining medicalization, it remains to be seen whether they view the problem in medical terms.

3. EATING DISORDER AS A “PROBLEM WITH SOCIETY OR CULTURE”?

Before reporting on the terms that were included in the index of endorsement for medicalized terminology, I briefly describe reactions to the phrase “problem with society or culture.”¹ There was little variability; 47 of 50 respondents agreed with the idea that eating disorders were indeed a problem with society or culture. It was not included in the medicalized terminology index because of this lack of variability and because it did not elicit discussion of medicalization or related themes. As noted in Chapter 3, some respondents (Alyce, Margaret) resisted medical conceptions and contrasted them with sociocultural ones,

¹ I also excluded the term “lifestyle.” Respondents interpreted this term in different ways, often compatible with medicalized notions (because an illness changes what you do in daily life thereby changing your lifestyle). Because early answers did not address my research questions I did not consistently ask respondents about “lifestyle” during data collection and do not report the results.

but this was unusual and in any case was not elicited by the vague term “problem with society or culture”.

A clear majority of those who approved of the phrase linked it to cultural norms of thinness. Some also thought that having a plentiful food supply could encourage eating disorders, because food was something that could be refused (by restricting) or wasted (by bingeing or purging). Relatedly, some thought that the problem of obesity encouraged eating disorders because of widespread messages encouraging dieting and fitness. It may be possible to define and discern a more fundamentally social view of eating disorders that contests a medicalized viewpoint, but this sort of view was not obvious in the interviews.

The phrase “problem with society or culture” did not elicit discussion of medicalization or related themes. Indeed, they spoke about social and cultural causes in ways that were compatible with medicalized models held by respondents; as shown in Chapter 3, cultural and social pressures were already part of their causal models and co-existed with diverse other factors. Indeed, the hospital-based eating disorder program included a regular media-watching component. For Mary, an inpatient, this media component encouraged a critical consciousness of the portrayal of women and body size.

Of the three who did *not* endorse the concept of eating disorder as a “problem with society or culture”, two interpreted the question to be about whether it is a problem *in* or *for* society. They both thought that obesity was more of a problem (Vanessa and Yvette). In other parts of the interview, however, they too had spoken about pressures to be thin.

4. MEASURING MEDICALIZATION: REACTIONS TO FIVE TERMS

In the interview, I asked respondents to respond to seven terms that could be used to describe eating disorders. I selected five from the seven terms presented as most relevant to perceptions of medicalization: psychological problem, mental illness, brain disease, physical illness, and choice. I chose these terms for their face validity. The first four are medical terms because they suggest problems for which a person would receive help in a healthcare setting – treatable disorders, in Conrad’s language - as opposed to being a choice for which the person is held responsible, or another understanding of deviant behavior such as spirit possession.² Three of the four terms include the words “illness” and “disease,” placing them in the medical realm. “Psychological problem” is arguably the least medicalized term of the five. Yet it does seem more medicalized than simply the word “problem”; Isabelle made this case when she contrasted “problem” to “psychological problem” because the latter “attributes the problem to something”. (She spontaneously compared the two; this was not something I asked respondents.) The last term, choice, is clearly not a medical concept; it implies that the person’s behavior is subject to volitional control rather than being a condition for which one needs help. A clear rejection of “choice” constituted greater medicalized thinking than otherwise, for this analysis. My categorization of “more” and “less” medicalized viewpoints depends on the assumption that all five terms are relevant to medicalized understandings. If this assumption is accepted, then examining respondents’ answers will shed light not only on whether they have medicalized views of eating disorders (what terms seem appropriate to

² While spirit possession may seem very far from the respondents’ worlds, some respondents referred to their eating disorder as a “demon”, “monster” or “entity” that speaks, yells, growls and roars. Such externalized metaphors resemble spirit possession. In addition, a few respondents spoke about their eating disorder as a “sin”, suggesting that medicalized language coexists with other ways of talking about problem behavior.

them), but also how they would define medicalization for eating disorders (how they reason about the appropriateness of terms).

Table 4.1 provides an overview of respondents’ endorsement or non-endorsement of medical terms. I reversed the coding logic for “choice” because the rejection of that term connotes greater medicalization. The medicalized categories are shaded in Table 4.1 to clarify their similarity, despite different response coding. Disagreeing or in some way distancing oneself from a term is sometimes a matter of interpretation, as I did not ask respondents to choose among response options.

Table 4.1. Endorsement of five terms to describe eating disorders. Frequency (percentage) Shading indicates responses coded as more medicalized

	Psychol. problem	Mental illness	Brain disease	Physical illness		Choice
Agree clearly	39 (80)	26 (52)	14 (28)	19 (40)	Disagree clearly	14 (28)
Disagree*/conflicted	10 (20)	24 (48)	36 (72)	29 (60)	Agree*/conflicted	36 (72)
Total	49 (100)	50 (100)	50 (100)	48 (100)		50 (100)

*This category included anyone who did not clearly agree (with the more medical terms) or clearly disagree (with the idea of choice). For all terms, this included people who endorsed both options or expressed uncertainty or did not know. For medical terms it also includes people who said they would need evidence, or distanced themselves from the presented option.

The table above represents how people responded to the language presented in the interview. A majority agreed that eating disorders were psychological problems, and they were almost evenly split regarding whether these disorders were instances of mental illness. A majority disagreed that they should be seen as a brain disease or physical illness. Only one third saw the disorder as a clear matter of choice.

Reporting on agreement or disagreement with medicalized terminology is not the same as a description of a person’s underlying view. While in some ways acceptance of medical terms can be seen as a reflection of medicalization, it is more specifically a reflection

of medicalized terminology. I asked respondents to react to terminology, and their decisions about whether the term applied reflect not only what they thought eating disorders were, but also what the terms mean and whether it is desirable to apply them. Respondents' ways of reasoning about terms resembled each other, even when they did not agree about whether to endorse them. To illustrate how respondents thought through these issues, I now present their reactions to each of the five terms.

4.1 "Psychological problem"?

A clear majority of respondents endorsed the idea that AN or BN is a "psychological problem" (39 out of 49 who addressed the question). Forty-eight people were asked to react to the idea of their AN or BN as a "psychological problem" and one did so without prompting for a total of 49. Many simply stated "I agree", "definitely", "that's true", "that's valid" and I did not probe their thinking as much as those who said it was not. (I also probed more on answers about "mental illness", "brain disease", and "choice"). Below I describe themes of respondent answers, paying special attention to respondents who did not accept the term "psychological problem", as they may be the least medicalized group.

4.1.1 Not a "psychological problem." Of the ten who disagreed or distanced themselves from the phrase, eight disagreed with it at first and two agreed with it at first but later amended their answers. Most answers sounded like they were against medical views of eating disorders. One very subtly distanced herself in a way that suggested the term was not medical enough: Delia emphasized chemical imbalance and the biology of addiction (She was in the inpatient unit for AN, had a very strong existing narrative about her family's genetic addictive tendencies.)

Three (Alyce, Victoria, and Lynn) thought “psychological problem” implied biological causation or serious mental illness, a premise they rejected.

Like you’re bipolar. Which to me it’s more clear. There’s a genetic thing going on in your brain. And, you’re bipolar. And, there’s something “wrong with you”, in quotes. But, to me bulimia is not quite like that. (Alyce, R-BN)

Psychological problem I think of like, depression or, like, bipolar. And it’s things like that. So I wouldn’t put it exactly in that category. I mean I think it’s like, more like a learned behavior. It might develop into, like, an addiction or like, psychological thing. But I don’t know if it starts like that... like, depression and stuff I’ve heard it’s like, a chemical imbalance and things like that that are psychological. I don’t feel like being bulimic is because, like, I have a chemical imbalance. (Victoria, T-BN)

Lynn (R-AN), also described below said that it was an “emotional problem” rather than a “mental problem”; the latter would mistakenly connote “not seeing the world as it was.”

Three (Carol, Margaret and Lynn) characterized it as a response *to* a problem, rather than a psychological problem in and of itself. Carol (R-AN) described it as a “psychological response *TO* a problem” (emphasis hers); “It really feels to me like a psychological response to some problem that for some reason you choose this drastic way of trying to solve.”

Similarly, Lynn offered:

...to me it’s not the basic problem. It’s more like a symptom of some problem... I think that for me and I would think for some other people – other women. It’s that I wasn’t comfortable in my life situation at that point. And, I didn’t really know how to handle the changes that had happened in my life. How to handle the independence. How to make my own decisions and be comfortable with those decisions. So, it wasn’t – anorexia gave me something that I could really control. I could control what I put in my body. But, the issue might have been control. The issue wasn’t the food... It was more about adaption and assertion. I guess those are terms I’d use. Asserting myself. And then, adapting to the world that I’d made by asserting myself. (Lynn, R-AN)

Lynn’s view is much more akin to a model of normal development into adulthood, or, alternatively, feminist cultural change during the early ‘70s (described elsewhere in her

interview). Margaret's answer was similar, though she contextualized the problem more broadly; she thought of it as a response to women's oppression under patriarchy.

Two felt that "psychological problem" was inadequate because the fundamental problem was physical, though not medical. Melanie said that bulimia reflected a normal human urge to eat food because it is pleasurable, and that in a society with plentiful food people binged and had to compensate. Tammy (R-AN) said the problem arose when a person was overweight and made to feel bad about herself. These respondents distance themselves from "psychological problem" by mentioning a physical basis, but the physical aspect is not pathologized: it is normal human appetite in an abnormal context, and low self-regard due to social devaluation of people who are overweight. So it is less like a psychiatric disorder and more like a social problem because it is not located in the individual (see discussions of medicalization and feminism in Chapter 1).

Two other answers also ran counter to medicalization but could not easily be categorized into the above, though there were themes in common. Paula (R-AN) stated that an eating disorder was a "manifestation" of a psychological problem, not a psychological problem in and of itself. This answer is arguably a challenge to medicalization because the eating disorder is demoted to a symptom rather than a psychological problem, which would not warrant inclusion in the DSM. Vanessa thought the term "psychological problem" was too similar to mental illness or addiction, which implied permanence and stigma,

...you still get that "Huh. Something is wrong with you." Kind of label. Because it sounds so final. Like you're going to wear it for a long time. But with a phase you just feel like – like your kid. They go through their little phase. They throw a tantrum. Or they turn – that's the terrible two's. It's a phase they're going through. They feel like – you feel like, "Okay. It's just something I'm going through. But six months from now I won't have the problem." (Vanessa, R-BN)

Her comparison of an eating disorder to a child's phase, the "terrible two's", is diametrically opposed to the more medicalized responses, which sought to establish the seriousness of eating disorders and their deservingness of treatment.

4.1.2 *It is a "psychological problem."* In answering the question about eating disorder as a "psychological problem", many gave short answers and as noted above I did not probe carefully for this question. I touch upon some themes here, though in less depth than for those who did not endorse the term.

"Psychological problem" made sense to many respondents because it involved the mind, presumably as opposed to the physical. They said "it's mental", "it's all in my mind", and "it's a mind thing".

And, we've even in treatment learned to name the eating disorder. And, call it Ed. Or, whatever we want. And then, practice separation. Leading to divorce. So, that doesn't happen anywhere but in psychology. (Amy, T-AN)

Willa said that she thought "the psychological is really the only issue. And when you can fix that the weight will follow." Some respondents endorsed "psychological problem" but noted its serious physical consequences as well, including the psychological effects of starvation (Ingrid and Hannah). By doing so, they implicitly defined "psychological" in opposition to "physical".

Several approved of the term because it connoted a loss of control or inability to simply will one's behavior. Barbara describes how she still has unwanted feelings of guilt about eating even though she has recovered, and contrasts control with "something that clicks in" where you "can't completely break off of thinking about it."

I think that's probably the case. It began not a psychological problem. I had control of the situation. But by the end, like, the fact that I saw the scale at eighty and a half. And I was just like, "Wow. That's great." Like, that's terrible. And it's something that clicks in like – it's definitely psychological. Because like, you can't completely

break off of thinking about it. Years after and I'm recovered I still feel a little bit more guilty probably than the normal person if I gain weight and that sort of thing. (Barbara, R-AN))

Liana (T-AN) had a similar definition of “psychological” which I asked her to define because she had already used it several times throughout the early part of the interview: “That it becomes a coping skill that your brain automatically depends on. And, it isn't a choice. It's more of a reliance kind of thing.” Yvette, recovered from AN, described having to manage her “brain”, and “take my brain out of this” to make herself eat when she was hungry.

And so it was like, everything else about me knew I was hungry. But my brain was telling me “No. You're not.” And so I'd listen to my brain. And so that's what I say, like, as I take myself out of it. Like, I take my brain out of this. Even though like, I know I'm hungry. But my brain is telling me “Well you can just go another couple hours. It's almost dinner.” And so that's what I was saying. Like, I had to take that out of me. Because I mean that's still engrained in me. (Yvette, R-AN))

In addition, for some “psychological problem” appropriately conveyed the severity of eating disorders. They explained, “You can't just stop”, “it's much more serious than people think,” and “It is a very serious, severe, psychological disorder.”

Gillian specifically said that AN was psychological because it was not just environmental. Although she was the only one who answered in this way, I highlight it because it is a conceptual contrast to answers given by non-endorsers, such as Margaret, and illustrates the in-between status of eating disorders: neither medical disorder nor social phenomenon.

I think that a lot of people think that it's something that the mother did. Or, that society did. And, I think that lots of different things can influence it. But, I don't think that the person who abused me caused my eating disorder. I think that I was – I had a pre-disposition for it. I think that I used it as a coping mechanism. And, I think that it was mental. Not environmental. And, I think that it has medical sequelae. But, I don't think that it's a medical disorder. (Gillian, T-AN)

Although Gillian was answering the question about “psychological problem”, she brings up issues that are salient to the other categories as well. Her answer suggests the conceptual boundary work that is needed for defining eating disorders not only against a fully medicalized concept as “medical disorder” but also from a non-medicalized grounding in social influences. The picture is made more complex by suggestions of agency (e.g., “I used it”).

The themes identified above drew on several meanings of “psychological”, endorsing it as psychological because it was (1) mental rather than physical, (2) non-volitional, complex, and serious rather than a matter of choosing and controlling oneself, (3) mental rather than environmental. These themes were overlapping rather than mutually exclusive. Several of these themes will return in discussions of “mental illness” and “brain disease.”

4.2 “*Mental Illness*”? “*Brain Disease*”?

About half of respondents thought that “mental illness” was an accurate way to describe AN and BN (26 out of 50). Fewer endorsed the idea of “brain disease” (14 out of 50). The rest expressed skepticism, uncertainty or disagreement about the terms, which I coded as less medicalized than the alternative. Because respondents used similar reasoning for these two terms, I developed general codes and applied them to both terms whether or not respondents endorsed them. The reader will also notice some similarities with reasoning illustrated above for “psychological problem,” though answers to “psychological problem” were more diverse.

Most of the answers to the question about “mental illness” and “brain disease” could be captured by the same four codes (49 respondents received one of these codes for one or both answers). These codes accommodate the finding that respondents sometimes used the

same reasoning to argue for *and* against the idea that it was a mental illness *or* a brain disease (e.g., one respondent might say it is not a mental illness/brain disease because it is possible to recover and another that it *is* one because one never completely recovers). These codes illuminate not only whether respondents think the specific terms apply to eating disorders, but also how they define those terms and, more broadly, the meanings of medicalization for respondents who have been diagnosed with eating disorders. The codes were based on over 20 inductively generated codes, tentatively distilled to four general categories, then reviewed conceptually to make sure that any codes that were relevant to more than one category were subdivided and re-coded, if applicable (See Table 4.2 next page for further information on the inductively generated codes and how they map onto the four categories). I provide approximate frequencies for these four codes.

My choice of four general categories was influenced by my research questions (e.g., conceiving of mental illness as fixed or biologically based is salient to my questions on genetics). Respondents could receive more than one code; indeed many use a logic that ties these themes together, such that they may be seen as dimensions of “medical-ness”, often inseparable for respondents.

Table 4.2 Reasoning about whether eating disorders are mental illnesses and brain diseases

General code	Inductive codes on which general code is based
Dysfunctional, severe, serious vs. not	Delusional vs. not Life-threatening, serious vs. not Function in world vs. not Extreme vs. not In a psych ward vs. not
Consciousness, choosing, or control vs. not	In my control, conscious, choice vs. not Feels like something external to you vs. not Need help vs. handle it myself
Biological origins, treatment vs. not	Simple biological causation vs. complex causation Physical brain vs. not Neurotransmitters vs. not, chemical imbalance Simple biological treatment or cure vs. not Something you contract/catch vs. not
Static or fixed vs. dynamic and developmental	Inevitable, unavoidable vs. not Permanent or chronic or born with it vs. temporary Persistent vs. not Develops over time vs. birth/very young age Develops over time vs. happened in a moment Environment and experiences important

4.2.1 *It is a “mental illness.”* Those 26 who agreed that an eating disorder was a mental illness primarily did so by emphasizing that it was dysfunctional, that the person did not have control over it, or that it had a biological basis (n= approximately 9, 9, and 6, respectively). By bringing up these themes they appear to define mental illness by these criteria, and perceive eating disorders to be similar.

Respondents who discussed themes coded as “*dysfunctional*” spoke about a loss of connection to reality via delusions associated with eating disorders, though some asserted that there was less of this than with some other mental illnesses. With an eating disorder, “your views are distorted... all you can concentrate on is food and weight” (Delia, T-AN), and “it consumes all your thoughts... it’s just as severe as a lot of other ones that I think no one would disagree that are mental illnesses” (Eva, T-BN). Yvette and others focused on the distortion of body image in particular:

You have a different picture than anybody else has. And that was one of my main faults is mentally I saw myself as still that chubby little girl. Like, I didn't realize that I had gotten thinner than everybody else. All the other girls. Like, I had gotten so thin that it was like I didn't have any shape to me. But I didn't see that. Like, I still saw all the other girls and being like, "I wish I could be that thin." Like, they looked so pretty. (Yvette, R-AN)

Some who discussed dysfunctional themes argued that eating disorders are less dysfunctional than some other mental illnesses, such as schizophrenia. Wendy reasoned about it in a way that suggested that by some definitions, distorted thoughts about bodyweight would constitute mental illness, even if hospitalization were not required.

R: I guess because when people think mental illness you think of, like, somebody that needs to be in, like, a psychiatric ward or something. Or at least that's what I immediately think of. But if you think about like – if you think about it objectively what a mental illness is, then yeah. I guess it would be.

I: And what is that more objective way of looking at mental illness?

R: Like, just that you might have distorted thoughts about something. And maybe a distorted body image. Like, I understand now that I was too skinny when I was a hundred and three pounds. But I was really happy with my weight at that point. So distortion I guess in your mind would be classified to me as an illness. (Wendy, T-BN)

Ingrid (T-AN) also distinguished between people with eating disorders and those who are "hearing voices" or whose intelligence is impaired; "on the contrary," she said, "anorexics are extremely smart. And, very, very resourceful in keeping their addiction alive." Sarah (R-BN) noted that compared to some other mental illnesses such as schizophrenia and bipolar disorder, with eating disorders "you're not as far apart from reality that you cannot think about getting help."

The sense that the eating disorder was *not under the person's control* also justified classification as a mental illness. Respondents talked about the problem being in their brain - "there's problems with the way my brain is forming thoughts and reactions" (Jackie, T-BN), or being like an external force - "at least for me there's like, another voice. And I've heard it

compared to like, an abusive relationship... Somebody that's abusing you and pushes everybody else away and isolates you" (Mary, T-AN). Several spoke of not being able to choose to stop the behaviors, even when they themselves and others wanted them to. Wendy mentioned people who ask, "Well why don't you just eat?" and refuted the implied premise of choice:

...if the person was healthy in their head. They would want to eat normally. But obviously there has to be something wrong with someone's head to make them want to not eat or to make them want to eat huge amounts and then throw them up. Like, those are not normal healthy things to do. There's some kind of illness going on there. And I just don't see how you can really make a solid argument for it not being a mental illness. (Wendy, T-BN)

For many respondents, the lack of individual control overlapped with the sense that an eating disorder is a disease and dysfunctional.

Respondents cited or speculated about *biological origins* as evidence of mental illness. A few claimed that there was a biological origin, but most were coded here because biology was part of their definition of mental illness, even though eating disorders might not have that aspect. Petra speculated about serotonin:

I've read enough to know that there's like, lower levels of serotonin or higher levels or there's something in there. I think there's a mis-firing chemical in there that's not connected the right way. Because why can one person grow up in the same environment and not develop it and another person does? I think there's something mentally not connected there. (Petra, T-AN)

Rebecca wondered if scientists had "done studies to show like, chemical imbalances in the brain... to validate it". Olga (R-AN) felt certain that based on her experience with medication for a different disorder, her AN was related to the same "imbalance"; "some sort of chemical thing in your brain that's not quite right." Fiona (T-AN) said it was a mental illness even though you didn't have to be on medication permanently, like someone with schizophrenia.

Some endorsed “mental illness” but their definition seemed to imply a static, fixed state; by contrast, eating disorders were more *dynamic* because they developed and changed over time in response to the environment. This idea may not be separable from the idea that a mental illness is biological, because the dynamic, developmental dimension was usually brought up as a contrast to the biological idea. Wendy (T-BN) said that her answer would depend on the definition of “mental illness,” and after talking about the possibility of genetics and biological definitions of illness, she emphasized that for her, BN was not this: “I think it’s mostly just based in cognitive distortions. I think it’s mostly based on environmental factors.” Olga (R-AN) also contrasted a biologically based idea with something more dynamic “I guess whenever you say mental illness that makes you sound like you’re crazy or something. I don’t think you’re crazy. But just like depression and anxiety. I would consider some sort of chemical thing in your brain that’s not quite right. I would think that the anorexia is the same way. Now I don’t think – I definitely think there are outside triggers for it. So it’s behavioral too.” Environmental factors and “outside triggers” appear to be connected to “cognitive distortions” and “behavioral” elements, and both are contrasted to a biological conception. As I note later, the four coded themes work together.

In general, respondents frequently reasoned about mental illness by considering its resemblance or non-resemblance to psychosis, where psychosis was conceived as serious, uncontrollable, biological, and permanent in nature. For some, mental illness connoted psychosis, for others it did not. In addition, some viewed AN or BN as like a psychosis, and others did not.

4.2.2 *Not a “mental illness.”* Those who disagreed or were uncertain about whether an eating disorder was a mental illness also thought that a mental illness connoted something

dysfunctional (about n=11) or biological (about n=13), but believed that eating disorders did not meet these criteria. Those who disagreed with the idea of mental illness also tended to reject it because it sounded too static, fixed, or permanent (about n=11) whereas they saw eating disorders as something that developed and changed over time.

Many who rejected the term “mental illness” said that people with eating disorders were more *functional* than their picture of the mentally ill. Amy (T-AN) thought that “even when I do have my eating disorder present and all consuming, I’m functioning every day”. Barbara (R-AN) said she “was reacting to situations completely rationally. I wasn’t like, having outbursts or anything like that.” Alyce (R-BN) pointed out that unlike people with AN, those with BN are “not in denial. They know what they’re doing is destructive... dangerous and sick... They want to stop. And, they can’t.” Tammy, Beth and Thelma thought there was a continuum or spectrum of eating disorders, and (for Tammy) only those with “significant anorexia who are very OCD [could be] crossing over into a mental disorder,” or (for Beth) people who weigh “fifty pounds and go to the hospital and they’re almost dying.” As above, the idea of psychosis informs reasoning about whether “mental illness” applies to eating disorders; someone who can function, or is aware that what they are doing is destructive, is not psychotic and therefore not mentally ill.

Biological origins seemed to be part of the definition of mental illnesses, though like respondents described above they differed about whether biological origins applied to eating disorders or not. Like those who endorsed eating disorders as “mental illness”, there was uncertainty about whether eating disorders were biologically based. Other mental illnesses “could be caused by the wrong levels of serotonin or whatever other different hormones or substances you need” (Betty, T-AN), “brain chemistry and neurotransmitters” (Thelma, T-

BN), “chemical imbalance” (Mireya, T-BN), something “organic” (Nell and Lynn, R-AN) located in the brain. Sydney reflected,

it’s very difficult for me to accept that something’s wrong with my brain and I might not be normal in that sense. I prefer to think of it as a phase I’m going through that sense. I prefer to think of it as a phase I’m going through (laughing). That I’m normal. I’m just off in this little – this is something that I’m temporarily afflicted with. It’s not – when I think illness – especially in something like this where there’s no pill you can pop and just get cured. I feel like that’s something I have to live with for the rest of my life. And I don’t really want to put that label on it. Because that’s really daunting. (Sydney, R-BN)

I believe that Sydney was laughing at herself when she said she preferred to think of it “as a phase”, because earlier in the interview she had criticized her parents for seeing it that way rather than taking it seriously. Her discomfort with the term “mental illness” appears to be based on the fact that “there’s no pill you can pop” and the stigma of mental illness. Several others mentioned the fact that eating disorders are not treated with medicine, implying that biochemistry must not be relevant. Whereas, implicitly, for mental illness biochemistry and pills *are* relevant.

Several who rejected the term “mental illness” did so because it implied too much *permanence, fixity, and stability*. Again, there was overlap with other themes presented here. With mental illness, people had to be on medication their entire lives and could never truly recover, or they were born with the problem which implied that they would always have it. Vanessa preferred the analogy of a virus because they “run their course... then you’re well” (even though she did not perceive it to have biological origins). People who emphasized the role of the environment in the development of eating disorders were at odds with the idea of permanence, which was often linked to biology. If eating disorders developed in response to environmental influences, then they must not be mental illnesses. As Nell (R-AN) put it, “I

almost think of anorexia as a response to something that happened. Whereas somehow mental illness seems more like something that you're born with really.”

Loss of control implied biology and dysfunction for those who mentioned it. A person who is permanently dysfunctional for biological reasons cannot control her own behavior. Thelma links them together, and talks about her difficulty in knowing whether to apply “mental illness” to her BN:

[M]y mom is a psychiatrist. Like, that's her type of medicine. And so she deals with mentally ill people. And obviously the difference between psychiatry and psychology is psychiatry, like, yes she, like, meets with her patients and like, talks to them. But a lot of them are on medication. *Mental illness is like, a chemical thing* for a lot of people... I kind of like, changed my mind when I started thinking about it in that sense. Like, *whether or not it's something that you have any control over.*

I: Can you say how you changed your mind?

R: I started thinking about the term, like, mentally ill in the way that, like, my mother uses it. And that – the connotation for that for me is people who have just, like, *lost all touch with reality* because they're so sick. And *they can't help it.* It's not their fault. (Thelma, T-BN; emphasis added)

There are many more examples of how biology, permanence, dysfunction, and loss of control work together. These are dimensions of mental illness as a medicalized concept, generated from respondent perceptions. Eating disorders sit uncomfortably with other examples of mental illness, like schizophrenia, which is often contrasted to AN or BN.³ It may be useful to think of two versions of medicalized mental illness, a weaker and stronger one, with the stronger one involving biology, permanence/incurability, dysfunction/being out of touch with reality, and loss of control. Some equated only the stronger, psychotic version with the term “mental illness”; others thought mental illness could encompass weak and strong types of disorder. As they talked about eating disorders and the stronger version of medicalized mental illness, i.e., psychosis, there was a hint of what might be at stake: the ability of

³ The analogies respondents draw to other mental and physical conditions defines the conceptual boundaries of the disorder: it's like this, it's not like that, or it's like this but only in certain ways. Using other cases to reason about whether your own case fits the definition is similar to casuistry in philosophy.

respondents as agents to work toward recovery. To take one more example, from someone who endorsed the idea of mental illness, the capacity to “work on” your eating disorder implies that it is not permanent, nor biologically based, and not like some other mental illnesses.

I mean it is a mental illness. But, I think it can be fixed. It’s not something *I don’t think’s permanent*. I think it’s just temporary. And, you can fix your thoughts from it. So, it’s not like schizophrenia where you will have it for the rest of your life. And, *if you don’t take your medication*, it’ll come back. It’s something – I think anorexia can be fixed *if you work on it*.” (Fiona, T-AN; emphasis added)

When respondents think through genetics, they may think it implies this strong version of medicalized mental illness and an inability to work toward recovery.

4.2.3 *It is a “brain disease.”* Although far fewer respondents approved of the term “brain disease”, they used some of the same reasoning as for “mental illness”. While “brain disease” may seem far-fetched to apply to AN and BN, it is a term used by Thomas Insel, Director of the NIMH, in a letter featured by a prominent advocacy website (as discussed in Chapter 1). Only 14 respondents thought it was reasonable to call AN or BN a brain disease. I describe them using the same four codes as for mental illness.

Not surprisingly, the majority reasoned about *brain* disease by discussing *biology*. Some talked about serotonin and other neurotransmitters, “my chemistry,” neurons “mis-firing”, a genetic pre-disposition in which “something neurologically is different in an anorexic than another person that doesn’t have an eating disorder” (Reba, R-AN). Several used the term “brain disease” to encompass the brain’s reaction to eating disorder behavior. Rebecca (T-BN) discussed the effect upon the brain of repeated purging; “our brain models. So I think again initially it’s not a brain disease. But as you repeat and repeat and repeat the behavior you create new synapses in the brain that are in response to these chemicals.” Mary

spoke about how “the brain actually shrinks” as a result of malnutrition; Nell also spoke about how the “brain changes” in reaction to starvation. Irene and Ingrid endorsed the concept, but redefined brain to mean something more akin to the mind or the material component of the spirit.⁴ Thus, only a minority of respondents endorsed the idea of brain disease as a property of the physical brain, and a yet smaller subset thought the concept was more than a downstream effect of the eating disorder.

A smaller number reasoned that the apparent *permanence* of their eating disorder made “brain disease” plausible. Selena observes that her BN behavior has been difficult to stop, so a brain disease would make sense.

I think it’s sort of some kind of chemical imbalance or disease in the DNA. I’m not really sure. But because of the fact that even when I’ve been in a place where I was doing better. And it seemed like I should be in complete remission. I still felt like I needed to purge... But because when I’ve gotten better I still wanted to purge. I mean so it’s not – I mean I could make the choice not to. But it still felt like it was out of my control to do that. It felt like I just had some sort of imbalance or something. (Selena, T-BN)

Gena talked about a ‘glitch’ that could explain why she felt so self-conscious about her body even at a very early age, and why she ‘keeps going back there’:

Because, there’s got to be something to explain what happened when I was way younger. And, what happens with even the fact that I know that it’s something that I shouldn’t be doing. But, my brain keeps going back there type thing. There’s maybe a glitch. I don’t think that there’s like, ever going to be, like, medicine for it. Of course there’s no, like, eating disorder gene. Or, whatever. So, I would say yeah. But, I mean I haven’t read anything about that. So, I don’t know how competent I am in saying that. (Gena, R-BN)

Both Gena and Selena bring up genetics to convey their understanding of brain disease as permanent.

⁴The meanings of “brain” are interesting; people often use it to mean ‘mind’ but in a way that seems to externalize behavior; the ‘brain’ is often where respondents rhetorically locate the part of themselves that is not obedient to their will/agency. It is a way of externalizing agency without going beyond the body.

A few reasoned that because of their inability to control their own behavior, there must be a problem with their brain, making “brain disease” a reasonable possibility. Claire said “a lot of it’s out of my control. So, it must be at least partially mental, something wrong with my chemistry.” Fiona brought up genetics in answer to the question:

I’ve heard that about genetics and stuff like that. So, I do think that has some consideration. Because, it’s strange that both me and my sister both have an eating disorder. Because, if one was not – if it wasn’t somehow genetic, then the other person – the other twin could have probably fought not to have it. So, it’s strange that both of us got it. I do think there’s some genetic factor into it that makes it maybe a brain disease. Something that you can’t control. (Fiona, T-AN)

Again, there is the connection between the ability to control behavior and an origin in genetics or another biological characteristic. Selena’s answer about the persistence of her BN against her best efforts also demonstrates the connection between the ability to control behavior, persistence of the behavior, and its putative biological origin.

4.2.4 *Not a “brain disease.”* Most respondents did not endorse the idea that AN or BN were “brain diseases.” This included outright rejection of the term as “ludicrous” or “ridiculous,” as well as open-minded uncertainty about it (e.g., not having an opinion because it seemed like an empirical question). Therefore this category includes some people who were open to the idea.

For many, brain disease connoted something more physiological, anatomical, neurological and strictly *physical and biological* than was appropriate for eating disorders, which concerned the mind. Kathleen (R-AN) summarized it well when she said “It’s not physical in that kind of way”; even if there are physical components it would not be right to call it a brain disease. Several said it sounded too “medical” or “clinical.” While some

thought of brain chemistry,⁵ most said that “brain disease” sounded like a cancerous tumor or other growth, a deterioration, infection, lesion, aneurism, encephalitis, virus or bacteria “attacking your brain”. Some linked the term brain disease to neurological conditions like problems with myelin sheaths in multiple sclerosis, “depletion of certain neurons” in Alzheimer’s or Parkinson’s and Jackie (T-BN) contrasted this view to one that involves “mental processes”: “I think brain disease sounds like it’s a more physical thing going on inside your brain. Like cells attacking each other inside your brain. But, I don’t think that’s what it is. I think it’s mental processes.” Liana (T-AN) speculated that brain disease suggested something that would be visible on a “scan”, which was not the case for eating disorders; “I don’t think you could compare my brain to a normal eater’s brain. And, you would be able to tell any difference.” Willa dismissed the idea, “when I think of brain disease I think of I mean just pure genetics. And so I don’t think of it that way at all. I don’t think you get like, the anorexia gene kind of thing (short sarcastic laugh).” Gillian allowed that it might be possible to “have a tumor that’s at a perfect spot in their head that makes them have anorexia so it is a brain disease.”

The purely physical dimension implied a kind of treatment that many respondents found implausible. If it were a brain disease, it would require surgery, radiation, a medication, vaccine, or some implausible “magic potion”, or other interventions that were incongruous with eating disorders. Mireya linked the idea of brain disease directly to medicalization, and objected to it as a misunderstanding of the etiology and treatment of eating disorders.

⁵ Some thought of brain chemistry in connection to brain disease and rejected the term for that reason. Olga, who thought her own eating disorder is strongly related to brain chemistry, rejected the term brain disease because to her it connoted tumors rather than brain chemistry.

This is more, like, healing and about mental issues... basically what I think is like, that there's very much, like, a Western approach to, like, medicalizing everything and like, wanting to, like, cure. So it's you take a handy wipe and you clean the table and there's no more germs on it... There's no, like, way to completely wipe away and to remove something like an eating disorder. Because it's an action. And it's reaction constantly to your environment and to eating and your relationship with eating. And so it's, like, recognizing, like, why you did it in the first place and why you continue to do it. Which takes a lot of energy to think about it. Which is a lot more about healing, like, whatever is hurting or whatever is not resolved in your life as to why you continue to do a destructive behavior. (Mireya, T-BN)

Some wished that it were a brain disease, because this would entail a straightforward treatment. Beth said that if there “some MRI evidence” or real knowledge about “chemical imbalance,” “I would feel like that would maybe be good news. Since that means it could be fixed and predicted.” Alternatively, Carly thought if it were a brain disease that meant it could *not* be fixed; in her estimation, “brain disease is where you can't do anything about it because your brain is just deteriorating.” (This theme will be part of Chapter 6 on implications; if it's biological that suggests both permanence *and* treatability. You may always have the underlying problem but there is a treatment.)

Brain disease also implied too much *permanence* for some respondents. As conveyed by the description of the four codes in Table 4.2, this broad theme encompassed contrasts drawn between permanence and recovery as well as those between stasis and dynamic developmental interaction with the environment. Brain disease went too far toward permanence/stasis than some were comfortable with. Clearly these answers were related to the physicality of “brain disease” as well as the ability of the individual to control it.

Brain disease to me implies that there's some sort of inevitability to - like that you're born with it. That this is some sort of anatomical feature that - that's what I associate. Brain disease meaning this is something unavoidable. Not the product of a set of social conditions that I'm reacting to or being influenced by. It just that doesn't sit well with me. (Zinnia, T-BN)

R: I just think that to me is too medical. If it were as simple as it being a brain disease, well shoot. Operate. Get it out. But it's not. It's just – yeah. I feel like disease is too clinical. And yeah. It's too simple....

I: And can you say more what that means to you?

R: A medical issue means something that is not – I mean that's not environmentally impacted. So you could be in a room with your family. Or you could be with a room – be with your family at home or in a room full of strangers at the North Pole. And you would feel and act the exact same way. And I think eating disorders are SO environmentally charged. Obviously being at home with my family I'm going to behave very differently than at somewhere else with a bunch of strangers. Yeah. I just think there's too much of an outside impact on eating disorders and the way you behave and think and feel about yourself. (Sydney, T-BN)

Ideas about treatment were linked to ideas about recovery; “you can't fully recover from it.

Unless they go and take something out of your brain” (Yvette, R-AN).

It was possible to reject “brain disease” but embrace biological causation and an important role for environmental influence. Olga articulated a gene-environment interaction model, in which environment plays an important role, but only for those with a certain genotype or SNP (single nucleotide polymorphism, which represents a “difference in a single DNA building block”, National Library of Medicine 2010)

[I]t seems like Alzheimer's is something that no matter where you are or what your situation is it's you're going to get it if you have the genetic pre-disposition towards it. And like I was saying again. Like being poor in India versus being in an affluent country like the U. S. I think anorexia would be one of those things where it wouldn't be – just because you have a certain gene or a certain SNP or whatever you're going to get it. But you have, like, a higher chance of getting it given your social surroundings. Your environmental factors. I mean I would look at obesity the same way. I'd look at heart disease the same way. In that there's a lot of things in our environment that we could change to prevent it. But some people are never going to have a problem with it. And some people would have a problem with it just because of their genetic make-up. (Olga, R-AN)

Several thought brain disease implied less *individual control*. Paula, who was one of several who were open to the idea, speculated that it might help with stigma to think of it as a brain disease.

Can somebody show me on an MRI where there is something in the brain that's happening that then is manifesting itself in this behavior? I mean maybe it's possible that there is something going on. I mean because there's always sort of unexplored territory. And we find out that these things may be, like, a myth what we think now causes eating disorders. The same way that we've come a long way from "Oh. It's just a bunch of girls that want to be thin." Which was sort of like myth number one. Maybe – God only knows. They'll find, like, some kind of weird virus that causes... I mean they thought ulcers were caused by too much stress and coffee and type A behavior. And then discovered the H pylori. (Paula, R-BN)

Paula says it is possible that eating disorders could turn out to have a basis in the brain, just as ulcers were found to be caused by bacteria, and that this would help combat "myth number one", the claim that people with eating disorders just want to be thin.

The interconnection between biological basis and individual control is apparent in Gillian's answer. Brain disease sounds much more like a completely physical illness, "something that you should be in a hospital receiving radiation for" whereas an eating disorder can and should be "worked on" by the person with it. Gillian asserted that calling it a disease should not excuse the person from working on it, as they have some control.

So, I think that it's – well, yes. I think it is a disease. It is an illness. And, mental illness implies disease. I don't think that it's – I don't think that you can excuse thinking of it – use the excuse of "Oh. It's a disease." To excuse not trying in recovery. Like, if you say "I want to recover." You can't go into recovery and say "But, I'm not going to eat because of this disease. And, I can't help it. So, you cure me. And, I won't do anything..." I feel like there are parts of it that you can't help. And, I feel like you do lose a lot of control over it. But, I feel like you can make the choice to try. And, if you're eating – even if you can't physically hold down more than two hundred calories a day. If you're trying to mentally work on your thought processes. That's still trying. And, disease sounds like you're not even going to work on your mental stuff. Because, that's just your disease. And, you know you can't help it. (Gillian, T-AN)

Similarly, Willa connected individual control and the biological origin:

I think the difference is for brain disease I would think of that as something that you have no control over. And anorexia I mean you don't have control over it. But you can go get treatment and cure it and be done one day. Whereas with the brain disease just kind of my impression of that is something that you can't just go fix. At least not, like, psychological treatment. And so that's the difference in my mind. (Willa, R-AN)

To summarize, if eating disorders sit uncomfortably between choice and disease, brain disease is too close to the disease side of the spectrum. Mental illness was acceptable to more people, but even then many found it necessary to distinguish eating disorders from other mental illnesses that were more severe, biologically based, dysfunctional, or more difficult for the individual to control. Discussing mental illness and brain disease enabled participants to identify contrasts between a medical concept and eating disorders, thereby clarifying underlying definitions of both. Eating disorders are something that develop over time, and can be ameliorated or recovered from over time (though some were “on the fence” about whether you could fully recover or not) by means of the person’s own effort, rather than the physical intervention of a doctor upon a passive patient. Looking ahead to the chapters about genetic causation, there are hints that genetics are already part how some respondents make sense of both mental illness and brain disease. They can be viewed simply (e.g., Willa, Tammy) or complexly (e.g., Olga). Environmental influence and individual volition are at odds with a simple genetic or other biological conception. A subset viewed “brain disease” as an empirical question, requiring more research; they may be similarly open to the idea of genetic contributions. As suggested in the introduction to this section, the criteria respondents use to decide whether an eating disorder is a “mental illness” or “brain disease” tell us not only about how they view eating disorders, but also how they define these medicalized terms. Their responses could be approached from an opposite tack and used to show what mental illness means: a permanent, biologically based, severely dysfunctional state that is not under the individual’s control.

4.3 “Physical illness”?

Of 48 who were asked, 19 endorsed the idea that AN or BN were “physical illnesses”, 29 did not. Although these groups differed on whether to endorse the term, nearly all spoke about the physical effects of eating disorders. I considered endorsement of the term to indicate a more medicalized orientation but do not present quotations from those who endorsed it separately from those who opposed it. The difference consisted in whether to apply the term “physical illness” to the physical effects of eating disorders, which was not accompanied by as much deliberation as other terms elicited.

Thirty-four focused their answers on the physical *effects* of eating disorders: 14 said it was a physical illness for this reason, 20 said it was not. Petra stated simply, “it’s a mental illness that affects you physically” (coded as a non-endorsement). Those who endorsed it often said that although it did not start as a physical illness, it became one over time. Respondents listed a wide array of specific physical effects: damaged teeth, being weak and fatigued; feeling ill, feeling cold, disturbed sleep, losing hair, loss of menstrual period, osteoporosis, electrolyte imbalances, dehydration, cardiac problems, lanugo (fine body hair), and death. Delia (T-AN) observed that “it does a number on your esophagus and your bones and your teeth and your kidneys and your liver and all your organs. It really takes a toll on your physical body.” Mireya also mentioned her body’s adaptation to BN, identifying the ease with which she can throw up as “really dangerous physically ... [b]ecause your body shouldn’t be able to do that.” For some, “the physical is just a symptom” (Claire) and therefore it’s not a physical illness; for others the symptom or effect was part and parcel of the disorder, warranting the label of “physical illness.”

Although the most important way of reasoning about physical illness was in relation to the physical effects of eating disorders, several used reasoning of the kind presented in Table 4.2 here too. Irene rejected the idea, contrasting individual control with physical origins.

So, I guess it's not a physical illness because it's in my control. Whereas cancer is something that happens to you. You may be able to treat it. But, you can't say "I'm not going to get cancer." And then, not get it. Whereas somebody could say "I'm never going to vomit." And then, in theory never vomit. They have some control over it. (Irene, R-BN)

The implausibility of simplistic biological treatment and static conceptions that excluded a role for environment were also linked to physical illness:

I think it has physical symptoms definitely. But it's not just a physical illness in that you cannot just do physical therapy or pop a pill or again is something unaffected by environmental or psychological factors. (Sydney, T-BN)

I think it's more a set of actions as opposed to an actual condition of the body itself. A set of actions that's connected to sort of the mental side or the cognitive side. But it's fundamentally about sort of the actions that you – it's actions. Not a physical condition. (Zinnia, T-BN)

I don't think it's a physical illness. Because I think of physical illness as, like, you just getting sick. And maybe throwing up because you have the flu or something. That's what I think of as physical illness. I don't think it's a physical illness. (Yolanda, T-BN)

To summarize, a minority viewed AN and BN as physical illnesses. However, there was general agreement that eating disorders can result in physical health problems. Recalling one of the compound causal factors featured in Chapter 3, it was the sheer repetition of eating disorder behaviors that resulted in physical effects on teeth, bones, esophagus, heart, and brain.

4.4 “Choice”?

So far I have discussed how respondents view terms that connote medical conceptions of eating disorder. Now I turn to a non-medical conception of eating disorders: choice. Thinking of eating disorders as a choice places it in the volitional realm, something a person could stop if she or he simply decided to. A more medicalized approach would insist that “choice” is an inaccurate and insulting way of describing the phenomenon; diseases are not chosen. A less medicalized view would not object to the idea as much, and entertain the idea that people choose to have eating disorders. As with the other terms, I separated respondents into two categories, one with more medicalized views based on their committed rejection of the term “choice” (n=14), the other less medicalized (n=36). I classified those who said emphatically or consistently that it was not a choice, as having more medicalized views than others, even if they acknowledged that some aspects were chosen. The group with less medicalized views includes those who said it was a choice, or that it was both a choice and not a choice. The group coded as less medicalized was more ambivalent, and occasionally embraced choice. I probed extensively on the theme of choice, but classified them into these two categories based on their initial responses. (Answers here recall themes from the section on agentic language in Chapter 3.)

1.4.4.1 Not a choice. Of the 14 respondents who said it was not a choice, many were passionately emphatic about it. Because respondents’ experience of and perceptions of choice in eating disorders is central to this project, I present more and longer excerpts in this section than for previous sections. These excerpts represent an important subset of people with eating disorders. The five listed below were currently receiving treatment in the inpatient unit or the day program for AN or BN.

Oh, no. No. No. No. I don't think anyone with any eating disorder would ever choose to feel like they feel. That's an infuriating thing when people say that.... Because, they don't understand it. I mean who likes to throw up? Nobody. Who – nobody would choose to be as miserable as eating disorders make you. I mean it's such an isolating thing. You just feel completely alone. And, honestly you're your own worst enemy. It's miserable. I mean you should just be able to just be alone. And, be with your thoughts. And, just be safe. But, it's awful. And, I can't explain it to someone who has that mentality that it's a choice. Because, I mean nobody chooses any kind of illness. (Jackie, T-BN)

It's not a choice. No. I mean I can't ever see it being a choice for anybody.... I can't see how anyone would choose to have something like this just take over. Take over their thinking. Their way of life. And, something that's destroying you. But, you can't do anything about it at the time. You feel like it's something that you just have that. And, you just got to – that's become – that's you. That's part of you. But, you struggle each day with it. Because, you worry. You got to make sure the weight don't go up. Because, if it goes up, you're going to have to lose more weight. You're going to have to exercise more. It's just like – I mean it consumes your whole life. You're existing. You're not living. You're isolating yourself from everybody but maybe your close family. And, it's like you're in your own little world. (Karen, T-AN)

It's not. It's never a choice... That's probably one of my pet peeves is when people says "But, it's your choice." And, it's like, "No."... just like anybody else I think it's a choice to want to lose five pounds at the beginning. But, then I think with obsessiveness and with compulsiveness and rigidity. I mean I think genetics is a lot [of it]. And, your personality just makes it more, like, an extreme thing. Like, all or nothing. Like, I'm either fat or skinny. Or, whatever. Like, I think that's when it's not – I mean once you – I think all normal eaters have a choice to eat a little healthier. But, after that it's not a choice. (Liana, T-AN)

It's not a choice. Nobody would choose this. It's hell to live through. ... [People] think you have the choice to eat or not eat. But you don't feel you have the choice to eat or not eat. If it was that simple, I wouldn't be sitting here [in the inpatient unit] for the seventh time. And there wouldn't be clinics like this for people who have this disease. It is not a choice. It is something that you cannot – it's not like you go out and say "Hey. I believe I'll get anorexia today. And drive my whole family crazy and make my life miserable." It's not a choice. I think it's something that happens gradually over a period of time until it just snowballs into something that becomes what mine is today. Which is a completely horrible, nasty, terrible disease that I can't get rid of. (Petra, T-AN)

I don't think it is at all. I think it's an addiction wherever you're at on the spectrum. Like a way to control your life. ...[T]hat's the only way that I'm going to get better. Is to define myself as an addict. And, I do. And, it's an addiction if you're addicted to not eating. It's an addiction if you're addicted to food. And, I don't think any addiction is a choice. (Claire, T-AN)

At the simplest level, respondents' reactions to the idea that it is chosen hinge on the following proposition: (1) People choose positive rather than negative things for themselves, (2) eating disorders are negative, (3) therefore logically nobody would choose them. Petra and Jackie use the words "disease" and "illness" to describe eating disorders to make the case that nobody would choose such a thing; "nobody chooses an illness," as Jackie put it. Petra and others (not quoted here) pointed out that they would not be in the inpatient unit if they had a choice, and that the very existence of treatment centers proves the point. It is hell, self-destructive, makes you and others miserable, is not "enjoyable," to put it mildly (as Eva did). (At the end of this section on "choice" I address again the presumption that choice is only a choice if it's positive, and provide an analytic table to organize some of the results presented in this section, Table 4.5.)

Beyond this simple proposition, however, there are many distinctions drawn by the respondents above that merit a more finely grained analysis. To organize my discussion of respondents who argued against the idea of choice, I present conceptual distinctions drawn by respondents, with illustrations of each. These conceptual distinctions illuminate their reasoning about choice, what aspects of eating disorders are and are not choices, or what might make others perceive them as such. These are summarized in Table 4.3 and examples of each are provided, using the shorthand title provided in the left column. (People who were less emphatically against the idea of "choice" also mentioned these themes along with a few others, see Table 4.4.)

As they discussed these conceptual distinctions, respondents were aware that from other vantage points, their behavior seemed controllable and therefore a choice. Respondents mentioned family members, friends, and even healthcare providers who presumed that they

could stop if they would just listen to reason. As Jackie said above, “they don’t understand it.” Several respondents lamented such misunderstandings in answer to the question, sometimes angrily like Wendy: “That almost makes me very angry. Thinking about again those stupid people I was talking about that think “Oh. Why don’t you just stop?” Yvette suggests that nobody “in their right mind” would have AN, which indicates that only people who are in their right mind can be said to make choices.

I mean if you look from it, it looks like just a choice. Like, as my [adoptive] dad said “You can choose to eat or not.” But it’s not – like, no one would be anorexic if it was just a choice. Like, I don’t think anybody in their right mind would choose to just not eat and to be unhealthy and to be, like, dying or on the verge of dying. Like, I’ve had people tell me that I look like I was an Auschwitz survivor. Like, no one chooses to get that thin who’s in their right mind. Like, there has to be something else in the game. Like, definitely. (Yvette, R-AN)

Petra explained, “they think you have the choice to eat or not eat. But you don’t feel you have the choice to eat or not eat.” The other themes below show nuances of this basic idea, by breaking eating disorders down into components, some of which are or can be chosen and others that cannot.

Table 4.3: The finer points of choice I: Distinctions made by respondents rejecting “choice”

Choose actions/ behaviors, not eating disorders	Choosing to refrain from a specific action at one moment in time (possible, though it is hard), but cannot “choose” to stop feeling compelled to do it. Related: Can choose to stop behaviors; Can choose not to “listen” to the eating disorder but it is still there; Can choose to avoid “triggering” situations and prevent your own eating disorder behaviors, but not being able to directly stop the behaviors.
Choosing to start behaviors	Choosing to diet or purge initially, but not choosing to have it go out of control, for it to become an eating disorder.
Losing choice	Losing the ability to choose whether to diet, purge, etc. Not a choice because external or biological forces exert control over you, or because a habit takes you over.
Regaining choice	Becomes a choice through recovery; same action may not have been a choice earlier.
Choice without blame	Choices you can reasonably be held responsible for, vs. choices for which you ought not be. Related: Choosing among a constrained set of maladaptive coping strategies

Choose specific physical actions, not the eating disorder. Respondents drew a distinction between choosing a specific action – eating a meal, purging – and choosing to have an eating disorder. Thus, virtually nobody thought people actually chose to have an eating disorder per se. But they did think it was possible to choose to refrain from restricting, bingeing, or purging in a given moment. They spoke of choosing to restrict or purge as part of the development of the eating disorder, after which they no longer had as much choice. They also said that even in the throes of the eating disorder it was possible to speak of “choosing” to eat or refrain from bingeing and purging.

Even while experiencing an eating disorder it was possible to exert choice about physical actions in the moment.

...you can physically, like, control whether or not, like, you go to the bathroom and purge. But, I think in the sense that you lose control it's more of like a mental, like, emotional thing. Where, like, your anxiety just gets so bad. That, like, in the moment that's the only thing you can do to fix it. (Eva, T-BN)

Respondents defined the eating disorder more in terms of the “emotional thing”, the underlying urges or compulsions, not the physical acts.⁶ Thus, the eating disorder was there even if one didn't act upon it. Ingrid separates the choice to “listen to” the eating disorder, which tells her to restrict food, from the eating disorder telling her what to do, which she did not choose.

I don't have a choice. It's with me all the time. I have a choice not to think about it. I have a choice not to restrict. But, I know that the disorder is greater than the choices I make. I can make the disorder. I mean I can either not listen to my eating disorder and go ahead and make that choice. But, the choice I have is either to listen to it or not listen to it. One day I may decide not to listen to it. And, I'll go ahead and eat. But, then the remorse of eating and so on and so forth. Because, the eating disorder comes back and says “Oh. You fat pig. Now, you've got to go home and restrict.” That is not

⁶ This is interesting because physical acts are part of the official DSM criteria for AN and BN. There is no AN without restriction, no BN without bingeing and purging; having the thoughts but not acting upon them would be a different diagnosis or not receive a diagnosis at all.

a choice. It is a choice in the sense I don't have to listen to it. But, especially if it's chronic it's very hard not to. It's very hard. (Ingrid, T-AN)

According to Ingrid, the eating disorder is always there – she has no choice about that. Her only choice is about her behavior moment to moment.

In treatment, patients are held responsible for their eating-related behavior; this is consistent with Ingrid's account because the eating disorder is recognized but it is separated from the individual's action in the moment. Eva, Ingrid, and Mary (below) were all either inpatients or in the day program, confronted daily with three meals plus snacks. After eating, they and their fellow patients were “on obs” or observation, during which nobody was allowed to go the bathroom (to prevent secret purging). Mary expands on the theme:

I don't agree with that [choice] as the cause of it. I don't think anybody chooses to have anorexia. But, there is a part that you're responsible for choosing whether or not to eat at any given meal... I mean nobody chooses to have something that they're slowly killing themselves to do to manage stress or to cope with – using as a coping skill. But, once you start trying to recover, and you sit down to a meal, it is your choice whether or not you eat. (Mary, T-AN)

As a part of recovery then, the individual is responsible for consuming meals and complying with other rules. Thus, for Mary and others, it might be reasonable to use the word “choice” to apply to specific acts, even though other aspects of the eating disorder are not under their control. Presumably, Amy (T-AN) is referring to this when she says, “Once you get it, you can either choose to get better or not get better. That's the choice. That's the only choice.” It is not a choice to develop an eating disorder, but once it has emerged, choice can play a role in recovery.

As part of working on recovery, behaviors could be controlled through prevention and management. Claire, below, describes the difficulty of self-interpretation. She did not know whether to interpret a recent binge as part of the disorder or as an indirect choice by

“letting” herself do behaviors that led to it. She had recently completed the structured day program and was working on recovery as an outpatient. She speculated that if she had made other choices, the binge might have been prevented; she struggles with how much responsibility she should take.

If I choose like I did on Friday to not exercise. Which I know affects me in a really big way. And then, that in turn made it so that I didn't go and do what I wanted to do with the evening. And then, I didn't know what I was going to do with the evening. So, I like, ended up engaging in one of my pretty bad binge behaviors. And, I like – I hadn't done – I hadn't gone and been in the grocery store in a long time. And, I, like, went in the grocery store. And, was just, like, letting myself just look around and, like, fantasize about bingeing. And, that stuff is hard. Because, it's, like, it could be disorder. But, I also have control over it. And so, it's like, “Wow. You're just being really ridiculous right now.” But, am I being really ridiculous? Or, is it partially I can't help it?” (Claire, T-AN)

Claire narrates her choice not to exercise as the beginning of a chain of events that led to one of her “pretty bad binge behaviors.” She describes a dual agency, in which she is “letting” herself fantasize about bingeing at a grocery store. She is unsure whether to hold herself responsible – is she being “ridiculous” - or whether she can't help it, at least in part.

Choosing to start behaviors at beginning of eating disorder. Eating disorders did not begin with a conscious moment of choice, according to virtually all respondents (Willa is the exception). Yet respondents recalled and acknowledged having control over their behavior at the beginning of the eating disorder, or before it started.

Several made their point by mocking the idea that someone might “wake up one day” and consciously choose to have AN or BN.

It's not like people with anorexia wake up one day and say “Okay. This is going to be the day that I do not eat for the rest of my life.” (Amy, T-AN)

“It is not a choice....it's not like you go out and say “Hey. I believe I'll get anorexia today. And drive my whole family crazy and make my life miserable.” It's not a choice. I think it's something that happens gradually over a period of time until it just snowballs into something that becomes what mine is today.” (Petra, T-AN)

I just kind of slid into it. I was never happy about it. No. (Fran, R-BN)

It's not something that's, like, enjoyable. Or, they like, wake up every day and get like, really excited about it. Decide I'm going to do it. It's just – I guess it's kind of something that developed over time. (Eva, T-BN)

The conscious decision-maker, clear-minded and free to contemplate alternatives, who wakes up one day and chooses to have an eating disorder is contrasted with a less conscious and more passive person. Rather than a moment of decision there is a slower, more gradual process. In addition, such a “choice” is rendered implausible because it has such clear negative impacts (as noted above); such a choice suggests that the person's ability to choose is impaired thereby invalidating it as a choice.

However, respondents acknowledged that voluntary acts of weight control such as dieting and purging were present at the beginning. These acts may have been chosen, but this did not mean the eating disorder was. “[J]ust like anybody else I think it's a choice to want to lose five pounds at the beginning... I think all normal eaters have a choice to eat a little healthier. But, after that it's not a choice. (Liana, T-AN, quoted above) Fiona describes such initial choices as the “instant”, immediate things, which are more “innocent.” Then such a choice “kind of twists itself” and turns into an eating disorder that is out of control.

I don't think anyone really chooses to have anorexia. I mean I don't – I think it's more you don't choose it. It just kind of happens. Because, I didn't want anorexia. I would have never chosen this for myself. So, I think it's just, like, you choose the instant thing. Like, “Let me just lose a little bit of weight.” And then, it kind of twists itself. So, I don't think anyone willingly chooses it. It just kind of happens.... Initially I think you choose. I mean, like, you choose an innocent thing. But, as you start losing the weight, something in your brain isn't connecting right. And, it becomes obsessive. And, that's when you start to lose control of it. Because, it's no longer your decision. It's your obsession with it. It's an eating disorder that's controlling you more.” (Fiona, T-AN)

The idea of choosing to have an eating disorder is thus deconstructed by breaking the connection between initial acts of dieting and the eating disorder, whose hallmark is a loss of control and lack of choice. Eva (T-BN) defined the eating disorder by the loss of ability to control such acts: “it really, like, becomes bulimia kind of like, when you lose control. If you still have control of it, then I don’t think it’s really an eating disorder.”

Losing choice. How did respondents go from initially being able to control behaviors to an out-of-control eating disorder? Respondents had diverse theories, some more biological, others about repetition of behavior. The latter will be discussed when I turn to the more ambivalent, more choice-oriented respondents.

Biological forces helped explain the lack of control for a few respondents. Liana (already quoted) brought up genetics in connection with an obsessive personality. Reba alludes to genetic factors (which she had already mentioned earlier in the interview), life stressors, and to a lesser extent her “choice” to cope by restricting (discussed later).

According to Reba (R-AN), “It’s not really a choice. I think I was born into this family where there were eating disorders that were prevalent. And I don’t think I had much choice in the matter.” Fiona spoke of “something in your brain” that “isn’t connecting right,” followed by the effects of extreme weight loss on the brain:

Initially I think you choose. I mean, like, you choose an innocent thing. But, as you start losing the weight, something in your brain isn’t connecting right. And, it becomes obsessive. And, that’s when you start to lose control of it. Because, it’s no longer your decision. It’s your obsession with it. It’s an eating disorder that’s controlling you more. Because, things aren’t processing as well as they used to in your head as you lose weight. And, that’s where you become obsessed. (Fiona, T-AN)

The eating disorder takes control as the brain becomes impaired and the person becomes obsessed.

Regaining choice or the capacity to choose. If choice is involved in recovery, as several asserted, how does the person regain the ability to choose? Fiona above spoke of losing the ability to choose, or to be “rational,” as partly an effect of too much weight loss. Logically, she also asserted that regaining weight would help restore choice.

And, that’s why I think that at some point you gaining weight helps you get your thinking processes back to make you more rational. And, I think I have experienced that. Because, there’s certain things that’s like, “Why did I do that in the past? That was so stupid of me.” And so, I do think that gaining weight a little bit returns – makes you more rational when you think. But, as you lose it, you re-lose more common sense. (Fiona, T-AN)

With weight gain, the ability to think more rationally returns, which entails choice for Fiona. It probably cannot explain all of the loss of control, however, because losing weight to the point of affecting the brain reflects AN in and of itself (and recalls themes raised in the discussion of the term “physical illness”).

Wendy had a less biological account of how choice is regained: through realizing her own self-worth. This realization is won through a learning process; by counteracting years of learning she was worth little, her self-worth will be developed or restored and a real choice will become possible.

R: I think that it only becomes a choice once you learn that it’s a choice. Like for instance it’s not a choice until you realize that you don’t have to do it anymore. And then it becomes a choice.

I: How would people come to learn that it can be a choice?

R: Through treatment. And through realizing that people did wrong things to them. And through coming to learn your own self-worth. And through coming to learn how to validate yourself. And that you deserve to be validated. Just learning that you deserve to be healthy. I think it requires a lot of treatment before you can come to the place where you could honestly say that it was a choice. (Wendy, T-BN)

Regaining choice is clearly linked to recovery for some respondents, though my analysis did not assess the frequency of this perspective.

Choice without blame. Some respondents articulated a type of choosing that did not confer blame on the chooser. The choice to begin or continue eating disorder behaviors was so constrained that the person could not be held responsible. Wendy suggested that even though by some definitions people chose their behaviors they should not be held responsible.

You could make the case that it's always a choice. Because obviously it's always your choice whether or not you go throw up. Even when you're first starting out before you know any better. So I guess technically it's always a choice. But not in the sense of blame. Like, it's a choice in the sense that it's the best you know how to do. (Wendy, T-BN)

Reba articulated a similar view about whether it was a choice:

...maybe just in the sense of it's something that I picked up on early on in my starting adolescence that was my way of coping with life stressors. Maybe I could have chosen other avenues. But in that sense it was a choice maybe. That I chose anorexia over other ways of – maladaptive ways of coping. But for the most part I don't think I had a lot of choice. (Reba, R-AN)

The sense of choice without blame could require active “work” on the part of the respondent. Claire struggled with how to interpret her own behavior, and strove to find a way of achieving a sense of choice without blame. It was important that she not think negatively about herself, because this contributed to the eating disorder. She was more likely to feel negatively about herself if she saw it as a choice.

It's like, I can be like, it's a choice and it's not my fault. But, that's something that I'm still working on. But, I think I realize more now than before. I don't know. I can't – I have trouble drawing the line. Like, where is it a choice and where is it – where is it a choice and where is it, like, totally out of my control? And, I think it's healthier to believe that a lot of the negative things that I've done around food are not my fault... The bottom line is I need to think positively about myself. And, that's what OA [Overeaters Anonymous] is all about. Is like, “You need to think positively about you.” So, it helps me to think positively about myself if I think about it as a disorder and not my fault. (Claire, T-AN)

The “bottom line” of positive self-conception was more important than assigning responsibility and blame. By theorizing choice without blame, respondents ceded some

ground to the idea of choice while rejecting it as an adequate term to describe their eating disorders overall. This difficult balancing act, between forgiveness and responsibility, is central to the themes of this project, and informs how respondents consider the idea of genetic contributions.

4.4.2 Ambivalent or favorable about “choice”

I turn now to the diverse group of respondents who were more ambivalent or at times even favorable toward the idea of choice. This group includes people who say it is a choice, or that it both is and is not a choice. These ambivalent respondents did not argue as strenuously or consistently against the idea of choice, compared to those described above, whom I consider to have more medicalized understandings of eating disorders.

Although I have categorized these more ambivalent respondents as having a less medicalized view, they nevertheless echoed the themes described already by those with more medicalized views. The decision about whether to endorse the word “choice” seemed to be based on how best to frame a complex, mixed situation (e.g., Claire’s thoughts about “choice without blame”). Rather than provide quotes to illustrate the presence of each of the previously-described themes for this new set of respondents, I present a single quote that incorporates many of the themes above, by way of reminder.

Well, at first I believe it’s a choice. I had the choice to make myself throw up. And, I had the choice to start a diet. But, it wasn’t my choice to be born into an addiction family. And, because of that, I got out of control. Sometimes it’s not my choice. But, when I learn the skills to do something different, then I can begin to believe that I do have a choice. (Delia, T-AN)

This account of shifting choice across the “life course” of the eating disorder does not focus on the lack of choice, but rather the changing levels of choice.

Although answers for this more ambivalent group of respondents resembled the others, a few new themes emerged. The new themes are presented in Table 4.4 and each is closer to a view that a choice was made, compared to the group just described that rejected the idea of choice more definitively.

Table 4.4. The finer points of choice II: Distinctions made by respondents ambivalent about “choice”

Nobody is forcing me	It must be a choice because nobody is forcing me to do these things.
Seeking something good	Eating disorders are a problematic way of trying to achieve something good. It is a choice, but <i>not</i> for something negative.
Choice that is constrained, not free	It is a choice but there is something behind it, or there are very few options. It should <i>not</i> be seen as a <i>free</i> choice.
Choices become habits	Through repeatedly choosing to engage in this behavior, it became engrained and therefore less under my control. Habits are not really choices, though they may be the result of repeated choices.
No excuses; it’s a choice	Calling it a choice is appropriate because it makes the person take responsibility. Saying it is not a choice is a cop-out.

Nobody is forcing me. Some respondents who were more ambivalent about choice argued that it must be a choice because they were not being compelled by another person nor threat of death. This did not mean they saw it as a completely free choice, but it met a minimum standard for agency.

I don’t dispute that it appears to be a choice for most people. I mean it’s an outside substance that you’re putting into your body. You’re the one who’s doing that to yourself. Nobody’s forcing that on you. (Isabelle, T-BN)

I know that there are other ways. I know that I don’t have to do this. That, like, I’m not going to die if I don’t do this. Or I’m not going to – like my life is not at stake if I don’t do this anymore. I feel like I make a choice. (Zinnia, T-BN)

I think it is. Because I mean I know I could make the choice tomorrow to stop. And like, just force myself to. There’s nothing in me that’s like, making me have bulimia. Like, literally shoving my finger down my throat. Like, if I really, really needed to, I could stop. (Yolanda, T-BN)

Well I do think it is a choice. Nobody forced me to do that or continue to do that or anything. So yeah. But also addiction too. (Melanie, R-BN)

By the logic of these metaphors, if nobody is forcing them and their life is not at stake, it must be a choice. This is a low bar for choice and leaves out the sense that choices can be constrained or compelled, which the quoted respondents also noted.⁷

A choice that is constrained, not free. The more ambivalent group of respondents also spoke about constrained choices, elaborating further on the idea of choice without blame. As with the earlier group, the choice to engage in eating disorder behavior is constrained, compelled, or forced, making it not a free choice. They accept the idea of choice to a greater overall extent than the earlier group, but share similar reasoning by insisting that it is not a *free* choice. Margaret (R-AN) actually used the phrase “constrained choices,” to convey the effect of gender inequality on women (she mentioned having studied feminism and social sciences). Even if eating disorder may appear to others, and even to herself at times, as an individual choice, she maintained that “there’s constrained choices that women make. There aren’t that many choices. The consequences for getting heavy in society. And for speaking out. Being angry.” If women cannot speak out or be angry like men, and there are also consequences for gaining weight, there are fewer choices available, or fewer socially approved choices. “Choosing” an eating disorder is thus constrained by imperatives that channel the person toward some things and not others. Carly talks about pressures on people:

I mean it’s your choice to binge and purge. But there are usually some influences on you making that decision. Like some outside pressures or – mostly outside pressures. Which usually lead to mental or, like, inner pressures for yourself. . . I mean you’re the one that decides. . . . But you usually – depending on your mental capabilities and

⁷ Just because respondents used this logic did not mean that they believed it to be the full story. Nell used a metaphor that evoked life-or-death situations, but to a different effect. She compared someone with an eating disorder to someone who is “under the gun”, saying that decisions made in such conditions are *not* conscious and free choices. As she put it, “sometimes ‘choice’ makes it sound as though you were free to choose. And that you were free to choose another alternative.” While Nell’s reasoning is very similar to respondents who clearly rejected the idea of choice, she is ambivalent overall; she began her response by saying, “It really kind of was my choice in that I guess I knew what I was doing and things like that.”

your mental strength that'll determine if you can find those other ways or not. (Carly, R-BN)

“Outside pressures” produce “inner pressures” on the person, whose decisions and choices are influenced as a result. Carly theorizes that some people might be able to find alternatives to an eating disorder, depending on other characteristics, like “mental strength.”

Nell thought an eating disorder could be “chosen” as a way to avoid traumatic memories. This would not be a free choice because, as she says, “if I wasn’t dealing with all that kind of stuff, then I wouldn’t have to choose it.”

[I]t’s not a choice you would make if you didn’t have to make the choice. If you – if situation wasn’t so bad that you – that sort of like, “Well I’m going to choose anorexia. Or else I’m going to have to deal with the fact that I was beaten by my parents or raped by my boyfriend or something like that.” So in that way I’m choosing anorexia over something even worse. But if I wasn’t dealing with all that kind of stuff, then I wouldn’t have to choose it. I don’t know. Does it make sense?” (Nell, R-AN)

In a situation with no very good options, the eating disorder is “chosen” but not freely.

Seeking something good. As noted at the beginning of this section on “choice”, an important reason for objecting to the idea of “choice” is that nobody would choose something as bad as an eating disorder. It makes sense, then, that those respondents who incorporate the idea of choice into eating disorders may think of it as an attempt to achieve something good, even though the results are bad.

In a constrained situation, eating disorder behavior may be better than some even more negative alternative (this is similar to the idea presented by Nell above). The idea that an eating disorder is a problematic way of achieving a positive goal is similar to the idea that it is a way of trying to communicate something or cope with something, which has been presented above. Although one group argued against choice more fervently, the two groups

raised similar themes. Alyce offers that it may be possible that the eating disorder actually helps the person in a bad situation:

... I think you have to look at the degree of pain that they're living with. In their family. And, in their personal situation. And, only in the context of looking at the personal pain that they're feeling can you assess whether or not it's a good choice to self-medicate with food or some other substance. Because, maybe that's a good choice. Maybe that's what it's going to take to keep them going until they can get the hell out. So, I guess what I'm saying is sure. In some sense it's a choice. You have to look at the developmental age of onset. And, you have to look at the cost and benefit to the person in their situation. I mean they're not, like, a perfectly happy person choosing to binge and purge all the time. There's something driving that. And, there's a perceived benefit. (Alyce, R-AN)

Alyce characterizes the person with an eating disorder as someone who seeks the good, tries to preserve herself through means that serve her in a particular situation. Karen said it was a way to achieve love, not a "choice to be sick" nor "to do something detrimental."

And, the choice for me wasn't whether I wanted to have anorexia. It was kind of more "If I don't eat this, then I will lose weight. And, I'm going to look good. And then, I'm going to have – somebody's going to love me." It was kind of more like that kind of thought process. So, it's not a choice to be sick. Or, to do something detrimental.... It's a choice that you kind of get a little high from it or something. Because, you get some kind of positive feedback I guess. (Karen, T-AN)

Even for Willa, the only respondent who said she consciously chose to have AN, the choice was made to achieve something good, not bad. Other respondents said they may have chosen something, but it wasn't the actual eating disorder. By contrast, Willa says she did choose AN, "I feel like in a way I did choose anorexia. Because I thought it was this very glamorous thing." To make sure I understood, I restated it more conservatively, asking if she was saying she was conscious it could become anorexia and that was OK with her. She corrected me saying, "No. I think it was more of 'Let's be anorexic today.'" But she describes her choice not as self-destructive but as wanting the "good things" about AN and ignoring the bad ones.

...like, you don't wake up and say "Oh my gosh. I want to be in the hospital and in a wheelchair and weigh fifty pounds." But it's wanting kind of all the, like, good things that come along with anorexia and none of the bad things. So in that way kind of striving for it. Until you get to that point where you feel like – you suddenly realize there's so much bad that comes along with it it's not worth it. But I could definitely see it as a choice. (Willa, R-AN)

The group that was ambivalent or favorable toward "choice" thus theorized ways of understanding the eating disorder as an attempt to obtain something positive, which was compatible with the idea of choice.

Choices become habits or "second nature" over time. Through repeating chosen behaviors, the person develops a habit, which then has its own momentum. I discussed this in Chapter 3, in relation to causal factors that help to maintain or intensify the eating disorder, and in the section about agentic and non-agentic language. The repetition of behavior was also important as respondents talked about choice, and relates to the subsection on "Losing choice" described above for respondents who emphasized the *lack* of choice in eating disorders.

Respondents described how choices became habits using a variety of terms. Some spoke of them as "habits", such as Gena (R-BN): "I made that choice. However, when it got to the point where it was habit. And, that I could throw up without even trying. That was – it was no longer a choice. It was something that was completely out of my hands." A few spoke of them as a "pattern" you could "slip" into (Hannah, R-AN); "I don't feel that once you get into that pattern that it's a choice anymore" (Olga, R-AN); Melanie (R-BN) said it was a choice, "But also addiction too. Because... it becomes physical. If I don't have like, a Coke or something, I get a headache. So it's – initially it's definitely a choice. I think once you're in the throes of it it's more of an addiction or more of a physical thing." Choices could become something even more powerful than habit: a kind of "second nature" according to

Isabelle (T-BN): “you use it out of habit for so long that it really becomes second nature.”

Some respondents explored the process of choice becoming habit or second nature in greater length, and I quote them at length in the following paragraphs.

Thelma thought her BN was a choice, but because of the “compulsion” she feels the choice gets “a little bit more blurry” and “harder to define.” She explained that the sheer repetition of a behavior could produce a problem like OCD.

I learned in psychology class in high school that when they were making that movie “The Aviator” about the OCD, like, airplane mogul or whatever that the actor ... doing all these incredibly OCD things ended up picking it up. Like it’s something that can be kind of absorbed. And that it’s like, a real clinical phenomenon. And so I feel like repetitious acts kind of become, like, engrained. And so just, like, the longer it goes on the more of a just kind of like a – I guess I feel like I have to do it. And it’s just like, it’s like, something that’s in the back of my brain – mind. And it’s distracting. Because I can’t – I keep thinking about it. Like, if I’m out to dinner with friends. Like, it’s distracting. And it like, prevents me from like, enjoying the people I’m with and, like, having a good time. And if I just like, go do it, it’s like, a weight has been lifted off my shoulders. (Thelma, T-BN)

An actor playing someone with OCD developed OCD because he repeated the behaviors. She uses the words “absorbed” and “engrained”, which convey something external being taken in and becoming part of the being that took it in. These are metaphors for how history and experience make their way “into” the body, how social forces get “under the skin.”

Vanessa thought of the eating disorder as a choice initially, but that the repeated action of purging essentially taught her body a new sense of what is normal. As others have described, purging required practice, which Vanessa sees as a choice. But choice is compromised after enough repetition.

[A]fter being in it for a while it really takes you over. And it starts to control you. Because we don’t have that normal tendency to just be able to sit and throw up. I mean regurgitate. It’s just after time your body conforms to that upward motion. That it’s okay to do this upward motion. And I think the muscles or whatever desensitizes to stop it. So in that instance that’s why I say over time it takes over you. But you initially make that choice. ... while I was in that mode and doing it regularly it

became easier and easier. I mean when I first started I had to stick the toothbrush down my throat... And you get the gag reflex. And it still wouldn't come up. Then you do it again. And you do it again. And then it comes up more. But the more I did it the more that it was easier. I only have to stick the toothbrush down once now. And it would just flow and flow and flow. And that's what I mean by eventually it takes you over. Because your body gets used to doing certain things that it didn't do before. And it says "Okay. Well that must be normal. I do it every day. So let me just go ahead and do it. Because that's what I should be doing." (Vanessa, R-BN)

Mireya talked about the fact of having binged and purged as having set a powerful precedent that compels present action. She has gone over to the "other side", she has broken a "code", she has violated "normal" and even "sacred" expectations. Here, the past influences the present in a different way than simple repetition. In this remarkable passage she describes a kind of fall from grace, in which people obey a "code" of sharing food with each other and digesting it completely. She asserts that food shared with the family ought to be treated differently from "crap" food from McDonalds, but she has not respected that distinction.

It's really like a formidable force that's like, once, like, you break that kind of code that we all have is, like, we eat and then we digest and we go to the bathroom. Like, that's it. So it's like, once you break that code it's very hard to go back to something that's quote-unquote "normal". Because you can kind of eat anything you want whenever you want. And you don't really need to, like, really eat it. So it's like, that's what's really, really for me not a choice is that, like, I've already gone there to that other side. And I know how it feels. It feels awful and it feels great at the same time. And that feeling is so overpowering that that feeling will always be there. So then that choice sometimes is not really a choice.... [probing]... It's like, you break a pact with your body and with, like, society in general. ...Because I mean, like, everyone's like, sharing a meal together. Everyone – like, people in general. Society in general. And then throwing up you're undoing all of that experience with people. And you're basically negating all of that. ... because you're not actually digesting your food. And you're not accepting, like, everything that came with the food. Which is preparation. It's the love. It's the money. It's the sitting down together. I mean, like, I feel like basically I've eaten and purged everything from life. Like anything. Any situation. Like, nothing is kind of sacred. It's like sometimes like, I feel like, back in the day, I'd only, like, specifically go out to eat, binge, and purge. Like, I'll buy like, shit. Like crap. Like, McDonalds or whatever. But now it's like, I have basically done it at any time, anywhere, and any place. So it's just like, nothing is – it's like, a lack of

respect too. And for me it's very important to recognize when people make meals. But I'm also disrespecting it. (Mireya, T-BN)

Mireya's sense that she no longer has a choice is related to the power of past choices.

Bingeing and purging has removed her from the realm of normal human beings, who obey natural and sacred codes, who respect the food and love provided by others.

Zinnia spoke of a milder version of habit based on knowing that she could satisfy herself in a particular way; it made sense to use a sure method rather than risk the unknown. Bingeing and purging satisfy her, and she sees them as choices. But because of past experience, the choice to binge and purge are more "attractive", "comfortable" and "routine" than the alternative, so it is difficult *not* to choose them.

I feel like I make a choice. But I make a choice out of – the choice is more attractive because it's become comfortable and routine in a way... [probing] ... it's almost like if you are going to a restaurant. And you are – do you want to try something new? Or do you go with the thing that you do? If you're hungry, do you go with the thing that may not totally satisfy the hunger and you don't really know what that tastes like and all that? Or do you go with the thing that you know what it tastes like? You know that it's going to give you this feeling and that it's going to be satisfying in a particular – you already know how it's going to be satisfying. And you know everything about it in a way. Then it's much more easier to make that choice. (Zinnia, T-BN)

The choice is "easier" because similar choices have been made before and their results are known.

Happily, patterns, habits, addictions and even "second natures" could be reversed. Several respondents thought it would be possible to learn a new "pattern" and recover. Victoria, currently in treatment for BN, speculated that "if I, like, got in the habit of not always throwing up my food after I eat. Like, that would be like, learning a new pattern that I would be able to sustain if I did eventually live alone. But it's like, when I'm so much in the pattern of doing that it's, like, hard to break the pattern on my own." Vanessa noted that the

acquired second nature can be altered; “after you get away from it your body has the ability to go back and say ‘Okay. Hum. Yeah. That doesn’t feel right anymore.’”

No excuses: It is a choice. Some respondents argued that it was important to take responsibility for the eating disorder as a choice. As noted above, many stated that behaviors were under the person’s control, even if the eating disorder as a whole was not. Some respondents metaphorically externalized their eating disorder, as discussed in the section on non-agentive language, by comparing it to a monster, a demon, the “anorexia entity,” or an abusive husband or boyfriend named Ed.⁸ Externalizing the eating disorder implies that it is not a simple choice. The external force may be compelling the person to choose something, or may be making the choice for the person. Some respondents thought there were problematic implications for thinking of their behavior in this way; claiming that something was controlling them could seem like an irresponsible excuse.

Gillian’s discussion of choice includes the implications of choice for one’s own responsibility to control behaviors such as purging and restricting. Gillian (T-AN) brings up Ed, and distinguishes between his and her choices and responsibility. Even if Ed is the one making the choices, the person has a responsibility to become “aware enough” to realize it is Ed, not to listen to him, and not to let her body carry out his orders.

...your mind is so warped by the eating disorder. That even if you make the choice to restrict, it’s not really a rational choice. So, it can’t even be considered a choice anymore. Because, *it’s your eating disorder convincing you* it’s okay to restrict. So, actually I’m going to take back what I said earlier about that being a choice. Because, the more that I think about it, the more it seems to me like *it’s not you making a choice. It’s your eating disorder making a choice....* when I was in [the day program]

⁸ The name “Ed” is a personification of the eating disorder based on the acronym e.d., as developed by Jenni Schaefer in her book *Life without Ed: How one woman declared independence from her eating disorder and how you can too* (Schaefer & Rutledge 2004). This metaphor makes an analogy between the eating disorder and an abusive boyfriend or husband, and self (or the part of the self that would like to recover) is the abused female partner (eating disorder : healthy self :: abusive male partner Ed : female partner).

– we talked a lot about the book *Life Without Ed*... Where Ed is the abusive boyfriend. And, you can't blame it all on him. Because, you can still try and challenge him in things. But, he can sound very convincing. He can be very difficult to distinguish from yourself. And so, I think that a lot of the times if you aren't trying to seek recovery, it's not because you're making a choice to be anorexic or bulimic or NOS.⁹ *It's because Ed's making a choice for you. And, you're not currently aware enough to realize that it's Ed*... I think that you can't just blame it all on Ed. Like, "Oh. I did X, X, X because Ed told me to." Like, no. That can easily become an excuse. And, also like if Ed does something horrible. *Like, even if it was Ed. It doesn't excuse the fact that you were the one who listened to him.* Like, at this one point over about a month I had stolen approximately five hundred dollars from my dad to buy pills and binge food. *And, even though I can sit there and be like, "That was Ed's influence." That didn't mean that it wasn't me who had gone and done it.* And, I still needed to pay my dad back. I still needed to accept the consequences. Like, yes. It was Ed making that decision for me. But, I still have to pay the consequences for his actions. Because, as long as he's living in my head. And, *my body is the one carrying out* what he says. In a way I guess I'm listening to him. And so, I need to be punished for listening to him. (Gillian, T-AN, emphasis added)

Sydney also thought she should take responsibility, rather than blaming it on Ed. She spoke of feeling helpless, because despite using "reason" and "logic" with herself about why she did *not* want to buy binge food at the grocery store, she nevertheless went ahead with the purchase: "And then I'm like, "Swipe [the credit card]. Check. Yes. In the basket." Her eating disorder seemed to have a "life of its own", because she could "reason to the stars and the moon" about why she did not want to buy the food, but did so anyway. She preferred to see this as emotions and feelings getting in the way of logic and reason, rather than externalizing it as a metaphorical person like Ed.

Because I feel like that kind of *takes the ball out of my court* a little. It takes some of the responsibility off of me to think "Oh. This is just – this is the war I'm fighting against something else." And I have to take responsibility that – but I'm kind of letting it. Like, *I'm letting this person in.* I'm letting him through the door. I'm letting – like whatever this entity is I am making conscious decisions. And *I need to take responsibility and say "No." to some of these choices I'm making.* And to say "Yeah. Right now the easier choice would be to just go ahead and do something I know is going to be bad. Because I know it's going to cause me less anxiety. I know it's going

⁹ NOS means "Not Otherwise Specified", which as noted in Chapter 1 is an eating disorder diagnosis that captures people who do not meet all criteria for AN or BN.

to be easier to me emotionally.” But I need to take responsibility and say “No.” And to do the harder choice. And to sit with that being uncomfortable. And to sit with being – and I think just *making it completely separate from myself sort of absolves me of that responsibility in my mind and in my head*. Again I know it works for other people. But for me personally I need to not make it separate. Because then it’s too easy for me to think like, “Oh. Well this is all on that. *I don’t have to do with it.*” (Sydney, T-BN, emphasis added)

In short, a small number of respondents emphasized their own responsibility for their behaviors (in part to aid their recovery) and as a consequence they, more than most other respondents, state that their dysfunctional eating was indeed a choice.

To interpret and summarize my discussion of choice, I created a decision tree based on answers by both groups of respondents (Figure 4.1, next page). All respondents agreed the eating disorder was a bad thing. Normally people don’t “choose” bad things, so this means that eating disorder must not have been a choice. However, in respondents’ social context, eating *behavior* is usually perceived to be chosen and claiming otherwise might not be plausible; such an account might not be honored.¹⁰

¹⁰ I will return in the concluding chapter to Scott & Lyman’s classic definition of an account as “a statement made by a social actor to explain unanticipated or untoward behavior” (Scott and Lyman p. 46).

Figure 4.1 Decision tree for conceiving of choice and eating disorders

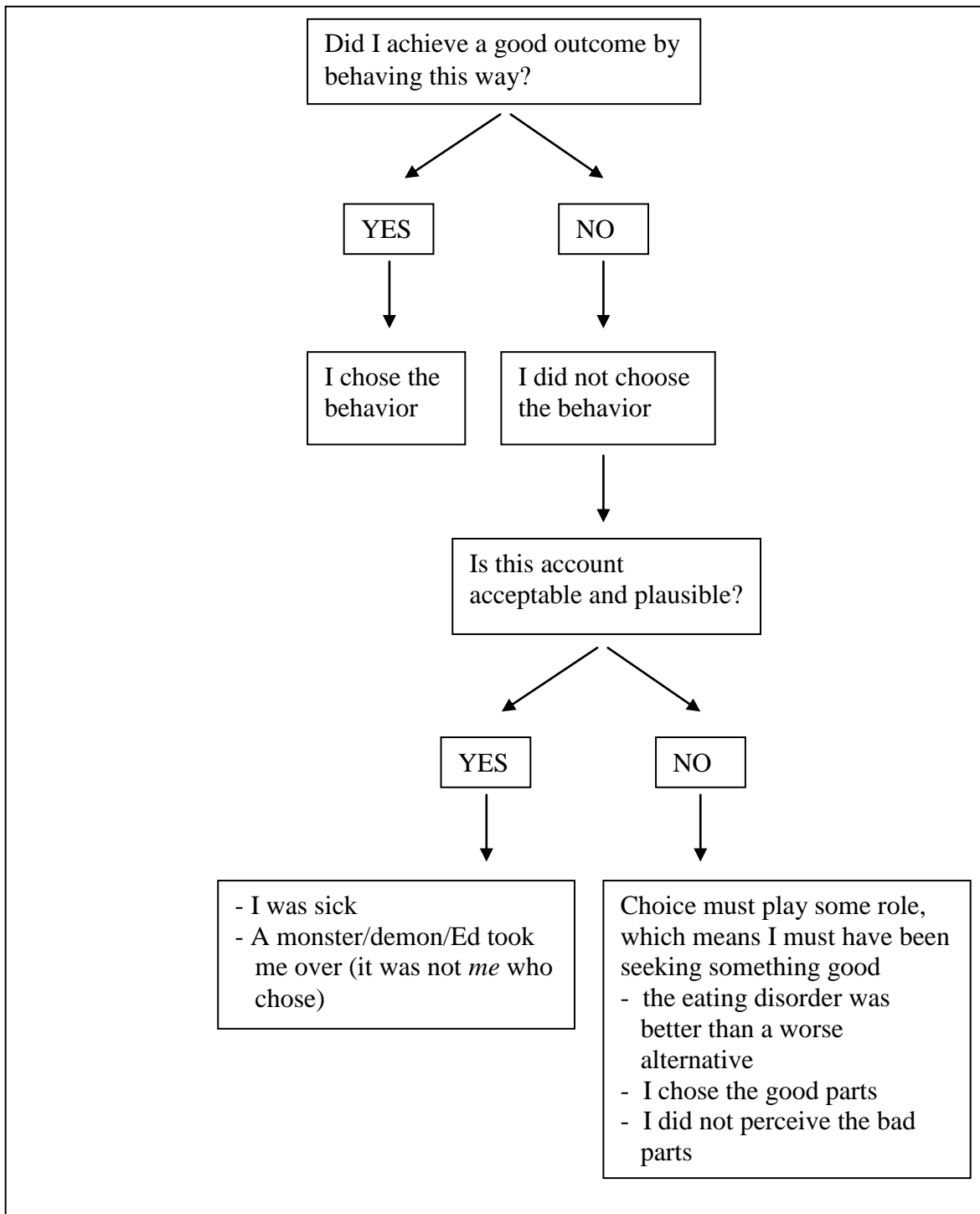


Figure 4.1 provides an organizing framework to make sense of the diversity I found in discussions of choice. This figure represents a logic of reasoning “backwards” from the

problem behavior; the deviant behavior requires some kind of explanation, which must be chosen from the available cultural options. If a medicalized account is available and likely to be honored by others, respondents may reject the idea that eating disorders are a choice. If it is not, respondents may struggle to describe how their “bad” behavior could have been a choice in some way, parsing aspects of the disorder that might have been reasonable to choose under certain circumstances.

In summary, I divided respondents’ reactions to the idea that eating disorders are a “choice” into two broad groups. Both groups mounted criticism against the idea, but one was more consistent and emphatic in its rejection, which I interpreted as a more medicalized viewpoint than that of the other group. In their reasoning about choice I identified five fine-grained distinctions for each group (see Tables 4.3 and 4.4). These included the separation between “choosing” to refrain from a particular act in the moment and “choosing” to have an eating disorder, the meaning of “choosing” when options are constrained and may seem worse than the eating disorder, and a distinction between initial behavioral choices and habits that become engrained. Figure 4.1 summarized and organized the material into a theoretical decision tree, focused around respondents’ shared understanding of choice as involving action taken toward a good outcome rather than a bad one.

5. OVERALL ENDORSEMENT OF MEDICALIZED TERMINOLOGY

As is clear from respondents’ reactions to the terms “psychological problem,” “mental illness,” “brain disease,” “physical illness,” and “choice,” a simple agree/disagree categorization is a superficial description of their thoughts. Furthermore, reasons for endorsement and non-endorsement of terms were not always substantively different. For example, with “physical illness” many people thought the eating disorder behaviors had

physical effects, but differed on whether the term “physical illness” was warranted for effects alone. Similarly, with “brain disease” some referred to the effects of eating disorder behaviors on the brain. Effects upon the brain were consistent with the idea of a “brain disease” for some but not others. Some also disagreed about whether the involvement of neurochemistry constituted a brain disease or not; for some “brain disease” connoted a “tumor” or other problem with the physical brain. Similarly, some people equated the words “psychological,” “mental” and “brain” and others drew distinctions among these. Some assented to “brain disease” because for them “brain” entailed “mind” but others contrasted the two terms. Similarly, some saw psychological problem and mental illness as synonymous, others did not. People made different decisions about whether the proposed term fit that content, even when they talked about the same concepts in their answers.

Yet, the index, described below, does capture how respondents felt about medical terminology. Even if they define and apply the terms in different ways, embracing or rejecting a term is one way of gauging respondents’ comfort with medical concepts. Despite the complexities described above, respondents had fairly clear views of whether these concepts applied to their disorders. To assess the overall degree to which they held medicalized views, and to facilitate analysis of possible correlates, I constructed an index based on respondents’ views as represented in Table 4.1.

5.1 Univariate analysis

To create an index, I summed the number of medicalized terms endorsed (see Table 4.1; 1 = medicalized view, 0 = not medicalized), namely, psychological problem, mental illness, brain disease, physical illness, and choice. (For choice, the “medicalized” answer was an emphatic or consistent message that an eating disorder is not a choice.) A total of three

respondents had not been asked about one of the five terms but the index was constructed as though they had heard the term and rejected it.¹¹

Table 4.5 Endorsement of medicalized terminology: Index and ordinal variable

Index (# terms endorsed)	Freq. (%)	Ordinal variable	Freq. (%)
0	7 (14)	Low (0-1)	17 (34)
1	10 (20)		
2	12 (24)	Medium (2-3)	22 (44)
3	10 (20)		
4	7 (14)	High (4-5)	11 (22)
5	4 (8)		
	50 (100)		50 (100)

Seven endorsed no terms, and four endorsed all. The modal number of terms endorsed was two. Of those who endorsed only *one term*, it was usually “psychological problem” (7 of 10). Of those who endorsed *two terms*, the most common combination was “psychological problem and mental illness” (5 of 12), followed by “psychological problem and physical illness” (3 of 12) and “psychological problem and brain disease” (3 of 12). Those who endorsed *three terms* more frequently combined “psychological problem, mental illness, and physical illness” (4 of 10) than the other terms. Those who endorsed *four terms* (all but one term), more often excluded “brain disease” (3 of 7) than the other terms.

I condensed the medicalization index to a three-part ordinal variable to maximize cell sizes and facilitate crosstabulation for bivariate analysis of this small sample. Those who endorsed all terms or all but one are coded as “high”, those who endorsed two or three are “medium,” and those who endorsed one or none are “low.”

¹¹ Claire was not asked about “psychological problem” and neither Gena nor Tammy was asked about “physical illness.” An alternative version that assumed missing answers to indicate acceptance was also constructed to check that statistical relationships were not dependent on this coding decision.

5.2 Bivariate analysis

Respondents varied in the extent of endorsement of medicalized terminology. What might account for such variation? I hypothesize that people with more involvement in medicalized settings will be more likely to endorse such terminology and test this using two independent variables. (1) I expect that those with more exposure to *highly structured treatment* like hospitalization will endorse more medicalized terms. This may be due to inculcation of medicalized concepts during treatment, or to the experience of a more severe disorder.¹² Likewise, people who received little or no treatment would be less likely to view

¹²Betty is a good example of someone who was **in the process of coming to see her eating disorder in medicalized terms** as a result of her debilitating experiences *and* receiving treatment from the inpatient unit. Betty was 64 years old and had been told nearly 30 years ago that she probably had AN “or borderline” but did not pursue treatment until several weeks prior to the interview; “I’ve always thought I could fix it on my own. Until I just got so sick I couldn’t take care of myself. So, that was the big difference.” She said she had not recognized it in herself because the descriptions of it always focused on very young women who feel pressured to excel and be perfect. During the interview she said she saw her eating problems as a “disease” but not as a “mental illness”. In the following excerpt, it is clear that her understanding of “disease” was recently acquired in response to events in her life *and* treatment experiences.

I: Thinking about all these different ways of looking at anorexia. How do you see it overall?

R: My problem? Or, just anorexia?

I: I guess your problem.

R: How do I see it?

I: Yeah. Like we’ve talked about psychological problem. Mental illness.

Brain disease. Physical illness. Choice.

R: Well, I think to me it’s just a way of life that I’ve chosen. And, I never looked at it as a disease until I got here. And, I didn’t look at it as any [problem]. That I wasn’t doing fine. Until last August.

Except I just thought it was the antibiotic’s fault. Or, this or that or the other. Why I didn’t feel good. I just never did accept the fact that it was because I was too thin...

Betty’s views of anorexia continued to change post- interview, and she contacted me later to amend her negative answer about “mental illness”.

R: I would like to revise my answers and say that anorexia is a mental illness.

[I: hold on, I would like to write this down. I imagine there is more to it than that. What made you change your thinking?]

R: Well this is a disease, I’m willing to accept that. But then why wouldn’t it be a mental illness to me. Because it affects every area of your life probably. I just decided I didn’t want to go on record thinking it was not a mental illness because I do. (Fieldnotes, July 1 2008, square brackets indicate paraphrasing)

Betty is actively **redefining her experience as a result of her experience in the inpatient unit, not only her experience of no longer being able to take care of herself**. Her wish to correct the “record” suggested to me that she had “learned” that “mental illness” was a better descriptor of her condition. Viewed from one perspective, this could mean that she is trying to be a good and compliant patient, accepting the treatment team’s perspective long enough to get out. But other parts of the interview suggest that her experience of feeling awful before and better now with adequate nutrition were an even more visceral form of learning and internalization of a medical viewpoint.

their behavior in medical terms because their experience was not as severe, or because of less exposure to medical concepts. A four-category variable (described in Table 2.3 in Chapter 2) was collapsed to two, representing those who had been in a highly structured program and those who never had. (2) I hypothesize that people currently in treatment will view their condition as a treatable disorder and therefore endorse more medicalized terms. Those who recovered may be less likely to endorse medicalized terms for two reasons: they may have had the eating disorder before it was medicalized or they may have revised their definition after recovering.

Both hypotheses were supported in bivariate analysis. Respondents who had participated in highly structured research programs were more likely to endorse medicalized terminology ($p=.001$) and those currently in treatment were also more likely to endorse such language ($p=.005$).¹³

Table 4.6 Endorsement of medicalized terminology by treatment experience variables

	Endorsement of medicalized terms			Total	Signif.: p-value*
	Low (0-1)	Med.(2-3)	High (4-5)		
Highly structured program					
Never	11 (44)	14 (56)	0 (0)	25 (100)	.001
One or more	6 (24)	8 (32)	11 (44)	25 (100)	
Recovery status					
Recovered	12 (48)	12 (48)	1 (4)	25 (100)	.005
In treatment	5 (20)	10 (40)	10 (40)	25 (100)	
Total	17 (34)	22 (44)	11 (22)	50 (100)	

*Fisher's exact test

The differences between groups are most apparent for high levels of endorsement of medicalized language; of the 11 in this category, all had been in a highly structured program and all but one were currently in treatment. Further analysis shows that being currently in

¹³ An alternative version of the index, re-coded as though the three respondents with missing information had heard the term and endorsed it, was used in the bivariate analysis with highly similar results. Results remained significant when the alternative version of the index was used ($p=.000$ and $p=.002$, respectively).

treatment and any experience of highly structured treatment are positively related (not shown, $p=.004$ using two-sided Fisher's exact test) and moderately correlated ($r=.44$) (Relationships of medicalized terminology endorsement with age, education, race, and diagnosis¹⁴ were also examined but none were significant.)

Experience with structured treatment in the past, or any treatment in the present, does increase the likelihood that medicalized terms will be endorsed by respondents. However, I cannot determine from these data whether the experience of treatment encourages more medicalized views, or if greater severity of illness leads to medicalized views and affects treatment-seeking (see footnote on Betty, above).

6. SUMMARY OF CHAPTER 4

In this chapter, I focused on how respondents understood eating disorders, as conveyed to me before I brought up genetics. There was consensus that eating disorders were problems generally as well as problems with society or culture, but disagreement about whether they were psychological problems, mental illnesses, brain diseases, physical illnesses, or choices. As respondents discussed whether and why a given term was appropriate to eating disorders, they revealed not only how they thought about eating disorders but also how they defined these terms. A majority said eating disorders were psychological problems and that they had physical effects, though there was disagreement about whether the term "physical illness" ought to apply to effects. Mental illness and brain disease frequently connoted psychosis for respondents, which entailed a permanent, biologically-based, severely dysfunctional state that an individual could not control alone. Half thought AN/BN was a mental illness but less than a third thought it was a brain disease.

¹⁴ To maximize cell sizes, age was divided into two groups as defined by the median age (30), education was divided into two groups (less than BA and BA or higher), and race was divided by white and non-white.

Respondents struggled with the idea of “choice,” identifying ways that eating disorders were and were not chosen. On the one hand, “choice” implied that the disorder was volitional and easily stopped, which did not ring true for most respondents. On the other hand, even those passionately against seeing eating disorders as “choices” identified aspects that could be “chosen” depending on how this was defined, and I presented the distinctions they drew in two tables. I synthesized their discussions of choice into decision rules governed by their underlying shared idea that choices are made to seek positive rather than negative outcomes. If eating disorders are negative, but conceptualization as non-choices or illnesses is not acceptable or plausible in one’s social context, behavior can be recast as a botched choice: an attempt at a positive outcome that was misguided or constrained.

Throughout my discussion of terminology for eating disorders, I divided respondents into two groups based on whether they endorsed medicalized terms (or rejected “choice”). I summarized their reactions to all five terms by creating an index of medicalized term endorsement, and found that some respondents rejected all medicalized terms, others endorsed all, and most endorsed or rejected some. Respondents currently in treatment, and those who had ever been in highly structured treatment programs were more likely to endorse medicalized terms, though the direction of causality was not clear.

Recalling the over-arching motivations of this dissertation as presented in Chapter 1, this chapter (along with Chapter 3) provides necessary background and context for respondents’ views of eating disorders before the idea of genetic causation was introduced. While all saw AN and BN as problems, some were not comfortable with phrases such as “mental illness,” and endorsement of medicalized terminology varied by treatment and recovery status. This variation suggests the possibility of further medicalization of eating

disorders, perhaps by means of genetics. In the next chapter, I explore reactions to the idea that genetics could play a role, including whether people with medicalized viewpoints are more likely to embrace this idea.

CHAPTER 5

THEORIZING GENETICS: MAKING CONCEPTUAL “ROOM” FOR NON-GENETIC INFLUENCES

As demonstrated in Chapter 3, respondents see eating disorders as developing over time with important roles for environmental influence and individual agency. Some notions of eating disorders presumed certain causes, such as media messages about feminine beauty, and specific phrases like “mental illness” connoted biological factors. In this chapter, I examine how respondents think and feel about one causal factor in particular: genetics. I find that theories of genetic contributions can be fit into the complex, developmental causal stories described in Chapter 3, particularly if respondents theorize genes to be less behaviorally specific than AN or BN, causing depression or addiction. I review their initial reactions, positive and negative, to the idea of genetic contributions, then present seven theories of genetic influence that were plausible to many respondents. Finally I reflect on how respondents combined genetic and non-genetic influences in their theories in ways that revealed a preference for complexity, indeterminacy, and recognition of the importance of the environment. I touch upon the implications of genetic theories in this chapter and explore these more fully in Chapter 6.

1. “POSITIVE” AND “NEGATIVE” ABOUT GENETICS: RESPONDENTS’ INITIAL REACTIONS

In this section I describe respondents’ initial reactions to the idea of a genetic causal factor. I introduced the topic by asking this general question: “Some say there may be genetic

causes for AN/BN. Have you heard this before?” The idea was presented in general terms to avoid specifying what the genes might be “for”.¹ I consider “initial reactions” to include all discussion of genetics before they were asked to imagine exactly how genes might influence AN or BN. Thus, initial reactions include spontaneous discussion of genetics and answers to a variety of questions and probes following the presentation of the topic.

The category “positive” includes people who are convinced that genetics play a role in eating disorders or identified positive implications; the category “negative” includes people who were skeptical about genetics or concerned about its implications. People who said they were neutral about and open to the idea were coded according to whether they talked more about negative or positive themes. Those who expressed elements of both categories were coded as one or the other based on my interpretation of their overall answer (e.g., there were more positives than negatives, or they themselves summarized their overall reaction as positive or negative).²

1.1 Overview of positive and negative reactions

Twenty-seven respondents had an overall positive reaction toward the idea of genetic contributors to AN or BN and 22 had a negative reaction (one, Natalie, was non-codable because I could not understand her answer³). Those who said it was the first time they had

¹ This information approximates the conclusions from a twin study; genes seem to matter in some way but it is not clear how.

² It may be possible to further subdivide initial reactions to capture those who were skeptical but positive (e.g., wish it were true but it’s not) and those who believed in it but wish it were not true. Respondents were not systematically asked to distinguish between their beliefs and feelings about genetics. A finer-grained analysis could take into account the kind of reasoning used (e.g., science-based), how convinced they were (e.g., open but not convinced), whether they began their answers by thinking about the potential implications (e.g., ‘that would be great!’).

³ At first Natalie (T-AN) seemed to say that she thought that doctors would probably say her sister had had AN also, but then emphasized the benefits of abstaining from food when undergoing stress, and how it is also a Christian practice she uses to clear her mind, so “abstaining from food is not such a bad thing” (so I would tentatively interpret this as leaning toward a “negative” reaction, because only doctors would see her sister as

heard of the idea tended to respond negatively (13 of 16). Most who had heard of it before brought it up themselves (28 of 33), usually with a positive spin (21 of 28).

Respondents reported hearing about genetics from a variety of sources. Some had learned in psychology courses about the possibility of genetic contributions to eating disorders, or to other conditions that seemed similar enough to serve as an analogy, such as alcoholism or depression. Several reported hearing about it from a healthcare provider, or inferring it from questions about family history in an intake interview; “I just assumed that there was some kind of connection. That there must be or they wouldn’t ask” (Margaret, T-AN). Several read about it, on the internet, or in books, or articles.

These initial reactions provide an introduction to themes developed in the rest of this chapter on the details of respondents’ genetic theories. The idea of genetics was not only a logical proposition to be evaluated on the basis of experience or other evidence. It was also something people could wish and hope for because of its positive implications, or reject for its negative implications. Chapter 6 will explore the implications of genetics in more detail.

having AN and she seems to be contesting the medicalization AN to some extent). Then she said heredity can establish a person’s “constitution” and their ability to “weather things perfectly fine” vs. not, and this could cause depression, and “the anorexia stemmed from the depression and the anxiety and the suicidal thoughts.” I understood this as a general predisposition that is funneled into AN. Then I asked what her reaction was to the idea that heredity could play a role in people developing AN, and she said “it doesn’t make sense”, and goes on to say “on the mental aspect, yeah. I think the anorexia could stem from the depression and anxiety. As I have gone through this process myself, I have learned that a lot of people have dealt with suicidal feelings. And life has become so overwhelming that they dealt with suicidal feelings. And I don’t know that that’s a – that was something that stemmed from heredity. I think it’s just life becomes so overwhelming for a lot of us that we just have difficulty dealing with life.” So it seemed like she was saying that heredity is not as important as environment, which sounds “negative”. Then I asked, “So, are you saying that it does make sense to you that a tendency to be stressed or depressed or anxious could be hereditary? But that anorexia and suicidal thoughts would not really be? Is that what you’re saying sort of?” She said “No”. Looking back, I wish I had used the word “constitution” instead of “tendency to be stressed or depressed or anxious” because I was trying to get at the distinction I thought she made. But further probing did not illuminate this; she went on to say “I’m thinking that they could both be the reason.” I did not understand this and asked, “That they could both be hereditary?” and she said “Yes” which would indicate a “positive” response. I was not sure how to understand this. Natalie appeared to have cognitive difficulties but with better probing I might have succeeded in understanding.

1.2 Positive about genetics

The 27 who embraced the idea of genetic contributions reasoned about it in a variety of ways. For about half⁴, a genetic contribution made sense based on observations of biological relatives; familial similarity was evidence for genetic similarity. Mary's sister had AN, as did Fiona's identical twin sister. Some, like Reba, observed multiple relatives with eating disorders.

Genetics. Big time. Because I think my mother's mother was anorexic... my grandmother was always rail thin. Didn't eat much. Pushed food on other people. And her death certificate did say anorexia... And my mother while she's never been anorexic has always been self-conscious about her weight... And then I have four sisters and three brothers. And I believe every one of us has some kind of an eating – maybe not a disorder. Two have been diagnosed with an eating disorder. Two other sisters. But everyone has some issue around food and weight. No one is overweight. One sister is still quite a bit under-weight. So I think genetics is a big link. And then when I have talked with cousins I have been informed that they have – that they are dealing with eating disorders. Anorexia and bulimia. So I think that's a really big factor in my family. (Reba, R-AN)

Others, like Nell, observed diverse traits in family members that she thought could be genetically connected to AN, perhaps by means of a “good little girl” gene:

It's not like anorexia runs in my family. But I think there are various – people in my family have had problems with food. And if it isn't food, then it's like they've, like, totally have used religion in a really weird way. So as far as I guess, like, the good little girl gene or whatever it is. I mean in some ways it's like there's some sort of something that a lot of people in my family have this problem. Some sort of related problem. (Nell, R-AN)

It was not necessary for a family member to have been officially diagnosed with an eating disorder to warrant a theory of genetic contributions; connections to the eating disorder were possible anyway.

For those without a family history of eating disorders, genetic contributions could also be plausible. Karen and Gillian (both T-AN) speculated that although they did not know

⁴ Amy, Ingrid, Liana, Mary, Petra, Deena, Joelle, Delia, Fiona, Sydney, Fran, Nell, and Reba

of a family history, there may have been ancestors with eating disorders who were never diagnosed. As Gillian put it, “up until the 1960’s there really wasn’t any way of recognizing it.” (More often, a lack of ancestors with known eating disorders was taken as evidence that sociocultural context was more important than genetic factors, described later in the section on negative reactions.) Some offered causal models to explain why they developed eating disorders even though others in their family did not. Ingrid and Gillian compared it to color-blindness and hemophilia respectively, suggesting a type of genetic transmission in which the condition was carried by other relatives without affecting them. Alyce (R-BN) was adopted and did not know whether genetic relatives had AN or BN, but endorsed the idea of a genetic contribution; genetics had come to seem like a good explanation for many other differences between herself and her adoptive family. A few respondents with no family history (Paula, R-BN, and Betty T-AN) concluded that genetics did not apply to them, based on an absence of family history, but that it might well apply to others.

Several respondents with positive reactions took care to distance their conception of genetics from a simplistic, direct, deterministic model of genetic causality. Genetic ideas were plausible as long as there was also room for non-genetic influences compatible with complex models of causality of the kind presented in Chapter 4.

[Y]ou don’t have a gene that says “You’re going to be an alcoholic.” Or, “You’re going to have anorexia.” I think you have a gene that says that you’re going to cope with things [by wanting] immediate gratification. Which way you go is one way or the other. Whatever you experience the most gratification [with] first is what your body kind of relies on. (Liana, T-AN)

For Liana, seeking “immediate gratification” was a method of coping to which she was genetically predisposed. Willa (R-AN), who believed that genetics played a role in her AN via her genetic “type A” perfectionism, rejected the idea of a gene for AN, which she had

introduced when answering the question on “brain disease” earlier in the interview: “I don’t think you get like the anorexia gene kind of thing [short sarcastic laugh].” Some emphasized the importance of environmental contributions and the difficulty of disentangling them from genetic contributions in a family context.

I’ve heard of studies with anorexia with, like, twins and stuff like that. In my own family there’s a high incidence of disordered eating. So to me there’s some truth in that. But like I said. I think there needs to be more research work done to separate nature from nurture. Because you grow up in a household with someone who may be biologically related to you. But you’re going to copy their behaviors. (Sydney, T-BN)

Despite observing that four generations of women in her family had eating disorders, Sydney speculated that genes, though relevant, were unlikely to be the whole story because the experience of living with relatives who have eating disorders could create an environment that encouraged eating disorders.

About half welcomed genetic explanations because they implied less individual responsibility for the disorder. Several thought that genetics could help validate the sense that it was not simply a choice, thus they could stop blaming themselves. Reading about genetics on the internet “kind of substantiated me not feeling like I caused it” (Mary, T-AN). Claire (T-AN) said, “It’s comforting because it helps to support that ‘this isn’t all my fault’ feeling.” Petra and Deena voiced similar ideas:

if it is a genetic thing, then I don’t feel like it’s as much my fault. I feel like I can at least say that I didn’t set out to cause this. That it is something that might be genetic... So it made me feel a little bit better about myself.... Not quite so responsible for being such a pain to everybody that they let me know that I am sometimes. (Petra, T-AN)

... I struggled ... with the whole thing of having to be this perfect person and have nothing wrong with me. And so, struggling with ... blaming myself. And so, I guess that kind of, almost, as silly as it might sound, kind of made me feel relieved a little bit. Like, “Well, maybe it is something in my brain that I can’t really control.” (Deena, R-AN)

It was like, “Ah. This helps explain it some.” Because, why did I do that? I didn’t know. Did I want to do it? No. But, I did it anyway. (Fran, R-BN)

I think that there might be [genetic contributions]. Just because I’ve struggled with it so long. And I mean it felt like a runaway train. And I’m a person that’s sort of gotten what I wanted. Like, I was able to do things when I really needed to. So for me I just feel like it couldn’t be that I choose to be like this. (Selena, T-BN)

These respondents welcomed genetic explanations because they made sense of their experience and removed some of their guilt. Selena added that genetics made her BN more like a “physical disease” and removed some of the stigma of mental illness.

But I think it is good to know there’s a possible link. Because I think people – with physical disease you can see physical disease. Mental disease you can’t see it. So people just assume that you’re making it up. They think “What? What is it?” They think you’re well. And so I think mental disease makes people feel very uncomfortable. It makes them feel like they can’t trust you. Or that you’re going to hurt them. (Selena, T-BN)

Just as genetics could provide evidence that it was not under the person’s control, the experience of being unable to control the disorder provided evidence that it was genetic; the two went together.

Another positive aspect of genetics was hope for new, more effective medical treatment and preventive interventions. Genetic contributions suggested that a “pill,” “drug,” or other medical treatment were possible and imminent, though even positive respondents were often skeptical about such forms of treatment. Some who found medication useful for their AN or BN, took their experience as evidence for a genetic contribution; perhaps the chemical imbalance targeted by the medication had a genetic origin.

1.3 Negative about genetics

The 22 respondents with an overall negative initial reaction to genetics as implausible or undesirable at times also articulated “positive” ideas like those above. Here I describe their

reasons for skepticism or concern, which were shared to a lesser extent with some respondents above as well. Many coded as “negative” said they were open to genetics if convincing evidence were provided, but sounded doubtful or critical of the premise.

A few considered genetics in the context of natural selection and evolution, and were doubtful that such a trait would have been preserved.

I hadn't heard it before. I guess I – like, I'd be pretty skeptical of it. Because, like, in Darwinism, like, survival of the fittest. Like, anorexic people wouldn't be as likely to survive. So they would be less likely to produce offspring that would also have that genetic defect.... I really do believe it's kind of a learned behavior. It's not something – it's not something that's natural. Like, you need food to survive. And to throw it up isn't natural or healthy. So it wouldn't be genetically favorable. (Rebecca, T-BN)

Thelma also saw no selective advantage, and doubted genetics were important.

if it's genetic, why would that still exist? Like, why wouldn't we have evolved beyond that because it's so counterproductive? Yeah. I don't know if I buy into that. (Thelma, T-BN)

Several respondents were skeptical because they did not see evidence of eating disorders running in families. Sarah said she had no known relatives with eating disorders, but felt this was inconclusive because they had also avoided exposure to environmental conditions that fostered eating disorders:

I certainly don't have any relatives who have struggled with anorexia or bulimia that I know of. But I also have all male cousins and all brothers. So I don't know. [My parents and grandparents] were all very, very poor. Like, which comes back to this whole environmental thing of abundance and wealth [as necessary conditions for an eating disorder]. And I don't think that they could have. They certainly couldn't have been bulimic. (Sarah, R-BN)

Zinnia and Vanessa both said that genetic ideas did not make sense because in a pair of identical twins, one might have BN and the other not, showing that it was not genetic.

[W]hat do you do when you find the person that has the same sort of genetic code? They have a whole different set of behaviors. I mean you can have the same DNA. But one person could be – one twin could be bulimic and the other doesn't. So how do you then reconcile the two? (Zinnia, T-BN)

I think of it as this is just something you do to get to something you want... because you can have two people. A sister. Twins. And they came from the same parents. Identical twins or whatever. And one could live in Florida in a tropical environment. And one could live up North. And one could be bulimic. And one can't. Well if they're really twins and everything, both of them should have the disease. And that's not the case. So to me it's just not plausible. (Vanessa, R-BN)

Vanessa had already spoken about how living in a tropical environment led to more time spent wearing skimpy clothing and more evaluation based on physical appearance.

Several were skeptical about genetics even though they had family members with eating disorders or similar behavior. Yvette laughed at the idea that her mother, who also had AN, could have "passed" the gene to her, saying "that's silly".

I don't think genetically my mom passed along, like, her X chromosome to make me anorexic or anything (laughing)... [I]t just doesn't make sense in my mind that it could be genetically – like, I guess, like, maybe I want to think that so if I have a girl,, like, I'm not passing [it] along. But I mean my mom has three kids. And I'm the only one who's been anorexic. So it makes sense in a sense if it's genetically passed along in my mind that – I don't know. Like, nobody – my mom's mom – nobody older than her [in the family] was ever anorexic. So it's kind of like all of a sudden she has it. Like, that doesn't – it's like there's nobody before her. So I guess, like, I don't see it being passed along genetically. (Yvette, R-AN)

For genetics to be a plausible causal factor, Yvette would need to observe several generations with AN or BN. Several respondents wondered aloud if certain relatives might have AN or BN, or tendencies in that direction, even without a known diagnosis. Irene reflected that if there were scientific evidence of a genetic contribution to BN, she would re-interpret her father's purging behavior as genetically related to her own.

For me I guess it would be a surprise because for so long I've thought that for me it wasn't genetic. But, I would probably believe it. If there was some kind of proof, I would go "Well, my dad DID vomit a lot. And, duh. Yeah. It probably was genetic." So, I would accept it. I would believe it if there was some proof. Because, there's no reason for me to disbelieve it. Especially since my father vomited. (Irene, R-AN)

Hannah (like Sydney described earlier) said that even with a family history, environmental factors were difficult to disentangle from genetic factors if one lived in the same household as genetic relatives. “[Y]ou can’t separate out the environmental and genetic influences. Because, of course it could run in families if your mom is demonstrating a pattern of something that all your kids see. Of course they’re going to eat like you do” (Hannah, R-AN) Margaret, who had several female relatives with AN, stated that because of this a part of her did indeed believe in a genetic explanation. But she was concerned about genetics overall because such explanations distract from social, particularly feminist, explanations (further detail provided in Chapter 6).

The clear importance of environmental factors mitigated the acceptability of genetic factors in a variety of ways. Several who focused on environmental factors noted that the changing prevalence of eating disorders historically cast doubt on the importance of genetics.

Well I don’t know. To me the bulimia and the anorexia stuff doesn’t have, like, a long enough history. Perhaps if it’s gone on before. But to me things only – this type of thing only really started in, like, the 1970’s. So to me that’s not enough time for, like, a couple generations to be able to determine that or not. So my initial reaction would be I’d be skeptical of that. (Melanie, R-BN)

Similarly, Thelma pointed out that “If it’s part of our gene pool, then why is it so much more of an issue now than in other times in history?” Mireya also thought that the rise in eating disorders was related to an increasingly excessive amount of available food over the last century, which she related to bingeing:

I don’t think it’s genetic. I don’t think, like, my predecessors were bulimic or could have been or had traits that allowed me to be. ... I would highly doubt that there were people in my family or in many families who had disordered eating patterns, like, to the extent that we have them today, like, a hundred years ago. I would hope not at least. I do think that, like, this is stuff in the last fifty years or eighty years ... this plethora of, like, available food everywhere all the time anywhere at whatever price. And too many options. Like, too much excess ... (Mireya, T-BN)

Carly (R-BN) thought that the cultural valorization of thinness militated against genetic theories: “it used to be that you wanted to be fatter in society... And I mean if people had those genes now, they would be – everyone would be that size.” Carly suggests that if eating disorders were genetic rather than due to cultural norms, there would be more consistency in body size across eras.

The centrality of environmental factors cast doubt on genetic explanations in a variety of other ways as well. Genes could seem irrelevant, unimportant, or incompatible with complex developmental narratives. For Irene, they seemed irrelevant; she already had a familial explanation that made sense.

I guess because I didn't come from a household that was happy. And, I was not happy and supported and healthy emotionally. And then, became bulimic. If that were the case, I could see it being genetic. But, to me it was so much caused by social things. To me it didn't seem genetic. There were just so many other things that seemed to be leading to it. (Irene, R-BN)

Irene's family history provided a satisfactory explanation; there was no reason to look to genetics for answers. For Barbara, possible genetic factors seemed unimportant in comparison to environmental factors. Even if there were a genetic factor, a “societal trigger” would be needed, which made it a societal problem.

I almost feel like in a way that could set people back in terms of dealing with anorexia. Because it's a societal problem. Like, maybe genetics makes it easier for you. It's like the same way people can be genetically pre-disposed to alcoholism. But if they are not ever around alcohol, there's no way they could become an alcoholic. And so maybe a person [is] genetically pre-disposed to anorexia. But if they were kept in a completely healthy environment. Like if they were – if someone's mother had anorexia. Then they moved them to, like, a remote village in, like, Central America. Where it's subsistence living. They wouldn't develop an eating disorder most likely. I think there need to at least be societal triggers. And I mean those societal triggers could cause it without the genetic pre-disposition. But in order for a genetic pre-disposition to manifest itself there has to be the societal triggers or, like, internal triggers or something to make it go off I think. (Barbara, R-AN)

Some respondents thought that genetic ideas were at odds with the idea that eating disorders develop over time in interaction with others in one's environment. Carol (R-AN) asked, "you're born with your genetic make-up. So, what happens later on that would then make you not want to eat? I don't know. It just seems kind of bizarre to me (laughs)." If genetics are there at birth, why wouldn't the eating disorder be as well? Zinnia shared the sense that experiences over time were more important than genes, and that talking about genes denied a fundamental reality of eating disorders, and behavior generally.

I mean who we are and what we do and what our behaviors are I believe are all sort of in reaction to or in relation to with sort of our broader social world. So to say that something is inherent or biological implies that it's *(holding her hands shoulder-width apart with palms toward each other, moving together up and down as though defining the sides of a box)* – it denies or erases that sort of set of social interactions that are – *(rotating left and right hands at the wrist as though receiving and handing things off to people beside her)* and exchanges that are taking place and informing how people see themselves or how people formulate behaviors and all that kind of stuff. (Zinnia, T-BN)

Vanessa and Wendy both expressed discomfort with the idea that something inside oneself like genes could be the source of an eating disorder. They preferred thinking of the disorder and its causes as external to themselves, and theorized that the only plausible genetic cause would be mutation from environmental toxins.

...Now maybe if somebody grows up around maybe a nuclear plant or something. And they have access to fumes that alter your – I mean just like Schistosomiasis and stuff like that. It's normally localized. Because your body is reacting to chemicals that you – or what is it? Things that are in the water that people are drinking. And it causes the elephantitis and stuff like that. Then to me that is a DNA kind of change. But it's being altered because of the environment. It's not because you were born to be that way. It's outside. So ... I would say no to that. I would not say that you're born with an innate gene that says you're going to be anorexic or bulimic. Or more prone to be... I just don't think that – I don't think that my Creator would have created people to be disposed to go through something like this. To inflict that kind of bodily harm on yourself. (Vanessa, R-BN)

These two respondents brought up external sources of genetic mutation only to theorize how genes could possibly produce an eating disorder; they did not think that such genetic explanations applied to them, nor virtually anyone else.

Some respondents thought genetic research and concepts distracted from some aspect of treatment and recovery. Gena (R-BN) downplayed their importance: “if there is, there is. If there isn’t, there isn’t. Either way it’s not a good idea... Either way eating disorders would be something that you’d have to work on” in order to recover. Several respondents rejected genetic ideas because these implied a simplistic biological intervention that was inconsistent with their idea of treatment.

And if you make it entirely a genetic – if you make it a genetic disease, they’re just going to prescribe drugs for it like they do for everything else in America. (Laughter)
And I just don’t think that’s the way to deal with it. (Barbara, R-AN)

(Several who were more positive about genetic ideas also rejected the idea of a pill.) Two thought that the search for genetic causes could obscure other more important factors. Jackie thought that genetic research might “waste resources” rather than focus on environmental factors and treatment.

Well, I mean I just don’t think it’s really important to waste resources on finding out if it is genetic or not. Because, it wouldn’t change the fact that it needs to be treated...I mean I think environmental factors are much more important. I think if people are aware of what environmental factors are triggers or causal. I mean I’m definitely as a mom going to be really, really looking out for it. And, part of that could be because I carry the gene. But, it doesn’t really matter if I do or don’t. Just because, I mean, that one thing is not going to be what causes my kids to get bulimia. (Jackie, T-BN)

Hannah used stronger language to convey a similar sentiment to Jackie’s.

I think it’s stupid. I really don’t think it matters if there’s a genetic link. Unless they’re going to be able to fix it. Which I think they could spend their money better. By finding ways to treat it effectively. And, yeah. They could go spend their money on treating dire real illnesses in infants. (Hannah, R-AN)

Thus, respondents evidently felt that an overemphasis or any emphasis on genetics could have negative consequences.

Although many respondents with an overall negative view of genetics liked the potential of genetics to reduce responsibility and blame, which was described in the section on “positive” views of genetics, several identified problematic aspects. Victoria and Thelma thought the relief of blame could be a “cop out” and provide an “excuse” for people who ought to be held responsible, even leading to fatalism about recovery.

I think that’s kind of – seems like a cop out to just be like, “Oh. I was just born this way. Like, I have an eating disorder because this is how I emerged from the womb (laughs). I was definitely going to have an eating disorder.” It just seems like – I mean I think we have, like, control over, like, our decisions. We’re not just, like, victim to our genetics... I mean if that were the case, it seems kind of like, hopeless if it was, like, a genetic thing. Like, you’re always going to have an eating disorder. (Victoria, T-BN)

I feel like a lot of times in medicine – especially with things that, like, deal with food. There’s an effort to, like, find science that, like, removes blame. Like, “Oh. People are genetically pre-disposed towards obesity.” Which is true. But at the same time, like, people – it definitely becomes an excuse for why, like, Americans are overweight. And Americans aren’t overweight because we have bad genes. Americans are overweight because we eat too much. And I don’t know. I mean I guess – like, I’m not closed – completely closed minded to it. (Thelma, T-BN)

Genetics could thus provide an excuse for people who want to evade responsibility for bad behavior, or even genuinely discourage people from believing they can overcome their eating disorders. These concerns about fatalism and irresponsibility suggest a negative aspect of genetics’ ability to reduce perceived responsibility for behavior, and will be taken up in more depth in Chapter 6.

Respondents evaluated the idea that genetics could play a role by considering the logic of the claim as well as its potential implications. With regard to the logic of the claim, respondents frequently reasoned based on whether other family members were known to

have eating disorders: the presence of relatives with eating disorders usually supported the claim; their absence was usually evidence against. Respondents were categorized as more “negative” if they interpreted the claim of genetic influence to be simplistic and deterministic, deemphasizing the important role of environmental forces; the importance of the environment was evident to respondents and suggested that any genetic influence would be complex and non-deterministic. The positive implications of genetic explanations included a reduction in perceived responsibility for the eating disorder and hope for scientific breakthroughs leading to more effective treatments, but some respondents also perceived negative aspects to these, because genetics could be a “cop out” or provide false hope that a pill would soon be available. Here I have described respondents’ initial reactions only. As the interview continued, respondents developed these themes and introduced new ones, to be explored in the next chapter.

1.4. Predicting responses to genetics: Bivariate analysis

In Chapters 3 and 4 I proposed five variables that could affect how respondents’ view genetic causal factors. These variables were based on how respondents spoke about the causes of eating disorders prior to being asked about genetics.

- (1) Theorizing a biological factor
- (2) Theorizing a disposition, such as personality type or mental illness, as a causal factor
- (3) Citing a morally charged environmental factor: abuse, trauma, or injustice
- (4) Displaying relatively more “gender consciousness” than other respondents
- (5) Degree of endorsement (high, mixed, low) of medicalized terminology.

As stated in Chapters 1 and 2, I also hypothesized that recovery status, diagnosis, and illness severity might also affect attitudes toward the idea of a genetic causal factor.

- (6) Being recovered
- (7) Diagnosed or primarily talking about BN rather than AN⁵ (because purging is thought by respondents to be a learned behavior)
- (8) History of purging⁶ (an alternative measure related to diagnosis)
- (9) Severity measured by proxy: involvement in highly structured treatment programs (because greater severity of illness would increase the likelihood of a highly structured treatment program; as noted earlier, this variable may also represent education received from highly structured treatment programs and its meaning is difficult to interpret). (An alternative version of this variable is presented in the table as “9-alt” and discussed below.)

I expected 1, 2, 9, and high values on 5 to be associated with positive reactions toward genetics and 3, 4, 6, and 7 to be associated with negative reactions. I also examined relationships between initial reaction and age, education, and race.⁷

I found that 1, 5, 6, 7, and 9 were significantly related to the respondents’ reaction to genetic involvement in eating disorder and 2, 3, 4, and 8 were not (at the $\alpha=.05$ level for a two-sided test, see Table 5.1).⁸ Age, education, and race were not significantly related to positive or negative reactions toward genetics (not shown).

⁵ This includes respondents with (1) a history of BN only, or (2) those with a history of both but a current diagnosis of BN or more focus on their BN than their AN in the interview.

⁶ This variable includes all respondents who reported BN over their lifetime, or purging (not including purging via excessive exercise). I created two versions, one based on interview data alone (n=30 with coded answers for “positive” or “negative” reaction) and the other including two additional respondents (n=32 with coded answers for “positive” or “negative” reaction) who reported vomiting, laxative use, or diuretic use on the eating disorder symptom questionnaire administered at the end of the interview (EDEQ).

⁷ To maximize cell sizes, age was divided into two groups as defined by the median age (30), education was divided into two groups (less than BA and BA or higher), and race was divided by white and non-white.

⁸ Tests of relationships for 2, 3, and 4 were not significant at the .05 level: theorizing a disposition (p=.199 one-sided test, .372 two-sided test), mentioning abuse, trauma or injustice (p=.578 one-sided test, 1.000 two-sided

Table 5.1 Initial reaction to the idea of genetics playing a role in eating disorders (N=49)
Significant relationships are bolded, $p < .05$ for two-sided test

	Initial reaction to genetics Frequency (Row %)		Total	Significance: p-value*
	Negative reaction	Positive reaction		Two-sided One-sided
(1) Biological factor				
Not mentioned	19 (63.33)	12 (38.71)	29 (100)	.003
Mentioned	3 (15.79)	15 (83.33)	18 (100)	.003
(2) Psychological disposition				
Not mentioned	10 (55.56)	8 (44.44)	18 (100)	.372
Mentioned	12 (38.71)	19 (61.29)	31 (100)	.199
(3) Morally charged factor				
Not mentioned	15 (45.45)	18 (54.55)	33 (100)	1.000
Mentioned	7 (43.75)	9 (56.25)	16 (100)	.578
(4) Gender consciousness ⁹				
Less gender cons.	10 (38.46)	16 (61.54)	26 (100)	.396
Relatively more gender cons.	12 (52.17)	11 (47.83)	23 (100)	.250
(5) Medical term endorsement				
Low (0-1 terms)	11 (64.71)	6 (35.29)	17 (100)	.022
Medium (2-3 terms)	10 (45.45)	12 (54.55)	22 (100)	
High (4-5 terms)	1 (10.00)	9 (90.00)	10 (100)	
(6) Recovered				
In treatment	7 (29.17)	17 (70.83)	24 (100)	.045
Recovered	15 (60.00)	10 (40.00)	25 (100)	.029
(7) Bulimia nervosa				
Anorexia nervosa	8 (30.77)	18 (69.23)	26 (100)	.047
Bulimia nervosa	14 (60.87)	9 (39.13)	23 (100)	.033
(8) Purging history ¹⁰				
No evid. of purging hist.	6 (31.58)	13 (68.42)	19 (100)	.155
Purging or BN	16 (53.33)	14 (46.67)	30 (100)	.115
(9) Highly structured treatment				
Never	17 (68.00)	8 (32.00)	25 (100)	.001
One or more programs	5 (20.83)	19 (79.17)	24 (100)	.001
(9-alt) Four-category treatment				
Little or no treatment	8 (80.00)	2 (20.00)	10 (100)	.002
Outpatient treatment	9 (60.00)	6 (40.00)	15 (100)	
One highly structured prog.	4 (36.36)	7 (63.64)	11 (100)	
Two or more str'd prog.	1 (7.69)	12 (92.31)	13 (100)	
Total	22 (44.90)	27 (55.10)	49 (100)	

*Fisher's exact test

test), and displaying relatively more gender consciousness (Narrower definition: $p = .444$ one-sided test, $p = .755$ two-sided test; Broader definition: $p = .250$ one-sided test, $p = .396$ two-sided test).

⁹ Broader definition used to maximize cell sizes: 23 respondents with apparently greater gender consciousness as opposed to 14. Similar results found for both versions of this gender consciousness variable.

¹⁰ Purging history based on interview, not the EDEQ. Both were unrelated to reactions to genetics.

Not surprisingly, those who had mentioned a biological causal factor (#1, often a genetic factor) prior to my bringing up genetics were more likely to have a “positive” reaction to the idea once I did. Only three of the 19 who spontaneously mentioned biological factors were coded as “negative” about genetics (Mireya, Margaret, and Melanie). Those who endorsed medicalized terminology earlier in the interview (#5) were likewise more “positive” about genetics; only one of ten who endorsed all or all but one of the terms did so was “negative” about genetics (Wendy). Those who endorsed one or none of the terms were more evenly split but tended to have a “negative” reaction. People currently in treatment (#6) were more likely to have a “positive” reaction than those who had recovered. With regard to diagnosis, I found that respondents with BN (#7, n=23) were more likely to be “negative” about genetics, but when seven respondents with a history of BN or purging were added (#8, n=30), the relationship was no longer significant.¹¹ Those who had been involved in highly structured treatment programs (#9) were also more likely to find genetic ideas plausible and/or appealing. This pattern applies across the four categories of treatment (the non-collapsed version of this variable) and suggests that “more treatment” rather than only “highly structured treatment” promotes positive reactions to genetics.¹²

¹¹ Of the seven additional respondents coded as having a history of purging but not having BN as their primary or current diagnosis, five had “positive” reactions to genetic ideas (Claire, Delia, Gillian, Karen and Petra), thereby weakening the previously observed relationship between BN and “negative” reactions. All five were current inpatients or recently discharged day program patients, that is, in highly structured treatment programs. As observed in Chapter 4, people who were treated in highly structured programs tended to endorse more medical terminology to describe eating disorders. They were also significantly more likely to feel positively about genetic explanations ($p=.001$, not shown). I examined the other significant relationships described above and all were rendered either non-significant or conditionally significant when involvement in a highly structured program was controlled for.

¹² Tests of the variable subdivided in different ways revealed that the difference may not hinge on experience of highly structured treatment. There were also significant relationships (1) when those with more than one experience in a highly structured treatment program were compared to the others and (2) when those with little or no treatment were compared to the others. These alternative indicator variables collapsed (1) the lower three categories and (2) the upper three categories, with p -values of .003 and .029 for two-sided tests, respectively.

To summarize, there was consistent evidence that medical experiences or attitudes correlated with “positive” attitudes toward genetics. Being currently in treatment, having received more treatment than others, or thinking of eating disorders in medical terms apparently make genetic explanations more plausible and appealing. Perhaps the “positive” aspects of genetic concepts only apply if the disorder has already been treated or perceived in medical terms; respondents who see it as an illness despite others’ skepticism might feel bolstered by genetic theories. Perhaps if medical concepts do not seem applicable to eating disorders, genetic ideas have fewer positive and more negative implications. Yet, some conceptions that I thought would militate against genetic conceptions did not appear to matter in these analyses (i.e., displaying heightened gender consciousness or citing morally charged environmental factors like injustice).¹³ I conclude that if genetics “go with” medical conceptions, differences of opinion about genetics may reflect differences about medical concepts of eating disorders.

2. GENES “FOR” WHAT? RESPONDENTS’ THEORIES OF GENETIC INFLUENCE

I have characterized initial responses according to the plausibility and appeal of genetic explanations for eating disorders. However, this window into how people with eating disorders initially react to my presentation of the idea of genetics sheds little light on how they thought genetics could work, and whether some kinds of genetic theories were more plausible than others. In this section I describe theories about genetics that respondents found plausible enough to entertain or endorse, with special attention to what the genes were

¹³ These were not assessed for the interview in its entirety; perhaps relationships would be different if they were. Coding for these concepts was limited to interview material that appeared before questions that might lead respondents to bring up these themes. Therefore the current coding is more likely to capture what is important to respondents with less influence from me.

“for” (e.g., what they predisposed the person to) and how these might interact with other, non-genetic influences to produce an eating disorder.

As presented in Chapter 3, respondents tended to have complex, developmental accounts of causation. If a question about genetic contributions was interpreted to mean that eating disorders were “genetic disorders” like hemophilia, most respondents decisively rejected the notion. But the same question could be interpreted to encompass a more complex model of causation to a respondent who had already thought about genetics in relation to eating disorders. Therefore, the initial positive or negative response does not reflect evaluation of identical content, as the meaning of genetics depended on their existing understandings. A respondent who assumed that my questions referred to a deterministic theory of AN and responded negatively on that basis might nevertheless endorse a more modest claim about genetic causality.

The list of theories below masks some important differences in respondents’ level of engagement with genetics and changes in their theories over the course of the interview. Most respondents (n=37) brought up genetics before I asked about it, and not only held pre-existing theories of genetic contributions to eating disorders, but could even recall how their understanding had changed since first hearing about it.¹⁴ Others were hearing about genetics for the first time from me in the interview (but always as a hypothesis under investigation rather than a new discovery), and invented theories on the spot only because the question was

¹⁴ This number included respondents who spoke about genetic influences on body size, personality, or other characteristics, as long as they connected it to eating disorders in some way. The respondent had to use variants on genetics, DNA, or inheritance. Simply mentioning a “predisposition”, “inherent personality trait”, being “born with” or having an “in-born” characteristic or a characteristic “running in the family” was not sufficient to be coded as genetic. How and when they brought it up is an interesting topic in and of itself. For example, Reba brought up genetics in answer to the very first interview question to explain what eating disorders are. Some brought up genetics in answer to question about “mental illness” and “brain disease” as the implausible extreme version of a biological oversimplification of eating disorders. Not surprisingly, genetics frequently came up in answer to the question about whether biological factors could play a role in the development of eating disorders.

asked. Respondents often came up with new theories as the interview progressed. For those who thought only in terms of simple genetic disorders, my questions about environment and personality often caused them to generate more complex concepts of genetic causation. Not surprisingly, the process of answering my questions made genetics come to seem more plausible to some respondents. For example, Yvette reflected at the end of the interview that simply by thinking about it, she had warmed up to the idea.

...the more I thought about it. “Well it does kind of make sense. Like, it is like a disease so to say. And genetics affect you. Like, you can’t really always help them.” And so it just – mental disorder sort of thing. Kind of like bipolar and stuff like that. Like, if any of that’s genetic, then it makes sense for anorexia to be genetic. And so it just – like, it just – it warmed me up to the idea... I’m definitely not like, “Oh yeah. It’s genetics.” Like, I’m just, like, not so much of a “Oh. Definitely NOT genetics.” (Yvette, R-AN)

Even though I continually referred to it as a hypothesis that some people have, rather than a new discovery, my interview questions made genetics seem more plausible, apparently. I anticipated this possibility and chose to exclude from the study people with current, untreated eating disorders because if the idea were disturbing they would not have a treatment provider with whom to talk about it.

The material below draws on initial reactions to the idea of a genetic influence, any spontaneous discussion of genetics, and responses to the following questions.

1. How *could* genes influence or help cause [E.D.]?
2. Would everyone with those genes develop [E.D.]?
3. Does everyone who has [E.D.] have those genes?
4. What makes more sense to you - genes for [E.D.] specifically, or genes for something more general that in turn influences [E.D.]?

5. One idea is that genes affect temperament or personality, which in turn can make [E.D.] more likely. What do you think of this?
6. If there is a genetic cause, do you think other causes would still matter?

These questions were crafted to balance two competing priorities: to avoid introducing content that might influence their answers and to provide some basis for comparing the plausibility of different theories even when the concept of genetic contributions was entirely new. For those who might only have thought of simple deterministic genetic causation, it was helpful to introduce the idea that non-genetic factors might make a difference, that they could propose genetic theories that were not “for” AN or BN, such as temperament (i.e., Q4 and Q5).¹⁵

Respondents offered diverse theories about what kinds of genes might matter for AN and BN (see Figure 5.1). Their theories are important because they get at what component of eating disorders might be biological as opposed to cultural or volitional. As discussed in Chapter 4, respondents reacted to “brain disease” and “mental illness” by associating biological concepts of eating disorders with the idea of permanence, a lack of dynamic development over time in interaction with environment, and a lack of individual control. When theorizing about how genes might influence eating disorders, genes that are directly “for” AN or BN also seemed to suggest too much stasis, and too little role for individuals; respondents tended to prefer a less determinative role for genes. Most respondents theorized genetic influence that went through the brain somehow, presumably because anorexia and

¹⁵ Questions #4 and 5 suggest more content than the others, and I did not ask these before asking #1-3. Question #1 is quite broad and allows for a wide range of theories involving genetics. Question #2 examines the importance of environmental or other factors in the development of the eating disorder. Question #3 addresses whether some forms of eating disorders could have nothing to do with genetics at all. Question #4 introduces the idea that genetic influence need not be “for” AN or BN directly, and provides a space for skepticism about this idea that may not have been aired earlier. Question #5 introduces one specific theory, the idea that genes for personality or temperament might indirectly make AN or BN more likely.

bulimia *nervosa* are psychiatric diagnoses (but there were interesting exceptions in which theorizing about genetics of the body went with resistance to the medicalization of eating disorders, see below).

In the following I describe their theories of genetic action. Table 5.2 (below) reports frequencies for seven theories of genetic influence on eating disorders, classified according to what genes were thought to be “for”, that is, the outcome or “phenotype” to which a genotype could give rise. Frequencies for theories of genes “for” AN and BN are limited to clear endorsements of the idea rather than simply mentioning it, because so many respondents took my questions to imply genes “for” AN or BN. Discussing genes “for” AN or BN did not imply an endorsement in the same way that spontaneously mentioning addictiveness or personality would.

Table 5.2. Respondents’ theories of the outcomes that genes could be “for” (N=50) (Frequencies and percentages do not add up to 50 and 100% because respondents offered multiple theories.)

Outcome or phenotype influenced by genes	Frequency (percentage)*
Anorexia nervosa, bulimia nervosa <i>endorsed</i> **	3 (6)**
General eating disorders or eating “issues”	17 (34)
One or more specific DSM diagnoses (or related traits)	22 (44)
Addiction, addictive personality	15 (30)
Non-specific psychopathology	28 (56)
Personality and other non-pathologized traits	33 (66)
Visible body characteristic	8 (16)

* Frequencies reflect answers to questions listed earlier, not later interview material.

**Most respondents referred to genes “for” AN or BN at some point in the interview, probably because the interview topic lent itself to such ideas. I did not code every occurrence; here I report on the minority that endorsed the idea of genes specifically for AN or BN.

Respondents offering multiple theories or theories that included multiple kinds of genetic influences were placed in multiple categories. Almost half theorized genes for a specific DSM diagnosis such as depression, and when this group was combined with those who theorized genes for addiction it comprised two thirds of respondents (n=32, not listed above).

Genes for non-pathologized traits or personality traits were also popular (endorsed by two-thirds of respondents), as were genes for non-specific psychopathology (offered by more than half and described in more detail later). Next, I describe each of the seven categories and provide examples of causal models that incorporated them. Along the way I briefly compare and contrast causal models, considering differences between the phenotypes theorized (e.g., pathologized or not, brain-based or not) and how causal models incorporate non-genetic influences (e.g., interactions). In section 3 of this chapter I will focus on respondents' preference for genes predisposing for general, broad phenotypes rather than genes specifically "for" AN or BN, and will draw comparisons between theories by focusing on the conceptual "room" they allow for non-genetic influences. Some theories were more compatible with complex causation and ambiguous agency than others.

2.1 Genes "for" anorexia or bulimia nervosa

Although Table 5.2 counts only three respondents who clearly endorsed genes "for" AN or BN, most respondents referred to genes "for" AN or BN at some point in the interview because they took my questions to imply such a theory. I did not count all such occurrences but nevertheless describe them here along with the three endorsers (Selena, Carol, and Fran) because my focus is on how people reasoned about genes "for" a given outcome, not how many did so. Judging from the low numbers who endorsed the idea, some respondents who implicitly endorsed the idea may have spoken about genes for AN or BN simply to answer to my questions, rather than reporting a belief to which they felt committed. For some the genetic contribution was plausible for other people but not for themselves, as they had no relative with an eating disorder. Some entertained the idea of genes "for" AN or BN even if in the end they preferred a different theory.

Some reasoned that because genetics seemed to matter for other problems or behaviors, they could for AN and BN too. Betty (T-AN) explained, “I get headaches like my daddy had. And, I have neck problems and back aches and stuff like my daddy had. My sister gets things that my mother had.” Amy (T-AN) reasoned, “over the years neuropsychiatry has linked depression to genes in the bodies. And, biochemistry and things like that. So, maybe anorexia isn’t all that different.” Petra argued,

...genetically you get things from your mother and things from your dad. And you come up with a child who for example enjoys music or art. And they have a parent who is really into music or art. Is that a part of the brain that is more dominant in that parent? And therefore they picked up that dominant gene? So, why not? If there is a genetic component to an eating disorder, why couldn’t that child pick up that little component in their brain? That could do it. (Petra, T-AN)

As already mentioned, some people who had a relative with the same eating disorder saw that as evidence for AN or BN as genetic.

A few who endorsed the idea of genes specifically for anorexia or bulimia nervosa reasoned from their experience of the disorders. Selena (in treatment) speculated that a gene specifically for BN made more sense than for something more general like anxiety, because pharmaceutical treatment for anxiety did not help with bulimia: “I mean they can give me Xanax. They can give me anything. I still wake up and throw up.” She notes that ancient Romans “did the same thing” and reasoned that is “definitely” a biologically-based disease “in the brain”, as opposed to a learned behavior. Yvette (recovered), though skeptical of genetic influences, said that if there were a genetic contribution for AN, it would be specific to AN, rather than contributing to AN *and* BN, because her experience of self-control in AN felt incompatible with her conception of BN and purging. (According to DSM categories, AN can include purging so her theory maps onto a very narrowly defined “subphenotype” of AN.)

How did genes ‘for’ AN or BN result in an actual eating disorder? As already touched upon in the description of people’s initial reactions to the idea of genetics, some theorized a fairly simplistic genetic transmission. Petra spoke of picking up a “dominant gene” (earlier) and compared it to the inheritance of eye color. Ingrid expanded on her analogy to color-blindness:

...you have the dominant and the recessive genes. Now, if you have two dominant genes from the parent that represent anorexia or represent mental illness or represent whatever. Versus one dominant and one recessive from the other parent. Chances are you’ve got three dominant and one recessive. That child is going to be born with that illness whatever it is. So, that’s how I think genes do play a part. (Ingrid, T-AN)

Theorizing genes specifically for AN or BN did not mean that social and volitional factors were unimportant. Some clearly thought environment and genes were important but did not specify how they might have worked together in their own cases. Fran believed that she inherited her father’s BN: “I would say my dad was the whole cause. From the genetics to his behavior towards me.” Fran, along with Selena and Yvette above, spoke at length about environmental causes, but not their interaction with genetic factors; their models of genetic and environmental causation may have been parallel co-existing models of causation. Some described an interactional model of causation, with genes being “triggered” or “activated” by some other factor, such as the environment or one’s own personality.¹⁶ Helen thought that genes might be “activated”, and “depending on the environment they may never be triggered.” Mary, whose sister had AN, said “there could potentially be one that determines whether or not you could develop anorexia. So, you’re kind of like I said pre-disposed to it. But, then you may never develop it unless other things line up. Like environmentally. Or, changes in your hormones. Or, just somehow figuring out that using

¹⁶ This resembles diathesis-stress theory, in which a constitutional vulnerability is expressed when stress triggers it. Although environmental triggers are more commonly theorized in academic literature, I include other kinds of triggers for the sake of brevity.

anorexia for like, stress or coping skills.” Petra also theorized that a genetic component for AN might “never develop” in certain environments. Amy talked about how competitive gymnasts genetically at risk for eating disorders could be helped by changing their environment, as when Olympic television coverage stopped listing heights and weights of athletes, which presumably constituted environmental triggers. “Individual” factors could also interact with genes for AN or BN such that temperament, personality, and even agentic choices affected whether a genetic predisposition would take effect. Petra mentioned personality traits, like being a “follower” or a “dependent” person, or someone striving “to always be perfect” that might make a difference in whether the genetic predisposition for AN would develop into AN (theories of genes for personality are described later). Barbara spoke of “internal triggers,” suggesting that the person’s thoughts and understandings could “trigger” AN. Carol had a complex dynamic model of causality that included agency, environment, and genes for AN. Carol integrated a gene for AN into her existing model of eating disorder as a way to express oneself and get needs met; she saw it as the way she, as a child, expressed her distress about growing up as a woman in a gender-oppressive family environment.

Genes for anorexia. Like, you and I both had the same genes for anorexia. But, my temperament and my situation was very different than yours. And, you – when something wasn’t what you needed, you were able to articulate it. You were able to get out of the situation. And, find ways to get your needs met. But, because of my temperament and situation, I wasn’t able to. So, then out of not knowing what else to do, I allowed the genes to then manifest themselves as anorexia. (Carol, R-AN)

Reframed slightly, Carol theorizes that genes for AN may have interacted with a combination trigger (i.e., passive temperament and a problem situation) to produce AN. Carol’s account also includes a passive version of action in which she “allowed” genes to manifest because there was no alternative. For Carol, a theory of genes specifically for AN could be part of a

dynamic model of causation involving agency and environment. Margaret theorized that her sister was protected from developing AN because “she had other stuff going on in her life that she cared a lot about.” This suggests some kind of beneficial interaction, in which the environment or some other “individual” factor protects against or compensates for a genetic predisposition.¹⁷

2.2 Genes “for” eating disorders generally or eating-related problems

Seventeen respondents theorized genes for eating disorders in general, or eating disorder-related food or body issues, rather than separate specific genetic contributors for AN or BN. They described genetic relatives with AN, BN, obesity, over-eating, “compulsive eating”, being “obsessed with food,” severe dieting and “yo-yo weight”, bingeing, vomiting, using laxatives, hoarding food, showing their love with food, obsessive need to control the area between neck and stomach, or some other “issue around food and weight.” This suggested a genetic tendency toward generic eating disorders or eating problems rather than a specific eating disorder.

Helen among others theorized that a genetic tendency toward eating disorders could be “manifested” in one of many specific diagnoses.

I see eating disorders as a spectrum. In the sense that you have so many different forms of the eating disorder itself. I mean different people exhibit it in so many different ways. There are people who have anorexia. There are people who have bulimia. There are people who have overeating syndrome. There are people who have eating disorder not otherwise specified. And, even with anorexia you have so many different types. I mean there are some people who have anorexia with bingeing tendencies. Or, anorexia with purging tendencies. And, you have bulimics with anorexic tendencies. And, without. So, I mean there’s so many different forms like that. (Helen, T-AN)

¹⁷ See Shanahan & Hofer 2005, for social context as compensation and its conceptual similarity to triggering, p. 67.

The variety of eating disorder behaviors was captured under the umbrella category of eating disorders, so genes might correspond to the entire category.

Environment was important in a variety of ways. It could determine whether the eating disorder developed at all, or just how “strong” it was.

...the eating disorder is strengthened by society telling you you have to be thin. Society telling you to look a certain way. By pressures around you. By you saying that you have to do this. You have to be more healthier by exercising. You have to – in order to be better as an athlete you have to be thinner. Like, that type of stuff. Society pressures. It just kind of supports the eating disorder. (Fiona, T-AN)

In Fiona’s formulation, people with the genes had a weak version of an eating disorder that was strengthened by social influences. For Victoria, environmental factors could also affect whether the eating disorder developed or not. Victoria, who was skeptical about genetics thought that they could affect one’s reaction to environmental causes.

...if I was born with the genetic make-up to be, like, prone to an eating disorder and someone else wasn’t, then I guess I’d be more likely to respond to those factors and developing an eating disorder than someone who wasn’t.... I mean just like playing on a sports team in college. And just the environment there. And my coach. And I guess reacting to it differently whether that’s your genetic make-up or not. (Victoria, T-BN)

Fran (R-BN) was already familiar with GxE interaction models for AN, which she articulated in terms of eating disorders generally:

They’ve traced it to genetics. And, the offspring of the male and female. And, the risk you’re at for it. Take two girls with pretty much the same make-up except for their parents. Stick them both in a gymnastics class. Competitive. If you’ve got one parent that’s eating disorder. And, none on this one. This one’s going to have a higher tendency to have an eating disorder. This one probably won’t. But, this one’s much higher risk.

Fran had learned about genetics of eating disorders from a University professor and presumably drew this account from a presentation she attended; she never mentioned

gymnastics in relation to her own BN and she did not apply this reasoning to specific elements of her own biography.

What directed a genetic predisposition for generic eating problems into a specific eating disorder? Mireya (T-BN) theorized that people could have different reactions to the same common genetic denominator, which in her case she thought had something to do with appetite and a tendency toward eating too much. Mireya speculated that her aunt, like herself, found it hard “to restrain herself and to control herself in front of food.” But the two dealt with the same “triggers” or “stimuli” differently; both overate, but only Mireya purged afterwards. A “trigger” such as a cake might “process similarly with both of us. Maybe that’s genetic. But the way that we both reacted towards how to deal with that situation is different. And I don’t think that’s genetic.” The physical reaction to food created a situation to which a response was required. This response could take different forms.¹⁸

Some respondents used quasi-agentic language to describe the funneling of a general predisposition into a specific eating disorder. Fiona (T-AN) spoke of her twin sister’s eating disorder as her sister’s “decision”, even though genetics played a role (and both attributions helped reduce the guilt she felt about having introduced her sister to dieting). She linked multiple eating disorders together by theorizing that all are centered on a desire to lose weight and differ only in the methods used. The desire to lose weight would apparently be supplied by genetics, but the way to accomplish it was chosen by the individual.

Because, I think it might not just be anorexia. That it could be any type of eating disorder. Like, they could end up eating too much or too little. They could take another way and exercise too much. It’s just the idea that they want to lose weight. But, I think the method is by our choice of how we do it. (Fiona, T-AN)

¹⁸ This is reminiscent of other developmental explanations for disease, in which an initial “insult” is compensated for biologically, and this compensation/adaptation becomes part of the problem.

Fiona theorized underlying genetic eating problems that are channeled in different directions depending on individual choice. She identified several potential traits to which genes could predispose a person (the various possibilities are italicized).

I think that you're pre-disposed to *certain thoughts*. Like your brain's wired to have – like my mom and her side of the family definitely have some type of eating problem. Because, *they eat too much*. And, *they can't control* what they eat. They just keep eating. They *don't know when to stop*. And, I think that there has to be something with *our appetite* that makes us that way. Something – it also could be *a coping strategy*. I don't know. It's hard to say. I just think that the possibility of developing eating disorder comes from genetics. But, it ultimately and it could be possibly our decision to whether we develop it or not. (Fiona, emphasis added).

Whether and how the individual acted upon the eating disorder involved choice and decision-making (and recalls distinctions made in Chapter 4 about not being responsible for having an eating disorder but being responsible for enacting eating disorder behavior).

Helen also used agentic language and integrated it with environmental factors. “I think environment plays a role in how a person chooses to manifest it.” I asked her how the choice was made, and she elaborated in ways that are familiar from Chapters 3 and 4, in which choice need not entail a conscious moment of decision-making but could be the end result of a series of experiences:

I don't know how the choices are made actually. I think if a person finds one thing that works for them. I think if it works for them in the moment. And, it gives them that – then, it does give them the positive feedback to return to it. And, I think that's actually very much random. Subject to chance. I mean if a person restricts. And, that does for them what they wanted it to do. Which was maybe at that day they needed to feel in control. And, they restrict by having that control. Then, that makes them feel good. So, that's more positive feedback for them restricting the next day. And, I think similarly if someone throws up. They feel this kind of cleansing, cathartic feeling. That could be positive feedback for them to do just that the next day. And, the next day. And, I think that's how it usually develops. It works for them one day. Then, they think “Well, hey. Why not? It could work for me tomorrow.” (Helen, T-AN)

The choice of a behavior to try could be based on chance, then the behaviors can “work” or not, provide positive feedback or not (such as a “cleansing, cathartic feeling”), leading to

repetition of the behaviors. Arguably, Helen's causal model could be seen as an interaction between genes and experience of behavioral reward; without this experience the genetic predisposition would not have been realized. However, Helen discussed the experience of behavioral reward to explain why one kind of behavior -- restricting -- was relied upon rather than another, thereby leading to AN rather than a different eating disorder. This suggests a different sort of interaction, in which non-genetic influences channel a predisposition in one direction or another, rather than triggering it. However one interprets it, Helen's theory was compatible with the dynamic developmental theories shared by many respondents, as described in Chapters 3 and 4.

2.3. *Genes "for" addiction and addictiveness*

Fifteen mentioned the idea that a genetic propensity toward addiction could contribute to AN or BN. I describe addiction separately from mental illness to reflect the language used by respondents (they spoke of "addiction" rather than using the DSM-IV language of "dependence"). Respondents thought of AN and BN as one kind of addiction, and theorized general genes for addiction shared by family members who had different kinds of addictions. These related addictions included alcoholism, drug "abuse" or "habit", smoking, addiction to sex, gambling, prescription pain killers, caffeine, and frequent church attendance.

Several respondents observed addictions to run in families with the eating disorder as one possible version.

... in my experience in families I've known or people I've known, their dad's an alcoholic. Their granddad's an alcoholic. Their mom's an alcoholic. And, they have a food addiction. And, it's just in the family. And, the women in the family [are] more into the food. Men [are] more into the alcohol. But, it seems so connected to me.
(Alyce, R-BN)

... I think also it's in part genetic, too. Not necessarily the disorder. But, the addictive component part of it. Because, my dad is an alcoholic. Or, was. He's a dry drunk now. But, growing up he was an alcoholic. Suspected drug abuser... And, my brother ended up with a drug habit of his own. I ended up with eating disorder. (Amy, T-AN)

I think that because – my mom's side of the family has a very addictive personality. And, their drug of choice is alcohol [or] drugs. Every one of my mom's nine siblings has had an addiction problem with drugs and alcohol. Anorexia. Or, gambling. Or, some kind of other addiction. So, I believe that I got the genes to have anorexia. (Delia, T-AN)

Respondents included a wide variety of behaviors under the umbrella of addictiveness.

The experience of AN or BN felt to some like addiction to a substance. Reba spoke of the “euphoria” and “high” she feels when restricting food:

There's something about euphoria that happens for me anyway it did and still does if I go too many hours without eating that is – it sort of keeps the vicious cycle going. The more I feel this sense of – this high. I want it to linger. And it – when I say it's a vicious cycle it's like it makes me want to keep on restricting to keep that sense of euphoria going. (Reba, R-AN)

Amy (T-AN) also spoke of a need for endorphins and adrenalin rushes in connection to exercise. Willa spoke of dopamine as being involved in multiple addictions, including her AN:

I would say just kind of addictive tendencies. I think of eating disorder a lot like that. And it plays off of dopamine in just the same way. And so I would say I mean anything from, like, smoking a cigarette to an eating disorder to alcoholism to drug addiction. And I don't think someone who has whatever addiction is just as likely to have any of the other ones. I think we all have our vulnerabilities. But it is a lot of the same thought processes. (Willa, R-AN)

The eating disorder was described as a way to respond to the addictive urges. According to Delia,

... a lot of this has to do with the need for something. The need. The urge. The temptation. Just like anybody who urges. They want to – they just want to indulge themselves with something to make them feel better. Like a hurting heart or something.” (Delia, T-AN)

Liana (T-AN) theorized that a genetic predisposition toward addiction was a predisposition toward a particular kind of coping by means of “immediate gratification” from a substance or behavior.

Non-genetic influences over time shaped which substance or behavior the person would turn to. Several respondents spoke of “choosing” AN or food as their addiction.¹⁹

How did they “choose” to enact or manifest it in one way rather than another? Delia noted that her negative experience with alcoholic relatives steered her away from alcohol, and her involvement in cross country athletics steered her toward weight loss.

I’ve seen my uncles drunk when I was younger. I’ve seen stuff happen when I was little because of their addiction. So, I always did think that that’s why I became this way. I’ve always felt that I didn’t want to be like them. But, I wanted to fill the gap in my heart and my head that I was missing. And, I chose the food. Because, it just kind of at the right time. At the puberty stage. And, at the – with the running cross country and stuff. It just hit me at the right time. And, that’s how I became addicted. (Delia, T-AN)

She used choice language reminiscent of those who spoke of genes for general eating problems; “I just chose food instead of drugs and alcohol. Or, gambling and sex.” Mary also spoke of “choosing” food instead of alcohol, prompting me to ask her how a person would choose.

I guess it would depend on what their background was. For me in particular I mean I experimented with alcohol when I was in my twenties. And, I got a DWI. And then, just got my head on straight. And, stopped drinking as much as I did. And, would just drink a little bit socially. And, I guess for me at that point in my life having a daughter and having that responsibility I guess I was just thinking it would be irresponsible of me to be drinking. But, yet it was okay to abuse my body by not eating... I kind of viewed it as I could be hurting [my daughter] if I’m drinking.

¹⁹ Amy expressed confusion about how to think through genetics. If AN is just one type of addiction, there would not be genes specifically for it but for addiction generally. On the other hand, she had heard of specific genes for obesity, which she thought of as another version of addiction. If there were specific genes for obesity, there might be for AN too.

I think if it – because anorexia is like an addictive type disease, it would make more sense to not say that there’s a general anorexic gene. And then, it’s kind of confusing. Because, it would make sense to say that there was, too. Because, there’s like obesity genes. So, maybe yeah. But, then maybe for someone who started out obese. Then, that doesn’t make sense. (Amy, T-AN)

Because, there could be the potential of me driving with her in the car. But, with anorexia I'm just hurting myself. Not really thinking about the fact that I'm still affecting all the people around me. (Mary, T-AN)

Mary's definition of choosing involved choosing against alcohol because that could harm others, particularly her daughter; AN seemed more acceptable because it only harmed herself. As discussed in Chapter 3, this is a constrained choice because she does not articulate a non-destructive option; it has to be one or the other. Liana's description of the development of AN rather than another addiction focuses on the body's growing reliance over time in one particular behavior rather than an individual choice:

...you don't have a gene that says "You're going to be an alcoholic." Or, "You're going to have anorexia." I think you have a gene that says that you're going to cope with things and want immediate gratification. Which way you go is one way or the other. Whatever you experience the most gratification first is what your body kind of relies on. (Liana, T-AN)

This account depends on experience and repetition over time, and resembles Helen's discussion of reinforcement of eating disorder behaviors through a positive feedback loop (at the end of the section on eating disorders in general). Such accounts seem compatible with the complex developmental accounts presented in Chapter 3 because they have important roles for environmental factors, individual quasi-agency, and dynamic processes over time.²⁰

Addiction-oriented genetic theories tended to involve a broad, non-specific need for some kind of gratification that was channeled into a particular direction. The general need led a person to "choose" or otherwise develop a *specific* method of gratification, which she arrived at over time in interaction with "environmental" and "individual" factors (see Chapter 3). (Some respondents held similar theories about genes for eating disorders in general, as described just above.) These non-genetic factors did not simply "trigger" or "activate" an

²⁰People may assess genetic explanations based on whether they already have an explanation that works. Delia said she had such a great childhood, so genetics make more sense. By contrast, Irene said there are so many environmental contributors to her BN that she wouldn't have thought of genetics.

underlying specific condition, rather, they shaped it into a particular form, such as alcoholism or BN. To use a genetic metaphor, a gene for a general predisposition could be compared to a pluripotent stem cell capable of developing into a variety of forms rather than one pre-determined form. The form it took depended on chance, on choice, on environmental constraints and opportunities, and a host of other non-genetic factors.

2.4 Genes “for” a specific mental illness or related trait

Nearly half of respondents (n=22) volunteered that a genetic predisposition for a mental illness other than AN or BN could make some kind of eating disorder more likely. The eating disorder was sometimes a way of coping with the negative feelings from the other mental illness, recalling Mireya’s reaction to the “stimulus” of a cake creating a problematic situation that required her to respond. This general theory of genetically-induced problems followed by non-genetically-shaped coping introduces yet another way that genes may interact with the environment to produce an outcome, but with even more diversity possible than the “trigger” model or the “specific manifestation” model. Usually respondents who theorized about genes for a specific mental illness mentioned depression, but some also talked about obsessive-compulsive disorder, panic disorder, anxiety disorder and bipolar disorder as possible contributors. (Several brought up chemical imbalances, which I categorized as non-specific psychopathology, below.) I included here adjectives such as being obsessive, compulsive, anxious, because they alluded to specific mental illnesses even though they were not discussed as disorders per se. All quotes in this section referred to genetics even when genetics are not mentioned; respondents were mentioning other psychiatric diagnoses in answer to my questions about how genes could make an eating disorder more likely.

The idea of eating disorders as a way of coping (discussed in Chapter 3) was compatible with the idea that genetically influenced mental illness created problems that instigated coping. The way of coping need not involve eating, but did for those with eating disorders.

Genes for something more general. It may be genes for like, depression. And, maybe that's how some people – I know, like, me. That's how I kind of coped with depression was eating. It was surrounding eating. Had a lot to do with that. So, yeah. I think just in general. (Deena, R-AN)

Let's say you have a gene that causes less serotonin to be produced. And so, you are in a less happy mood most of the time. And so, then you're more susceptible to depression. And then, the depression makes you more likely to cope by restricting your food intake or something. I mean I don't think it can cause the disease. I think it makes you more susceptible to it. (Hannah, R-AN)

And, like, I worry. And, I have panic attacks. And, I think a big part of the eating disorder is you worry. So, you need this comfort. I think alcoholism is a coping mechanism that people use for immediate gratification. And, I think eating disorders are the same. You want that immediate weight loss. I want to feel good right now. (Liana, T-AN)

I'm not sure that I buy it. But, there's this idea that genes affect whatever it is – the stuff that makes you happy. Dopamine or serotonin or something. And, that you might not be producing enough of that. ... You're unhappy. So, what do you do with your unhappiness? You become anorexic. As a way to – I don't know. Cope. (Lynn, R-AN)

It's like a chemical imbalance. Because the reason why you do it is because you have so much anxiety... I know that part of the reason why I do purge is because it helps me calm down. And then I'm able to do my work or to do whatever I need to do. Or I'm able to deal with something that seems harsh or whatever. But it helps me have a calming or like a soothing – it's very soothing. And that's part of it. (Selena, T-BN)

Genetically caused depression, unhappiness, anxiety or worry created a problem with which the person had to find a way to cope or soothe herself. The method of coping or self-soothing was not inscribed in the genes but arrived at by agents interacting with environmental factors over time. These respondents tied the negative emotion to a particular

psychiatric diagnosis, but others, described later, spoke about addictiveness and general psychopathology that could manifest in a variety of ways.

It was also possible to see the eating disorder as a “symptom” of another mental illness.

I feel like bulimia was for me a symptom of a larger problem. Which had a lot to do with anxiety and depression. Which I suppose – because, it wasn’t like I was just dealing with the bulimia at the time of recovery. I was dealing with what I felt like were larger issues too at hand. Which is I guess why I have that opinion. (Joelle, R-BN)

Joelle’s theory may be compatible with the idea of a coping strategy. If the eating disorder was a way of coping with the “larger problem” of anxiety and depression, it might be seen as a symptom, and terminating that coping method would confront her with the original problem anew.

Respondents often reasoned based on family members who shared a general trait but manifested it differently, recalling causal models involving genes for addiction or eating disorders generally. Speaking about her family, Isabelle describes obsessive compulsiveness, though without specifying a diagnosed disorder.

I think we’re all like, a little bit obsessive compulsive. I mean for me that manifests itself definitely in my bulimia. I mean for my dad it’s his running. For my mom it might be her commitment to work. She’s working, like, eighty hour weeks right now. So I think we’re all – we’ve all got that trait. And, like, I growing up, like, when I was in school like I always wanted to do really well for myself. Like, less for my parents and more for myself. (Isabelle, T-BN)

Or you have some kind of gene that makes you very compulsive and very, like, obsessive and obsessed with like, being perfect or something like that. So maybe there’s not, like, a specific, like, bulimia gene. But something that will lead to it. (Yolanda, T-BN)

Some respondents spoke about how environment and dynamic quasi-agentic processes helped to “focus” the general category into an eating disorder:

Like maybe you get anxiety. And then, your life influences make it so you develop a more perfectionistic sort of anxiety as opposed to a more helter skelter anxiety ... the influences from just the world in general. Or, your background. How you've grown up. Can mold a very general anxiety into a more focused sort of anxiety. Like, some people will develop anxiety that's focused towards panic disorder. Some people will develop an anxiety that's focused towards obsessive compulsive disorder. Some people develop it focused towards an eating disorder. But, I think it all stems from anxiety. I think they're all anxious disorders. (Gillian, T-AN)

Beginning with a predisposition for a wide spectrum of anxiety disorders, one's "background" or "the world in general" can "focus" or "mold" the anxiety toward eating or something else. Gena also described genes for depression that left ample "room" for environment, learning, coping and individual choices:

... I think personally I was born with a genetic pre-disposition for depression... Which caused me to not feel comfortable in my own skin. Which caused me to find different ways to cover up and run away from it. And, it just so happened that I met people and lived in a small little country bumpkin town where people didn't care if you didn't eat lunch... Like, you're not born knowing how to tie your shoes. And, you're not born knowing how to brush your teeth. I think it's kind of one of the things that you learn.... (Gena, R-BN)

Genetically predisposed to depression, Gena as agent finds ways to deal with it such as self-starvation, which nobody in her small town stops her from doing presumably because they are not aware of warning signs for eating disorders. She sees this development as a quasi-agentic learning process rather than the fulfillment of genetic programming for AN or BN, and she notes that she could have turned to cutting instead; "you can choose to mess yourself up in a multiple of ways." Thus, a predisposition to a mental disorder leads to a coping response, which may be abnormal eating behavior.

2.5 Genes "for" non-specific psychopathology

Over half of respondents (n=28) theorized genes for something broader and more general than the other conditions mentioned so far. This could be non-specific, generic psychopathology, a chemical imbalance or a common genetic factor for multiple specific

mental illnesses, addictions, and even personality types. (Genes for non-pathologized personality and temperament are discussed in the next section.) As might be expected from findings in Chapter 3, these causal models left ample room for non-genetic influences; genes did not dictate what kind of behavior or disorder would be “activated” or “manifested” over time as they interacted with other forces.

Some specified the diverse mental illnesses to which such a predisposition could give rise. Most who mentioned multiple specific disorders spoke of depression and anxiety, but OCD, PTSD, bipolar disorder, and addictions were also mentioned. (See examples in Figure 5.1.) For some, this general genetic predisposition could also manifest as a temperament or personality, suggesting that the genetic predisposition affected a continuum from normality to pathology rather than a discrete category of mental illness.

Figure 5.1. Combinations of specific conditions theorized to arise from a genetic predisposition for general psychopathology

Gillian: depression, anxiety, post-traumatic stress disorder,
Ingrid: depression, manic depression, obsessive-compulsive disorder
Liana: obsession, panic attacks, alcoholism, addictions, and perfectionism.
Mary: depression, alcoholism, any addiction
Natalie: depression, anxiety, “mental instability”, “tendency to struggle and worry and stress”
Rebecca: depression, anxiety, cutting, needing a “sense of control”
Mireya: depression, anxiety, manic depression
Alyce: depression, obsession, manic depression, addiction (all theorized to relate to serotonin)
Gena: depression, cutting and other self-harm, different “debilitating pathologies”
Olga: anxiety, depression, obsessive-compulsive disorder

To take one example, Ingrid clarified her understanding of genetic contributions to AN as giving rise to multiple conditions, potentially.

[A]norexia falls under the umbrella of two or three other mental illnesses. Depression. Manic depression. OCD. The attending illnesses as I said. They seem to work together. You can have one without the other. But, it seems to be that they all seem to interplay ... I didn't mean it as a gene per se for anorexia. I meant that the mental illness as a whole. Anorexia included. (Ingrid, T-AN)

Ingrid theorized an “umbrella” category for “mental illness as a whole” that also included depression, bipolar disorder, obsessive-compulsive disorder, and AN.

Some theorized genes that gave rise to an unspecified, clearly undesirable and pathological state. Some respondents already quoted referred to general problematic states of mind, as with a “hurting heart” or a “gap in my heart and my head” that drives an individual toward some kind of addiction. Gena, who had rooted her own eating disorder in depression, noted that “there has to be something in you that feels not complete.” Nell, who had spoken about AN as an addiction, offered a similar general predisposition: “I don’t think you can have – you have a gene for blue eyes. But I don’t think you have a gene for anorexia. I just think you have a gene for ‘you’re going to have some problems’.” A predisposition to “problems” allowed a large amount of room for non-genetic forces to shape an outcome.

The general state of experiencing pain, incompleteness, and problems was attributed to a chemical imbalance by some people. I coded statements about chemical imbalance and neurotransmitters as examples of general psychopathology because a chemical *imbalance* is by definition abnormal but is not specific to a mental illness. Brain chemistry was a logical entity that could link genetics to eating disorders and other conditions for several respondents.

I think that it gives people certain structures in their mind. Certain ways that the chemicals fire. Or, neurons fire. Or, chemicals just sort of interact. Different levels of serotonin and dopamine. And, I think that that can actually probably set somebody up for a lot of different mental illnesses... I think that if there is a genetic component related to eating disorders, it’s probably going to be very similar to the genetic components for most other mental illnesses. If not identical to. Beyond psychotic disorders. (Gillian, T-AN)

Olga explained how a chemical imbalance could produce a general lack of wellbeing, with which a person might need to cope using an eating disorder. She spoke of brain chemistry producing a “generalized ‘something’s wrong’” feeling.

[S]o if there’s not serotonin in your brain, then you start to feel like you’re – something’s wrong. I mean that’s the – serotonin is the feel good, everything’s okay drug. So if you’re not getting enough of that in there, then something’s wrong. And that’s where I see the anxiety coming in. And I don’t know exactly what triggers the anorexia. But I know once you’re in it there’s a lot of anxiety about gaining weight. Getting fat. So you have to control something. Just like with obsessive compulsive. ... [I] think that’s where the genetics comes in. Just a generalized 'something’s wrong'. And then you pick something to make you feel better about it. (Olga, R-AN)

Without enough of the naturally occurring chemicals that cue a person that “everything’s okay”, one must “pick” something in an attempt to feel better. This was reminiscent of a theory already mentioned in which a predisposition to eating problems or depression could create a problem with which the person needed to find a way of coping. The specific style of coping was developed through experience over time.

If a genetic predisposition presented the person with a problem, non-genetic factors shaped the content of the problem and how to solve it, in ways similar to those already described above. In addition, some spoke of a genetic vulnerability to environmental stressors and triggers. According to Barbara, environment could interact with a predisposition to general psychopathology was by altering one’s sensitivity to environmental “triggers”, according to Barbara.

I mean I learned a little bit in, like, a basic psychology class about how like, chemicals in the brain regulate like, emotional responses and things like that. And so I mean if there’s something in your genes that controls, like, mental processes. If, like, something is off about those genes. Like, I could see how you could become pre-disposed to that. It makes you more vulnerable to those societal triggers hitting home. (Barbara, R-AN)

“Societal triggers” are more likely to hit “home” for someone with a genetic chemical imbalance. Gena articulated a similar idea; she was predisposed to have some kind of “psychological crap” which made her more vulnerable to stressors:

Like genetically the way that I’m built, I’m kind of pre-disposed to having some psychological crap going on. Like, my family history. And, with, like, small life stressors it doesn’t take much for that to happen for me. (Gena, R-BN)

Respondents thus found a variety of ways to incorporate genetics into their vision of complex causality and ambiguous agency. Put very generally, respondents imagined genes to have a direct effect on the disorder, with other forces activating or shaping the outcome, or an indirect effect, in which genes led to problems that required coping.

2.6 Genes “for” non-pathologized traits

Well over half of respondents (n=33) also theorized genetic predisposition to personality types, temperaments and other traits that were not necessarily connected to mental illness. I separate them from general psychopathology because the genes are “for” something that is not inherently pathological, such as a need to feel in control, an achievement orientation, shyness, or a high level of sensitivity. I included traits like low self-esteem even though they are usually seen as negative because they were not diagnosable mental illnesses and were often mentioned as a type of personality and temperament.

Because one of my standard questions addressed the question of genes for temperament and personality, I have more information about whether and how respondents found this idea plausible. Twenty-one people brought up the genetics of personality, temperament, or other traits without my asking about it, and more approved of the idea when I did (some also rejected it). In this section I only include discussion of personality when it was connected to

genetics; many respondents discussed personality and similar traits in non-genetic terms as well.

Some respondents reasoned that their personality, temperament or other traits might be genetic based on comparisons with their family members. As with other genetic predispositions above, some respondents identified family members who shared similar traits, and drew the conclusion that the similarities reflected a genetic inheritance. Interestingly, others saw evidence for genetic contributions in *differences* between themselves and their siblings.

I mean I think genes factor in your personality type. Because, like environmentally with the same parents and stuff you still have different personalities. Like, I have a different personality from both my sisters. And, we were made from the same two people. And, grew up in the same house. So, that's what leads me to think that genes have to be involved. (Mary, T-AN)

I mean because again you look at my sister. She was raised in the exact same environment. And is totally different. So it's got to be something genetic. (Willa, R-AN)

By this reasoning, if two people grow up in virtually the same environment, their differences must be attributed to genetics. (Alyce, who was adopted, had used similar reasoning about the differences between herself and her adoptive family; differences were attributed to genetics. See "Positive about genetics" section.)

I identified three loosely-related groups of traits: those related to control, to low self-assertion, and to greater sensitivity to the environment. I grouped perfectionism with control because respondents frequently spoke about perfectionism in terms of control over body weight or shape. Most of what respondents said about personality and temperament could be fit into these categories, but some also mentioned impulsivity, recklessness, anger, and needing an independent identity as genetically predisposing themselves toward eating disorders.

I defined *control* very broadly, to include respondents who spoke about rigidity, perfectionism, and being resistant to change. Because some respondents connected these themes to “type A” personality, achievement-orientation, high standards, ambition, and competitiveness I include these here too.

Like, it’s more about the sense of control than anything else. And like that that can be the stem. If you don’t feel like you have control over other things in your life, you can control your eating or your cutting. Or like that gives you a sense of control. So I guess things similar to that maybe. (Rebecca, T-BN)

Some theorized a “control gene” (Amy) or a “gene that, like, helps encode for, like, control issues. Like, a need for control or something in your personality”. (Hannah) Some respondents linked control-related traits to AN specifically.

Like, because a lot of the people who have eating disorders have similar personality traits. That’s what people in (the day program) say... Like rigidity. And, a lot of fear. And, desire to control your life. And, control what happens to you. And, like, perfectionism. And, I think that’s more for anorexia... although bulimics I think sometimes do, too. (Claire, T-AN)

Well, I think I had, like, a typical personality for somebody with anorexia nervosa. The type A perfectionism. Like, ‘always on the go’ kind of thing. (Liana, T-AN)

Some respondents said it was hard to change their control-oriented personalities, even though they wanted to at times.

I’ve always been a person who’s very resistant to change. Very resistant to change. And, I don’t want to be that way. It’s just the way I am. So, I definitely believe that there’s something genetic about that. (Beth, R-AN)

I’m definitely a perfectionist and type A personality. And I think a lot of that has to do with your genetics. I’ve tried to be a type B. I’ve tried. I used to teach stress management. And I used to be standing there thinking “Oh. I could never do this.” So I’ve tried really hard. But I just have that personality. I can modulate it. I can make – I’ve gotten a lot better over time letting things go. But I think that’s just something that you’re born with. I mean my mom said I was always like that. (Olga, R-AN)

The trait was resistant to change, something you were born with, not something you could control, and therefore compatible with genetic ideas. It could be “modulated” or managed, however.

Other respondents spoke about genetic traits that I gathered together as *low self-assertion*. I included references to passivity, dependence, people-pleasing, taking the role of “victim”, not wanting to “take responsibility”, being shy, quiet, reserved, “low key”, “defenseless”, or “introverted”, as well as traits related to low self-esteem, low self-worth, feeling insecure, and internalizing criticism. Respondents brought these up in response to the question about genetics of personality and elsewhere.

If you have a passive aggressive personality. Or, if you have a personality that is self-defeating. You have a tendency to hide behind physical symptoms. That I see. Such as being quiet and reserved. And, you let yourself be victimized so to speak. Or, “Why me? Why me?” And, you don’t fend for yourself. You’re more defenseless. And so, you take on the little role of the victim. And, you won’t eat. Or, you do that. Versus say other people who are loud and raucous and so forth. The meat-eater types. (Ingrid, T-AN)

For Ingrid, not eating went along with not asserting oneself, in contrast to “meat-eater types” who are “loud and raucous” and presumably more self-assertive. Thelma offered that a “nervous” or “insecure” person would be more likely to care about his or her physical appearance than someone who didn’t care, making an eating disorder more likely.

...like if you were, like, a nervous person or, like, an insecure person. ... I mean because [by contrast] you definitely, like, meet people who just don’t care. Like, it’s just not something that’s important to them. And that’s partially environmental. And it’s partially just, like, who they are. Like, part of their genetic make-up that makes their brain, like, operate in that certain way. And so you have that aspect. Whether or not it’s important to someone. Whether or not they care at all. (Thelma, T-BN)

Non-self-assertive traits accompanied control-oriented traits for some respondents. Karen (T-AN) identified these traits as potentially genetic and making an eating disorder more

likely: “People who are perfectionists. People pleasers would be kind of the same way. People who don’t like to take on responsibility. Who are more dependent upon others.”

A third broad family of traits involved *sensitivity* to stressors and other environmental influences. Nell said that some people, like herself, are more “sensitive to stuff” and therefore need to find ways to soothe themselves. She also linked this to chemical imbalance and addictiveness, but by talking about sensitivity she also gave the trait a more neutral, less inherently pathological meaning.

I mean the only thing I can really think of is the idea that your brain doesn’t produce enough serotonin or something like that. And that would be... like, a way to – your body soothes itself. And if you didn’t have – if I didn’t have that. And probably my kid wouldn’t have that either. And would therefore rely on some other something to get that soothing that most people get naturally. So that might be passed down from generation to generation. ... And so you’re so sensitive to stuff that other people just wouldn’t be all that sensitive to. And so then you’ve got to figure some way out to buffer yourself. And so then an eating disorder or something else could be a way.
(Nell, R-AN)

Nell’s causal model resembles indirect models of genes causing problems that elicit coping, discussed earlier. Natalie also drew a contrast between herself and people who are less sensitive to the environment, those who “weather things perfectly fine”:

It just seems some of us are just extremely more sensitive than other people. I think this deep sensitivity or being over-conscious – because, it’s just like you’ll find that these people are trying to be over-achievers with their lives. In some ways trying to do the right things with their lives. And it’s like – And this is almost impossible sometimes. It’s hard to make good sound decisions. And I think we’re prone to worry ourselves so about it. To the point that it overwhelms us. And then, things begin to work on you mentally. And I think sometimes we’re prone to just be real, real sensitive. (Natalie, T-AN)

Natalie spoke of sensitivity in connection to depression and worry, but also noted that it can lead to people “trying to do the right things” and achieving, making this genetic trait not as necessarily negative as a genetic predisposition for a mental illness. Referring to the death of a mentor, Selena (T-BN) said “There’s something in my DNA that caused me to not be able

to get through things like that...”; her sensitivity to “really bad stress” impels her to turn toward bingeing and purging, because “it helps me cope somehow”. Being particularly sensitive was contrasted to being “resilient”, “hardy”, able to “resist”, not succumbing to “pressure” from external forces.

...some people are resilient. And, I think that would be a genetic type thing. They can – bad stuff can just kind of bounce off them. Or, they see it. And, it doesn’t really – they just intrinsically know better. And, somebody who has this genetic risk factor would not necessarily know better. (Beth, R-AN)

Well, I think it could make you more susceptible to environmental influences. Or, less able to cope with stress or something. ... Maybe just a gene for, like, decreased ability to cope with stressful life situations or something. What’s that word? Not hardiness or something. Like, there’s, like, some trait that people have that makes them more able to, like, resist. (Hannah, R-AN)

Maybe someone that has a genetic susceptibility to, like, pressure from, like, our culture and the media. And, maybe, like, other people, like, their family or friends. And then, having, like, a low self-esteem. Those kind of could all fit together and equal bulimia. Or, anorexia. (Eva, T-BN)

The environmental influence to which the person was sensitive could be general like stress, or content-specific, as with pressure from “our culture and the media” toward thinness. Stress here is not necessarily a trigger that activates an underlying predisposition. Rather, if a person is genetically “sensitive” to her environment, she could simply be more malleable and responsive to her environment; the sensitivity need not become stress nor result in a pathological phenotype. Usually, however, respondents spoke in terms of problems caused by such sensitivity.

All three groups of non-pathologized traits were compatible with the complex compound causality and ambiguous agency described in Chapter 3, but they differed from the above theories in one important way: they did not presume pathology. In theories about control, low self-assertion, and sensitivity, respondents imagined genes that were not by

definition problematic or pathological. As with predisposition to addiction and depression and other conditions, environmental influence could channel a general genetic predisposition toward eating disorder behavior rather than something else. For example, Willa thought that her “intense” “type A personality” might have turned to obsessive studying or even cheating had she been raised differently, and currently she continues to have the same personality without having either problem (Willa). Arguably, what is important for theories about personality and the like is that they are not inherently or by definition pathological; one can have the personality without having problems. Genes for a personality trait do not assume pathology in the way that genes for a psychiatric diagnosis do and may be easier to reconcile with recovery experiences because the genetic predisposition may theoretically be redirected toward non-pathological ends. Willa explicitly preferred thinking of genes for personality rather than genes for mental illness and incorporated the ongoing influence of those genes even while recovered.

I very much accepted the fact that that is the kind of person I am. I’m very much a perfectionist. And very much ... achievement oriented. So I guess for me I just know I need to keep it in check and not have my identity come from that. So I think just because I’ve owned it that doesn’t bother me. (Willa, R-AN)

Willa’s non-medicalized view of AN is expressed in her theory of genetics for personality (Willa endorsed only one of the medical terms in the index, Chapter 4). One of the objections to medicalization is that social problems come to be located in the individual body rather than in society (see Chapter 1, e.g. Conrad and Schneider 1992, Thompson 1994). If genes are “for” a pre-formed entity that is diseased, there may be less room for the idea that complex developmental processes, environmental circumstances, and individual agency led to the disordered outcome; the person may seem fundamentally diseased or flawed. By

contrast, genes for personality or sensitivity may be more compatible with the idea that one is not fundamentally flawed or diseased.²¹

2.6 Genes “for” visible physical features

A minority of respondents (n=8) theorized that genes for visible physical features might predispose people to eating disorders. All theories described so far concern individual dispositions, pathologized and not. As discussed in Chapter 3, enduring dispositions shape a person’s reaction to diverse situations. In the material presented so far it is apparent that respondents find genetic influence plausible for many such dispositions. Although theories vary by how inherently pathologized the dispositions are (e.g., mental illness vs. personality), all involved individual psychological dispositions. By contrast, seven respondents theorized something quite different: they speculated that genes might matter for eating disorders by affecting some visible aspect of the body: body size (six respondents) and female body (one respondent). Some held these theories in conjunction with theories about dispositions. For one respondent (Margaret), genes for the XX chromosome was a way to reject medicalized definitions of eating disorders in favor of feminist social understandings.

Those who mentioned genes for body size reasoned that having a large or curvy body would be interpreted as a problem for which dieting or purging is a solution, thus setting the stage for an eating disorder. Isabelle recalled, “One thing that contributed I think somewhat indirectly to my bulimia was my early experiences like in adolescence just feeling too big and feeling like I was so much larger than the other kids. And that’s I think a very obvious genetic factor.” Melanie also stated that “people who are more prone to being larger or just naturally having a larger body mass. ... Maybe they’re more prone to an eating disorder like

²¹ Unless those traits are disparaged; Joelle described herself as a “recovering introvert” which suggests quasi--pathologization of introversion.

bulimia. So then your genes would be somewhat responsible for that.” With a large body, one might be “more likely to feel like that was what you needed to do is have weird diet things going on” (Joelle). With a naturally thin body, one was less likely to attempt weight loss using methods that lead to eating disorders; such people are “born with the genes to make them skinny all the time. And they can eat whatever they want” (Carly, R-BN). Someone whose “genes were such that she had slower metabolism and could put weight on at a younger age is much more likely to struggle with an eating disorder just because of that natural – suddenly you realize ‘Oh my gosh. Food affects my body shape.’ ” Tammy (R-AN) spoke of having “fluffy genes” for overweight that, in conjunction with over-eating, led to obesity, which was central in her narrative of developing AN. Several who offered these theories nevertheless expressed uncertainty because they had met people with eating disorders who had never been overweight. The underlying model for a theory of genetic predisposition for large body size is quite indirect. For example, in a context with plenty of food available, a genetic predisposition for being large is more likely to be realized. If one is in a social context where having a large body is interpreted negatively by others, one is more likely to monitor one’s body and seek ways of losing weight that work in one’s social context.

Most who spoke of genes for body type also mentioned genes for mental illness, personality, and other psychological dispositions. Thelma theorized an interaction between genes for body size and genes for personality. She had theorized genes for being a nervous or insecure person for whom body image is important, and suggested that such a person who also tended to gain weight (as with hypothyroidism) would be more likely to develop an

eating disorder compared to the same kind of person who was naturally very thin (as with hyperthyroidism).

Like, body image is important to you [because of a genetic predisposition toward nervousness or insecurity]. But you have a hyperthyroid. Then like, it's something you don't have to think about. It would never be an issue. But if you have, like, hypothyroid. And it was really important to you. And you could eat just as much as your friend sitting next to you... Then I feel like it would be, like, conducive. Because you'd feel, like, cheated. Like, that's not fair. Like, I should be allowed to eat just as much. But I also don't want to be fat. (Thelma, T-BN)

Purging one's food would make sense as a response to these two predispositions, according to Thelma.

Some respondents pointed out that genes for a large physical size were only a problem in environments that valorized thinness. Joelle specified that a larger body type could “pre-dispose [people] *in this society* to behaving in certain ways” (emphasis added). This suggests a gene-environment interaction, but as noted above the causal pathway would be quite indirect. Carly thought of eating disorders as closely related to goals for physical appearance, which she defined in societal terms. Indeed, Carly could not imagine how genes for anything other than physical size could contribute to an eating disorder. I asked her specifically about the idea of genes for something psychological, and she responded skeptically.

I just don't see how it would relate to the psychological factor. Because I mean it used to be that you wanted to be fatter in society.... [T]hat was a way long time ago. But that's what people wanted to be like. And I mean if people had those genes now, they would be – everyone would be that size. ... I mean I just can't see how like my mom – like, her – if her brain is telling her to make herself throw up because she's not skinny enough. Like, I can't see how that would transfer over somehow. Like, something in her brain that is making her think differently of herself. I just think it's something that society has an influence on it. That society determines how you're supposed to look. (Carly, R-BN)

Carly was skeptical that there could be genes related to the attempt to achieve a particular physical appearance. If there were, she reasoned, those genes would be consistent across historical eras and there would not be fluctuations in ideal body size at different times. Although recovered, she is the only respondent who was neither treated for nor discussed her BN with a healthcare provider afterwards (and although she endorsed the terms “psychological problem” and “mental illness” to describe eating disorders, it sounded like she had only just begun to think that way).

One respondent, Margaret, asserted that genetic influence works solely through gender socialization based on *sex chromosomes*: having female sex chromosomes (XX) puts one at risk for an eating disorder in a U.S. context because of gender socialization. This was a self-conscious and explicit rejection of genetic theorizing, which she viewed as a challenge to the centrality of gender inequality in the development of eating disorders. Although before and after this section of the interview she said she thought genetics were relevant for her own AN, she balked when asked to imagine how genes might have influenced it.

...So trying to think of how genes would play out. That’s where I just stand on the other side and say “No. It’s a ridiculous explanation. It can’t play out. It won’t. The only genetic component is being female. And it’s the XX chromosome. That’s what the cause is.” So that’s how it plays out. You’re born a female. And then you get raised here.... again I see it as social. How women are taught to be and see themselves and identify and become the kind of people that shrink (laughing) and don’t weigh a lot. (Margaret, R-AN)

Her theory of eating disorders is based on the idea that eating disorders are a “coping mechanism” and “a way of dealing with something in your life”, and that women are more likely to see it as an option or “model” for themselves; whereas for men “[i]t’s not a coping mechanism that they’re trained to see as something that they could just go do.” Margaret’s genetic “theory” was offered in playful resistance to the idea of genetic influence on eating

disorders and her tone of voice and laughter suggested that this was not to be recorded in earnest as a genetic causal model but as a rejection of genetic causal models. Considering it for the moment as a genetic causal model, it resembles theories of indirect effects of genes for body size because much intervenes between genotype and phenotype. There is a kind of gene-environment interaction because genes that determine femaleness interact with cultural definitions of femaleness, but Margaret is not suggesting that all U.S. women have eating disorders. Rather, this interaction is the foundation for other social processes that lead toward eating disorders, suggesting indirect causation. Margaret alternated this resistance to genetic explanations with an apparent belief in them and an appreciation for how they would give women relief from the blame and stigma of eating disorders. (Margaret's interview will be discussed with more detail in Chapter 6 on implications.)

For respondents who theorized genetic influence in terms of body size or sex chromosomes, these characteristics created a situation that had to be dealt with through dieting or purging, which made eating disorders more likely. Several stated that the situation was only defined as a problem in societies that valorized thinness. By not theorizing a psychological genetic predisposition, Carly and Margaret in particular framed the issue as fundamentally societal. Arguably they preserved a space for an agent whose reaction is not defined or influenced by genetics. This agent is confronted with a genetically large body, whose value is determined by its context. Although her body has been influenced by genetics, her responses have not. A person whose reactions are not deemed to be genetically influenced may seem more free and agentic. By comparison with a genetically-influenced agent, her reactions may seem less like an outgrowth of pathology or idiosyncratic personality trait, and more like a reaction to social conditions. Furthermore, her reactions

have the potential to be justified, meaning they were appropriate or understandable given the situation, rather than excused or otherwise explained by what she brought to the situation. For Margaret in any case, theorizing genes for female sex chromosomes (as a joke or not) constituted a resistance to locating the problem in the person, which genetics threatened to do. By locating the problem in the society's definition of acceptable body size, Margaret's agency, and the warrant for social critique, are augmented. Genes for body size and sex may offer the most "room" for societal and choice-based explanations for respondents. Indeed, theorizing genetic influence on AN via sex chromosomes was for Margaret a way to resist genetic theories and place social construction of gender at the center of eating disorder genesis. Body size and sex were theorized by respondents as predisposing only in particular contexts; in a society that values thinness, the larger body is a problem. In a different context, the same genes would not have propelled someone toward an eating disorder.

In summary, respondents offered diverse theories for how genetics might influence the development of an eating disorder. Most theories could be fit into the seven categories above. The categories varied in specificity (i.e., genes for AN or BN vs. genes for something more general), pathology (i.e., genes for psychiatric diagnoses vs. genes for non-pathologized traits), and body "location" (i.e., genes that referred to brain-based operations vs. visible physical features). I have described all of them as types of genetic theories, whether the genes are "for" depression, personality or female sex. Some readers may argue that only genes specifically for AN or BN ought to count as genetic theories of AN or BN. If this were the case, only 6% of respondents (3 out of 49, discussed in the next section) endorsed such genetic theories. However, researchers who study the genetics of eating disorders do not limit their search to the diagnostic category, and may theorize genetic influence on sub-

categories of a diagnosis (e.g., AN with restricting only) or theorize genetic influence by defining AN or BN as part of a spectrum that includes other diagnoses or personality characteristics such as those mentioned above. Most respondents' views are compatible with those of genetic researchers who target personality, addiction, obsessiveness, anxiety, depression, and other conditions or traits without reference to AN or BN. In the following section I examine whether respondents viewed a specific or general genetic influence as most plausible.

3. NOT BY GENES ALONE: A PREFERENCE FOR COMPLEXITY AND INDETERMINACY IN GENETIC CAUSATION

I have described the content of respondents' theories, organizing interview material according to what genes are theorized to be "for." For each category I provided examples of how genetic and non-genetic contributors to eating disorders were combined. There were many theories of an indirect, rather than a direct genetic cause for eating disorders, often powerfully shaped by diverse non-genetic factors. Many respondents held multiple theories simultaneously. In order to assess which theories they preferred, I now present answers to a question that permitted comparison across respondents, namely whether they thought a specific or more general gene was most plausible. I also provide a rank ordering of theories according to the role afforded to non-genetic influences (Figure 5.2).

3.1. Respondents preferred general categories and indirect causation

Most respondents speculated that if genes mattered, they would be genes "for" something more general than AN or BN. Respondents used multiple theories over the course of the interview but when asked, "what makes more sense to you, genes for [AN/BN] or genes for something more general that influences [AN/BN]", 38 of 49 said that something general was more plausible. An additional three rejected the premise of the question (Carly,

Margaret, and Vanessa), leaving only eight who responded that it could be genes for AN/BN or *both* specific and general genetic influences. Of these eight, only three clearly preferred and believed in the idea of genes for AN/BN (Carol, Fran, and Selena). Because there was so little variability in their endorsement of a general, rather than specific, predisposition, I did not statistically analyze relationships to other variables (such as those presented in Table 5.1 on initial reactions to genetic ideas).

I turn now to respondents' more focused reasoning about why genes for something more general than AN or BN were more plausible. (For reasoning about why genes specifically for AN or BN made sense to some people, see the prior section where theories of genes "for" AN and BN are described.) Reasons for skepticism about genes for AN and BN have been alluded to earlier in the section on "negative reactions" to genetics. Arguments here echo and build upon themes presented there, particularly that genes "for" AN or BN did not adequately take into account powerful environmental forces.

For many respondents, genes for AN or BN were antithetical to their conception of eating disorders as developing over time in interaction with other influences and with some degree of agency. Genes for addiction, low self-esteem, depression, and other less specific conditions provided more conceptual room for the complex causality and ambiguous agency described in Chapter 3. Eva responded to the question saying, "Definitely for something more general. I don't think there's like, a bulimic gene." A "bulimic gene" implied too large a role for biology, and not enough for agency (behavior, choice, psychological issues) and environmental influences. Eva continued:

I don't think – if there was a bulimic gene, then I think you'd be able to like – I don't know. Because, I guess it starts out it's not, like, biological. It's more of, like, a behavior. And, I think for a lot of people they kind of like – there's, like, a reason why. And, it's usually, like, environmental. It's not something that, like, their body is

making them do. I guess it's like a conscious choice in the beginning. So, I don't think that's genetics...it's not entirely like – that it's not, like, biological. Like, I think of it more as, like, a psychological issue. And, I just think it's a lot more complicated than just having a gene and that being, like, the sole cause of it. Because, I don't think – I mean if it's, like, a susceptibility. And then, not – it's like there's another part of it that has to go into the equation to equal that someone has bulimia. You can't say that it's all genetics. (Eva, T-BN)

Eva had theorized that genes related to personality and sensitivity to the environment could have influenced her eating disorder, but genes specifically for BN sounded like a claim that it was “all genetics” with genes as the “sole cause of it”. Liana similarly understood genes “for” AN or BN to be highly deterministic.

R: A gene for anorexia? No.

I: And, tell me why. Just explain it on the tape.

R: Because, there's so much more that goes into it. I don't think you can look – when I think of a gene – which I could be wrong. And, I hated chemistry anyway.

(Laughter) But, I don't think you could look at a child's DNA and say that this person is or is not going to develop anorexia.

I: And so, that's what a gene for anorexia would seem to imply then. That it's definitely going to happen.

R: Yeah. (Liana, T-AN)

Liana rejected genes for AN because they seemed deterministic, which she termed “DNA genetic”, but accepted a more probabilistic form of genetic influence, which she termed “personality genetic.” Genes for personality and addiction made more sense to her. Willa made a similar distinction between conditions that were “genetic” vs. those that were “genetically triggered.” Thelma and Rebecca both compared the idea of genes for BN to genes for obesity, saying that they were implausible because behaviors such as over-eating were far more important. As Rebecca put it, “Yeah. Maybe I'm more likely to have bulimia. But that's not really why I'm puking up my food.” (Implications for perceived responsibility will be discussed in Chapter 6.)

Related to the idea that non-genetic factors were of great importance, several respondents said that AN and BN was defined by behaviors too specific to be potentially controllable by genes. It involved more cultural learning than a genetic disease would; as an analogy, Isabelle pointed out that there would not be a genetic predisposition for baseball.

...ultimately I mean it's an illness that manifests itself in a behavior. And like, I don't – I think that there are very few actual behaviors that are genetically, like, pre-dispositioned. People aren't more likely to play baseball over, like, basketball or something. It's, like, there are genes that, like, are going to make someone more athletic. But they're not going to determine which sport they do. (Isabelle, T-BN)

I think a lot of it has to do more with your environmental factors leading you in that direction than it is something that – it's kind of like you don't - you learn life skills throughout your life. Like, you're not born knowing how to tie your shoes. And, you're not born knowing how to brush your teeth. I think [an eating disorder is] kind of one of the things that you learn. And, if you're born with a gene that prohibits you from eating. Or, something like that. Like, if there was some medical disease where you had a strange aversion to food. Or, like, caloric intake. Then, I feel like that would be something completely different than anorexia. (Gena, T-BN)

Anorexia seems to be a really refined behavior. And very complex. And I just don't know that our genes are quite that good. And I just think there's cultural things and societal things. And I just – anorexia is caused by a lot more than just genes. Than just that. I don't think you can have – you have a gene for blue eyes. But I don't think you have a gene for anorexia. I just think you have a gene for you're going to have some problems.... Because I think that there are so many cultural variations that are required to be just right in order to get anorexia. And it just doesn't seem as though our genes are quite that specific. (Nell, R-AN)

AN and BN were defined by complex, learned behaviors, which were unlikely to be encoded at a genetic level.

In addition, one respondent pointed out that because she had personally experienced both AN and BN, they were unlikely to involve separate, unique genes.

Just because number one I think that a lot of people are like me. We see both eating disorders in the same person. Or people manifesting different anorectic behaviors as well as bulimic. I think that it would be – if there was a gene for anorexia and a gene for bulimia, it would be very odd that we would be having both. And then also – I don't know. Sometimes people are both concurrently. And it seems a lot of life

situation things that are – that interplay with which behaviors are manifested at which times. (Sarah, R-BN)

“Life situation things” influenced the kind of behavior more than genes.

To summarize, most respondents were skeptical of genes specifically “for” AN or BN, usually because such a theory appeared to deny the importance of environmental factors in shaping eating disorder behavior. If genetics are tied to eating behavior directly, as opposed to depression, addiction, or perfectionism, it suggests less of a role for non-genetic influences; the predisposition could have been channeled toward cutting, gambling, or scholarly achievement instead. To be sure, theories of genes “for” AN and BN also involved non-genetic influences, but the AN and BN is pre-formed and “waiting” until the right environmental conditions are present. It may also imply less of a role for individual agency; the AN or BN is a pre-packaged entity rather than a collection of specific behaviors learned or adopted by an individual actor.²²

3.2. Making conceptual “room” for non-genetic influences

Drawing on interview material and conceptual distinctions presented above, in this section I reflect on how respondents theorized genetic influences to work. I pay special attention to the relative roles of non-genetic influences. I approach this goal in two ways: identifying ways that respondents combined genetic and non-genetic influences across the “genes for what” categories, and ordering the categories according to how much conceptual “room” they leave for non-genetic influences (Figure 5.2).

Four general ways of combining genetic and non-genetic influences. Respondents envisioned interplay between genes and non-genetic influences in a variety of ways, to which

²² Perhaps the same applies to the conception of disease as opposed to choice. Sometimes the disease is conceptually divorced from the behaviors even though the diagnosis is based on the behaviors; i.e., when the eating disorder is “yelling” in one’s ear but one tries not to listen nor obey.

I alluded as I presented the seven categories. I simplify and summarize the findings above by suggesting three broad conceptual groups: gun-trigger interaction models, general-to-specific manifestation models and indirect-effects models. In addition there were indications of a fourth way of combining genes and non-genetic influences as independent factors because interplay was not mentioned.

Table 5.3 Respondents’ theories of gene-environment interplay

<i>Theoretical model</i>	<i>Illustration of model</i>
Gun-trigger interaction model	“Genes load the gun, environment pulls the trigger”
General-to-specific manifestation model	Genes for general addictiveness were shaped by environment to manifest as BN
Indirect-effects model	Genes led to something, and that something was defined as a problem or acted upon in problematic ways depending on environment
Independent-effects model	Genes and environment influence eating disorders additively rather than interactively

The first general way of integrating genetics with non-genetic factors was by means of a *gun-trigger interaction* model. With regard to the first, a few respondents used variations on the metaphor, “genes load the gun, environment pulls the trigger.” This metaphor has been widely used to explain how genetics and environment are important for complex conditions; in opposition to a deterministic genetic model, genes only become important under certain environmental conditions. Fran, quoted earlier, described such an interaction model:

Take two girls with pretty much the same make-up except for their parents. Stick them both in a gymnastics class. Competitive. If you’ve got one parent that’s eating disorder. And, none on this one. This one’s going to have a higher tendency to have an eating disorder. This one probably won’t. (Fran, R-BN)

In the environment of a competitive gymnastics class, those with a genetic predisposition to an eating disorder are more at risk than others. Petra used the gun-trigger metaphor to convey a similar idea:

I've heard it said many times. Genetics is the gun. Society pulls the trigger. And I believe that a hundred percent.... What it means to me is genetically I'm already predisposed to this disease because of the addiction cycle in my family. And I think society pulled the trigger. (Petra, T-AN)

The metaphor suggests a predisposition to a specific disorder that only becomes activated when the conditions are right. For Fran the condition is eating disorders, for Petra it is addiction.

The second kind of model, which I have termed a *general-to-specific manifestation* model, theorizes a broad theoretical predisposition that is shaped and channeled into a specific phenotype by non-genetic forces. This is also an interaction model in that non-genetic influences are important in the development of the disorder, but differs conceptually from the *gun-trigger interaction* model in an important way. Whereas the *gun-trigger interaction* model explains whether a phenotype develops or not (e.g., specific phenotypes such as AN, or general phenotypes such as an eating disorder for Fran or an addiction for Petra), this *general-to-specific* model explains how a general phenotype takes the form of BN for Fran and AN for Petra. In a gun-trigger interaction model for AN, there is a specific pre-formed AN “bullet” in the gun that will either be released or not when the trigger is pulled; the bullet represents AN, rather than eating disorders in general, or addiction, or another general condition. Because my focus is on how respondents theorized genes to matter for AN and BN rather than other conditions, a gun-trigger interaction model for AN presumes a gene for AN, though respondents mentioned gun-trigger interaction models for more general phenotypes. I propose that a *general-to-specific manifestation* model is conceptually distinct

from a gun-trigger interaction model for this project.²³ The idea that a genetic predisposition for a generic condition could “manifest” in a particular form enabled respondents to combine genetic influences with social and other forces in complex dynamic causation. Respondents who had a predisposition to a general condition such as addiction could “manifest” it with alcohol, gambling, or eating disorder behavior depending on environmental constraints and opportunities, norms and values, and personal preferences. Respondents spoke of similar processes in connection with anxiety, depression and generic eating disorders; these were channeled, funneled, or molded into specific forms in ways compatible with complex causation and ambiguous agency featured in Chapter 3.

A third way of combining genes and other influences was to theorize an *indirect-effects model*, in which genes create or contribute to a problem to which the individual must respond. Genes help create the problem, and the eating behavior is an attempt to solve it, which leads to an eating disorder (and yet more problems). The attempted solution is shaped by environmental influences and agentic processes interacting over time, and respondents described it in terms of learning, coping, self-soothing or gratification. This basic causal model was mentioned in most of the seven categories presented above, for example, Mireya’s reaction to the “stimulus” of a cake, Gena’s genetic depression, Nell’s sensitivity, Joelle’s curvy body, Margaret’s sex chromosomes. Unlike a simple interaction model, this *indirect-effects model* is a sequential, unfolding process: genes create a problem which evokes a coping response which is repeated if it is gratifying, which in turn creates new problems to be coped with, and so on. The genes are far more distant from the phenotype than genes “for” the eventual outcome.

²³ It is interesting that a general category like addiction or eating disorders could be “triggered” but not manifested in specific form, leading me to wonder in what sense it exists; is it a theoretical construct only or could it be detected with some effort, as with an endophenotype (see Chapter 1).

Fourth and last, some respondents spoke of genetic and non-genetic influences without attempting to integrate them into a single causal model: an *independent-effects* model. Here, genetic and other causal factors do not interact but exert influence independently. Such a model was harder to detect because respondents were unlikely to specify that two independent factors were non-interactive; this model is theoretically present when explicit talk about interaction or other interplay is absent. Selena may have held such a model: she alternately spoke of traumatic experiences in her early childhood and having a “DNA defect” in her brain, without trying to reconcile or integrate the two. She had come up with separate theories for why she was more debilitated by stressful events compared to others, but did not integrate the two explanations. It is also possible that for Selena, causation was either genetic *or* environmental.²⁴

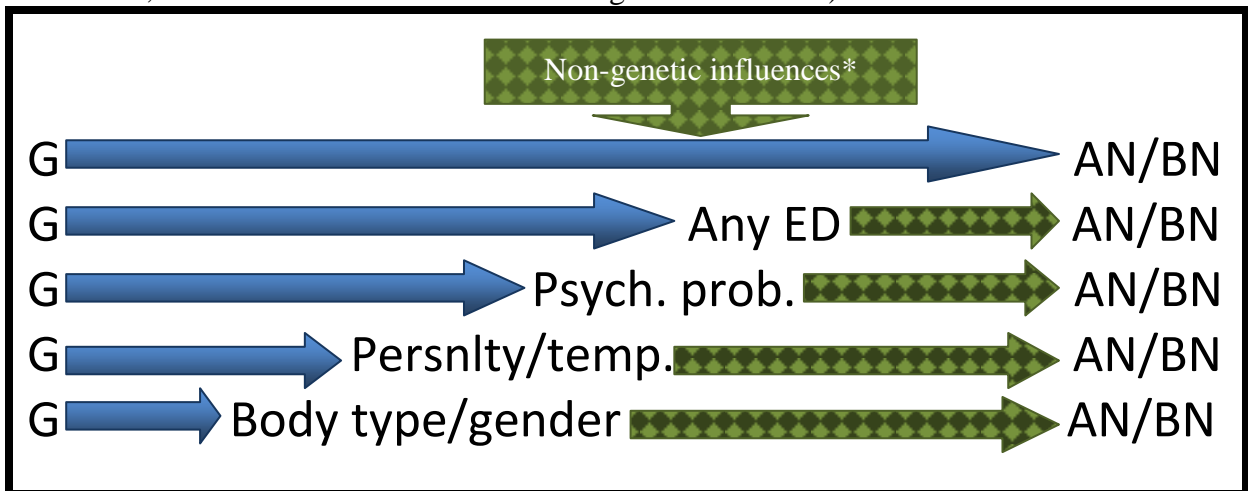
These categories do not exhaust the variety I encountered in these interviews, but describe several common ways of combining genes and environment. These were present across multiple “genes for what” categories and provide a way of thinking more generally about the role of “individual” and “environmental” factors when genetics are also involved. I turn now to another kind of overview across theories.

Rank-ordering of theories. Figure 5.2 illustrates a rank ordering of genetic theories according to the conceptual “room” for non-genetic influences. All theories of genetic influence involved some kind of interplay with non-genetic influences, usually environment.

²⁴ For Fran, who thought both genes *and* environment were important and clearly described an interaction model (regarding a gymnastics class, see just above and section on “eating disorders in general”) it was not clear whether she applied the interaction model to her own life experiences. When Fran stated the following, she may have been thinking either of an independent factors or an interaction model; it is not clear: “I would say my dad was the whole cause. From the genetics to his behavior towards me.” She discussed her father’s own purging as evidence of his own BN, which she thought she inherited from him, as well as his monitoring of her body size when she was a child. Both were important but the interaction concept was never applied to specific details of her life so she may have seen them as operating separately (despite articulating a theory of interaction involving gymnastics).

This ordering draws upon concepts relevant to diverse causal models (just described) as well as what the genes were “for”. I depict genetic influence as a blue arrow leading to five “genes for what” categories above, reduced from seven by combining the categories of specific mental illness, addiction, and general psychopathology into one category as “psychological problems.” To avoid the suggestion that respondents think that *only* genes are responsible for each category, I also depict other, non-genetic influences acting upon all five categories, as a green/checkerboard square and downward arrow. The horizontal green arrows convey the essence of the graph: there is arguably more conceptual “room” for non-genetic influences as one goes down the list.

Figure 5.2: Room for non-genetic influences in genetic theories (Blue arrows = genetic influence, Green/checkerboard arrows = non-genetic influence)



*These non-genetic influences interact with genetics (blue arrows) for all five categories.

Theoretically, genes “for” AN or BN limited the amount of play possible in the final outcome; if genes are for AN or BN, many aspects of the behaviors are pre-programmed once the genes take effect. Genes “for” a generic eating disorder allowed somewhat more play because environmental or other forces were theorized to shape the predisposition toward AN, BN, over-eating or other problems with eating. Genes “for” psychological problems (e.g., depression, addiction, having problems) leave yet more “room” for those non-genetic

influences that channel the person toward eating-related psychopathology. Genes for personality or temperament leave even more room because they do not presume psychopathology; even if the genes are expressed, other forces determine if the individual will actually experience a problem. Last, genes for body type or gender may presume the least genetic influence of all, because even when they take effect, thoughts and feelings are not necessarily affected thereby. This figure is a very simplified version of respondents' theories, but useful as an organizational device. Most respondents theorized in terms of "psychological problems" (n=37) followed by "personality" (n=33) (see Table 5.2 above for frequencies of other categories).

4. SUMMARY OF CHAPTER 5

In this chapter, I examined respondents' initial reactions to the idea of a genetic causal factor, and how they imagined genes could potentially influence eating disorders. First, I provided an overview of their "positive" and "negative" reactions, categorized according to positive-negative valence and perceived plausibility of genetic influence on eating disorders. "Negative" viewpoints characterized genetic explanations as simplistic and deterministic, with an inadequate role for environmental causes. "Positive" viewpoints of genetics often involved the perceived reduction of responsibility for the person with an eating disorder, as well as hope for scientific breakthroughs in the future, such as genetically-based treatments. People who already saw eating disorders in medicalized terms (Chapter 4 index) were more likely to hold "positive" views, as were people currently in treatment, and those who had received more treatment than others. The appeal of genetic explanations may thus be greater for people already favorably inclined or experienced with treatment.

I then described how respondents imagined genetic influence to operate in eating disorders. Their theories allowed a great deal of room for agency and environmental influence, with most respondents finding genes specifically for AN or BN less plausible than genes for something more general. I identified six more general categories: genes “for” general eating disorders or eating “issues,” specific psychiatric diagnoses or traits, addiction or addictive personality, non-specific psychopathology (e.g., not tied to a specific diagnosis), personality and other non-pathologized traits, and visible body characteristics (e.g., body size and sex). In these more general theories, genes were not tied to eating disorder behavior directly, thereby leaving substantial room for agents to interact with environments over time. I identified several ways that respondents combined genes and environmental influence, including the following kinds of interaction, broadly conceived: an environmental “trigger” metaphorically releasing a genetic disease bullet; a more indirect form of interaction in which a genetic predisposition for a general condition is shaped into a specific manifestation over time (to explain why a person turns to AN rather than alcoholism or gambling, for example); and a very indirect form in which genes simply create a wide range of phenomena (physical, emotional) that may or may not become a problem and to which agents may or may not respond by developing an eating disorder. Finally, I condensed respondents’ theories about genetics and environment into five categories organized in order of how much conceptual “room” they allowed for non-genetic forces to shape an outcome. I concluded that theories involving genes “for” AN or BN left the least room for the play of social forces and agency over time, and indirect theories involving visible body characteristics left the most. Respondents’ indirect and interactive genetic theories were compatible with their complex, dynamic explanations for the development of eating disorders, as described in Chapter 3

before genetics were introduced into the conversation. In these theories, external factors (e.g., beauty ideals, traumatic events) interacted with internal factors (e.g., personality, coping preferences) over time. Respondents were able to incorporate genetics into these developmental, meaning-rich narratives; genetic factors did not have to entail oversimplification. Their theories of genetic influence were also compatible with those of genetics researchers, who may investigate genes “for” personality as well as genes “for” phenotypes as narrowly defined as AN-with-restriction-only. But do such indirect theories encourage a more medicalized view of AN and BN? What do respondents perceive to be the implications of genetic influence for themselves and others with eating disorders? I turn to this topic in the next chapter.

CHAPTER 6

“SOME SORT OF LARGER FORCE AT WORK”: PERCEIVED IMPLICATIONS OF GENETICS FOR INDIVIDUAL AND SOCIAL RESPONSIBILITY

In this chapter, I examine the implications of genetic influence for perceptions of eating disorders and their causation. As should be apparent in the previous chapters, respondents were pre-occupied by the idea of eating disorders as a “choice.” What effect did genetic ideas have on the perception of eating disorders as volitional behaviors, chosen or at least controllable by the individual? The ubiquity of social factors in respondents’ causal models leads to a similar question: Did genetic forces reduce the perceived importance of social forces? In Chapter 1, I proposed a number of potential implications for the geneticization of eating disorders, including greater medicalization of eating disorders and impacts on perceptions of individual and social responsibility. I examine these three in turn, beginning with medicalization.

I will focus on interview material from the last section of the interview to discuss implications, but draw from the entire interview as relevant. When initially coding the full interviews, I had tagged material relevant to personal control, stigma, and good or bad implications. I drew on this previously identified material and supplemented it by reviewing the final section of the interview beginning with the question about a hypothetical media campaign. This question was asked of 49 or 50 respondents, and usually framed in general terms:

Imagine there were a media campaign to publicize the idea that genetics play some role in [AN/BN]. What effects do you think that would have?

Some respondents spoke about reactions by the general public, but all were asked how it would affect people with the eating disorder in question, and probed about potential good and bad effects. Then I asked all respondents about a hypothetical genetic test, with wording guided and approved by a researcher who studies genetic influence on eating disorders (Bulik 2008, personal communication):

Because both genes and environment play a role, it's not likely that a genetic test could predict whether a person would get 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 get an eating disorder. Would you want to know?

Frequently I would refer to respondents' previous answers about the importance of both genes and environment when asking this question to reinforce that this test did not imply deterministic genes; (e.g., "Because both genes and environment play a role, just as you were saying about addiction..."). After discussing why they would or would not want to know, I asked whether they would prefer to find out they had the genetic predisposition or not, and why.¹ I followed up with questions about the good and bad things about knowing one had a predisposition, and whether it would change how they understood their experience, how they talked about it with other people, and how they thought about treatment, recovery, and biologically related family members.²

¹ These questions about genetic testing revealed whether respondents would prefer to think of their case as genetically influenced, not whether they would want to know about a predisposition in advance of developing a disorder. Those who responded as though the question were about *predictive* genetic testing were redirected to talk about themselves as they were at the time of the interview, with a known history of AN or BN.

² At the end of the questions about genetics, I asked some respondents whether knowing genetics played a role would change how they viewed their own responsibility, and the idea that genetics could make the eating disorder seem less like the person's fault. All respondents had already brought up the theme of control by this point, and this question did not redirect their thinking.

Respondents reasoned about the implications of genetic ideas by drawing on simpler models of genetic influence than they had presented as most plausible (see Chapter 5). Even though respondents thought genetic influence was likely to work in an interactive and/or indirect way over time, with important roles for environment and individual agency, when they considered the implications of genetic ideas for people with eating disorders they used far simpler models of genetic influence. A few drew comparisons to simple, deterministic genetic explanations such as those for Huntington’s disease. Most retained an important role for non-genetic influences and in general their models were somewhere in between such simple models and those they had described earlier. I interpret respondents to be (1) drawing on a cultural knowledge of what genes usually mean to the general public (particularly with the media campaign question), and (2) interpreting some questions to connote a simpler model of causation than theirs. The very existence of a media campaign could suggest a powerful genetic influence, otherwise there would be little warrant for such a campaign. A genetic test of predisposition could suggest specific genes “for” AN or BN, a theory that was not adequate to capture respondents’ complex, dynamic, and often indirect models of genetic influence (see Chapter 5).

1. “LEGITIMIZED AS AN ACTUAL DISEASE”: GENES HEIGHTEN THE MEDICALIZATION OF EATING DISORDERS

If eating disorders are to some extent “contested illnesses”, despite their inclusion in the official listing of psychiatric disorders, do genetic ideas make them seem more like real illnesses? Does geneticization beget medicalization? In Chapter 1, I proposed that genes could “heighten” or further the existing official medicalization of eating disorders, such that medicalization could continue even after inclusion in official medical classifications such as the DSM. I elaborated existing models of medicalization in Table 1.1 by including biological

and not-clearly biological columns and argued that complete medicalization is the combination of biological and pathological components. The geneticization of eating disorders places them in the biological column, but to provide evidence for the proposed conceptual model this biologization would have to increase perceived medicalization. To assess changes in perceived medicalization of eating disorders, I developed two coding rules, one conservative and the other more expansive.

To assess whether genetics made eating disorders seem more “medical”, I was careful to define “medical” in a way that first excluded then included biology and biological treatment (thereby creating the conservative and then more expansive criteria). Conrad does not explicitly define “medical” as “biological”, even though that is usually the connotation. As noted in Chapter 1, the move from “badness” to “sickness” can take place even if a disorder does not have a biological origin or treatment; inclusion in the official listing of psychiatric disorders is used as evidence of official medicalization even though that work is agnostic about causation (i.e., the de-medicalization of homosexuality was achieved by its removal from the DSM, and it is increasingly discussed in biological terms even though it is not medicalized). For a conservative definition of medicalization, a respondent saying that genetic causation makes the eating disorder seem more biologically based or biologically treatable would not be sufficient evidence of perceived medicalization.³ Including biological causation or treatment could set too low a bar for medicalization because genes are biological phenomena and biological treatment would logically follow.

³ Conrad left the term “medical” undefined in his definition of medicalization, relying perhaps justifiably on shared cultural knowledge for the contexts he researched: “Medicalization consists of defining a problem in medical terms, using medical language to describe a problem, adopting a medical framework to understand a problem, or using a medical intervention to ‘treat’ it” (Conrad 1992: 211). Applying this definition to mental illness certainly connotes a biological basis to many readers, but I am concerned that it would constitute too easy a standard for medicalization.

On the other hand, as noted above, biological factors are the usual connotation for “medical,” so I therefore approach the definition of “medical” cautiously⁴ by using two sets of criteria corresponding to narrow and expansive definitions. An overview is provided in Figure 6.1 below. The narrow definition is based on two themes that do not depend on biological aspects: unambiguous *medical language and concepts* (e.g., words like “medical” and “disease”, and comparisons to uncontested disease like cancer) and the perceived importance of *treatment by a healthcare provider* of any kind. Most respondents (n=37, or 74%) spontaneously brought up one or both of these themes. These two themes correspond to concept of “sickness” as opposed to “badness” or another non-medicalized definition of eating disorders and loosely correspond to the pathologizing dimension of Table 1.1. The expansive definition accepts as evidence for medicalization spontaneous mentions of biologically-based treatments in connection with genetics. Nearly all respondents mentioned one of these three themes (n=47, or 94%). I illustrate the three criteria in turn and identify some countervailing themes pitting geneticization against medicalization.

Figure 6.1: Genetics and the medicalization of eating disorders: Findings using two definitions

Criteria used to assess medicalization	Narrow definition	Expansive def.
1. Genetics inspire medical concepts and terminology	74% (37/50) mentioned at least one of these two	94% (47/50) mentioned at least one of these three
2. Genetics locate problem in domain of health professionals		
3. Genetics suggest biologically based treatments		

⁴ I used a similarly cautious approach when developing the medicalized terminology index in Chapter 4. For the five-part index, the four terms that connoted medical terminology either connoted treatable disorders, included the words “disease” or “illness”, or both. Those terms were psychological problem, mental illness, brain disease, and physical illness. The other term in the index was “choice”, which was treated as a term that indicated *non*-medicalized viewpoints.

1.1 Genetics inspire medical concepts and terminology

Genetic explanations made eating disorders more like medical disorders to a majority of respondents. That finding makes it apparent that eating disorders are contested illnesses. Although my present focus is on medical concepts and terminology, many quotations touch on the theme of treatment, the second dimension of medicalization discussed in this section, and perceptions of individual choice or control, discussed in the next section on implications. I grouped material about medical concepts and terminology into these themes: genetics make eating disorders seem more “legitimate”; more “real”; more like a “disease”; and more like specific, uncontested diseases (e.g., cancer). Interview questions were framed very generally and did not use terms like “disease”, “illness”, or “medical” and thus did not lead respondents to consider medicalization. (When presenting quotations in this section I *italicize* the relevant medical concepts and terminology; italics do not reflect respondents’ emphases.)

Eating disorders as more “legitimate”. Several respondents thought genetic ideas could help convince others who did not see eating disorders as “legitimate” or “valid” disorders. Against a background of contestation, in which the reality of eating disorders *qua* disorders is questioned despite official inclusion in the DSM-IV, genetics makes them more legitimate. Melanie described BN and other mental illnesses to be “legitimized” as “actual disease[s]” if genetics were found to play a role.

Well I think if you, like, look at different types of mental illnesses like bipolar or depression. I think it’s, like, in the last twenty years it’s been more, like, *legitimized as an actual disease* that people – at least a lot of people anyway recognize that if you have that, you can take medication for it. It’s not something that you can just snap out of or something. I think that’s – certainly everybody doesn’t believe or get that or something. But I think it has helped some to talk about it as a *clinical disease*. (Melanie, R-BN, italics indicate emphasis added for all quotations in this section on medicalization)

“Legitimizing” BN as a disease involves the recognition by other people that it is “not something that you can just snap out of”, meaning it is not under the person’s control so treatment is warranted (the second dimension of medicalization, discussed later). Hannah also felt that if she knew she had a genetic predisposition, it would make it seem more like a “legitimate disease” to other people as opposed to being “crazy”, which apparently did not connote disease.

Because, then I could explain it better to people who ask me “What happened?” And, . . . then, I could, like, be, like, “Well, I had this gene that made me more susceptible.” And then, I could add the other stuff. Whereas now it’s just sort of like, “I don’t really know why it happened to me and not to everybody else that went through hard stuff.” Because, I didn’t go through that bad of stuff. Like, I don’t know why other people didn’t react the same way I did. So, that would help explain the story. . . I guess if I say it, it seems more like a *legitimate disease*. If I don’t say it, then people are just like, “Hum. Is she crazy?” (Hannah, R-AN)

Genetics would help to justify why Hannah developed AN even though she “didn’t go through that bad of stuff” and might have been expected to escape it. Isabelle apparently thought that healthcare providers did not take it seriously enough: “And so when you talk about genes and biology, I mean, I think that’s going to make the medical community pay attention to it a little bit more. And help maybe *validate it* in the eyes of medicine as well.” If eating disorders seemed illegitimate and invalid, genetic origins would shore them up and command attention.

Eating disorders as more “real”. Several respondents thought genetics would make eating disorders more “real” to themselves and others, as though there were doubt about their existence. I interpreted these doubts to center about their reality as entities; if eating disorders are real then they are, by definition, disorders, as listed in the official DSM which reflects

their medicalization. Lynn thought that people with a genetic version of AN would see it as “real” rather than the opposite, which was being “crazy” or a “bad person”:

Well, then they’ll say ‘Oh. I’m just not crazy. *It’s real*. ... And, that I’m not a bad person because I’m doing this.’ Yeah. This is, like, not functional. But, it started with something *real*. (Lynn, R-AN)

Tammy also thought genetics would confer more reality on AN:

I think it would [help people]. I’m not a freak. I’m not a control freak. *This is something real*. But the steps – knowing it’s *real* or knowing it’s a *genuine disease process* and not knowing it doesn’t make treatment easy. (Tammy, R-AN)

Natalie (T-AN) suggested that others would be more understanding if they saw it “as something that is maybe passed from one generation to the next. That it’s, like, *it’s there*. It’s *something that does exist*.” Delia (T-AN) said that her family would be “*more accepting of the disease*... Because, it’s genetic. It’s a genetic thing. I have no say in that.” She already saw it as a disease herself, but her family would accept it as one if they knew genetics to be involved. Zinnia’s (T-BN) view of eating disorders would change from the idea that they were a collection of “behaviors” and a “symptom of something” to being “a causal factor as opposed to the end. The outcome.” Although she does not use terms like “real”, she conveys something similar with the term “causal factor”; rather than BN as a name given to describe a collection of symptoms, BN would be an entity with causal power rather than an effect or outcome. Olga thought genetics would be comforting because it was a “real reason” for developing an eating disorder and went on to explain that doubts about AN’s reality were “something I fight with.”

I would feel like there was a definite reason. That it wasn’t just me being stupid or silly or whatever. That I actually had a *real reason*... That’s something I fight with. But I’m pretty sure with the anorexia *it’s real*. It was real. I don’t know. (Olga, R-AN)

In the midst of uncertainty about the reality of AN, genetics helps make the case that it is real and the person with AN wasn't just being "stupid or silly." If genetics are involved, eating disorders were in the body, not just "in their heads."

From the opposite direction, finding out that one's own AN or BN was *not* genetic could suggest that it was *less real* than for those for whom it was. Gillian was concerned that her own AN, which she did not think was genetically influenced, would come to seem less real if genetics were publicized as a causal factor for eating disorders. Her reasoning reflects a similar understanding to those described above.

I: ... what was your reaction to the idea of genetics as making – as playing a role somehow when you first heard about it?

R: Well, initially I thought "Well, no one in my family has it. So, does that mean *I don't really have it?* And, *it's all in my head...* I would be very distressed. Because, I would think that meant on some level I was making it up. Which bothered me.

(Gillian, T-AN)

Victoria raised a similar concern:

R:... And then if I found out that I didn't have the genetic make-up, then it would be like, "Well why do I have an eating disorder?"

I: What would it make you think?... Or feel.

R: That it was, like, all my fault. And *this [BN] wasn't even something.* (Victoria, T-BN)

Someone without a genetic explanation seemed more open to the charge that she was "making it up" or that it was all her fault. If it were genetic it seemed to have more reality: it was something, it existed, as opposed to being invented or caused by the person.

Eating disorders as "disease", and people with them as "sick". Respondents speculated that the involvement of genes would increase the perceived similarity of eating disorders to diseases or medical conditions generally. Mary (T-AN) expressed that genetics would make her feel less responsible for her AN: "Just that *like any other kind of disease* that it's always been inside me. And, there wasn't anything I could do about it. And, like I

said. For me everything just lined up perfectly to cause it....” Respondents said that genetic ideas would make others “more likely to see it as a *mental disorder*” (Joelle, R-BN), a “*mental defect or disease*” (Selena, T-BN), or “more of a *brain illness* [or] *mental illness*.” (Helen, T-AN) Fiona (T-AN) speculated that “it would make people more aware that these are people who need help and are *sick*”. Carly (R-BN) imagined that “I would kind of be like, ‘Oh. It’s not my fault. I mean *it’s a disease I have*. I can’t get rid of it....”

Claire and Karen noted that although genes could be expected to make eating disorders more medical to other people, it would not change their own thinking because they already saw them that way. Their answers reflected a similar logic to other respondents.

I: And, does that idea of anorexia having a genetic causal factor change how you see anorexia at all? If it were true?

R: No. Because, I still think of it as something that’s like is a *disease*. I think of it just like a *physical disease*. So, yeah. Either way. I mean *some physical diseases are genetic. Some aren’t*. (Claire, T-AN)

I don't think it could change how I feel about the last ten years. Because, *it’s a mental and physical illness*. And, it just took control over me. (Karen, T-AN)

These respondents assumed that conceptual change would be in a medicalized direction, even though their own conceptions would not change.

Some respondents expressed concern that genetics would make eating disorders seem too much like a medical condition. They thus shared the belief that genetics would medicalize eating disorders but objected to such a change. Sydney thought that genetics would make it more a “medical thing” and was concerned that this would ignore the complexity of the disorder, because “it is not just a disease”. Clearly, her concern stems from an expectation that others would begin to see it more like a disease.

I think a biological link or sort of discovery in that field would make it *more a medical thing*. Which hopefully could lead to a drug or something that might be able to help it. So I’m totally in favor of that discovery (laughing). But at the same time I

understand that it's very hard to separate out. Because *it is not just a disease. It's NOT a disease*. It's a very complex cycle of mental and physical collection of symptoms. (Sydney, T-BN)

While Sydney anticipated future pharmaceutical cures (with tongue in cheek, perhaps), Willa reasoned that genetics for AN would make it seem like an incurable "brain disease" and therefore implausible.

I guess I really don't see it as that likely. Because to me then it's much more of like a *brain disease* like we were talking about earlier. It's something that can't be helped really. Can't be cured. Maybe you deal with the symptoms. But you can't really do anything about it. And I don't think of anorexia like that. (Willa, R-AN)

When responding to earlier questions about the terms "mental illness" and "brain disease", Willa had brought up genetics spontaneously: "I really don't think it's a mental illness. When I think of a mental illness I think of something that's more like genetic. Or like schizophrenia or mental retardation or something like that." However, Willa did believe that her own genetically influenced "type A" and perfectionist personality contributed to AN. In Willa's estimation, genetics for AN specifically could medicalize AN, but not genetics for personality, suggesting the importance of the content of genetic theories for conclusions about medicalization. Even if Willa herself does not find genes "for" AN plausible, others might, and if they did she evidently would expect them to perceive it in a more medical way than otherwise. Wendy (T-BN) thought that if genetics were involved, others would unreasonably expect the person to recover quickly, "Because that's how *medical, like, diseases* that are, like, genetic and stuff like that are. Like, if there's a cure for a lot of *diseases*. Like, it's like a shot. You get a shot. And you're cured." Genes would make eating disorders too much like "medical diseases", in Wendy's view. The overlap between medical terminology and medical treatment is apparent here, as in other quotes from this section.

Genetics increases resemblance of eating disorders to uncontested diseases. Genetic ideas also prompted respondents to analogize eating disorders to specific uncontested diseases. For Selena, genetics would make eating disorders seem more like cancer or diabetes.

But I think if I realized that it wasn't just environmental. If it was *really a disease like cancer or diabetes* or something. I mean if you had diabetes, wouldn't you go and get treatment? Like, this one you think you're doing it to yourself... I just thought of it like, "This is a weight thing. This is not, like, *a disease.*" If it had that label. Like, I didn't have a choice. Like this just developed. I think I would be much more likely to go and seek treatment. (Selena, T-BN)

Breast cancer and diabetes are in opposition to the idea that BN is "a weight thing" or something one is "doing" to oneself. Asked whether a genetic causal factor would change how Paula talked about it to people, she likened it to chicken pox:

I can see it becoming something that is spoken of more in the terms of other just sort of things that you get (laughs). Like, diseases. Like, "Oh. I had the *chicken pox* when I was a kid." "Oh. I had bulimia when I was a kid." That societally might not be then associated with some kind of character defect... Sort of de-personalized. (Paula, R-BN)

Respondents also likened genetic eating disorders to how they or others would view gangrene (Lynn), schizophrenia (Fiona), Alzheimer's (Olga), autism (Zinnia), mental retardation or ADHD (Petra). (Some who spoke in medicalized terms also compared it to non-diseases, like homosexuality, eye color, and musical skills, suggesting that genetics do not have a single meaning.)

Some respondents thought genetics made it seem more like a disease that had not been chosen than a disease that had. Vanessa and Yvette made a distinction between diseases you choose and disease you do not. A disease that is not chosen more closely resembles uncontested diseases like cancer. Therefore I interpret this as evidence that genetics made eating disorders more like a disease.

If it's genetic, then I think that that would imply that the disease is not one that you choose. It would be viewed more like *cancer*. It's just you have no control over it. And yes – and I think people would be more apt to feel sorry for you and have more empathy for you or sympathy. Because you can't empathize unless you've been through it. Have more sympathy for you if you have the disease than not. Because now I think when people find out you have the disease it's just they're not sympathetic to you. Because they feel like, "You chose to be this way. You can correct it. This is just you." But if you say – if you prove it's a genetic thing, then it's not. It takes that away. (Vanessa, R-BN)

I don't even know how you would go across trying to help out somebody with a *genetic disorder*. But I think it would just change their outlook of the disease in general. Whereas, like, when I was diagnosed with it, like, my outlook was "This is a disease that I chose to get." Like, it's almost like if I knew someone had AIDS, I chose to sleep with them knowing they had AIDS so I would have AIDS. Like, it was kind of like, that's how I looked at it. Like, I'm choosing to have anorexia. It's like I went and bought it. Now I have it. Like, and so I feel like it would change how they felt that they got it so to say. (Yvette, R-AN)

The common sense meaning of a disease is that it is not chosen; this idea was articulated by Jackie and quoted in an earlier chapter. To choose a disease would call into question the sanity of the person, underscoring this common sense meaning.

Comparisons to disease sometimes focused on the idea that the eating disorder was something one has, rather than something one is. The disease was something external to the person that ought to be resisted, rather than being a part of the self. This idea is clearly linked to ideas about individual control, as well as the logic of pursuing treatment from professionals if genetics are involved. As Melanie put it, if her BN is genetically caused then "I'm fighting it. Not myself." The disease is external to the person, not the person's fault nor part of the self.

Well that [idea of a genetic causal factor] might be a positive thing. To have a kind of a focus... like if you have a certain type of breast cancer. If you have something. You can – I would think that you can put all your energies into doing everything you can to beat this cancer. Knowing that you can't control everything. And knowing that. But with the eating disorder maybe if you had this, could focus your energy. You know you have this gene. You can have more of a plan of attack. And have a more focused

way to deal with it... you could kind of put [it] outside of yourself. It's not myself I'm fighting. It's this gene. (Melanie, R-BN)

For Melanie, genetic origins made BN *less* like a part of herself, and more like a separate disease entity that she could not directly control but could fight against, like cancer. Yvette similarly linked genetics to the sense that AN was a disease that affects an individual, rather than the result of an individual's choice not to eat.

I think it might help society's view of it. Like, that *it is actually, like, a disorder and a disease* and not just somebody's choice to not eat. So I think then people might start, like, worrying about it like any other disease (chuckles). Like, "Oh my gosh. I could catch that." Like, "I could get that from my mom" (said in a dramatic, serious tone of voice). I don't know. I think that people would look at it differently in general... I guess, like, genetically I would view it as, like, Alzheimer's or something that you develop... Instead of like now it's like, I feel like people look at it like media caused this. Or the girl wanting to be thin has caused this. Like, so I think maybe it might take, like, some strain off of, like, negative attitudes towards it maybe. Towards the individual. Whereas they look at it as *like a disorder and a disease that has AFFECTED this individual*. (Yvette, R-AN, capitalization reflects respondent's emphasis.)

If genetics are involved, AN can seem like a disease that powerfully affects the person rather than being the result of choices she made.

1.2. Genetics locate eating disorders in the domain of health professionals

In addition to genetic influence making eating disorders seem more like legitimate, real diseases (the proposed first criterion of medicalization), it also made treatment for eating disorders seem more necessary (the second criterion). Genetic causation suggested that professional treatment was more appropriate and necessary. In keeping with my conservative definition of medicalization, expectations for new biologically-based medicines or procedures were excluded, but featured later as part of the more expansive definition. Even though common sense says that such treatments are more "medical," I excluded them in order to focus on the more general idea of whether genetics made eating disorders seem more

like a disease that needed treatment, regardless of the type of treatment. My interview questions did not lead respondents to associate genetics with professional intervention; they did so spontaneously. Although I asked whether they would expect the discovery of a genetic influence to change treatments for eating disorders, this was not the first time I had brought up treatment. In addition, when I asked about the hypothetical genetic test I did not specify a context, clinical or otherwise, that would necessarily tie genetics to medical conceptualizations. The sub-themes described below overlap with each other and with material illustrating the first criterion.

Treatment is warranted. Some respondents made general statements about the need for treatment. Isabelle stated that the idea of a genetic causal factor could “medicalize” BN, which she defined as “an illness that requires medical treatment” (similar to Conrad’s definition). Viewing BN in this way would help reduce the shame people feel when it is interpreted to reflect a “personal flaw,” a theme I will return to when I broaden my analysis to perceptions of individual control and responsibility.

R: ... I think it could also actually help them to accept themselves a little bit more. Like, “Oh. This isn’t like – this isn’t, like, just due to, like, a personal flaw in me. Like, there are actually scientific reasons why I have this.” And I think it could, like, lessen the shame that people feel in regards to their own behavior. So I think in that sense it could be positive. And also whenever you talk about genetics I think that – I mean I do think bulimia needs to be *medicalized* a little bit more.

I: Can you say more what you mean by that?

R: Yeah. Like, I just think that it does more need to be looked at as *an illness that requires medical treatment*. And I mean that can be a variety of medical treatment. But I mean I think it involves treatment both from, like, MDs who also are, like, more like the psychologist or psychiatrist nature. But also I mean from, like, family physicians. I think it’s really important for doctors, like, just more – less mentally inclined doctors to be aware of that as well. And so when you talk about genes and biology I mean I think that’s going to make the medical community pay attention to it a little bit more. And help maybe validate it in the eyes of medicine as well. (Isabelle, T-BN)

If “genes and biology” are important for BN, it would “validate” it as something for healthcare professionals to treat and to pay more attention to, rather than to neglect as a personal failing (the implied alternative). Joelle also said genetics can make people with BN see it as a “mental disorder” that needs to be addressed by a “professional” or “someone who knew more.” Thinking of it as genetic would encourage someone to seek help, just as they would for another condition such as heart disease or cancer.

...it might be good in some contexts for people to be able to know there was a root cause to this. And, that maybe they would actually be more likely to *see it as a mental disorder and something they should see a therapist for*. ... I feel like when people think of things that are genetically pre-disposed, they think heart disease and cancers. And, those are the kinds of things that you put on forms. And, that are talked about. And, that *you discuss with doctors*. And, recognizing it as a problem that you have that is genetic I feel like would make it – I don’t know. Somehow easier I feel, like, to discuss with a professional if you wouldn’t have initially. (Joelle, R-BN)

Melanie also thought that if BN were “legitimized as an actual disease” or “clinical disease” it would make it easier for people to “seek help”, rather than being expected to “just snap out of” it.

Also asked to consider good and bad implications, Mireya linked “medical treatment” to reductive medicalization, in contrast to “holistic” and psychological conceptions of BN.

I guess good is, like, if it was really genetic, then there’d be, like, a huge surge of interest in it and research. And maybe there would be, like, *medical treatment* that could cure it. I don’t know. And negative is like that *you medicalize it and you think that you can cure it through medicine or through gene therapy* or whatever. And you neglect kind of more of the mental psychological aspects of it... Why it would be negative? To kind of neglect the psychological aspects of it? Because I don’t think – because for me I don’t think it’s genetic. And I think it’s a disorder that’s very much individual by individual. And if you think of it as a genetic thing, then you kind of gloss over all of the different specific factors that kind of fed into for lack of a better word everyone’s disorder which are very different from person to person. And I think in order to really, like, heal somebody with an eating disorder you really need to think of it holistically and not like as a medical disorder that’s genetic. That’s purely genetic. And so that’s why. (Mireya, T-BN)

For Mireya the term “medical” implied a biological intervention like “medicine” or “gene therapy” as opposed to psychological treatment. Wendy also thought it would seem less like something to talk about with friends and more the exclusive domain of doctors.

I would probably bring it up less, honestly. Because I would feel like other people could help me less. Because if it was just this genetic part of me, I would feel like *only doctors could help me*. As opposed to being thought patterns whereas friends could help me. (Wendy, T-BN)

Carly thought people with BN “would want to go to, like, a psychologist more to talk about it... To see what would – in their brain would cause them to be like that.” If genetic, eating disorders would thus be in the domain of treatment professionals.

Help is justified because you cannot change it alone. Several respondents reasoned that because genes were too powerful to overcome on their own; additional help would be needed. Betty, in the inpatient unit for AN, said a genetic causal factor would help “rationalize” or “justify” the expense and trouble of professional treatment.

R: I mean I would be more accepting that I did have an eating disorder if I knew it was genetic. Or, could be. And, *I would go get some professional help* or the in-hospitalization program. Because, I would not think that I could do it by myself. Or, not expect myself to be able to do it with no help....

I: Can you say more about why something genetic would be harder to overcome on your own?...

R: I just think it would be. I mean anything that you were born with that you didn’t cause somewhere along the way. I think I would accept the fact that yes I do have it. But, shoot. My mother had it. Or, my daddy had it. And, maybe *I better get some professional help*. Maybe I can’t do this by myself. Because, my coming here I thought “Oh, my goodness. I don’t want anybody to pay that amount of money. Why can’t I do this myself?” But, if it were genetic, I would think “Aha. I can rationalize or whatever. Justify going to this expense. Because, it’s something that’s definitely going to be a problem for me. Because, I’ve inherited it. (Betty, T-AN)

Obtaining inpatient treatment was easier to justify if the condition “were genetic” than if it was something she could “do” by herself. Mireya also thought genetics encouraged a medical conception for people with bulimia.

R: They might think it's not in their will power or in their hands. That it's outside of their ability to change anything. That it *needs to be treated*. Because, like, a genetic disease or a genetic disorder or whatever.

I: So if you say genetics plays a role in bulimia, it sounds like a genetic disorder?

R: Yeah. Like, that it's outside of your ability. Like, it was outside of, like, your choice. It wasn't your choice. It was, like, in your genes that you were going to have bulimia. So therefore it's not your fault or it's not your – it's not external. It's within your make-up. Your genetic make-up. So I think people would think that they – I think if people think it's genetic, then there's, like, they feel like there's nothing that you can really do. Unless there's some real, like, *medical treatment* for it. (Mireya, T-BN)

There is nothing *you* the person with the eating disorder “can really do”; only “real” medical treatment can make a difference. Fran, who recovered decades ago by treating BN as a bad habit and resolving to quit, just as she had quit smoking, thought that she would have turned to professionals if she knew she had a genetic predisposition.

I would have probably *sought more professional help*. And, I'd probably been digging into it a little more deeply. Going “*Is there some kind of medication? Or, is there something I can do to stop these tendencies since it is genetic?*” Instead of just diving into it and taking care of it myself. (Fran, R-BN)

Yvette also imagined a greater role for doctors, which warranted coding under the conservative definition of medicalization. She also envisioned pills, injections, and an endless hospital stay, but these biologically-based interventions did not constitute medicalization under the conservative definition⁵ and so are discussed in the next section on the expanded definition. Rather, it is the more active role for healthcare professionals in treating eating disorders that suggests medicalization for the respondents just described, as well as in the following passage.

I mean in my mind if doctors are trying to prove that it's genetic, then they would then turn to trying to prove to find some type of pill or some injection to have to cure

⁵ As noted before, genetic causes logically imply biological treatment, but this alone does not constitute evidence of medicalization because that may be only a shift in the *kind* of treatment, not its appropriateness or quantity. My conservative definition has focused on conceptions of the eating disorder and seeking professional help, but has excluded the seeking of biologically-based treatment because inclusion in the DSM constitutes medicalization but comprises both biological and non-biological causation.

it. So then I feel like it would not be so much of an active on the anorexic recovery as much as *just passive while letting the doctors drug them* or do whatever... So I guess, like, I feel like that's the type of recovery instead of like, "You need to look and mentally understand why you don't want to eat." Like, I feel like it would be "*You just need to go to the doctor. And they'll handle it for you.*" So I think that to me when I hear genetic that's what I think. That they're going to then try to find is a pill that cures it. (Yvette, R-AN)

With genetics, the patient's role would be more passive than otherwise, and the doctor's role more active. Many respondents expressed skepticism about pills and other biologically-based treatments in ways that echoed Yvette's point. Even those skeptical about medical and/or biological treatment clearly link genetic causes with more professional treatment, supporting the broader point that genetic causes help to medicalize a condition.

Support from others to seek treatment. If it were genetic, one would expect family, friends, and even strangers to support treatment-seeking. Selena said she would be encouraged toward treatment if her BN were genetic and therefore re-conceptualized as a "disease" by herself and others. She speculated that if it were genetic, she would no longer view it as "just environmental" and a "weight thing" and neither would the people around her.

But I think if I realized that it wasn't just environmental. If it was really a disease like cancer or diabetes or something. I mean if you had diabetes, wouldn't you go and get treatment? Like, this one you think you're doing it to yourself. You think you don't have a choice... because, like, I had a friend who was seeking a treatment, like, in Texas for – it was, like, a cancer. But it was – there was some innovative treatment. And I was, like, "I'm so proud of you for doing that. That's awesome." But for myself I would – I've never done that. Because I didn't think of it like that. I just thought of it, like, "This is a weight thing. This is not, like, a disease." If it had that label. Like I didn't have a choice. Like this just developed. I think I would be much more likely to go and seek treatment. And to say "Oh. I have the gene that causes this. And I've had a problem. And I'm seeking treatment." And I think people would be a lot more accepting of that. Because I've found that even at work and school people when they find out you have an eating disorder are like, "Why are you doing that to yourself? It's not like you – why would you do that?" Like if you had diabetes. And you found out your co-worker had diabetes. You'd be like, "Oh my God. Let me

help.” But people don’t think of bulimia the same way. They think “Why do you stuff yourself and then throw up? That’s so gross.” (Selena, T-BN)

Selena expected people to be more supportive about seeking treatment for a genetic condition, again suggesting a link between genetics and professional intervention. Carly, who never received treatment for BN, imagined that her family would mobilize to support her to do so.

I think that my parents’ knowing and my, like, grandparents or, like, my brother knowing it would be more supportive. Because they would do everything possible to make it easier for me or for me to get help. (Carly, R-BN)

Gena speculated that her parents might understand better that she “needed some help”, rather than accusing her of trying to get attention or pretending she was sick.

But, I think that they [parents] would look at me differently... it might explain some things for them. Instead of them saying like, “She is doing something else.” Type thing. Like something else to gain attention. Something else to say that she’s sick. I feel like it might explain some things for them. And, make it a little bit easier to understand that I might need some help. Or, that I might have needed some help. That they just kind of either neglected to see. Or, didn’t want to see. I think that it might help them the way that they view me. (Gena, R-BN)

Lynn, who had a non-medicalized view of AN and credited feminism in helping her to recover, imagined that if she knew AN were genetic she would be encouraging others with apparent eating disorders to get help at a hospital.

But, I guess if I knew it, it might make me more activist. And, ... not overlooking it in other people if I saw it... Just it seems that makes it more serious. And, that’s wrong that I would think that way. But, it kind of – it would make it more of a disease. And, it’s like if you saw someone going around with gangrene, you would say “Hey. *You need to get to a hospital.*” (Lynn, R-AN)

Lynn likened genetic eating disorders to something as visible and uncontested as gangrene, which would indubitably prompt her to urge them toward a hospital.

More monitoring over a long period of time. Some respondents specified that they would expect medical monitoring and treatment over a longer period of time if the condition were genetic, perhaps even for the rest of their lives.

Especially if you're health minded knowing that you had a genetic pre-disposition for that may push you into some type of evaluation. Or at least some type of continued follow-up that kind of monitored your health. Much like diabetics need to go to the doctor every six months. And, like, they need to check their blood sugar. Is there some type of monitoring that could be in place that you would present with to your physician as, like, impending signs of a heart attack? Or left arm pain. Well do you find yourself not being able to eat at this time, this time, this time, and this time? If you do, maybe we need to investigate that. Have you lost more than ten pounds since the last visit? And there's no physical reason like diabetes or hernia or ulcer or something like that. So I think there could be cues for health care providers to act on. And I think that would be very helpful. (Tammy, R-AN)

The motivation toward long-term monitoring was sometimes driven by fear of relapse; recovery was less certain if the condition were genetic.

Well I think if the gene is there, there's always going to be those consequences of maybe going back to relapse very easily. I'm thinking that if the gene is there, then they'll probably have to be monitored. Seeing a doctor on a regular basis or something just to keep it in check. It's just like diabetes or anything else. I think you need to be monitored. Be kept in check. (Natalie, T-AN)

... I like to think that it's over and completely done. I guess there would be the concern for relapse. And there would probably be more attention on just maintenance – to just maintenance therapy and maintenance. Which probably isn't the worst idea. Because I mean people do relapse. Whether there's a genetic pre-disposition or not. People do relapse into their behaviors. But I think that if there were a definite genetic link, that we would worry more about not fully being able to recover. Because it would be this pre-programmed thin thing. (Sarah, R-BN)

If it were genetic, these respondents would imagine seeking “maintenance” treatment, to be “kept in check” and monitored. This is not just a new form of treatment but more treatment over a longer period of time.

To summarize, genetic involvement in eating disorders prompted three-quarters of respondents (n=37) to bring up one or both themes from my conservative definition: (1)

medical concepts, terminology or comparisons to uncontested diseases like cancer, or (2) perception that treatment by health professionals would be more appropriate. Respondents who brought up biologically-based treatments without these two themes were not included although it is clear from the quotes that biologically-based treatments were commonly mentioned as a consequence of genetic causality.

1.3. Genetics suggest biologically-based treatments

Under an expanded definition of medicalization with three qualifying themes (medical concepts, health professionals, and biologically-based treatments), ten more respondents were added, for a total of 47 respondents (94%).⁶ Imagining pills or gene therapy to treat eating disorders may also support the idea that genes promote medicalization because only healthcare professionals could administer such treatments. When Amy (T-AN) was asked about good or bad effects from the hypothetical media campaign, she immediately imagined new biologically-based treatments: “They would, like, probably try to produce more medications. Like, there’s a whole line of meds for depression. A whole line of meds for bipolar and schizophrenia. They may try to develop one for anorexia.” Some would have welcomed such a possibility: “Well, if they found a genetic link, then hopefully they could find some kind of treatment early on. Or, some kind of – I don’t know. Gene therapy or something.” (Deena, R-AN) Others were more cautious: “But, if it’s genetic, I don’t know. What are you going to – I guess you take a pill. I don’t know. Or, you take out the gene. I mean I don’t know. That’s kind of scary.” (Irene, R-BN)

1.4. Contrasting themes: How genetics might not medicalize eating disorders.

Several respondents who appeared to perceive increased medicalization also suggested genetics might counteract aspects of medicalization (e.g., whether an eating

⁶ Amy, Rebecca, Thelma, Yolanda, Deena, Gena, Irene, Kathleen, Margaret, and Reba.

disorder could be treated at all if it were genetic). In addition, three respondents made no mention of anything that could be interpreted as evidence of perceived medicalization, whether under a narrow or expanded definition (Jackie, T-BN; Liana, T-AN; and Emma, R-BN). It is possible these three would have endorsed the idea, if the question been posed. Here I briefly describe themes that mitigated medicalization by means of genetics.

Some respondents cast doubt on the idea that genetic causation would warrant more treatment. They specified that treatment was *less* likely to be pursued if eating disorders were thought to be genetic because the condition was permanent and the situation hopeless. As will be seen in the section on the various implications of genetics for agency and control, one possible response to genetics was precisely the opposite of help-seeking: giving up on the attempt to fight or cure the disorder because it was seen as a permanent part of the self. Yolanda imagined that some might say, “it’s part of who I am. So I’ll just accept it.” Yet, when I asked her what she might say to someone who drew that conclusion, she made a comparison to genetically influenced cancer.

I would say “If you got cancer, you wouldn’t try and – you wouldn’t just be like, “Oh. Well that sucks.” You would try and, like, go get chemotherapy and do what you could.” So, like, just because something’s genetic it’s still unhealthy. And you should still, like, if you can try and fix it. And there’s plenty of people that have got – that have just, like, gotten over bulimia. (Yolanda, T-BN)

If genetics prompted someone to give up, Yolanda would compare it to an uncontested disease that also involved genetics. Despite articulating a theme that ran counter to medicalization, Yolanda also compared it to a physical illness.

Only one respondent clearly stated that genetics made BN seem *less* like a disease, not more. The permanence of genetics made BN seem less like a disease and more like a part of the self to Wendy (T-BN): “I would have no hope of getting better. And I would give

up. *I would see it as a part of me instead of seeing it as a disease that can be fought.*” Her implicit model of disease seemed based on temporary infectious disease, caught from a source outside the body and expected to run its course and leave the body completely. Elsewhere in the interview she stated that genetics would make BN seem like something only doctors could treat, so she was counted among those who perceived greater medicalization. Wendy thus rejected the idea that genetics made BN more like a disease but did link genetics to medical intervention.

For a few respondents, medicalization depended on what the genes were “for”; some genetic theories would *not* imply that eating disorders were more like a disease. As noted at the beginning of this chapter, respondents often did not apply the theories they found most plausible when considering the effects of a media campaign and a hypothetical genetic test. Willa, quoted earlier (in the section “Eating disorders as ‘disease’, and people with them as ‘sick’”), thought that only genes “for” AN specifically would prompt her to think of it as a disease, whereas genes “for” perfectionistic personality would not.

Some respondents seemed concerned that a focus on causal origins was counter-productive because current treatment ought to be the focus, suggesting that genetics could distract from treatment. For example, Jackie’s (T-BN) initial reaction to the idea of genetic influence was “it doesn’t really matter either way. I mean people either have it or they don’t. So, it should be treated the same.” She used the words “illness” and “disease” to describe her BN, but also said that she struggled with self-blame and the perception others have that it is a choice. Although she stated that genetics would help others see that it as non-volitional, she did not say genetics would make it seem more like a disease (though she might have, if asked). It is possible that she was resisting this idea because it was so important for her to see

it as a non-choice that deserved treatment regardless of the cause; that genetics *should not* matter, even if they might to some people. This logic was similar to that of others who perceived medicalization.

To summarize, most respondents (74%) thought that genetics either increased the resemblance of eating disorders to other diseases, made treatment by healthcare professionals seem more logical, or both. Under an expanded definition that includes mentioning biologically based treatments, nearly all respondents (94%) thought geneticization would promote medicalization. As Joelle put it, “when people think of things that are genetically pre-disposed, they think heart disease and cancers. And, those are the kinds of things that you put on forms. And, that are talked about. And, that you discuss with doctors.” This constitutes strong evidence for assertions in Chapter 1 that geneticization of eating disorders would encourage or heighten their perceived medicalization; respondents thought it would change their or others’ perceptions in this direction. Although respondents may have objected to medicalization, or felt ambivalent or skeptical about some aspect (e.g., pills for AN), genetic ideas nevertheless elicited such ideas. To engage more deeply with the underlying logic of medicalization for eating disorders, I now turn to the perceptions of individual control and responsibility.

2. GENES VS. CHOICE: GENETICS AND PERCEPTIONS OF CONTROL AND RESPONSIBILITY FOR BEHAVIOR

In Chapter 1, I speculated about several possible implications of genetic ideas for perceptions of eating disorders (summarized in Table 1.2), in addition to the possibility that geneticization could heighten medicalization. Which of these implications were important to respondents, if any, and how? As several respondents articulated above in connection with

medicalization, genetics made the eating disorder more like a real, legitimate, valid disease entity that actually existed and less like something a bad or crazy person invented, or something for which she could be blamed. This broad theme is certainly related to medicalization; indeed, the anticipated “bright” and “dark” sides of medicalization included relevant positive (reductions in blame and stigma) and negative effects (“dislocation” of responsibility) (see Chapter 1). But I found that respondents spoke at greater length and with greater interest on the general theme of perceived control over eating disorder behavior than whether it seemed like a treatable disease. Their interest in perceived control and responsibility for eating disorder behavior went beyond medical concepts to include searching examinations of their own agency and responsibility for behavior that was alternately agentic, non-agentic and quasi-agentic (see Chapter 3).⁷

Many of the implications respondents identified were possible to organize using a two-by-two conceptual table organized around perceptions of control when genetics play a role. This table will be presented in the next section (Table 6.1). In this section I introduce themes that are important in order to understand the table, namely to establish the central idea that genetic influence implies reduced control over behavior; that people “can’t help it” to some degree. Many diverse implications flow from this general point, which the table will organize. In this section I aim to establish that the central idea elicited by the possibility of genetic causality is that a person is less able to control her eating disorder behavior. If it is genetic, it is less controllable.

⁷ I understand the reduction of perceived control conferred by genetics to motivate the perceived medicalization. Alternatively, medicalization could be at the root of the perceived reduction in control; if it is a disease then it must not be something I can control. Thinking about implications in terms of the broader category of control enables me to include alternatives to medicalization, which were often simultaneously held by respondents, including the idea that genetics implied that it was hopeless to treat the disorder, that genetics could seem like an excuse, and occasional comparisons to *non*-medical genetic explanations for homosexuality. Organizing my analysis at the more general level of control enabled me to make sense of diverse implications.

2.1. “Not something I chose”: Implications of genetics for perceptions of individual control and responsibility

Throughout the previous chapters I have discussed themes related to the broad category of control. In Chapter 3 I discussed respondents’ self-understandings of agency, lack of agency, and what I termed “quasi-agency”. I found that most respondents saw eating disorder behaviors as a complex product that could be controlled by the individual at different times or in different ways but could also be out of control. In Chapter 4 I described respondents’ reactions to the idea of “choice”, and presented several aspects of eating disorders relevant to control and responsibility: choosing to diet or purge initially, but not choosing to have it go out of control, choosing to refrain from a specific action at one moment, not being able to “choose” to stop, and feeling compelled to do it. Genes could “validate” these assertions: a genetic predisposition made it go out of control, caused a behavior to become obsessive. Genes could even contribute to the problem to which eating disorders was a response or a coping strategy; in Chapter 5, several theories of indirect genetic predisposition held genes responsible for causing an initial problem, to which the eating disorder was a response.

Respondents’ views should be seen as well-informed speculation about what people with eating disorders would experience if they found out about genetic influences. All questions were phrased in hypothetical terms. Answers encompass respondents’ ideas about their own reactions, those of other people with eating disorders, and a host of others (e.g., relatives, friends, general public, doctors). Because the implications and consequences often hinged on what others would think genetics implied and whether they would “buy” the idea of genetics reducing responsibility, these data represent respondents’ understanding of the

public's understanding of genetics.⁸ The socially shared meanings of genetics were inextricable from the imagined implications of genetics for respondents. Even though respondents were able to apply non-deterministic genetic reasoning to their own histories, others might not be as savvy and so the specter of deterministic genetic disorders was alive and well (also because of their interpretations of my questions about hypothetical genetic testing and media campaigns, as discussed in the introduction to this chapter).

In this section I flesh out the idea that genes implied less control, responsibility, and blame for people, and the related perception that if genes are involved it makes an eating disorder harder to overcome. Because I have already explored medicalized logics at length above, I spend less time on the latter, in the hope that it was adequately delineated there. Respondents thought the involvement of genes in eating disorders would be interpreted as a claim about the level of individual control over the eating disorder. Some did not think it ought to, but nevertheless perceived that it would. Genetics reinforced the idea that it's not a choice, and tended to encourage a redefinition of behavior from bad to sick. The person was not being "selfish" (Jackie), is not a "nut case" (Deena), is not "crazy" (Hannah, Nell, Irene), and her problems were not simply one's own "disordered thinking" (Barbara) because they were something more than "just you" (Karen).

Although some respondents argued that blame was not or ought not be apportioned and some hypothesized that genetics could *increase* blame (alternative viewpoints are addressed at the end of this section), all spoke about the implications of genetics for perceptions of individual control. Most respondents thought that explanations that involved genetics would reduce the perceived responsibility of the person with an eating disorder,

⁸ Thank you to Michael Lynch for this formulation; this is a study of the public understanding of science (PUS, an area of study in Science and Technology Studies) as well as a study of the public understanding of the public understanding of science (PUPUS: public understanding of the public understanding of science).

resulting in less self-blame and blame from others. I organized interview material related to perceptions of individual control and responsibility into diverse interrelated themes and summarize them in Figure 6.2. Some respondents expected skepticism about genetic absolution from responsibility but nevertheless expected the claim would be interpreted along those lines.

Figure 6.2 Themes related to genetics and perceptions of individual control and responsibility

- “A harder case”: Genetic disorder overwhelms individual control
- “I’ve got science to back me up here”: Defending against the presumption of choice
- “More sure that I wasn’t choosing to do it”: Less self-blame
- “There are people who have a valid reason. And I’m not one of them” (on not having a genetic predisposition when others do)
- “Yeah, right”: Public perception of genetics as an irresponsible excuse

“A harder case”: Genetic disorder overwhelms individual control. As alluded to in the section on medicalization, genetics made an eating disorder seem like something that an individual would need professional help to overcome. This need for help supports the general point of this section, that genes suggested less capacity for the individual to control her eating disorder behavior. In addition, the permanence of genetics suggested that the disorder would also be permanent, and there was nothing the individual could do about it.

[I]t might make it seem harder for me to get over it than, like, more than anything else.... Just because, like, if it seems like you’re battling a lot more. Like, I guess I can battle an addiction. And I guess I can battle these environmental factors. But, like, battling your genes sounds really hard (laughing). So I guess that’s the only thing it would really change. (Rebecca, T-BN)

I’d rather find out that I did not have them. Because, I think that would make it easier. Make me know it will be easier to get over. Instead of thinking “Oh. This is such a tremendous thing. I don’t think I can do it.” (Betty, T-AN)

Well yeah. It would make me feel like I had a, like, not as good a chance for recovery if it was in my genetic make-up... I mean if it’s, like, in my genes, then that’s something, like, that’s always going to be in my genes. Even if I get better for a little

while. It might be something that, like, is always like a tendency to go back to.
(Victoria, T-BN)

Genetics made it seem like something one would always be returning to, against one's own wishes.

I have this notion that it's harder to cure than – and, that may not be accurate. It probably is not accurate. But, that somehow this idea that it is genetic. You get it. Then, it somehow seems to me like it's going to be harder case. It's going to be harder to come out of it. (Lynn, R-AN)

If genetics made it harder to overcome, those few who had recovered without treatment looked back and felt more impressed at their ability to do so, including Lynn who credited feminism.

I: And, would it change anything about how you view the importance of feminism in your recovery if you found out genetics had anything to do with it?

R: No. Because, I mean if anything, it would strengthen the idea of feminism. The idea that that philosophy – that world view gave me a motivation to change. (Lynn, R-AN)

If I had it, I would be pretty impressed with myself for being able to stop without some sort of assistance or help or something like that. Because I feel like having it genetically you would – it would be really tough to stop yourself and have different images about yourself. (Carly, R-BN)

Genetics thus connoted greater difficulty for individuals in controlling their thoughts and behaviors. Although only some respondents brought this up, I interpret this as essential to the underlying logic that genetics implies less personal control, and hence less responsibility and blame.

“I've got science to back me up here”: *Defending against the presumption of choice.*

Genetics helped make the case to others that eating disorders were not choices. As demonstrated above, usually this meant they were more like diseases, but I focus here on the perceived effect of genetic explanations on blame from others. Eva and Joelle thought it

would be easier to talk about with other people because they would understand that it was not something they should be held responsible for.

I guess I might be more likely to bring it up. Just because I feel like that kind of places even more of the blame, like, off of you as a person. And, maybe just to kind of try to reinforce that, like, it's not something, like, I chose. And, like, there is a genetic component. And, like, I think that maybe would help some people see that, like, it's out of your control. (Eva, T-BN)

I feel like it would be an easier story to tell if you could start it off with "In my family genetically we have this problem."... maybe I would worry less about people being judgmental. Because, even though there's definitely still that same element of choice and accountability, just being able to attribute it to genetics definitely would hypothetically be anticipating probably less judgment there. (Joelle, R-BN)

Isabelle thought of an acquaintance who had expressed skepticism about her BN in the past, suggesting that it was merely a matter of willpower. For this kind of person, genetics might help convince him.

Like maybe a little bit less shame.... Like saying, like, to my friend who tells me that, like, it's just a matter of will power. "Hey. Here's this research. Like, look at this. Like, I've got, like, science to back me up here. Like, don't be so judgmental." I mean but again he's a really rare case. Like, there are not very many people I know like that. But to those, like, kind of skeptics in that sense I guess it [would] be validating to know that sort of thing. (Isabelle, T-BN)

Science could "back me up" that it was not a matter of willpower. Similarly, Liana (T-AN) felt it gave her "proof" that would be useful in explaining her situation to others; "I probably would bring it up and say 'Well, I was genetically pre-disposed.' I mean it kind of gives me, like, a proof that it wasn't, like, a decision type thing." Selena thought it would help her father and others understand that if she could stop doing it, she would.

I think it would really help. Because I think that people – like when my father found out I had this he said "Well just stop doing it." He said "Why would you do that to yourself?" I said "I don't know." I mean I really wish I did know. Because I don't know why. And it's gross. I mean why would you do that? And then you hide it. But I think that people would understand it a lot more. Because just like with any other disorder. People think "Well you did something to yourself. You did something that made you have it. Either you had a bad lifestyle. Risky lifestyle. Or you did

something that caused you to have it.” But I can’t think of anything I could have changed. And I’m a very responsible person. So I’m thinking “Well when people blame you for it. And they’re like, “Well don’t do it. Just don’t do it.” Well if I could not do it, why would I?” It’s gross. Messes up everything. I mean it’s like I don’t really feel that good. It causes me damage. I mean why would I do that to my body? Is it because I’m depressed? Is it because I have anxiety? I don’t know. I mean I’ve tried everything. (Selena, T-BN)

Similarly, genetics would bring “relief” to Petra because it would reinforce the idea that she was not to blame, in contrast to what others had repeatedly told her.

Well I think that it would make a lot of anorexics happy. Because we would at least feel like we weren’t totally responsible for being the bad person of the family. The black sheep of the family. Where we’ve caused this on our own. I think it made a lot of us feel relieved too to know that we’re not just totally crazy people. That it’s something that we really truly can’t help. Which is what we’ve been saying all along. People just didn’t want to believe us... I think it would change the way society thinks about people with anorexia. I know people I work with – they don’t – nobody understands. Nobody understands. And if they could post that up and say it was genetic, I think there’d be a lot more acceptance of people who have eating disorders. Whereas now we’re sort of shunned by it as sort of freaky.... So if it was posted up as being genetics, maybe they would understand “Well it’s not their fault.” No more than somebody who had autism or mental retardation or dyslexia or anything like that. It’s not their fault. Or ADD or ADHD. People tend to have a lot of patience with people that have ADHD. Because it’s something not right in their brain. And they can’t help it. Well if we could get it out that it’s genetic, maybe then people would understand that it’s not really our fault. (Petra, T-AN)

Gena likewise felt pressured to explain her behavior, and thought genetics might make it easier for others to understand. She compared eating disorder behavior to punching a wall and imagined how genetic ideas could help someone understand it.

... say if you punched a wall every day. People ask you “Why do you punch a wall every day?” “I feel bad. I’m punching a wall.” They’re like, “Stop it.” If you have a genetic pre-disposition to punch walls, then ... They might understand more why you punch a wall. And, be more open to hearing and kind of understanding and trying to help you. Instead of more of like, just like, “Stop it. Just stop it. It’s just simple. Go, type. Find something else to do.” (Gena, R-BN)

Genetics would help explain the behavior to others in a way that conveyed the difficulty in controlling it; genetic influences sounded like irresistible forces. Liana (T-AN) put it simply

when she speculated that her parents might stop blaming her, and conclude that “this wasn’t her choice. This was just part of her body.” As should be apparent in previous chapters and in the above material on medicalization, most respondents were concerned about how others judged and blamed them for their behavior. Genetics provided evidence to convince others it was not a choice.

“More sure that I wasn’t choosing to do it”: *Less self-blame*. Respondents theorized that genetic explanations would reduce self-blame as well as blame from others. As noted in Chapters 3 and 4, there was a lack of certainty about agency; some aspects of the eating disorder could seem like a choice, choice was more relevant at certain times, choices may have been made as a way of coping, etc. Genetics could provide reassuring evidence for themselves that they were not choosing to have an eating disorder.

Genetics would help defend people against others’ judgement and blame, thereby helping to convince themselves. The expectation of blame from others went along with respondents’ own uncertainty about the extent of their responsibility for their eating disorder behavior; their self-interpretations were affected by those of others. Gillian linked this directly to blame from other people, reasoning that if people tell you your behavior is intentional, you might start to blame yourself.

I think that people would start thinking that – they would stop doubting whether or not they chose it. They would stop doubting whether or not they’re just doing it for attention. Because, after a while if so many people tell you that you’re just – or, you hear so many times that people with anorexia are just seeking attention. You start to wonder if you are. I think that it would definitely alleviate a lot of those concerns.
(Gillian, T-AN)

Hannah illustrated the ambivalence of agency by performing it as two very different voices, one strong and adamant, the other weak and uncertain. By doing so, she conveys that she was

of two minds; sometimes she felt confident she was not choosing but at other times she had doubts.

...my family thought that ... a lot of the anorexia behaviors that I did were my choice. Which I alternated between being really angry about that. I mean (in a strong voice) "I'm not choosing to do this." And, feeling extremely guilty. And, being like, (in a soft insecure voice) "I am choosing to do this. Am I doing this? How come I'm such a terrible person?" I feel like if there were a gene, I would be more sure that I wasn't choosing to do it. And, that would be good probably. (Hannah, R-AN)

Hannah's ambivalence and self-doubt was linked to others' evaluations of her; genetics made her more sure that she was not "choosing" to have AN. This self-doubt was linked to others' evaluations of her.

Respondents observed themselves judging others, and applied the same judgements to themselves. Rebecca felt critical of a co-worker for smoking and being too "weak" to quit. She applied the same criticism to herself, but thought genetics might help her be less hard on herself.

...I'm really hard on myself. Like, "God. I'm such a weak person." Like, I don't know. Like, I worked with a woman who made really crappy money. But she would still buy cigarettes. And I was like, "Why don't you just quit smoking? Like, such a - money down the drain." But so, like, I guess I viewed her as, like, a weak person for not smoke - for not trying to quit or not quitting. And I think I view myself in the same way sometimes. Like, why can't I just stop? So it'd be nice to know that part of it was in my genes. But I mean it's still something I should definitely strive to get better. (Rebecca, T-BN)

Evaluations by others, and one's own evaluation of others, informed one's own self-evaluation as deserving blame or not.

Respondents also struggled with self-blame without mentioning others who blamed them. Joelle was uncomfortable about the idea that her own choice was involved. She noted that choice did play a role at the beginning of the eating disorder, and hoped that the continuation of the eating disorder was not conscious, nor a mere habit. Elsewhere in the

interview she had brought up the importance of being “accountable” about one’s eating disorder. Genetics would remove “accountability” because it was “some sort of larger force at work”, beyond the individual.

Accountability. I guess I don’t want to be wholly accountable.... Because, I definitely feel like – it’d be scary to think that – I don’t know. Because, I definitely can pinpoint, like, making the choice to maybe initiate that behavior. I’d hate to think that I was always consciously doing it or doing it out of habit. And, that no part of that was maybe – I don’t know. Some sort of larger force at work. I guess. Who knows how the brain and body really work? (Joelle, R-BN)

Carly had blamed herself for causing the BN which in turn caused problems with her competitive swimming; if genes were involved she would not have expected she could stop bingeing and purging whenever she wanted to.

I think I would feel better about myself. And I kind of would blame my poor performance in swimming and that time in my life on my genes. And yeah. I just wouldn’t feel as bad about it about doing it.... Because I would – not knowing that I had those genes I’d feel like I had more control over it. And I could have stopped whenever I wanted. I mean I had the choice of being able to stop whenever I wanted. But with the genes I was kind of – I know that I’m, like, pre-disposed to it. And I have less control over it. (Carly, R-BN)

Barbara and Irene spoke of the “relief” they and others would feel if AN or BN were thought of as genetically influenced.

I mean it might be a little bit of relief. Because I think even myself after I recovered I realized – I was kind of like, “How did I do that?” Like, “What was I thinking to do that to myself?” type thing.... But just criticizing yourself for, like, leading yourself down that path. (Barbara, R-AN)

Well, I think it would be very helpful to know that it’s genetic. Because, if it does give them some relief. Less guilt. If they feel less guilt. That’s part of the recovery. Feeling 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. (Irene, R-BN)

According to Irene, only a “freak” and a “failure” would choose such a “messed up thing”; knowing that genetics played a role would be a relief.

“There are people who have a valid reason. And I’m not one of them.” Some respondents speculated about what would happen if they took the test and found out they did *not* have a genetic predisposition. Theoretically, some people with AN or BN could have developed it without having the genetic predisposition that had been linked to it. In this scenario, respondents maintained the belief that genetic predisposition confers less blame on those who have it, but examined the logical corollary: that those whose case is not genetic are to blame. If eating disorders were understood to be genetic but one’s own case was not, it could seem even more like a choice by comparison. Most respondents (n=32) said that if they were to undergo a genetic test for predisposition in the future, they would prefer to find that they had the predisposition. (Eleven preferred to find out they did not have the predisposition and seven had no preference or could not choose.) Of those who preferred a positive result, several thought that not having the predisposition would suggest that choice played more of a role in their case than for others.

I don’t know. I mean I suppose, like, it would be nice ... to have the pre-disposition. Because you’d be able to say, like, “Oh. It’s not my fault.” And then to not have it. Then they would just kind of – if they had identified it, then it would just kind of be like, “Well there are people who have a valid reason. And I’m not one of them.” (laughing). (Thelma, T-BN)

My initial thought was “It would be better not to. Maybe I would be less likely to have problems in the future.” But, then I went back and thought “Well, what would that then mean for me having problems then? Would that mean that more blame should be placed on myself and my choices? Or, on environment?” And, I like to think that – I don’t know. There was some internal mechanism at work to some degree. Even if it was all chemical and having anxiety problems and that sort of thing. So, yeah. I think I would rather it be positive. (Joelle, R-BN)

... the initial thought is almost the exact opposite of the good one. Thinking that “Well, what if it’s not genetic for me?” And then, thinking that that means that it must be a choice for you. Despite whatever sort of experiences you’ve had. (Gillian,R-AN)

... I think I'd rather find out that I did have them. Because, if I didn't, then this is just something that's ALL stuff that I've done to myself. (Jackie, T-BN; capitalization reflects respondents' emphasis)

I think it would just be like a lose-lose situation. So I can't really say one or the other.... Just, like, not having a choice. And, like, knowing I was going to have an eating disorder or have a hard time not having an eating disorder. And then if I found out that I didn't have the genetic make-up, then it would be, like, "Well why do I have an eating disorder?"... It would just make me think ... That it was, like, all my fault. And this [BN] wasn't even something. (Victoria, T-BN)

R: It's funny. Because, I just took that information [about genetics] when I heard it and went "I have that genetic make-up." So, I just took it and said "I do." I had never thought that I might not have it.

I: Well, then it might be interesting to ask what – how would you react if you found out "Well, no." You did not have that high genetic risk.

R: I think that'd probably be a shock to me, too. Because, it has helped explain it to me why that happened.... I'd probably feel like, "Well, damn. It was more my fault than I wanted to acknowledge." After I found out it was genetics, I went "Ah. This helps explain it more." (Fran,R-BN)

If genes play a role in others' eating disorders but not their own, respondents would interpret that information to mean "more blame should be placed on myself", "it must be a choice", "that's ALL stuff I've done to myself", "all my fault", and "more my fault"; others have a "valid reason" but not them. I found this interesting because the availability of a "valid reason" for some people with eating disorders could increase suspicion of those without one, even more than if nobody had a "valid reason". The guilt-reducing genetic explanation would actually magnify guilt for people who arrived at their eating disorder by non-genetic means, even if their experience of the eating disorder was the same. It is as though once genetics enter the picture, there are no other plausible explanations for a loss of control.

"Yeah, right": Public perception of genetics as an irresponsible excuse. People who saw eating disorders as under individual control and responsibility might reject the claim about genetics. Indeed, talking about genetics might make the person with an eating disorder look like she was avoiding responsibility and making excuses. Several respondents

examined the possibility that the genetic excuse might be seen skeptically and cause a “backlash” against people with eating disorders for trying to blame their problems on genetics.

...the ideal family would be like, “Okay. How can we help you?” And, look for the best, like, geneticist, scientist, or whatever. To figure out what’s the best treatment. Now, my family. They probably wouldn’t care.... It would be like, “Okay. You’re still making excuses.” (Amy, T-AN)

I still think there would be some people that would be skeptical. “Oh, yeah. Blame it on something else.” Rather than the person taking responsibility for it. (Mary, T-AN)

... I think it would be a tough sell. Like, I think people would be really skeptical. They’d be like, “Whatever.” I mean I think so many people still do think it’s a choice. And they’d be like, “Oh.” People would be making comparisons like, “Oh. This is like saying that, like, because it’s genetic reasons that people become crack addicts or whatever.” And I just think people would be like, “Yeah. Right.” (Isabelle, T-BN)

Gillian thought that genetic explanations for complex conditions might have lost their effectiveness because they were perceived to be over-used; “some people would be like, ‘Oh. They’ll say anything’s genetic nowadays.’...And, they would discount it.” Claims of genetic contributions to alcoholism and obesity had been met with skepticism and even “backlash”, making some respondents cautious about such claims for eating disorders.

Well, the way I’m thinking about it is when they came out saying “Alcoholism is a disease. Alcoholism is a disease.” How did that work? Did that help? Well, maybe. I think some people kind of grudgingly said “Oh. He’s drunk because he has a disease.” But, there was a huge backlash. “You shouldn’t coddle those people. They don’t have a disease.” ... I mean I think it worked both ways. There was definitely backlash. “Those people are just weak-willed and bad.” (Alyce, T-AN)

R: I think there’d be, like, a lot of skepticism. But like also – I mean I guess skepticism is the best way to say it. Because I mean they have said that thing about, like, obesity. Like you can be genetically pre-disposed to obesity. I think people kind of were like, “Yeah. Well whatever.” (shrugging, half-laughing).

I: Can you say more about what you’ve observed with obesity?

R: Yeah. Just like – I don’t know, like, personally. Like, maybe you have a heavier build than people. But if you eat five hamburgers, you’re still going to be fat. Like, maybe you don’t have, like, the tall slender build. But I tend to think you’re stuffing your face more than you should anyway. Like, that doesn’t cause you to be three

hundred pounds. So I think people would say that about bulimia. Like, I don't know. I would say that about it if I heard that about bulimia. Like, "Yeah. Maybe I'm more likely to have bulimia. But that's not really why I'm puking up my food." (Rebecca, T-BN)

Genetics for obesity could seem like an irresponsible excuse for people who "eat five hamburgers"; so would genetics for BN. According to Irene, such explanations could seem particularly offensive to parents, who had already endured the hardship of their child's eating disorder and would resist the idea that they had passed along genes that contributed to the problem.

I: Imagine there were a media campaign to publicize genetic causes of bulimia to the general public. What do you think the effect of that would be?

R: (Gasp followed by laughter.) I could see mothers being like, "Yeah. Yeah. Yeah. You're going to blame it on me." All that stuff. Like, "It's not genetic. I'm not going to take that, too. We have enough problems around your bulimia..." (Irene, R-BN)

Those respondents who anticipated skepticism about genetics still assumed that genetics would be taken as a claim of reduced control and responsibility, thereby supporting the general point that genetics would be interpreted in this way.

A few respondents articulated ideas in contrast with the prevailing expectation that genetics would reduce perceived responsibility. If someone did not feel blamed to begin with, genetic relief from blame was not welcome, either because it seemed unimportant and irrelevant (several respondents) and could actually increase blame (for one respondent).

Isabelle pointed out that others blame people with eating disorders out of ignorance, and that for them, providing a genetic explanation would be beneficial to reduce blame.

... they may be likely to be "Oh. Well we can kind of, like, forgive this a little bit more now that we know that it has this genetic component." It might make it a little bit more acceptable to them. But as far as people who have, like, a really good understanding of the illness and certainly myself, like, it wouldn't really make any difference at all. (Isabelle, T-BN)

Like Isabelle, some who felt they did not need genetic relief from blame recognized that this was the meaning of genetics likely to be understood by most others, whether they accepted it as true or not. For one respondent, Wendy, genetics could actually confer more blame by locating the problem in the individual with an eating disorder rather than an external source. Wendy understood her eating disorder to be a response to extremely restrictive and controlling parents, who did not allow her to express her emotions and frequently punished her unfairly. She understood the eating disorder as a way of coping with this harmful environment, and recovery as a process of re-learning how to handle emotions and value herself. She did not see her BN as her fault.

I: And some have said that talking about bulimia as genetic helps make it less like the person's fault. What do you think about that?

R: I think of it opposite.

I: Tell me more about that.

R: I just – well I see it both ways. Like, I can see it from both ways. I could see how it would be less your fault. Because it would be less your fault if you were one of the people that are like, “Why don't you just eat? It's your choice.” Then genetically it would make it less your fault. But if you're a person like me who's like, “It's not your fault anyway. It's really complex. It's caused by the environment.” Then it being genetic would make it more your fault. Because it would make it more individualized and less due to the environmental factors. (Wendy, T-BN)

As Wendy said, if the person is already being blamed, genetics may lessen the blame because they are not under her control. But if the person were not being blamed, genetics could increase the blame by locating the eating disorder in her rather than elsewhere. Wendy was the only one to articulate this view, though others shared a similar view that genetics would not or should not matter to anybody who understood eating disorders; individuals with eating disorders should not be blamed in the first place, so genetics ought not make a difference.

In summary, the vast majority of respondents perceived genetics to imply a reduction of individual control and responsibility for behavior. I found this general notion expressed in a variety of ways, beginning with the idea that a genetic eating disorder would be harder to recover from (“a harder case”), and less compatible with individual control, responsibility, blame and related themes. Respondents focused on the reduction of anticipated blame from others, changes in how one might perceive one’s own control and responsibility, expectation of more blame for those who do not have a genetic predisposition, and skepticism about genetic explanations as “excuses” to evade responsibility for behavior. Whether genetic influence was a good thing or bad thing depended on whether it was seen as a valid or invalid excuse, and whether it was accurate to describe the case of a specific individual.

2.2 *Now what? Implications for agency and future action*

Although respondents generally welcomed a decrease in blame and self-blame, they were concerned about the implications for agency related to eating disorders. What kind of agency did they have, if genetics were responsible for the eating disorder? If genetics made them unable to control the eating disorder in the past, what did this mean for present and future action? Does removing blame also remove some kinds of power or agency? I will explore other logics of action that have only been hinted at thus far, among them medicalized agency (e.g., treatment-seeking) and helplessness.

I began this chapter by showing that most respondents thought the geneticization of eating disorders could make them seem more medical (real diseases, calling for medical treatment). I then stepped back to consider the broader theme of genetic implications for individual control and responsibility, because all respondents spoke about it, often at length, and frequently without explicit reference to medical concepts or terminology. Now I build

on the idea of control as the basis for a classification of the diverse implications identified by respondents, focusing on implications for agency and future action (Table 6.1.) I define agency loosely, as before in Chapter 3, to mean the individual exerting control, making decisions, choosing, or taking action, as opposed to being the passive receiver or product of other forces. I use the word agency interchangeably with control, choice, and volition, to convey the capacity for voluntary action (Marshall 1998).

To introduce the themes of the table, I focus for the moment on Gena, whose repeated use of the phrase “can’t help it” revealed the diverse meanings this claim can have for agency and action. The inability to control could be both positive and a negative because it removes blame but also power over one’s own behavior, or the perception of that power. Gena illustrated how the phrase “can’t help it” could be used in these different ways. Here she suggested that people might “give up the fight”.

I think obviously it’s going to be different for everybody. I think the worst thing that could happen would be that people would give up the fight. That they would say “This is how I’m always going to be. This is the way my body is. And, this is just kind of the way it’s going to happen. And, I can’t help it.” (Gena, R-BN)

Genetic explanations might make people think “I can’t help it”, lose hope and stop trying to overcome the disorder. The logic of action here would be fatalist: why try at all if it is impossible. However, if there were things a person could do, saying “I can’t help it” could become an excuse that people “use” to avoid hard work.

I think that the thought pattern [is] something you might not be able to help. However, the behavior choices you can learn to help. And, I think that the negative outlook would say that “I can’t help it. So, I’m going to keep throwing up. And, I’m going to keep doing this excessive behavior or this extreme restrictive behavior. Because, I can’t help it. And, I’ll use that as an excuse.” (Gena)

The logic of action implied by genetics as an “excuse” is that the person can control some aspect of the disorder but they are “using” genetics to avoid that responsibility. Gena also

envisioned that “I can’t help it” could be a way to counteract expectations that the person “change it today”; it would inspire compassion and refigure the eating disorder as something for which help and patience, rather than blame, was appropriate. This meaning of “I can’t help it” supports a logic of action that seeks help for one’s disorder.

If you want to go extreme positive. It would help people understand... that at least the thought pattern is something you really can’t help. And, it won’t be this extreme [expectation] like, “Well, this is something you can just change. Change it today.” Like, some people, like, that I’ve talked to. They’re like, “Sorry. Like, I’m telling you you’re beautiful. So, what’s the problem?” And, if there was this media campaign pushing the idea that there was more of a genetic link. I’m like, “This is something genetic. I can’t help it. Please understand that it’s not something that I’m doing on purpose. Help me try to correct my behaviors. But, as far as the thought, know that this is something that I can’t do anything about.” (Gena)

Gena is careful to separate which components of the eating disorder are legitimately her responsibility and which are not. She deemed genetic relief from responsibility negative if it wrongly excused people from legitimate responsibility, or if it led to hopelessness about their condition. Genetic relief from responsibility was positive if it encouraged a view of the person with an eating disorder as deserving of help.

I found that many of the perceived negative and positive implications of genetics for people with eating disorders were possible to organize in the conceptual table below.

Respondents’ concerns or appreciation for the idea of genetic causality depended on (1) whether eating behavior actually could not be controlled by an individual in reality and (2) whether a person believed it could not be controlled. The columns correspond to the first; the rows to the second. Within the cells of the table there are positive and negative implications depending perhaps on whether there is an alternative entity (i.e., a healthcare provider) who can control it, but I do not use this dimension to organize the table.

Table 6.1. Implications for direct control over behavior given a genetic influence

	If eating behavior CANNOT be controlled	If eating behavior CAN be controlled
And a person believes it CANNOT be controlled	Individual correctly believes and behaves as though <i>she cannot control her behavior</i> 1. MEDICAL CONTROL 2. PARADOXICALLY-IMPROVED INDIVIDUAL CONTROL 3. ADJUSTING TO HELPLESSNESS	Individual wrongly believes and behaves as though she has <i>less control than she actually does</i> 4. LOSING CONTROL THROUGH OVERVALUATION OF GENETIC POWER 5. USING GENETICS AS AN EXCUSE TO ABANDON CONTROL
And a person believes it CAN be controlled	Individual wrongly believes and behaves as though she has <i>more control than she actually does.</i> BLAMED UNJUSTLY FOR LACK OF CONTROL	Individual correctly believes and behaves as though <i>she can control her behavior.</i> USING SELF-CONTROL TO KICK THE HABIT

I present results for the types of agency listed in the first row of the table (numbered 1-5); the second row is implied by the first and will become clear by contrast. In the top left cell, corresponding to an individual correctly believing and behaving as though she cannot control her behavior, I placed medical control, paradoxically increased individual control, and adjusting to helplessness. With (1) *medical control*, the individual recognizes a true inability to control her behavior and seeks medical help. With (2) *paradoxically-improved individual control*, the person recognizes a true inability to control her behavior but finds that this belief paradoxically enables her to better control the behavior. This individual control could take place outside of treatment but was usually talked about in connection to treatment; less self-blame enabled more ability to work on the problem in treatment. (3) *Adjustment to helplessness* referred to an individual recognizing a true inability to control her behavior and

adjusting to this idea by giving up on medical treatment and/or individual effort because there was no hope of recovery.

Implications in the top *right* cell differ from the left because they assume that genetics do not actually remove control to the extent believed by the agent. The respondent describing such an implication is skeptical about the actual power of genes in this regard, and assesses the implications of an agent's belief in that power with the background assumption that the power has been overvalued. If the individual actually can control her behavior, but believes or behaves as though she cannot, she might abandon efforts she could have made toward controlling the eating disorder, thereby (4) *losing control through an overvaluation of genetic power*; a self-fulfilling prophecy. Such a person genuinely believes herself to be helpless, as with #3, but the difference is that she is not helpless in actuality; genes don't really have that kind of power. This wrong belief discourages her from action that might have helped. Using (5) *genetics as an excuse* is similar to #4 but involves a professed belief about helplessness in the face of genes, a belief that may not be genuine. Those who heard about genes might use them as an excuse to avoid working on a difficult issue; behaving as though genetics make her helpless, but she is not in reality. This table captures variation presented by respondents and my attempt to make theoretical sense of it. The next five sections will illustrate these consequences of beliefs about genetics and control (all from top row of Table 6.1).

2.2.1. *Medical control (1)*

In this form of agency, the behavior is redefined as a disease, the person is sick and cannot be held responsible, therefore the logical action is to seek help from qualified professionals. Even if a person lacks *direct* control over her behavior, she may gain *indirect*

control by seeking professional help. Similarly, within a treatment context or 12-step group, patients might be held responsible for how they respond to their disease and behavior, as with the responsibility to “challenge” thoughts in CBT, to earn privileges in an inpatient setting and to continue coming to Overeaters Anonymous meetings. Agency is re-focused toward a different goal – seeking professional help, challenging self-destructive thoughts, avoiding situational triggers – because the agent is no longer deemed capable of direct control over behavior. Results presented earlier to describe medicalization are relevant to describe this form of agency or action, but below I present additional quotations to illustrate the conceptual reworking of agency and responsibility, as well as the new forms of treatment respondents imagined.

Genetics removed responsibility for eating disorders, which could encourage treatment-seeking. If the person was no longer responsible for the behavior, she could seek treatment without feeling ashamed or wanting to hide her behavior.

...there was a lot of blame. And it was an impediment to me getting treatment. Because I felt like, “I should be able to overcome this. I should be able to fix this. I know what I’m doing wrong. I just need to stop doing it.” So I guess that if there were [a genetic causal factor] – and I don’t think that there is one. I guess I should preface it. But if there were. ... I think it would take away a lot of the blame and maybe the shame and reach more people. (Sarah, R-AN)

Well it could be a positive thing. I think that’s happened somewhat with alcoholism. That people get a message that this is part of someone’s genetic make-up. They always – they’re more pre-disposed to that. And I think that’s – I think that that’s helped in terms of people who struggle with alcoholism. Maybe it’s lessened their guilt or something. Or helped them feel less guilt. And hopefully they would feel more open to solutions or to alternatives. And maybe that would be the same for someone struggling with an eating disorder. It would say “Oh. Maybe this isn’t all my fault. So let me lighten up a little bit and see if I can get some help or something. And not be so secretive.” It might help with secrecy. (Melanie, R-BN)

Respondents who were not to blame for the disorder would be more likely to seek help. If they saw themselves as agents who could and should control the disorder, they would not

take action to seek help because it was their responsibility and it would expose them to judgement from others. But if they saw their agency to reside in the seeking of help, rather than the enacting of eating disorder behaviors, it would make sense to take action by seeking professional help. Indeed, like Selena's friend who sought innovative treatment for cancer, others might even be proud of them for doing so.

Several respondents envisioned new forms of medical control as a result of genetic discoveries, including pills, injections, and even gene therapy. Several disparaged the idea of medication for AN or BN as implausible "magic potions" and "magic pills", but others appeared sincerely to hope they would one day be available. Whether the treatment was biologically based or not, the concept of medical control offered a role – the sick role – for respondents, whose agency could be exerted by seeking treatment, but not by directly controlling the behaviors themselves. These forms of treatment were occasionally rejected for implying too passive a role for the "patient", as Yvette had attested when she imagined eating disorder patients becoming "just passive while letting the doctors drug them" (presented earlier in section 1.2).

2.2.2. *Paradoxically-improved individual control (2)*

Another form of agency respondents envisioned involved *improved* individual control over behaviors, despite the importance of genetics for those behaviors. I have termed this "paradoxical" because conceiving of a behavior as out of one's control seemed to confer greater control, yet this was not due to a relocation of control as with medicalized forms of agency, described just above. Several respondents thought their agency to control their behaviors would be enhanced by thinking of them as genetically influenced. The removal of

guilt and blame reduced stress and bad feelings, and eliminated the need to prove the validity of one's eating disorder, making it less necessary to engage in the behaviors.

Less guilt and blame mean less reason to turn to eating disorder behaviors. Several respondents said that the removal of blame would reduce stressors and bad feelings that made the behaviors worse. Feeling guilty about the disorder gave Paula “even more reason to” engage in her eating disorder.

I think that, like, I've talked about this for the societal view of it and the sort of stigma that goes along. I think the misconception that it is a choice. And sort of even a stubborn choice. Of children and women choosing to do this horrible, hurtful thing to themselves and their families. It's a little difficult not to internalize some of that and feel that you are at fault for not being able to stop. Or stop earlier. Or not have adopted behaviors in the first place. And of course then it's just a catch twenty-two. Because those – that just gets bundled into the bad feelings you already have. Like, sort of after my parents found out that I had the eating disorder. Then it was like, “Oh my God. Now I'm really – I'm disrupting the family. And they're all unhappy. And so I have even more reason to.” So I mean I think it might help in those cases for someone to hear. “Okay. This is not your fault that this is happening.” (Paula, R-BN)

Helen and Tammy also thought that self-blame contributed to the eating disorder.

I: Some have said that talking about anorexia as genetic helps make it less like that person's fault.

R: I can see that.

I: What do you think about that?

R: I would probably agree with that. Because if you're obese, it's your fault. If you're skinny, it's your fault. If you're obese, you eat too much. If you're skinny, you just don't eat enough. You got to eat these kinds of foods. So our society is always kind of doing that. If you don't make enough money, obviously it's your fault. Not the fact that you have to support other siblings. Or if you don't have the money to do the things that you want. So our society I think as a whole is so quick with the fault. And we need something to blame sometimes to have a better feeling for ourselves. 'It's not really my fault.' But if your perception of yourself is very, very, very, very negative, I think there could be some healing with knowing that some of your problem was by design. (Tammy, R-AN)

I think it would help a lot of people. I think it would just strengthen their recovery. And, realize – it would take – it would help them more. Because, they would not blame themselves so much. I think a lot of people with eating disorders have a tendency to blame themselves. And, I think there would be a lot less of that if they saw in the media this actually might have genetic components.... if you don't blame

yourself, then you're not angry at yourself. It lessens the degree of just that discontent you feel towards yourself. It frees you to then question "Okay. Well, where did this stem from? It's not my fault." It takes away that blame. And so, I think people are – they're encouraged to think a little more outside of what can be the other reasons for my eating disorder. And, just help them a little more on their path to recovery... could have a lot of effects. I mean if it's really a genetic – if there is a genetic component, then I think it just liberates them a little more. They might be at more ease with themselves. And, maybe even it might have the effect of them being able to connect with family and friends more. So, that's how I see it. (Helen, R-AN)

If the eating disorder were a way of coping with stress, guilt and blame from others about that method of coping were a secondary source of stress. Genetics could reduce that additional source.

I think I would have a more positive sense of recovery. Because again it would relieve a lot of my guilt. Which would relieve me. Take a lot of stressors off of me. It would make me less frustrated maybe. Because I would know that it's not really my fault. Whereas I still feel like it's my fault no matter who tells me it's not my fault. I just think it would – it's just like somebody pre-disposed to breast cancer. They didn't ask for it. It's not their fault. I think it would make people more compassionate toward me rather than frustrated with me. And that would give me a lot more support. And the more support I had, the easier it would be to recover. (Petra, T-AN)

Relationships with others could be improved if it was clear that the person was not to blame, thereby relieving some of the pressure of the eating disorder. Claire thought that the idea of genetics could inspire her toward working on recovery. The type of work she envisioned was not simply going to a medical provider to passively receive treatment, but the kind of treatment (or in her case, Overeaters Anonymous) that required individual effort, of the kind that might (paradoxically) suggest that the person did have some control over the behavior.

R: It's comforting because it helps to support that 'this isn't all my fault' feeling.... It's like people with obesity can work on themselves and put effort forth. And, know at the same time and gain inspiration at the same time from knowing that part of it for a lot of people is genetic. And, not a question of will power or inability to do certain things like some people say it is. Yeah. It's just thinking of it again as not all the person's fault.

I: And, you said with obesity that people can still work on it.

R: Yeah. It's not like it should give you an excuse for like, "Oh. It runs in my family. What are you going to do?" But, like, you still got to work on it. Because, it's still

your disease to handle and get over. But, you can draw inspiration from thinking like, “But, this isn’t all my fault.”... Inspiration to get well. (Claire, T-AN)

Another reason why thinking about the eating disorder in terms of genetics could paradoxically enhance control was that it would let the person ease up on trying to prove that the disorder was real. Only one respondent articulated this view, but it seems conceptually important. Genetics would provide validation and legitimacy, as discussed earlier, allowing the individual to stop trying to prove that it was real by becoming dangerously thin.

R: ... Because, then they feel more validated in what they’re feeling. And, it’s not – it’s almost not as competitive anymore. Like what I was saying about the DSM-4 criteria. Feeling like you had to fit the anorexia criteria. If those criteria weren’t there, it wouldn’t be so much of a rat race to see who could be the sickest. (Both laugh)

I: Is it a rat race?

R: Well, I know that when I was in in-patient, it seemed like a rat race. [The day program] not so much. But, in-patient everyone was trying to eat the littlest. Take the longest. Cut the food in the smallest pieces. And, it was just frustrating and triggering for everybody. And, I think the most progress definitely gets made in [the day program]... I think that people would just realize that it’s not something that they thought “Oh. Well, I’m just going to be anorexic now.” That there were other things contributing to that. And, that if it’s genetic, then that girl who’s sitting next to you cutting her food up into little bits has a genetic cause, too. And, you have a genetic cause. You all have genetic causes. So, you don’t have to battle for who has the worst genetic cause... I mean it’s almost like an equalizing factor. Like, yes, it will still be the competition in terms of who’s thinnest and who eats the least. That competition is not going to go away in the atmosphere where you’ve got more than one anorexic in a room. But, I think that it will be an equalizing factor... Equalizing in that you realize that you all have this one thing in common beyond the fact that you have the eating disorder. It’s, like, that you can’t start thinking “Oh. Well, that girl has a real eating disorder. And, mine’s fake. Because, I’m not as thin as she is.” You all have real eating disorders. Because, it’s genetically caused. It’s not that you’re making yours up and she’s not because she’s thinner than you. (Gillian, T-AN)

Gillian postulated, based perhaps in part on her experience of a healthcare worker telling her she did not look thin enough to have AN, that extreme thinness indicated a more valid and real problem and that it was desirable on the inpatient unit to have a real rather than a fake problem. In a context where AN and BN are contested illnesses, visible life-threatening emaciation provided proof of their reality. Genetics could also provide such proof, in a way

that was less harmful to a person's health. Gillian's eating disorder was apparently exacerbated by a need to prove that it was serious; if it did not have the reputation as a "shallow disorder" (her words), she would not be driven to prove otherwise. Thus, a reconceptualization of her agency as genetic paradoxically would improve her ability to exert her agency, because an important motivation for the behavior would be removed.

2.2.3. *Adjusting to helplessness (3)*

If genes meant a person "can't help it", she might lose faith in any form of effective action.

The extended quotes from Gena that introduced this sub-section on agency and action pointed to this possibility. Respondents offered fatalistic hopelessness as a possible reaction to genetics in eating disorders; if it were genetic, there was little reason to exert agency in connection to the eating disorder at all.

Well because it's sort of, like, I imagine this media campaign goes out. I don't know. Maybe also on the part of some young girls they just might think "Well." If they are already actively bulimic, then "Shrug my shoulders. I can't do anything to change this. Because this is again a pre-determined thing." (Paula, R-BN)

Genetics would make the eating disorder seem permanent; no recovery was possible and the behaviors would never stop.

R: ... just that you maybe wouldn't ever be able to fully recover... I mean maybe if, like, I had a genetic pre-disposition, just kind of there'd just be something that I would never be able to, like, get out of my head completely... it's always something that I'd, like, constantly think about the rest of my life.

I: You'd be thinking about having the genetic pre-disposition?

R: Or, just like the other thoughts with bulimia. Like, about, like, body image and, like, weight concerns. And, I don't know. I guess I feel like then if I did relapse, it would – I would maybe just kind of attribute that to genetics. And, maybe it would make it even harder to, like, get back on track. Because, I'd just be like, "Oh. Just another thing. Like, I don't know if I'll ever be able to, like, stop." Type of deal... I guess that was just kind of one example of maybe something that maybe you couldn't change [thoughts about body image, weight concerns]. I guess that's kind of one of those things that I feel like is going to be the hardest to overcome. And, that's one of the things I feel like will stay with me for the whole – like definitely the longest. Probably the rest of my life anyways. But, I just feel like it kind of maybe, like,

removes some hope that, like, you can get rid of all the symptoms and get, like, completely better. That, like, if you do have a genetic susceptibility that like – because, there’s some kind of thing, like, engrained. Something engrained in your mind that, like, makes you think those thoughts. Or, makes you think in a certain way that you’ll never be able to change. (Eva, T-BN)

Although Eva expects to continue working on it, genetics might make her give up hope that the thoughts will stay with her “for the rest of my life.” Some compared it to genetic diseases about which nothing could be done.

R: Well, good things would be like I said. Feeling a little less crazy. And, like it wasn’t as much my fault. And, not the fault of the environment as much. Although it is the environment. But, it would also make me feel a little helpless. And, vulnerable.

I: Any more about that?

R: Well, just like people who everybody in their family has had a certain disease. Or, they’re pre-disposed to something. It makes you just feel like out of control a little bit. Like something bad. You’re not healthy. And, you can’t do anything about it. So, that’s not – that wouldn’t be a good feeling. (Beth, R-AN)

... makes me think of the Huntington’s chorea. People have wanted to know so that they could plan their life. And this person who is in her early forties I think in New York went against her mother’s and family’s wishes and had this genetic testing done. She has Huntington’s chorea. She has that strong gene. And it’s a worse type than her mother has. So instead of helping her live her life to the fullest she’s developed, like, depression and this kind of thing. But anorexia I think can be avoided with the right recipe. So who is it that would be successful in taking the right approach to anorexia? When you think of the reality TV shows like Intervention. With some of those folks there’s, like, no help for them. There’s no hope for them. I might as well give up. I’m genetically pre-disposed. Because they’re not going to hear. It’s like obesity. We don’t hear those other factors. We’re going to hear “This is what I’ve got.” So it depends on where you fall in the spectrum of the disease process I think as to what your reaction would be. Would you live a healthier lifestyle? Or would you just get depressed and say “I’m damned anyway.” I think that you would see both extremes. I think that you would. (Tammy, R-AN)

Lynn thought that even if some cases were significantly influenced by genetics, it might be better not to publicize this fact, because it could be applied too broadly. She thought people tended to be “helpless” when confronted by genes. This might be appropriate for people with “super serious” genetically-influenced eating disorders, but not for others, whom she speculated would constitute the majority.

Well, if it really is genetic, it is genetic. But, if we don't know, then I wouldn't want to see it publicized. Or, wouldn't want there to be campaigns about it being genetic. Or, if it's genetic for a very small number of people but not the majority of people. Because of this idea that in the face of our genes. Sometimes people in the face of their genes think they're helpless. And, in some ways too once you say it's genetic, somehow that makes it really more serious. And, it's not super serious for everyone. It's not going to rob you of years of your life or rob you of your life for most women. I think it's something that a lot of women come out of it. (Lynn, R-AN)

Lynn suggests that some people might wrongly believe it was genetic and interpret themselves to be powerless when really they might have recovered. This idea introduces themes for the next two types of agency I examine, in which genetics are *wrongly* believed to exert influence over behavior, leading to negative consequences.

2.2.4. *Losing control through overvaluation of genetic power (4)*

Believing that genes were powerful forces in controlling one's own behavior could have its own effects, regardless of their actual power. In this sub-section and the next, I focus on the top right cell of Table 6.1, implications for action and agency that reflect an overvaluation of genetic power: believing that control was diminished when this was not the case. For this concern to make sense, there had to be skepticism by the describer about the actual power of genetics to reduce individual control, even if the excuse were accepted by others (i.e., the right column). Respondents were concerned that beliefs about the power of genetics could discourage people from trying to exert what control they could, leading to more eating disorder behavior, according to respondents. This could be an honest belief (this sub-section) or a dishonest excuse (next sub-section), but either way the result was more eating disorder behavior.

Genetics threatened to redefine agentic behavior as non-agentic, thereby changing beliefs about what could be accomplished, according to respondents. If eating disorder behavior is an uncertain mix of volition and non-volition, and genetics strengthens the sense

that it is non-volitional, it could change expectations about what is possible for individuals to do. If they did not expect to be able to control some aspect of the eating disorder that they probably could if they tried, they might not attempt to do so. If others no longer expected them to be able to control it, and genetics became an acceptable “excuse,” that would also lead to more eating disorder behavior (examined in the next sub-section).

The idea that genetics played an important role in one’s eating disorder could make eating disorder behaviors seem more non-agentic than before. Some respondents expressed concern the respondents would forego control over things they could have controlled successfully. I introduce these ideas by focusing on one respondent in particular, Fran, because she expressed this idea most vividly. Other respondents shared similar views to some extent and are presented afterwards. The sense that the behavior is re-defined as less agentic was also true for the three sub-sections above, on medical control, paradoxically-enhanced individual control, and helplessness, but here and in the next sub-section this redefinition is deemed by respondents to be unwarranted; in reality genes do not remove as much control as suggested. These respondents were concerned that genetics could mistakenly diminish agency.

Looking back on her recovery from BN, Fran said that if she had known genetics were important, “It would have been harder for me to have stopped it. There’s not a doubt in my mind. Because, I would have locked in on that thought. That I can’t stop this. I can’t prevent it. This isn’t me doing it. This is genetics.” (Fran) Genetics would make the behavior seem determined, such that efforts to stop or prevent it are misplaced because she is not the agent: “this isn’t me doing it. It’s genetics.” However, Fran said she had recovered without treatment by viewing her BN as a bad habit, like smoking, which she had already quit “cold

turkey”. She had spontaneously mentioned genetics while talking about her “decision” to stop bingeing and purging, before I had introduced the topic.

[I] just decided “I’m not going to do this anymore.” Had I known as much back then as I do now. That it’s genetics.... But, had I known all that playing against me, it would probably have been more difficult to have stopped the behavior.... Because, if it’s a habit, I’m extremely strong. Strong willed. Stubborn. My husband would tell you. Because, I’m mulish. Stubborn. And, if you tell me I can’t do something like breaking a habit, I’ll prove you wrong. So, I was able to stop that. (Fran)

Fran understood herself to have recovered by thinking of BN as a habit, and said she would not have attempted if she knew she had genetics “playing against me.” I asked her to elaborate on this idea later during our discussion of genetics, and she compared stopping BN to avoiding weight gain after surgery.

If I had thought – if I’d known that it could be from genetics, in the past I would have probably said “Well, I can’t change.” But, because I knew at the time that it was a habit, I could break a habit. Damn it. I’ll show you. I’ll break a habit. But, if it’s genetics, what are you fighting against? Something that you can’t change? Or, it’s kind of like when they tell you after a surgery. “Well, you’re going to gain weight.” Okay. That explains why I am [gaining weight]. So, it’s a little easier to swallow. But, it also is a little bit of an excuse, too. It makes it a little easier to accept it. So, you keep putting on a few pounds. Whereas, like, I remember when I had a hysterectomy when I was forty-one. And, somebody said “Well, of course you’re going to gain weight after a hysterectomy.” And then, I read “No. Not necessarily. You can if you don’t do what you need to do to prevent it.” Okay. So, if I gain weight, it’s my own fault now. It’s not the surgery’s fault. So, I nipped it in the bud and got it back under control. But, if you give me a good excuse for why something’s happened, I can fall back on it easily. Like somebody with a thyroid problem. Well, you can’t help it. You’re going to gain weight. But, then when you give them the meds. And, explain to them “Okay. This will help you.” But, then they don’t have – it’s not giving a total green light. It’s saying “Hey. We can pull some of this back.” (Fran)

Fran theorized that in order to achieve something difficult, a person has to believe that it were possible or she would not try. According to Fran, if people are told after surgery to expect weight gain, they will not try to avoid it as much as someone who was told that she could “nip it in the bud”. If weight gain is “my fault”, the person is expected to rise to the challenge. If it is framed as “the surgery’s fault”, or a thyroid problem, or genetics, it runs the

risk of giving the person “a total green light” to relinquish control. Fran theorized that by defining the behavior as a bad habit, she was able to control it.

Having introduced the themes of this sub-section, I now turn to other respondents who articulated similar thoughts. I have grouped them according to shared sub-themes to highlight conceptual distinctions, though these sub-themes also overlap. All are connected by the idea that genetics help to redefine agentic behavior as non-agentic, thereby changing what people actually do. Their conceptions of agency change the actions they would take, according to these respondents.

Too hard to stop if it's genetic. Several other respondents also noted that genetics might discourage people from making the effort to overcome their eating disorders because it seemed too hard. Eva said that genetic ideas would “give them, like, one less reason to try to get better”, and “not work as hard at recovery as they could.”

... maybe the people would maybe feel, like, kind of helpless if they had it. And, maybe would give them, like, one less reason to try to get better. Because, they wouldn't feel like there was anything they could do. Like, it was their genetics. And, maybe not work as hard at recovery as they could. Just kind of discredit that. And, think that they're, like, terminally ill. And, they were never going to get better.... I don't really think it would do me any good to know. And, I think it would do – I guess it might do more harm than good. Because, I feel like it would just be one more reason to kind of give up on getting better. I just think that I was never going to get better. Because, now I know that I have the gene for it. There's nothing I can do to fix that. It's just kind of like set in stone. Because, I guess you can kind of change, like, your behavior patterns. But, you can't change your genetics. (Eva, T-BN)

“Giving up” meant not doing the “work” needed to accomplish recovery. In this passage, Eva suggests that there is work people could do, and genetics would discourage them from doing it. This sentiment is different from the helplessness she expressed about overcoming eating disorder thoughts (described above). Such reasoning was shared by others who talked about helplessness, and the need for professional intervention.

Less responsibility if it's genetic. Like Fran, several other respondents thought genetics would remove responsibility for choices and reduce the motivation to work on them. Victoria said it took “all the responsibility off of us”, and that because her BN started as an “active choice”, her recovery would need to as well; “you have to want to” recover.

R: ... I think it would have a negative effect. Because I mean I guess if you try hard enough, you can probably trace anything back to genetics. And it takes, like, all the responsibility off of us. And, like, the choices that we make. Or having responsibility for our actions in our life ...

I: And so responsibility is important in what ways?

R: I mean just that the choices we make. I mean when I started having an eating disorder it was, like, a very, like, active, like, choice. I mean and so I think even, like, recovering from an eating disorder – like, you have to want to. And you have to, like, make different decisions. And to do that you have to, like, I mean take some responsibility. I mean whether or not – I mean at this point I do feel like it's a little bit of, like, an addiction and a pattern. But I know there's still things I could do.
(Victoria, T-BN)

Petra was similarly concerned about the removal of responsibility. Indeed, she said that her BN was prolonged because she thought it was “biological” and “in my genes” and therefore could not control her bingeing and purging.

I: And, how did you feel when you first started to think of it as biological? Not just the environment. But, genetic and biological.

R: 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.”

I: So, do you think it affected your disorder? Your behavior?

R: I think it prolonged it ... when I became bulimic. And, started to learn more and more about how the genetic factor lied in there. I kind of felt like, “Well, I don't have to give up bingeing and purging. Because, it's biological. My mom's addicted to pain killers. She didn't stop. Why should I? I don't have to. It's in our genes. My brother's on heroin. He doesn't stop unless he's locked up. Why should I work on myself?” So, it kind of made it – prolonged it. I think if I would have thought to myself “You can do this. This is something that you can turn around. You may have developed it biologically. But, that doesn't mean you have to live with it forever.” If I would have known that years ago, I think I might have worked harder at getting better. (Delia, T-AN)

Delia said that she would have fared better had she understood biology as contributing to the development of her BN, rather than as a permanent aspect of herself. Like Victoria, Delia said it would be better to think of BN in volitional rather than biological terms “if there were no genetic factor, they might think ‘It’s up to me to get better.’... [B]ecause it’s a behavioral attitude. It’s you did it yourself. You kind of produced it yourself. So, therefore you can make it go away yourself.”

More likely to repeat the behavior if you know it’s genetic. Redefining the behaviors as non-agentic could also make it easier for occasional eating disorder behavior to be repeated and then turn into an eating disorder. Melanie said that if people began to believe that their body’s natural tendency was toward purging, they might just accept it as inevitable.

Well I think you could also have somewhat of a negative effect in terms of people wondering if they have – I don’t know. People maybe who would, like, [who] tried that a little bit. I hasten to say tried. Like dabbled in that a little bit. And then they get this message that well they would hear as “Well if you tried it, you’re probably a bulimic.” Or “You’re more pre-disposed to that.” Or “It’s in your genes. So you might as well go ahead. Because you’re naturally to do that.” I don’t know. That sounds really hokey. But it could have a negative impact like that.... Kind of reinforcement message that they’re going to do that anyway. (Melanie, R-BN)

The idea that the behavior was genetically influenced could make it seem like evidence of an already-existing disorder even before the disorder had developed. Like Melanie, Thelma thought people might self-interpret the behavior as non-agentic: “if I’m doing it, then it’s out of my control”. If “it’s just something genetic” then people would struggle less about the “decision” to purge, which is also related to the theme of responsibility described just above.

R: I think one possibility would be that “Well it’s not my fault. Therefore, like, if I’m doing it, then it’s out of my control. Like, this isn’t a decision. This is just something that – like, I can’t help it.” And I think, like, removing that element of accountability would definitely, like, be negative... I think, like, it would make more people do it. Because it wouldn’t be their fault. It wouldn’t be a decision that they were making to engage in this activity. It would be something that was out of their control. And without that element of choice. Because I mean it’s a difficult choice to make.

Because you just have to – I don't know. It says a lot about, like, the way you think about yourself. And what, like, your – what's important to you. And if it's not your fault, then you don't really have to worry about that as much.

I: So you're saying it's a difficult choice to purge? Or it's a difficult choice to not?

R: Yeah. It's like a – yeah. It's a difficult choice to decide that this is something that you're going to do like a habit or an act. Like it's – yeah. It's a hard decision to make. Because of all, like, the connotations and implications that we talked about earlier... Because I mean if you don't have to think about it as a decision. Like, as you sit down and think about it. Like, if you don't have to say "Should I do this? Or should I not do this?" If you don't even have to think about that. It's not like whether or not you make the choice. It's just something genetic. You're genetically pre-disposed to do this. It's out of your control. Like you don't have the option of making this choice. (Thelma, T-BN)

If purging were no longer a decision but something thought to be out of one's control, people might stop thinking about it and just do it.

Predisposed people will expect to develop an eating disorder. Several respondents also volunteered that people who had never had an eating disorder but found out they were predisposed would also be more likely to develop one because the behavior seemed like something one could not control. I did not ask about predictive testing, and these ideas came up in response to questions about finding out one's genetic predisposition after the eating disorder had already developed. Betty said it might make children feel "pre-destined" and "accept" that they would develop AN.

Well, if I had everybody tested. If there were a test. And then, one of my grandchildren thought "Well, I'm just pre-destined to have this thing." And, they might accept it. And, just go on off and do anything they wanted to. Whereas if they didn't know, I think they would grow up more normally and not worry about it. (Betty, T-AN)

Victoria imagined that it could encourage agency related to medical control (represented in Cell 1) or might "put the thought in my head" so she was "succumbing to my genetics".

It could have been helpful. Because I would have, like, been more aware of being susceptible to an eating disorder. So made different decisions. But on the other hand it might have just, like, put the thought in my head that I might develop an eating

disorder. And I might still go down that path just knowing – just, like, succumbing to my genetics I guess. (Victoria, T-BN)

Barbara thought a teenager who knew about her predisposition to AN and wished to lose weight might be more likely to restrict her diet because she thought it would be “easier” for her than others.

But as a teenager they say “Oh. Well you’re genetically pre-disposed to anorexia.” If someone’s dissatisfied with their weight, they could be like, “Oh. Well maybe that’s a quick fix. And maybe it’s easier for me to lose weight. Because I already, like, have that genetic pre-disposition to restricting my calories.” And I could just see that spiraling out of control. (Barbara, R-AN)

Believing one’s body to be capable of extreme caloric restriction might encourage behavior in that direction, according to Barbara.

Expect relapse if genetic. Recovered people might feel more at risk of relapse, thus making a relapse more likely, according to some respondents. If the behaviors were redefined as non-agentic, people might be more likely to treat them that way, such that their expectations about relapse led to a self-fulfilling prophecy.

I think knowing that I would maybe down the line if I had like a – like those urges came back. And they were really, really strong. And I couldn’t do anything about it. I would kind of be like, “Oh. It’s not my fault. I mean it’s a disease I have. I can’t get rid of it. I can do it for a little bit.” ... I mean I could start doing it just a little bit. And like, “Oh. I’ll get better in a little bit. I’ll just do it for a couple days or a couple weeks. And it’ll go away like a cold.” And stuff like that. But then it may go down a different route. And I wouldn’t be able to stop it. It would just be too strong. (Carly, R-BN)

This idea is similar to the theme of helplessness, but here respondents are assuming that the person could have exerted some control and are not actually helpless. Some respondents said that knowing their eating disorder had genetic contributors would make them feel stressed and “at risk”. This theme is indirectly related to the redefinition of agentic behavior as non-agentic; if you are “at risk” and “vulnerable” to a behavior it made some people think you

might be more likely to do it. Feeling at risk could generally add stress as well as specifically “plant the idea” of relapse, thereby leading to eating disorder behavior. Barbara thought it would add to her stress and make her focus on and interpret her thoughts and behavior as early signals of a relapse, rather than ignoring them or seeing them as normal.

R: It would concern me a little bit. Because I would like – right now I’m relatively confident that I don’t ever have to worry about a relapse. And that would maybe make me more cautious again. And make me – just add more stress when I get really overwhelmed and I start to, like, be a little concerned about weight. Then I’d be like, “Oh my gosh. Like, I really hope this isn’t happening.” Which doesn’t happen. And I’m stressed. I just know I’m stressed. And it’ll be fine again. But [if] I knew I had a genetic pre-disposition, I’d be probably a little bit nervous about kind of crossing that threshold that triggers it again. Whereas right now I think that I’m relatively emotionally recovered. And so, like, I think it’d have to be really, really incredibly extreme circumstances to get me down that path ever again.

I: So you’re saying that it might make you worry more about it. Do you think it could change whether you actually do relapse to start thinking of yourself as having a genetic pre-disposition?

R: I think it might. Yeah. Like, I mean just planting the idea in my head that it’s easier, like, could make me start thinking like ... I mean I think it would yeah just concern me. Because I’d be worried about if I started to think about it too much if I would start leading myself down that path. And just, like, knowing that it might be a genetic pre-disposition would make me worry about it more when I was worrying about all these other things. And I’m already under an incredible amount of stress. And now I’m thinking “What if I relapse?” And all of a sudden, like, I’m not hungry one day. And I think that I’m relapsing. And I just, like, basically lead myself into that cycle all over again. (Barbara, R-AN)

Barbara speculated that her self-management as a recovered person would be disrupted by the knowledge that genetics had played a role. She described how it would increase her worry about AN and cause her to “lead myself” toward AN again. Emma also thought it would “make me stressed out about now,” it would “be on my mind more.”

It might give me a little bit of – just ease off a little bit on “You did something you shouldn’t have been doing.” So, like, there’s a reason or whatever. Not that there wasn’t a reason. But, somehow kind of justify. But, it would make me stressed out about now. Because, I still – not that I act on it. But, I feel vulnerable to food. So, that would stress me. It would make me feel at risk. And so, like, thinking about it’s going to happen more. When now, I don’t think about it’s going to happen as much. Like, I don’t think – it would be on my mind more. (Emma, R-BN)

Emma implies indirectly that genetics could make her more likely to act on the behaviors, she would be more “stressed out about now” if genetics threatened her with relapse. For a different reason, Vanessa thought that “re-opening” the “closure” she felt about her BN would threaten her with relapse as well: “I think it would be a blow. It would be a mental blow to some. It would be a stressor. It would be a stressor that could ultimately lead you back down that road. I really would feel that way.”

2.2.5. Using genetics as an excuse to abandon control (5)

Genes could be used an excuse to justify persistence in eating disorder behavior, even if there were something that could be done about it. As has been described, respondents brought up genuine hopelessness, which was represented in Cell 1 when that hopelessness was a reflection of a real inability to control the eating disorder, and in Cell 2 when it was a mistaken interpretation that caused the person to forego action that might have helped. Now I turn to a related concept, consciously “using” genetics to pretend to others that one is hopeless in order to avoid hard work: genetics as an excuse. Here, respondents raise the possibility that people with eating disorders would “use” genetics to convince others, and perhaps themselves, that change was impossible and efforts were futile. Several point out that having genetics available as a plausible excuse could discourage them from trying.

There is much overlap with themes described in the sub-section above, but here I focus on using genetics as an excuse, in which the person with an eating disorder is figured as someone who is trying to convince others, and maybe also themselves, of a dubious proposition. They are behaving as though they do not have control, though they may not believe this as much as those just above. Using genetics as an excuse implies that some part of the behavior is in the person’s control, and genetics provides a cover for it, thereby

enabling the person to slack off in her efforts. Gena alludes to the idea that having an excuse makes it “easier to explain” – an idea shared by those who welcomed genetics’ ability to relieve blame – but could also “add to an excuse” for “essentially killing myself” with the eating disorder.

Like with the media campaign ... I can’t lie and say that there’s a little part of me that would make it a lot easier to explain. If I didn’t always feel like I was justifying myself to everyone. That I can say “Look. This is the way I’m made. I can’t fix it. Deal with it.” But, that’s also – I mean I can’t lie and say that that wouldn’t make my life a little easier. But, at the same time it also would add to an excuse that I have to be essentially killing myself. (Gena, R-BN)

Fran’s observations above about weight gain after surgery play on a similar theme. If weight gain is the “surgery’s fault” then she will not try to avoid it, but if it is “her fault”, she will make the effort. Having an excuse available for use could affect one’s action; if one is uncertain about one’s ability to do something, the availability of an excuse could make the difference between action and inaction, particularly with a difficult task.⁹

Having an “excuse not to get better” reduces blame but also discourages hope; the excuse could disempower, according to Delia.

...knowing that you’re not to blame like I said would give most people an excuse not to get better. Because, they wouldn’t be able to control it. But, they have to believe they can control it in order to get better. So, it’s tough. I don’t know that one. I don’t have an answer for that. (Delia, T-AN)

Rebecca thought the removal of blame could be “freeing”, but at the same time be “very rationalizing”, meaning that it could justify throwing up to herself and others.

⁹Fran theorizes the “green light,” which is signaled by others, to affect one’s actions. She theorizes an actor who responds to cues about what is possible from others around her, suggesting a reflected agency. If everyone else treats it as objectively true that weight gain after surgery is inevitable, or that genetically-influenced eating disorder behavior is impossible to control, the actor will believe this too and act (or not) accordingly. This could be called the “Dumbo effect”, after Dumbo the elephant who can fly only with a feather in his trunk (I am borrowing an analogy to a similar phenomenon from Watters 2010). This is a good example of the social construction of reality because treating something as possible or impossible affects what people actually do, which only strengthens the sense that it is possible or impossible.

It might be kind of freeing for people. . . . It might be, like, easy for people to say “Well, like, I do have an eating disorder. But, like, part of it – like, it’s not all just coming from me.” Like maybe it wasn’t entirely my choice to start throwing up my food. So that might be kind of freeing. But I think it could, like, also be, like, very rationalizing. Like, “It’s in my genes I’m going to throw up my food. All right.” (raised shoulders and arms to indicate helplessness as though saying, “what could I do?”) . . . Yeah. So I can see it having both those effects. (Rebecca, T-BN)

Similarly, Victoria said she knew “there’s still things I could do” so talking about genetics would be like saying, “ ‘Oh. It’s not my fault I have an eating disorder. It’s genetic. I can’t help it.’ Like, I just think that’s a little bit of, like, a cop out.” Gillian thought some people could “just stop trying to recover. Because, now they have an excuse to be sick forever.” Cop-outs, excuses, rationalizations, and crutches imply a person who is self-indulgently choosing not to work on a difficult task. Sarah used the metaphor of a “crutch”, where genetics would let people say “this is the way I am,” rather than working hard to change.

I would hope that it wouldn’t – I mean I just would hope that it wouldn’t make people – allow people to just put it all on “It’s genetic. And this is how I am.” And not even think for recovery. And not even try for recovery. But I guess I would worry that that could be a potential harm. That people would not even try to get better. And it would be kind of a way to just say “This is the way I am.” Because it is hard to change once you’re in those patterns and have those behaviors. It is very hard to stop. So I would worry that people might use that as a crutch and not try to make those difficult changes. (Sarah, R-BN)

Several respondents thought that people who already wanted to give up would use genetics to justify doing so. Amy envisioned people “choosing” to have AN and feeling bolstered or reinforced by the idea that genetics play a role:

.. if people know that there’s a genetic link, they may just say – if this is a person that’s going to choose to have anorexia anyway. So, they would just say “Okay. I’ve got the link. So, why not just go for it all the way?” (Amy, T-AN)

Carol said people might “use” it if they wanted a “reason why they should die”.

R: If people used it as “Oh. Well, it’s my genes. There’s nothing I can do about it.” And, either didn’t seek help. Or, refused help. And, used it . . . to be as self-destructive as possible. . . . [S]ince people die from this, I can see that if you truly want to die that

you will – and, if you’re going to do it in this slow drawn out way. That sure. I could see some people wanting more, like, reason why they should die....

I: Like, what would the logic be?

R: Well, this is who I am. This is my genetic make-up. So, this is my destiny. I’ve started this journey. And, I’m finishing it this way.... I think we take every situation. And, we use it in some way that works for us. Whether it’s helpful or destructive.... Saying “Well, there’s nothing I can do about this.” Using it as a death wish I guess. (Carol, R-AN)

Carol theorized an agent seeking death who “uses” her situation and genetic ideas “as a death wish”. Fran, quoted at length earlier, thought that the availability of genetic ideas as a way to explain behavior could provide a “green light” for the person and “ammunition” against caregivers who want them to change their behavior.

It might give somebody a green light to “I can’t help it.”... If it’s a patient that doesn’t have the desire to improve, it would give them their ammunition against a caretaker trying to help them. “I can’t help it.” (Fran, R-BN)

Having the ammunition available made it possible to use against a caretaker; a resistant patient would presumably draw upon whatever was in her repertoire. Genetics would add to that repertoire. Tammy also thought it would give people a way to shift the blame.

We’d all have a reason to – we’d all have something to blame. Because we’re always looking for something to blame. Instead of trying to address – it’s, like, obesity is McDonalds fault. Anorexia – it’s my genetic pre-disposition. So don’t try to help me. It’s my genes’ fault. So we’re looking for something to blame instead of addressing some of the issues that impact causation. Yeah. McDonalds is not the best place to eat. And I don’t even call it real food. But I don’t have to go in there and eat everything either. They do have healthy choices. (Tammy, R-AN)¹⁰

Respondents who are concerned about excuses fear that genetics could change what counts as an acceptable explanation for behavior, what kinds of accounts can be honored. They perceive danger in loosened standards of accountability and responsibility, though many also perceive a welcome relief from blame at the same time. Changes to the socially agreed-upon norms of accounting for unusual and problematic behavior loosen the constraints under

¹⁰ Tammy also criticized society for always trying to blame people in a quote presented above; I interpret her to be conflicted about how much responsibility people have for over-eating.

which people are operating, with negative consequences according to these respondents.

With regard to eating disorder behavior, some feared it would make people more likely to engage in the problem behaviors due to reduced constraints.

That would probably give me that crutch. Or, that excuse. “I can’t help it.”... I think it 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)

Vanessa, responding to the question about whether she would want to know the results of a genetic test for predisposition, clearly linked genetic predisposition with excuses that would encourage more of the behavior.

No. [*Laughing, and the 'no' sounds very definite and almost reproachful or surprised.*] I wouldn’t want to know that. Because I think I would use it as an excuse to go back more easily. I could say “Oh. Well I have the gene for it, now.” And so I’d use that as an excuse to do it. And so no. I wouldn’t want to know that. (Vanessa)

Respondents described in this section were concerned about the effects on behavior of the availability of genetic accounts. The availability of genetic excuses seemed to give a “green light” to behavior that ought to be seen as a problem. This suggests that stigma has a positive side: it is a form of social control that discourages problem behavior.

To summarize, genetic forces were perceived to reduce responsibility for people with eating disorders. If their condition were influenced by genetics, they had less individual control and ought not be blamed for the behavior. The meanings of genetics for eating disorders varied for respondents, but all were operating in a context where people with eating disorders were often held responsible for their behavior, and genetics connoted powerful determinative influences rather than minor contributors to a dynamic multi-layered causal story. It is not surprising in this context that genes would provide a way to communicate the

lack of control, agency, and volition that most respondents experienced. The perceived implications take into account others' potential skepticism about genetic claims (e.g., when genetics seems like an illegitimate excuse, or other examples from the right column of Table 6.1); as we have seen in Chapter 5, respondents were themselves critical of specific powerful genes "for" AN or BN. Because genetic relief of past responsibility had potentially troubling implications for future agency, I examined five implications for agency or action regarding control over eating disorder behavior in Table 6.1.

Based on the perceived implications for future agency vis-à-vis eating disorder behavior, I conclude that care should be taken about what kinds of behavior are defined to be genetic and thus uncontrollable, as these affect expectations about what is possible to do and what actions are likely to be taken. In a context where respondents are held responsible and blamed for a complex behavior that feels non-volitional at times, emphasizing a factor that is clearly not in the individual's control provides a defense against blame. My respondents interpreted genetics in this way, though at other historical times and in other places something else might have served (e.g., the media, spirits, bad mothers, traumatic experiences, etc.). The effectiveness of genetic explanations as tools to reduce the perception of control and responsibility depended on whether genetics were perceived to be powerful enough to reduce individual control over eating disorder behavior. Genetics were not the only factors beyond individuals' control; environmental factors played a role in every respondent's account of eating disorder causation. Were environmental factors insufficient to counteract blame in respondents' social settings? I now consider the perceived implications of genetic explanations for the role of environmental factors.

3. GENETICS VS. ENVIRONMENT: IMPLICATIONS OF GENETICS FOR ENVIRONMENTAL CAUSATION

If eating disorders are a complex product of social forces, individual agency, and genetic contributors, overemphasizing genetic sources threatens to minimize social explanations as well as choice-based explanations. In Chapter 1, I had speculated that geneticization, like medicalization, might make social and political problems seem like individual problems located in the body, with the effect of diverting attention from important environmental causes. Did respondents identify this as a problem too? Did they consider the effect of genetic explanations on social explanations? If so, what were the consequences? While most respondents spoke at greater length about the implications for perceptions of individual responsibility, several nevertheless identified both positive and negative implications of genetics for environmentally based understandings.

3.1 “You can kind of choose to ignore culture”: Environmental causes less powerful than genes

Several respondents were concerned that environmental explanations were not honored by others as legitimate accounts for their eating disorder behavior. Several used the term “real”, just as above when speaking about genetic explanations making AN and BN more real, legitimate, and valid as disorders. Here, I interpret “real” to refer not to the status of eating disorders as disorders, but to the status of environmental forces as powerful and clear, rather than “squishy”, “foo-foo”, or “soft science.”

... [genetics is] possibly the least effective of all causes to target in terms of public awareness. But, it’s also the simplest. People will accept it. It’s genetic. It’s research based. It’s real. It’s scientific. And, they won’t accept something foo-foo like gender roles or something squishy. (Alyce, R-BN)

I think I would talk more openly about it. Because I think that thinking that genetic explanations just carry more weight and legitimacy to them... I think I think of it similar to, like, homosexuality is a choice or is it biology? But when we talk about

sexuality as biology instead of social or something that you choose. That you're off the hook. You don't have to explain anything. So I think the same with eating disorders. (Margaret, R-AN)

I'd just feel better knowing that there was more of a cause than just environment, surroundings, and the way I was – [that I] had to go through what I did with my dad. I think it would just make me feel better to know that there is another cause. (Karen, T-AN)

... I guess the good thing would be just the certainty of it. I don't know. I guess things just seem more concrete if they have some sort of basis in genetics that's almost reassuring.... Because, I feel like with psychology the flaw in psychology is the theoretical part of it ... the definitions change. There's a lot of debate on environmental causes versus genetic causes. That's where the soft science bit comes in.¹¹ So, I feel like there being a genetic link just makes psychology a little bit more real. (Joelle, R-BN)

Some respondents thought individuals were held responsible for their reaction to environmental factors, making environmental explanations seem like unconvincing rationalizations for individual choices for which the person was responsible.

Well, you can kind of choose to ignore culture. But, if it's genetics, it's a little bit harder to ignore. (Fiona, T-AN)

It would probably make me feel more guilty [if I did not have a genetic predisposition].... Because, if I didn't have a genetic cause, then I could have been more in control of my environment... I guess just not being influenced by all the dieting and media. (Mary, T-AN)

...[I]t would just be nice to say "Okay. This wasn't just me reacting really crazily to the environment. I actually had a pre-disposition to it." (Willa, R-AN)

R: ...Because, if it's not all environment, then I don't have to blame myself for the environment I was in. Because, many times I'll think "Oh. Suppose I lived in a different country. Would I still develop the eating disorder?" But, if that genetic component was present, then yeah. Probably I would have. And, I think so in that way, yeah. It probably would be a little easier for me.

I:... It sounds like it feels better to think of it as "Well, it's genes that I couldn't help." Rather than "It's environment that I couldn't help."

¹¹ Earlier Joelle had talked about stigma surrounding psychological problems but also psychological explanations themselves: "My mother and my father actually both a little wary when I told them I was going to major in psychology. Like there's still that sort of not really seeing it as branching off into more of the hard science that it's becoming. Like to them it's still a whole lot of Freud and silliness and sort of being – I don't know. What's the word? Self-indulgence. I feel like there's an element of self-indulgence."

R: Yes... Because, I think with environment to some extent you can control it. I mean you can leave one place. And, go to another. And, see how things work in that sense. But, with the genetics I mean that's always present. That's always going to remain a part of you. So, that's a little easier for me. (Helen, T-AN)

These respondents theorized that individuals could “ignore” culture, stop themselves from being “influenced by” media, and “control” their environment by moving away from it. If they were reacting “crazily” to their environment, that was their fault, not the environment’s. This implies that genes are uncontrollable, but environment or one’s reaction to environment is controllable.

3.2. Consequences of removing focus from environmental factors

Some respondents discussed negative, positive, and ambiguous consequences for shifting blame from society and family to genetics.

3.2.1. Bad to remove focus from environmental factors: Society and family “off the hook”

A small but conceptually important minority was explicitly concerned about genetic explanations distracting from social factors, such as media representations of women and abuse by family members. Genetics would remove focus from harmful social forces, resulting in less collective responsibility for and pressure upon these forces to change.

Margaret (R-AN) spoke at length about the importance of keeping environmental factors in mind and provides a good introduction to these themes. She appreciated the capacity of genetics to remove individual blame, which “comes from seeing it as something you dreamed up yourself and as something that you created for yourself.” However, genetic explanations are not “good in the long-term”, because they do not address “the bigger issue” and will “keep us from dealing with what I see as the more relevant explanation. Which is that it’s a social problem.” A genetic explanation would be “too easy”, and a “way of getting

around” gender inequality. I quote her at length because her analysis is so directly related to the themes of this chapter.

I think that anything that takes the pressure off of women to blame themselves for this, to feel like they themselves created this problem, is good to them personally... But I think it’s a sorry substitute for dealing with it as a social problem. And that the way we construct gender and what it means to be a woman and all those things – that that’s what needs to be dealt with. And so I think for society at large it doesn’t help. But for individual women it probably does help to give them some other explanation. Because we don’t have the other explanation. We don’t have this treatment of eating disorders as a social problem. If we had that, then I would say that we don’t need the genetic explanation. And publicizing it I think makes it too easy to ignore that it’s a social problem. (Margaret)

Like those respondents described just above, Margaret did not believe the social explanation of eating disorders is widely accepted; “we don’t have the other explanation” available as a cultural tool, perhaps, so in that vacuum genetics are an inadequate and “sorry” substitute.

Ignoring social responsibility for the problem could make the problem worse, according to Margaret and others. She and others spoke of the importance of keeping society on “the hook” and under “pressure” to change.

But I think that the negative effects are that it takes us all off the hook. We’re off the hook for trying to see this as a social problem that we all contribute to every time we buy a magazine that shows women in bathing suits or shows – whoever - Nicole Ritchie. “Look. She had a baby. And now she still weighs a hundred and two pounds.” But those things that keep focusing our attention on women and their shrinking bodies. Or bodies that should be ever-shrinking. Having these genetic explanations makes it too easy to ignore what I feel is the bigger problem. Which is that we are socializing women to shrink. They think they’re supposed to shrink. And it takes our attention off that bigger problem. (Margaret)

I think some people would – it would kind of take the pressure off, like, other causes like the media. And, I think some people would just be like, “Oh. It’s just genetic.” Like there’s nothing else we can do. Like they’re going to get it no matter what they hear or see on TV. Or, anywhere else. Like, what anyone tells them. Like they’re going to get it no matter what. I think a lot of people would think that. And, just maybe not focus on some of the other things they could do to try to prevent it or decrease frequency. (Eva, T-BN)

...I feel like that takes pressure off of society to change. Just kind of it has nothing to do with the fact that we idealize thinness. It's just these girls are screwed from the beginning. Then there's no responsibility there. (Willa, T-AN)

Genetic explanations suggested that social forces were irrelevant or at less important than otherwise, thereby threatening to remove social responsibility, including individual responsibility to resist harmful social forces, with the result of decreased pressure to change.

Paula also noted that a decreased awareness of the "choppy waters" through which adolescent girls must swim would prevent parents from doing what they could to support their daughters.

On the one hand I would be happy that some sort of stigma and weight of these myth type things would be lifted. On the other hand I would worry. Because I do think that there are these other factors that come into play. I would worry that parents everywhere would just breathe a sigh of relief and think "Great. We don't have to worry about this. Because it's all pre-determined because it's genetics." It's like eye color... I guess I still feel like adolescence is sort of this, like, choppy waters that need – the girls need help navigating their way through. So hopefully that media campaign [about genetics] would be combined with "Oh. And by the way there's still – we're still figuring all this out."... the hazard is still there. I wouldn't want parents to think that it's not. (Paula, R-BN)

To navigate around "the hazard", one needed to be aware of it; awareness of harmful social factors thus not only helped to ameliorate them but also to prevent their negative consequences.

Genetic explanations also ran the risk of drawing focus away from abuse or trauma from family members. Alyce was concerned that in addition to letting society "off the hook" for the increased prevalence of eating disorders, it could also let parents "off the hook" for abusing their children.

...it would also neglect so many causes that need it. Other factors that I think need attention as well. And, almost let them off the hook. "Well, you can molest your daughters. It's genetic that they have bulimia." Because, I'm not making this up. I have sponsored a lot of people [in Overeaters Anonymous]. And, sponsor means you lead them through the steps. And, you talk to them a few times. And, they start to

come out with their sexual abuse. And, if you say “Oh. It’s genetic.” You just let a whole slew of things off the hook. I think. And, you don’t answer the question of “Why are one out of three or whatever college women bulimic now?” When in other cultures and other times this was not the case. (Alyce, R-BN)

If genetics are important, then “you can molest your daughters” and ignore other social factors.

Genetic explanations could disrupt and unsettle respondents’ understandings about the work they had done as part of treatment or recovery. Irene stated that genetic ideas would change how she talked about her BN with other people by making the abuse less central.

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)

Vanessa thought genetic ideas would disrupt the closure she felt about her experience with BN (she was briefly quoted above as well).

I mean with me thinking I started this because of ... my peers or my friends. I started this because I was labeled as big in comparison to my mom. I started this – and then after all of these years I’ve done all this healing and closure with these things. This is why. This is why. And then you come over here and tell me “It’s because you’re genetically disposed.” Well that means I got to go back and re-open all of that stuff again and try to get closure and re-think all of it all over again. That would be me. I was like, “Okay. That’s not fair.” (Vanessa, R-BN)

Vanessa thought the disruption of “closure” could be a “mental blow” and a “stressor that could ultimately lead you back down that road.” Carol, whose account of childhood AN was centered on the dismal prospects for growing up female in her family, thought genetics would be troubling.

I have worked really hard over my life at trying to make sense out of and deal with and create a life that worked for me and the whole issues of being female and that. And so, would I want to say that all my hard work could be in vain? (Laughs.) No. I wouldn’t want to say that. (Carol, R-AN)

Wendy also thought a genetic explanation could jeopardize the work she had done in treatment, particularly in relation to her parents, who created an environment that led to her BN as a coping strategy.

I: How do you think family members would react to the idea that you had this genetic pre-disposition?

R: They might be glad for a simple explanation.

I: How would you feel about that?

R: I would probably roll my eyes at them.... It would just be, like, defeating of all the work I've done to conquer it. It would be like, minimizing all my efforts. (Wendy, T-BN)

The work of overcoming eating disorders would seem to be “in vain” or minimized if genetics were an important factor. In many ways, then, genetic explanations would remove focus from familial and social explanations in ways that were harmful.

Some thought genetics would lead to less talk about social causes and about their eating disorder with other people. This is directly related to the idea that genetics removes focus from social causation, and indirectly related to the idea that awareness of eating disorders as a social problem is important. It removes eating disorders from the realm of social awareness and conversation because it seems to be in the domain of science and professional care providers.

I think people would be a lot less curious about it.... Because they would just say “Okay. It's another scientific thing that I can't really understand.” Whereas, like, I got lots of questions from friends about it whenever they found out about it. (Barbara, R-AN)

I would probably bring it up less, honestly. Because I would feel like other people could help me less. Because if it was just this genetic part of me, I would feel like only doctors could help me. As opposed to being thought patterns whereas friends could help me. (Wendy, T-BN)

The geneticization of eating disorders would remove it from public discussion and relegate it to expert domains, according to these respondents.

3.2.2. *Good to remove focus from society and family*

Respondents also identified good consequences for removing blame from family members and shifting focus from societal explanations. Talking about media causation felt like a way of stigmatizing eating disorders by trivializing them and stereotyping people with eating disorders as preoccupied with their looks, rather than suffering from a disease.

It would make it more valid of a disease. . . . Just that it's not some stereotyped White girl disease. That there's actually a cause of it. And, again that I didn't choose to have it. . . . Yeah. I think that's, like, what people think of and what media portrays. And, you even see stuff in movies about – like in *Mean Girls* they show the girls at the popular table. And, they were sitting eating, like, six Skittles. And, that was their lunch with a diet Coke. And, they were the popular girls that were cheerleaders. But, I already feel like for me I'm not the typical stereotype. And, I developed it when I was older. So, I already feel like the exception. . . . I always feel like I have to explain. I never felt like I was conceited or anything like that. Or, thought I was better than anybody else. But, that's kind of like the stereotype. (Mary, T-AN)

Instead of like now it's like, I feel like people look at it like media caused this. Or the girl wanting to be thin has caused this. Like so I think maybe it might take, like, some strain off of, like, negative attitudes towards it maybe. Towards the individual. (Yvette, R-AN)

In addition, several respondents welcomed the idea that genes could lessen parental blame.

I think that my family would have an easier time not blaming themselves. Because, I mean you can't help what genes you pass on to people. I mean you can choose not to have children. But, you can't really, like, pick and choose your good ones. So, I think that would be helpful to my family to really feel like you really didn't do this to me. (Jackie, T-BN)

I think it would give people peace of mind. I mean it would take some of the blame off certain people. Because I know my mom felt like am I going through this because of something she did? And for her to hear a scientist on a radio campaign or on CNN say "Oh. We've now determined that bulimia is due to a genetic gene in a person." It would alleviate stress from people. It would switch the blame. Because nobody wants to feel responsible like they've done something to cause a person to fall into anorexia or bulimia. (Vanessa, R-BN)

I think my mom would be relieved actually. I think she would sort of be like, "Oh. Good. There's a reason why this happened. And, like I wasn't a bad mother. I didn't cause this." Because, I think that she wondered a while. Like, "What did I do wrong?"

Why is my daughter doing this to herself?” Which I explained to her many times. It’s like, “Mom. It’s not you. It’s just me.” (Hannah, R-AN)

.... it would be nice for parents I guess. Because I feel like for parents who are thinking “Oh my gosh. What did I do wrong?” it would be kind of nice to know “Oh. Okay. I actually didn’t do anything. My daughter was already – had the genes for this.” And so I think it could be good in that way. But also really frightening. Because then parents start to feel like, “Oh my gosh. Well then does this mean she’s not going to recover?” (Willa, R-AN)

Genes removed blame from parents not only because their treatment of children seemed less important, but also because their treatment of children might also have been caused by the genes they passed on.¹²

... we can maybe see environmentally that parents maybe behave a certain way towards food or diet. Or, have problems. And, think of them as having issues with choice and making bad choices and showing bad behaviors for their children. But, it takes a completely different element if they too are – have something that they can’t help going on. Which I think is nice. Because, I feel like it’s – I don’t know. A little easier to correct [with medication] (Joelle, R-BN)

Joelle is certain that her BN was improved by medication, and because her mother and grandmother also benefit from the same medication, she hypothesizes a genetic link between them. For Joelle, maternal “slip-ups” that contributed to the eating disorder may be partly due to her mother’s own struggle with genetically-influenced psychological problems.

3.2.3. *Ambivalence about removing focus from parents*

Some respondents were conflicted about whether removing focus from parents was good or bad. On the one hand, parents ought to take some responsibility, on the other hand, they might feel overly guilty.

... my first reaction was I thought “Oh. Well that will be my mother’s reason for my being anorexic is that it’s genetic. And that she won’t take any responsibility for her part in it.” That’s what I thought. And then I kind of got over that quickly and said “Well maybe I do feel some relief that it’s not all about the ways that I was raised.” (Reba, R-AN)

¹² This is called passive gene-environment correlation (Jaffee and Price 2007, p. 2).

Although Reba is concerned her mother might not take responsibility for her role in Reba's AN, she apparently values getting "over that"; responsibility is not supposed to rest there.

Sydney also felt her mother played a role, but did not want her to feel guilty.

My mother would probably be resistant. Or she'd be really guilty. She would totally feel guilty about it. "Oh my God. I gave you this." Which that's totally what happened (laughing). Now that I think about it. But I think – but at the same time if I was able to logic and reason with her. "This is not something you gave me mom. This is something you couldn't control. This is something." So it might absolve guilt for some people. Yeah. I think it depends on the family. (Sydney, T-BN)

Sydney laughed when she pointed out, "that's totally what happened"; though she does not want her mother to feel guilty about genetic influence, she does think her mother's behavior contributed. Earlier she described her mother's own apparent AN and "disordered eating" as important in the development of her BN.

Selena is poignantly ambivalent about whether it is OK to see her parents as part of the problem; after all they did the best they could. Earlier she had spoke about her mother's illness and death as a traumatic experience, and the emphasis placed on appearance by both parents, particularly her father. On the one hand, she would welcome a genetic explanation to remove guilt from her father.

Well I would love to think it was completely genetic. And that because my parents – I think my father especially has felt very guilty about did he do something to me? Hurt me or something. Because I mean he's like, "God. I mean I can't imagine why." So he's thought – he's said to me openly "I think I was too hard on you. I think I pushed you too hard." So I think he in his older years feels like he's responsible for me, like, having this problem. So I think it would help to know that it's just something in your DNA. (Selena, T-BN)

On the other hand, she does think parents can contribute to their child's eating disorders, and that if it's simply a genetic condition for which medication can be taken, parents might not change their problem behaviors. By discussing her father in this context, it is clear that she sees him as part of the problem.

Well if they thought they could just take a medication or something, maybe they wouldn't alter their parenting style. And I do think that my dad – no matter what the genetic factors are. He certainly made it worse. Because I'm not saying I blame him. Because I think he did the best he could. But just the way he is about women. And the way he is about his controlling nature. I mean he didn't help. I can tell you. I mean he didn't help me in any way. But in his mind I think he thought he was helping.... Like, my parents put me on a diet when I was five years old. That was a little rigid. That was a little bit strange. And I think to myself "Maybe if they just let me alone, I probably wouldn't have gotten as bad." I don't know. But there was so much focus on food and size and appearance and all this outward stuff. Recitals, pageants, and all that stuff. It's like, why didn't they just let me be? Just let me be a normal kid. (Selena)

It is a challenge to see parents as flawed and responsible for some part of the eating disorder, and Selena would "like to get them off the hook" and genes would help her do that.

I would think that if there was a genetic link, I probably would have it. Because I just feel like my problem has been so severe. That if there is any kind of defect, I've got to have it. Because I do think my parents tried. I would like to let them off the hook. I mean not that I do blame them. But I would. Because my mom couldn't help that she got a disease [and passed away]. She couldn't help that. And my dad couldn't help it that he had to raise a family. I mean so it's like, nobody did anything wrong. Everybody did what they thought was best. (Selena)

Irene also displayed some ambivalence. She would like to see her mother's abusive behavior as stemming from a mental illness, whether genetically-influenced or not, which would enable her to feel better about the relationship.

3.3. Brief reflections on genetic explanations for agency and future action regarding environmental factors

There were a number of interesting logics of action that stemmed from shifting responsibility from society and parents to genetics. As seen above, the idea that genetics played a role led most respondents to think that seeking professional help was a logical action, particularly if biologically-based treatment were available. If environmental factors were of primary importance, it was logical to try to change the environment. Margaret and others touched on some of the logical actions that flow from the idea that cultural and

familial factors matter: raised awareness of how one contributes to the problem, talking about one's own history with others, keeping people and institutions "on the hook" if they are responsible.

But if it was purely genetic and something that was out of your control. And you could just take this pill and be better. Then I think that'd be great. Like it's – I don't know. Maybe, like, comparable to treatment of depression. Like, sometimes people are depressed because their lives are terrible. And they need that to change. And then sometimes people are depressed because of off body chemistry. (Rebecca, T-BN)

On hypothetically finding out she did NOT have a genetic predisposition, Alyce said, "I would feel much more optimistic in some way. Like, "Oh. If everything that caused me to do that was social and familial and cultural and whatever, we can change those things." (Alyce, R-BN) It seems obvious, and is supported by earlier excerpts, that if environment were thought not to matter, it would no longer make sense to do these things. Indeed, they might seem like forms of denial that the illness is serious, ways of stereotyping and stigmatizing people with eating disorders, and to wrongly blame parents.

For many respondents, even if genetics were of primary importance it was also important to change the environment. Perhaps surprisingly, some thought the idea of a genetic causal factor would inspire *more* attention to the environment. When considering the idea of genetic testing, many expressed that if they found out they had a genetic predisposition they would be even more careful to provide an environment that did not promote eating disorders for their children. Some spoke of counteracting harmful social forces and being careful about what they communicated to children.

Well, I mean genes are only one part of the equation. Like, the environment definitely still matters. And so, like, if you know you have that risk factor, then, like, I think the only real reason you would want to know that is that your parents or whoever or you can make sure your environment is as conducive as possible to not developing it. So, you can make sure not to read those magazines. And, to be careful to make sure that when you're stressed out, you are still making sure you sleep enough and eat enough

and get proper amount of exercise and everything. Take care of yourself. I feel like you still have to take care of yourself. Even if you know you have a risk factor for something. It's the same. I mean yeah. (Hannah, R-AN)

... there's probably a lot of people that assume that it's something teenage girls do because of X and Y. Who would benefit from the knowledge that it goes a little deeper than that.... It might be beneficial for parents to be able to maybe examine themselves a little bit more closely. And, maybe recognize risky behaviors before they turn into problems. I don't think my mother ever realized that I took to heart some of the things that she told me. (Joelle, R-BN)

Knowing that eating disorders had a genetic contribution also involved changes to the environment in the form of awareness of and monitoring for early signs of eating disorders, as part of a medical logic of action.

Well, there'd be more discussion about it. People might look at people they know. And, say "How are you doing? What's going on with you?" If there were adults in the household who had been anorexic, maybe they'd take more care with their children on this issue. (Lynn, R-AN)

Amy thought that a genetic causal factor could inspire social change beyond the family context. Publicizing the idea of genetic contributors to AN could change how modeling and athletic competitions were conducted because officials would endeavor to prevent AN deaths by relaxing their focus on weight.

... to me it would make sense if they were to say "Okay. We've identified a disorder. A genetic link to a disorder that could potentially end up in a mass amount of deaths so to speak." And so, they would want to do everything in the world to try to prevent that.... it would be like in some countries. You have to be a certain amount of weight to participate in runway shows. You have to maintain a certain amount of weight to even stay in competition for sports and things like that. And, not that coaches or people in industries would know who or who doesn't have an anorexia gene. But, it would be a generalization to where they would say expect for everybody when they're younger to kind of have it. And, the comments wouldn't be like, "Well, if you lost maybe five pounds, you would look a lot better in this act." (Amy, T-AN)

I perceive in the above a logic of action that connects genetics to environmental change via medicalization. If genetics matter, then eating disorders are a disease that ought to be taken seriously, so all contributors including environmental factors should get more

attention too. This was compatible with the idea of gene-environment interaction. Again, I am basing this on hints in the interviews and to understand this better, future research would need to be more systematic about asking if genetic contributions would be likely to change how people think and act upon environmental factors. There is a lot of evidence that genetic factors would encourage people to see eating disorders as something for professionals to handle and therefore seek treatment. Many respondents spoke about biologically based treatments – a pill, gene therapy, an injection, an operation – as logical to expect if genes played a role, as far-fetched as many of them thought this to be. Genetic contributions also encouraged treatment even if there was no biologically-based intervention, probably because of the above logic. Helen (T-AN) described cognitive behavioral therapy as working on the environmental factors. I asked her to explain why:

It's environmental in that the change comes when you start looking at your life in a different way. When you start looking at those thoughts. And, you recognize you can control the way you act. And, you can control what you do with those thoughts. So, I think when you just have that awareness, that really does make all the difference.
(Helen)

“Looking at your life” is looking at your environment, presumably, and becoming aware of your thoughts and actions.

Similarly, it was possible to justify “environmental” treatment if one’s condition had genetic origins. Selena had spoken repeatedly about her father and others who “love her to death”, and suggested that they might be adding to the problem, though she did not want to blame them for her BN. Seeking intensive treatment for her eating disorder would get her away from people who “enable” her BN but this seemed like something she should be able to do by herself. If BN were reconceived as genetic she could receive treatment without feeling guilty, just as though she were receiving treatment for cancer or diabetes. Yet, the treatment

she described was recommended because it would change her environment. Therefore, reconceiving of the eating disorder as genetic would help Selena justify seeking treatment that focuses on her environment.

Well in the crises that I've had each time somebody would say to me "You need to go to, like, Renfrew or somebody, like, here and just stay. Because the structure of that will help you. And the people in your life who love you to death somehow probably enable you. Maybe they don't try to. They try to help you. But maybe the structure of going and doing that would help you." And I've never really been open to that. Because I thought "I can do this. I don't want to be away from my own setting. My own life." But I think if I realized that it wasn't just environmental. If it was really a disease like cancer or diabetes or something. I mean if you had diabetes, wouldn't you go and get treatment? Like, this one you think you're doing it to yourself. You think you don't have a choice. (Selena, T-BN)

If it is just her environment, she believes "I can do this". But if it is genetic, then she reconceives it as a disease that was not in her control, which enables her to get help with the environmental factors. Paradoxically, it is the very conceptual exclusion of environment from genetics that enables this respondent to seek treatment that addresses the environment precisely because her problem is reconceived as genetic.

In summary, although environmental factors were ubiquitous in respondents' causal stories, they were a less central preoccupation than individual agency when genetics were considered. Some respondents held themselves responsible for being affected by their environment, and some could identify reasons why de-emphasizing environmental factors might have good effects. A conceptually important minority spoke at length about the troubling consequences of getting society and family "off the hook." Some respondents said genetics could actually prompt more preventive attention to the environment, because of a theorized gene-environment interaction, or even just because genes made the condition seem more serious and commanded attention to all potential causal factors, environment included.

4. SUMMARY OF CHAPTER 6

In these interviews, the involvement of genetics promotes a more medical conception of eating disorders in a social environment that contests their reality as illnesses. More generally, genetic ideas prompt consideration of the line between individual responsibility and irresponsibility for behavior that is complex and difficult to explain. Genetics are a metaphorical tool to convey the non-volitional aspects of eating disorder behavior. Genetic accounts may or may not be honored by others, but they are taken to mean that individuals are less able to control and therefore less responsible for their behavior. Respondents perceived genetic ideas to have consequences for action, from propelling people toward treatment to providing a handy excuse for getting out of treatment. If an eating disorder is an irreducible mix of factors that feels non-volitional at times, genetics is helpful in conveying non-volitional aspects but may artificially narrow conceptions of contributing factors and how to stop having an eating disorder. Reducing stigma may, ironically, threaten active attempts at recovery. Letting environmental factors “off the hook” seemed disturbing and counter-productive for some respondents, but reassuring and helpful to others. Genetics works as a cultural tool to convey a need for compassionate help in respondents’ social contexts, but it may have negative side effects, such as decreasing a sense of agency or control in combating the disorder and de-emphasizing environmental forces.

CHAPTER 7

DISCUSSION AND CONCLUSIONS

The principal aim of this study was to understand the meanings and implications of genetics for people with eating disorders. No previous study had examined what people with eating disorders thought about the idea of genetic influence on AN or BN. In this chapter, I will summarize the main findings, identify their implications more generally, note some of the limitations of the study, and suggest future directions for this research.

In Chapter 1, I set out several interrelated research questions, all revolving around perceptions of genetic influence and its consequences. I wondered whether genetic ideas would make eating disorders seem more like medical diseases, and whether respondents who already saw eating disorders as medical diseases would welcome the idea of a genetic influence. What were the positive and negative aspects of highlighting the role of genetics in the development of eating disorders, and did they resemble those I identified before conducting this research (as summarized in Table 1.2 of Chapter 1)? Did respondents already think of eating disorders in genetic terms? Did they find the idea of genetic influence plausible, and if so, what role did they imagine genes to play (if any)? My study illuminated all of these questions.

1. Summary of the main findings

To summarize, I integrate across chapters to identify three over-arching findings: geneticization and medicalization appeared to reinforce each other, respondents perceived

genetics to have implications for individual responsibility and future action, and respondents imagined genes to work in complex, dynamic ways that went beyond “simple” and even “complex” models described in Chapter 1.

1.1 Geneticization and medicalization reinforce each other in eating disorders

In the introduction, I proposed that conceiving of eating disorders as genetically influenced would heighten their perceived medicalization. Eating disorders had already been officially medicalized by their inclusion in the DSM, but I argued that if medicalization is a continuum, the perception of biological causation could heighten or intensify it. I found that additional medicalization of eating disorders was possible, given the variation in endorsement of medicalized terminology described in Chapter 4. I presented strong evidence for the idea that geneticization promoted medicalization in Chapter 6: when discussing the implications of genetic ideas, three-quarters of respondents spontaneously mentioned one or both of my criteria for a narrow, conservative definition of medicalization (i.e., disease concepts and warrant for treatment) and nearly all (94%) spontaneously mentioned these or a third criterion (i.e., biologically-based treatment). Respondents thus thought that the idea of a genetic influence would prompt themselves or others to conceive of eating disorders in more medical terms and to act on them accordingly. Thus, geneticization promoted medicalization.

I also found that medicalization promoted geneticization, meaning that people who already viewed eating disorders as a disease were more likely to respond favorably to the idea of a genetic influence (Chapter 5). In addition, people who had received more treatment and those currently in treatment were more likely to endorse medicalized terminology and react positively toward genetics. Indeed, the medicalized terms “mental illness” and “brain

disease” frequently connoted biological causation to respondents (though some rejected the terms precisely for this reason) (Chapter 4).

1.2 Perceived implications of genetics for respondents resemble those mentioned by experts

Respondents’ perceived implications largely resembled those I identified in Chapter 1. Like the experts I cited, they had concerns about reductionism, distraction from social causes, and fatalism (though few respondents anticipated additional stigma). They hoped that genetics would make others take eating disorders more seriously, treat them with greater compassion, and recognize their need for assistance.

I found that respondents were particularly preoccupied by the meanings of genetics for personal responsibility, volition, and agency as perceived by others and themselves. This focus on responsibility was shaped by existing understandings of eating disorders as “contested illnesses,” in this case, more like choices than medical diseases. In the view of respondents, genetic influence reduced the culpability of the person with an eating disorder (and, to a lesser degree, culpability of her family and society). Respondents’ interpretations of genetics were tied up with what others thought eating disorders involved and what genetic influence might mean to them. As anticipated and described in Chapter 1, respondents took into account their social context where eating disorders were perceived to be volitional – people asked them, “why don’t you just stop?” – rather than diseases the affected person could not control. The presumption of volition was troubling to respondents, and in Chapter 3 I noted that most ideally wanted others to think of the eating disorder with sympathy as a sickness or as a struggle. In Chapter 6 I highlighted the perception that genetics implies a person with an eating disorder “can’t help it,” with implications for how the respondent was perceived by others, as well as how she perceived herself.

The difficulty of understanding and describing their own role in eating disorder behaviors clearly informed their interpretation of genetic influence; genetics provided a way of conveying that the behaviors were not simply volitional. In Chapter 3, I explored the multiple, perhaps contradictory, ways respondents spoke about their own agency in eating disorder behavior, distinguished “agentic,” “non-agentic,” and “quasi-agentic” language, and found that all respondents who used “agentic” language also used one of the other kinds. Thus, for respondents, eating disorder behavior was not simply a choice and their language implied that choice was either constrained or non-existent in some ways and/or at some times. In Chapter 4, I showed that respondents struggled to define the ways in which AN and BN were and were not volitional “choices,” with some rejecting the term entirely and others rejecting certain interpretations of the term. By articulating the “finer points of choice,” (see Tables 4.3 and 4.4) respondents revealed the complexity of agency, and the potential appeal of genetics as a way to explain behavior that felt out of control.

Genetics enabled respondents to identify “some sort of larger force at work,” a force larger than themselves that prevented the free exertion of their agency. As they considered the implications of genetic influence for themselves and others with eating disorders, much turned on the idea that genetic influence conveyed the idea that one “can’t help it,” as described in Chapter 6. Genetic ideas had the potential to reframe the agent from “bad” to “sick”; rather than perversely “choosing” to harm herself with an eating disorder, with genetics she could be a healthy, normal agent grappling with an illness. As was seen in Chapter 4 (particularly Figure 4.1), respondents thought “choices” were by definition in pursuit of good rather than bad and self-destructive ends. If eating disorders were contested illnesses, they could not confidently assert that they were under the control of an illness;

sickness would not be accepted as an explanation for self-destructive behavior. However, if genetics could make eating disorders seem more like illnesses to other people, some respondents appeared to think they might be able to retain a sense of their healthy agency despite the illness. The transformation from “bad” to “sick” would be accomplished if others in her social context began to perceive eating disorders as an illness, which respondents thought would be encouraged by genetic ideas (see above on medicalization). (Genetics were not the only way to accomplish the rehabilitation of agency; as suggested in Chapter 4 respondents could also rehabilitate their agency by describing past choices as botched or impaired attempts to seek the good.)

If respondents sought to explain their behavior by attributing it to “some sort of larger force at work,” genetic forces were apparently “larger” than social forces. Although environmental influences were ubiquitous in interviews and virtually all agreed that it was a “social or cultural problem” (Chapter 4), these influences apparently did not adequately convey that people “can’t help it.” Indeed, some felt additionally blamed by social explanations because they seemed to reflect their individual weakness, vanity or extreme conformity to cultural ideals; a better person would not have succumbed. Social and other “environmental” forces did not seem to pose a challenge to the idea of eating disorders as “medical”; respondents who cited morally charged environmental causes (trauma, abuse, or injustice) or who spoke more than others about gender-related issues, were no less likely to endorse medicalized terminology (Chapter 4). Even Margaret, the respondent who explicitly resisted genetic explanations because of their potential to remove focus from gender inequality, thought that genes were more effective in her social context for removing blame, even though she wished it were not so. Even if environmental explanations were not helpful

for removing blame, they were nevertheless valuable and central to respondents' narratives about eating disorder causality, and several respondents were concerned about letting these forces "off the hook" if genetic explanations were foregrounded.

Given that genetics promoted medical conceptions of eating disorders and reduced the perceived responsibility of the individual and social forces, it is not surprising that respondents identified changes in what kind of actions one could or should take toward eating disorders. Genetic loss of control (i.e., the idea that genetic involvement made people less able to control their behavior) could be seen either as a plausible and justified understanding (an accurate, reasonable conclusion to draw from the idea of genetic influence) or an implausible and unjustified understanding (because the genetic influence did not preclude individual control over behavior). If genetic loss of control seemed justified, respondents speculated that people with eating disorders would be more likely to take medical action, or to become hopeless, or (paradoxically) to gain greater control over the very behaviors that genes were supposed to control. If genetic loss of control were implausible and unjustified, claiming a genetic loss of control could seem like an excuse given to evade responsibility for controlling behavior, or a mistaken belief that discouraged attempts to do so. People might genuinely believe they were unable to control their behavior, or they might cynically pretend they were unable to, with the same result for both: further entrenchment of the eating disorder when it might have been stopped. These implications for action are shaped by the taken-for-granted knowledge that eating disorders are often considered to be choices; otherwise the idea of an "excuse" would not make sense. Thus, the idea of genetic loss of control elicited from respondents a wide array of desirable and

undesirable potential consequences for future action, especially with respect to treatment seeking and efforts to get well.

1.3 Beyond “simple” and “complex” genetic models

In the introductory chapter, I described “simple” and “complex” genetic causal models, and wondered which kind respondents might hold, if any. I found far more complexity and dynamism than was captured in my description of “complex” gene-environment interaction models. As briefly noted in Chapter 5, most respondents (37 of 50) spontaneously mentioned genetics in connection to eating disorders. However, respondents spoke about the development of eating disorders in ways that could not be reduced to a gene-environment interaction (what I had referred to as “complex” in Chapter 1), much less Mendelian causation (what I had referred to as “simple”). By speaking about causes such as the valorization of thinness (Chapter 3) – in which physical body size, its social meanings, and efforts to control it by individuals coalesce into a single irreducible compound causal factor – respondents far exceeded the complexity of gene-environment interaction models. I was not surprised that environment would be important even if genes were involved, but the centrality of a temporal dimension (in coping and the repetition of behaviors) implied a more developmental perspective than I had seen in discussion of the genetics of eating disorders. In addition, I found respondents were able to incorporate genetics while also leaving significant room for themselves as agents interacting over time with environments (theories of interactive and indirect roles for genes described in Chapter 5). Indeed, I believe that their discussions of genetic testing indicated that the “gene-by-environment interaction” version of complexity actually constrained their imaginations, probably because it seemed to imply genes “for” AN or BN. However, simpler models of genetic causality were also on their

minds, and in their initial reactions to genetic ideas (Chapter 5) and discussions of public perceptions of a media campaign or the implications of genetic test (Chapter 6), they expressed concern about reductionistic interpretations of genetic causality that could exclude environmental influence, agency, or the temporal dimension. Diverse models of genetic influence co-existed as respondents imagined implications of genetics for their self-conceptions, others' self-conceptions, and others' evaluations of them.

2. Contributions

These three findings contribute to scholarship across multiple disciplines. I focus on contributions to literature on geneticization, medicalization, and the classic theory of accounts in ways that are relevant to both social psychology and sociology of culture. Results also inform the following areas but I do not discuss them here: stigma for mental illness, health behavior change, recovery narratives, and public understanding of gene-environment interaction.

2.1 Medicalization and geneticization

My findings provide empirical support and individual-level detail for the argument by Shostak et al. (2008) that geneticization for a condition that has already been medicalized encourages yet more medicalization. Shostak and colleagues argued this on the basis of a case study comparing the different effects of geneticization of depression, homosexuality, and susceptibility to environmental toxins. As noted in Chapter 1, the idea that medicalization might be heightened even after official adoption has not received much study. My findings suggest mechanisms for this process: physical origins rather than moral responsibility for behavior, expectation for new treatments (“genetic optimism” as described by Conrad 2001), and perception of increased severity of the disorder that warrants

biologically-based treatment (supporting Phelan 2006). By focusing on a medicalized condition defined by *behavior*, this project also illuminates potential limits to medicalization when compared to conditions such as depression; some respondents were resistant or skeptical about genetics and expected others to be too.

My findings on medicalization add to the study of medicalization as a continuous process and illuminate aspects of negotiation processes “on the ground” for individuals. Genetics seemed to promote medicalization, and likewise medicalization seemed to promote geneticization. Taking a step back, and considering geneticization as something that may or may not happen for a given condition (Cox & Starzomski 2004) there were factors that promoted and discouraged thinking of eating disorders in genetic terms. On the side of promotion, genetics helped combat the perception of volition and resultant stigma surrounding eating disorder behavior by providing a way to convince others and themselves that it wasn’t simply a “choice.” Alternative explanations for why eating disorders were not simply a choice, such as culturally-based and feminist explanations, appeared less powerful to respondents; social explanations did not counteract the accusation of choice and for some seemed to add yet more blame and stigma. Armed with a genetic explanation, some respondents felt more able to expect compassion from others and to justify treatment, even treatment not based on genetic information. Alleviating parental guilt via genetics also appealed to some respondents. Other factors militated against geneticization. Respondents were concerned that blaming genetics would seem like an irresponsible excuse to others. Respondents were also cautious about the potential for some genetic explanations to ignore environmental factors. Last, because there is no genetic test nor genetically-guided treatment there are limits to the geneticization of eating disorders.

In general, I expect that my findings of mutually-reinforcing medicalization and geneticization, along with some resistance and skepticism, are likely to be relevant for other quasi-medicalized problem behaviors such as addiction to alcohol or substances, over-eating, gambling, or conduct disorder. Genetics may provide a way of explaining the development of these behaviors and the difficulty of stopping, and strengthen claims about the medical status of deviant behavior as behavioral disorder rather than choice or habit. Eating disorders are a good case from which to generalize, for three reasons. First, many respondents thought of their disorder as a form of addiction, so their reasoning might be used by those with other addictions. Second, the diversity of behaviors in eating disorders map well onto diverse other behaviors, both those that involve taking things into the body and those that do not: bingeing involves such consumption and may therefore be conceptually similar to over-eating and consumption of an addictive substance; purging and restricting do not involve taking things into the body and may have some conceptual overlap with “addictions” to other such behaviors (e.g., gambling or shopping). Third, there is diversity within eating disorders with regard to the sense of control and stigmatization of specific behaviors: for respondents, restricting connoted more self-control than bingeing, and less embarrassment than either bingeing or purging. If a genetic explanation could be applied to a behavior like restricting, it seems even more likely to be applicable to other behaviors that feel out of control or shameful.

My findings show that for laypersons, geneticization may happen in different ways for a single condition, because respondents theorized diverse genetically-influenced causal pathways. Genetic influence on AN or BN is not tantamount to genes “for” AN or BN, neither for genetics researchers (Kendler 2005) nor respondents. Some researchers studying

genetic influence on eating disorders have conceptualized it as part of a more general phenotype relevant to multiple diagnoses, such as “negative emotionality” or “obsessionality” (Bulik et al. 2007). Such theories are compatible with respondents’ ideas about personality and general psychological problems.

I found that respondents’ theories of genetic influence ranged from genes specifically “for” AN or BN to quite indirect causal pathways, genes “for” outcomes that were unambiguously pathological or not, and genes “for” physical attributes as well as psychological characteristics. Respondents seemed to care how the genetic causal pathway was specified, not only because some pathways were more plausible than others but also for what they meant about the disorder and recovery.

By focusing on eating disorders, this study draws attention to important distinctions among types of geneticization for diverse medicalized conditions, whether behavioral or not. Studies that focus on testing for predisposition for single gene disorders (e.g., Novas & Rose 2000, Marteau and Richards 1996) or predisposition for disorders that involve both genes and environment or risky behavior (e.g., Senior et al. 1999, Marteau and Weinman 2006, Harvey 2009) are different from my study because they involve *specific genes* known to contribute to the condition, *individual knowledge* of personal genetic information, and address predisposition *before a condition has developed* rather than afterwards. In my study, all respondents already had a diagnosis, there was no concrete information about personal genetic predisposition, and the claim of genetic involvement in eating disorders was based on twin studies, with no identification of a particular stretch of DNA (Hedgecoe’s 2002 definition of geneticization). Yet, even in the absence of personalized genetic information, most respondents used genetic ideas to talk about their disorder and its development even

before I introduced the topic, suggesting that geneticization is relevant here too. My study helps map a less-explored territory, somewhere between these studies of people with known genetic risks and studies of the general public's understanding of genetics (e.g., Condit 2006, Shostak et al. 2009, Phelan 2005, Singer et al. 1998); my respondents are applying genetic ideas to their diagnosis without concrete personalized information.¹ It makes sense, then, to study genetic ideas even in the absence of clinical information simply because respondents are using them and, as my study shows, they can be part of complex negotiations about medical status, individual responsibility, and social responsibility.

By focusing on how people already diagnosed with eating disorders think about genetics, my study considers ideas about responsibility and agency -- familiar themes in studies of geneticization -- from a novel vantage point. Many studies of geneticization suggested or showed that new responsibilities and obligations came with identification of one's own genetic risk for a disease (e.g., Lippman 1991 and 1998, and later Novas and Rose 2000, Arribas-Ayllon et al. 2008). The susceptible person had new ethical obligations, including responsibility for managing health and reproductive behavior in light of new information. For example, Lippman (1998) was concerned that in the context of prenatal testing for Down syndrome, this redirection of agency and responsibility seemed to offer more choices but would in the end constrain mothers' choices and hamper public health efforts. Novas and Rose (2000) also observed that information about genetic predisposition for Huntington's disease created new responsibilities and prompted particular kinds of action, which they contrasted to previous expectations of passive resignation to a genetic fate.

Harvey (2009) and Marteau and colleagues (2004) examined diseases that also involved

¹ Some research has addressed the perceptions of people with high familial rates of depression (Laegsgaard et al. 2010), bipolar disorder (Meiser et al. 2005, 2007, 2008). I plan to do a more thorough literature review to identify whether there have been studies similar to mine for other physical and mental health conditions.

genetic predisposition but could be exacerbated or prevented through individual diet and exercise behavior (e.g., hypercholesterolemia). Unlike monogenetic disorders, where individual action could not prevent the outcome, in these more complex conditions individual agents were not limited to preparing for, managing, and preventing future generations from inheriting the condition; through changing their behavior they could plausibly prevent or alleviate the condition. These more complex conditions bring to the foreground different concerns about responsibility and agency in relation to a known genetic predisposition because one's own behavior can influence the outcome.

My study concerns yet a third kind of condition, for which individual behavior is incorporated into the definition of the condition and thus suggests a different relationship between genes, behavior and responsibility. The criteria for diagnosing AN or BN include behaviors as well as the results of behaviors. In AN, the “*refusal to maintain bodyweight*” (emphasis added) is listed along with its results, very low bodyweight and absence of menstrual period. In BN, most of the criteria involve behavior, namely binge eating and “inappropriate compensatory behavior” (e.g., vomiting). By contrast, a diagnosis of heart disease is not defined by current over-eating and under-exercising even if these behaviors may have contributed to the outcome. In heart disease, over-eating and under-exercising would be categorized as “lifestyle” behaviors that individuals can control, not part of the disease. If genes play a role in a condition that is defined in part by behavior, it suggests a third potential relationship between genetic predisposition and individual agency. Figure 7.1 provides an overview of my reasoning about three kinds of conditions, simplified to focus solely on genetics and behavior rather than more complete causal models.

Figure 7.1 Genes, behaviors, and responsibility in three different kinds of conditions

<i>Causal factors</i>	<i>Outcome</i>	<i>Responsibilities of the agent</i>
Genes →	Monogenetic disease	Managing, preparing for, and preventing inheritance of disease by future generations
Genes → Behavior →	Complex disease with genetic component	Changing behaviors to prevent disease (e.g., diet, exercise)
Genes → Behavior A → Behavior B →	Complex disorder defined by behavior and with a genetic component	Sort out responsibility by identifying which behaviors are “genetic” or “non-genetic” Manage “genetic” behaviors Change “non-genetic” behaviors Hybrid forms of responsibility

If behavior is the very problem that is explained by genes, one cannot think of behavior and responsibility in the same way as for solely genetic diseases, or for diseases where behavior influences but does not constitute the outcome.

My study sheds light on how respondents make sense of genetics and responsibility for what I argue is a third category of disorder. If genes influenced their behaviors, that meant that to some extent they “couldn’t help it,” and thus had less responsibility for causing or controlling the problem. Yet they were also held responsible for behaviors by themselves and others, despite genetic influence. The implications of genetic influence identified in Chapter 6 resemble responsibilities in both simple monogenetic diseases and complex diseases in which behavior contributes to the outcome. As with a monogenetic disease, my respondents identified a responsibility for seeking professional assistance to manage the disorder (though they also thought some might resign themselves to genetic fate) (see Table 6.1). And as with a complex disorder in which genes *and* behavior contribute to the outcome, respondents spoke of the potential for genetics to be used irresponsibly as an excuse to persist in unhealthy behaviors, implying that behaviors could be controlled. Arguably, respondents also identified a hybrid form of agency that corresponds to this third category of

disorder: the improved ability to stop a behavior precisely because one is no longer blamed for it.

2.2 *Changing cultural repertoires of accounts*

In order to understand the conceptual inter-relationships of responsibility, agency, and genetics in eating disorders, I place them in the context of historically changing cultural repertoires of accounts. In their classic article on accounts,² Scott and Lyman (1968) theorize an account as “a statement made by a social actor to explain unanticipated or untoward behavior” (46) which helps to maintain social order.

An account is a linguistic device employed whenever an action is subjected to valuative inquiry. Such devices are a crucial element in the social order since they prevent conflicts from arising by verbally bridging the gap between action and expectation. Moreover, accounts are ‘situated’ according to the statuses of the interactants, and are standardized within cultures so that certain accounts are terminologically stabilized and routinely expected when activity falls outside the domain of expectations. (46)

Accounts³ are provided when behavior is unexpected or deviant and they are standardized within cultures, suggesting that they may be different in different places and times. Scott and Lyman note that variation across time and cultures ought to be expected not only because behaviors that are questionable in one culture may not be in another, but also because different communities will use and accept different accounts. As suggested in the

² According to a review of the theory of accounts (Orbuch 1997), newer writing on accounts places “far less emphasis on the construction of accounts to justify unexpected or disrupted social interaction” (p. 456) than earlier writing, broadening the concept to include stories generally. I am using the theory of accounts in the narrower, older way, as do some contemporary sociologists who deal with “excuses” (e.g., Throsby 2007, Arribas-Ayllon et al. 2008).

³ This is not the only framework that could be used. Recent work by Boltanski and Thévenot (1999) on regimes of justification is relevant, as are several older theories (e.g., C. Wright Mills (1940) on vocabularies of motive, Weber on motive, Sykes and Matza (1957) on techniques of neutralization, Hewitt and Stokes (1975) on disclaimers). Biological accounts can be fit into other sociological literature as well, including medicalization (Conrad and Schneider 1992), stigma (Link and Phelan 1999), and the sick role (Parsons 1951).

introduction, genetic ideas are relatively new additions to a cultural repertoire or toolkit, specifically a repertoire of accounts.

Respondents imagined genetic accounts to be useful for explaining behavior that they and others were troubled by in order to restore a sense of themselves as acceptable, healthy members of a social order. My study calls attention to the historically changeable repertoire of accounts – genetic accounts have been available only relatively recently – and how that repertoire may affect individual action. Historical changes in repertoires of accounts – that is, the set of accounts honored in a particular place and time – connect macro-level cultural change to micro-level interaction and negotiation about behavior and action. Scott and Lyman note that among mid-20th century Italian-American male immigrants, “uncontrollable sexual appetite” was accepted and expected as an excuse for extramarital sexual behavior, as well as avoidance of being alone with a female relative (Scott & Lyman 1968 citing Gans 1962). It is easy to imagine the availability of such accounts would enable and constrain some kinds of behavior.

Based on my findings, I suggest ways that genetic accounts are important not only for maintaining social order, but also for action and conceptions of agency. If honored, genetic explanations can smooth conflict by accounting for undesirable behavior, but also produce social expectations that constrain or encourage new actions. According to respondents, the availability of genetic accounts for behavior would affect their behaviors, by changing expectations of what was possible to do without medical help, for example. Most interesting to me was that genetic accounts for behavior were capable of changing self-conceptions of agency. Knowing that an account would be honored made it easier or more acceptable to engage in the behavior, according to those who spoke of “excuses” (right column of Table

6.1). With an account at the ready, some actors might “get away” with behaviors more easily. Interestingly, the availability of an account also made it easier for an actor to stop the behavior, according to several respondents. Some who felt blamed and guilty about the disorder thought that a genetic account would enable them to re-interpret their behavior as non-volitional and themselves as less culpable, which would enable a more positive self-conception that appeared to confer the ability to act differently. Perhaps the account worked internally, to restore an internalized version of social order in which they were acceptable members of society rather than stigmatized, deviant outcasts who were “bad” or chose to be sick. Once redeemed, they were able to take action, as though the account were a way of rehabilitating their agency.

Extrapolating from my findings and reflecting on other cases of geneticization outside medical contexts, I perceive a growing potential for genetic accounts to re-categorize what people do from agentic “action” to biologically-based “behavior.” Behavior is thus somaticized, (to borrow a term from Rose 2007⁴), manageable by an agent but not willed or volitional. This transformation applies to not-currently-medicalized phenomena as well. The idea that homosexuality is genetic has been used to fight against the idea that it is a chosen deviant lifestyle that can and ought to be “cured” (Brookey 2002). The idea of genetic influence on political attitudes has been suggested as a way to resolve conflicts, or “to mute societal divisions” between conservatives and liberals (Alford, Funk & Hibbing 2005: 165) by re-categorizing differences not as moral but as temperamental and genetic: “Recognizing that our political antagonists probably have a different genetic predisposition to people, life,

⁴ Rose (2007) argues that the self has been “somaticized” from a psychological to a neurochemical self: “... over the past half century, we human beings have become somatic individuals, people who increasingly come to understand ourselves, speak about ourselves, and act upon ourselves – and others – as beings shaped by our biology. And this somaticization is beginning to extend to the way in which we understand variations in our thoughts, wishes, emotions and behavior, that is to say, our minds” (p. 188).

human nature, and politics may serve to ease frustrations and, eventually, to improve communications across the chasm.... [V]alue exists in recognizing that intransigence is not the result of willful bullheadedness but, rather, genetically driven differences in orientation” (p. 165). I am curious about (and disturbed by) the geneticization of political commitments and behaviors, not only because (1) differences are framed as permanent and biological, but (2) conflicts are framed as potentially resolvable precisely through recognition of this biological permanence. I see a similar theoretical move with eating disorders, and wonder what the effects of such thinking are, in social interaction.

3. Limitations of the study

My study is limited in a number of ways. I cannot generalize findings from this convenience sample to the broader population of people with eating disorders. The sample over-represents college graduates and current college students compared to the U.S. population. The sample excludes people with current eating disorders who are not in treatment, primarily to ensure that if non-recovered people found genetic ideas disturbing they would have ready access to a treatment provider. Respondents in treatment for eating disorders may have been disproportionately aware of genetics because they were drawn from a hospital-based program at a research university, as opposed to a private therapist or Overeaters Anonymous group.

In addition, the comparison between people in treatment and people who had recovered was not ideally achieved by my sample. Most people in treatment had been through more treatment than those who had recovered (see Table 2.4, Chapter 2). Therefore I was not confident that I could disentangle separate effects for current involvement in treatment compared to lifetime experience with treatment. I did not attempt multivariate

analyses for this small sample and opted to interpret current treatment as relevant to overall treatment experience.

Because I presented hypothetical scenarios and asked respondents what they or others might do *if* genetics were widely publicized or a genetic test were available, I have little knowledge about what they would *actually* do. A longitudinal study of respondents' reactions to individually tailored genetic information, or of their changing understandings following a publicity campaign about genetics over time, would better address the actual effects of genetic ideas on behavior. Similarly, an ethnographic study involving observation and interviews with respondents at more than one time might have afforded a view of actual behavior, rather than reports on hypothetical behavior.

My study is also limited because I am not certain which understandings of genetic influence go with what implications. When respondents spoke about implications, they drew on simpler models of genetic causality than when they imagined how genes could most plausibly influence an eating disorder. These simpler models were not always explicit and I only rarely probed about them and their connections to their more complex models (which they had described first). I do not know if the implications of the simpler genetic models also applied to more complex models. Maybe some implications only matter if genes “for” AN or BN are theorized, rather than genes “for” depression or personality. To answer this question in future research, it would be helpful when asking about hypothetical scenarios to ask respondents what kind of genetic theory they believe is implied by the scenario, and whether they find it plausible.

Another potential weakness of the analysis is its focus on segments of the interview rather than each interview as a whole. Taking the interview as a whole might have led to

identification of respondent “types,” differences, and resistance to ideas rather than descriptions of the central tendency and range of themes brought up during a segment. I divided my analysis by interview segments because the interview guide introduced progressively more specific content that shaped what respondents spoke about. While this approach might be seen as a weakness, its advantage was to distinguish clearly between spontaneous conceptions of causality and elicited thoughts about genetics, to name one example. These content “landmarks” provided the boundaries for each of the four chapters: Chapter 3 included material that appeared before their reactions to specific terms for eating disorders (e.g., “mental illness”); Chapter 4, material before I brought up genetics; Chapter 5, material prior to questions about how genetic influence might work; and Chapter 6, material preceding the introduction of hypothetical scenarios (i.e., media campaign and genetic testing). Through statistical analyses in Chapters 4 and 5 I linked material across interview segments, but additional synthesis will have to await future analysis.

4. Directions for future research

In the future I hope to analyze these existing data more broadly and deeply. I would like to focus on how respondents conceive of environmental influences on behavior: gender-related environmental influences, resistance to genetic explanations as distractions from social factors, the idea that some people may be genetically more sensitive to environmental influence, and the responsibility some felt for having been influenced by their environments. An examination of these themes would tie well to theories about geneticization as a force that individualizes responsibility for health and removes it from collective responsibility (Lippman 1998, Conrad 2007) and to recent genetic theories about “plasticity” genes that make individuals more susceptible to both enriching and harmful environmental influences

(Belsky et al. 2009). A more focused and detailed comparison of respondents' understandings of environmental and genetic influence with frameworks used by social scientists and biomedical scientists would identify the nature and extent of overlap, which would contribute to studies of science communication. Future analyses could also interpret the full diversity of perceived implications, beyond those centered on individual and social responsibility, to respondent expectations for future treatment, genetic testing, and stigma. Finally, I could assess whether the main findings of this dissertation vary by respondents' diagnosis and treatment experience (current treatment and extent of treatment).

Future research on genetic accounts would benefit from ethnographic, observational methods to study people with eating disorders in interaction with others, such as clinicians and relatives. The implications of genetics for eating disorders had much to do with guilt, stigma and blame, which necessarily imply other people's judgements. Observing genetic account-giving and identity negotiation in "real" time and space would provide a fuller picture. Respondents provided information about their own perceptions of others' understanding of genetics, but without observing interactions with others and the meanings of genetic explanations for them, it is difficult to assess actual effects. How do nurses and other care-providers make sense of genetic explanations and do they apply them to their patients or clients? How do genetic accounts fit with different treatment models and philosophies? Especially important, how do friends and relatives interpret genetic accounts, particularly those biologically related to the person with an eating disorder?

Family understandings are potentially important not only for learning about how genetics are understood in an important social context, but about how parents' understandings of genetics may affect children. Research on gene-environment interaction

for anti-social behavior (e.g., Caspi et al. 2002, Frick and Viding 2009) suggests the possibility that children might one day be genotyped for the purpose of intervening to improve their environments, thereby preventing problem behaviors or conditions. Even without a testable genotype there could be genetic or gene-environment interaction concepts that affect parents and how they think about and act upon their children's problem behavior. Research will be needed to illuminate parents' understanding, acceptance, and adaptations to such ideas, the consequences of new conceptualizations over time, and how to communicate ideas about genetics in ways that avoid some of the pitfalls my respondents identified. Longitudinal ethnographic and interview studies would be ideal for identifying changes in genetic understandings and their effects over time for families.

Longitudinal follow-up with respondents in this study of eating disorders would also shed light on the future of medicalization and geneticization for this complex condition. It is possible that specific genetic contributors for eating disorders and related conditions will be identified in the future. Will such a finding further promote medicalization for eating disorders? If genetics are found to be relevant only to one subtype of eating disorders, how will this be understood by respondents with various diagnoses? It is also possible that specific genetic contributors will never be identified. Will there be a subsequent re-invigoration of social causal attributions, i.e., greater environmentalization as opposed to biologization? How will future findings or non-findings about genetic influence on eating disorders be presented in the media and interpreted by people with eating disorders and those who treat them? Studying such developments over time would enrich and extend this study and I look forward to continuing this line of research.

APPENDIX
INTERVIEW GUIDES

The interview guide for respondents with a current diagnosis appears below. The guide for recovered respondents included the same questions but was edited to reflect past diagnosis and is not reproduced here. Guides for men and people with binge eating disorder were identical to the guide below.

Introductory Script

- Review purpose: To understand how people diagnosed with an eating disorder think about eating disorders, including the causes and risk factors, how much control people have over it, and what kinds of treatments make sense. There are no right or wrong answers, I just want to learn how you personally think and feel about these issues.
- This interview should last about 1½ to 2 hours, including a questionnaire that I will give you to fill out at the end.
- Can skip questions if you feel uncomfortable answering.
- The interview will be audio-recorded and transcribed only with your permission.
- The transcriber will not know your name and will not put proper names and other potential identifiers in the transcript. When I write about these interviews I will take out any remaining identifying details and give you a different name.
- I am not a clinician but if you become distressed I will help you get to one. Here is a list of resources that I'm giving everybody to keep in case it's useful at some point (Give list of resources)
 - (For UNC) I am not affiliated with UNC Eating Disorders Program. I do not have access to your medical record and I will not in the future. I will not talk to providers about your treatment nor attend meetings about your treatment.
- I will keep your name and contact information separate from any information you give me in this interview or follow-ups.
- Do you have any questions?
- You can decide about re-contact now or at the end of the interview.
 - Recontact: At the end of the consent form there is information about recontacting you in the future. You don't have to agree to that in order to do this interview; they are two separate things
- Sign Consent Form.

OK to begin the recorder now? (Begin recorders)

First I'll ask a few easy questions about yourself and then more open-ended questions about your experiences and opinions about eating disorders. Then I'll ask you about how causal factors for eating disorders including some specific causes. Again if you are uncomfortable answering any of the questions please just let me know and we can skip them.

1. BACKGROUND

First, I just want to ask some quick background information and then we'll turn to your experiences and thoughts about eating disorders.

How old are you?

How would you characterize yourself in terms of race and/or ethnicity?

How many years of school have you completed? (degrees)

What is your marital status?

Do you have any children? How many? What ages?

Do you have a job?

(If not, or in full-time treatment program) what was your last job/ job title?

(If so) What is your job/ job title?

And just to confirm, when we talked earlier I think you said you had been diagnosed with [DSM term]... is that right?

(If not recovered) Is that your current diagnosis?

Have you ever had another diagnosis? Which one?

2. PERSONAL EXPERIENCE WITH ED

What is your **current understanding** of [E.D.: relevant eating disorder]? (What it is)

CAUSE

Going back to the beginning, can you tell me how it started?

What are some causal factors that you think led to your [E.D.]?

Causal factors, Risk factors, contributing factors

Why do you think it started *when* it did?

Why do you think *you* developed it as opposed to other people you knew?

Do you think it could have been prevented in some way?

Has it come and gone or been pretty steady?

Are there things that make it better, or worse?

[If time is short, skip to BMI questions.]

Can you walk me through how you **first got diagnosed and treated**?

Age

Context – voluntary seeking treatment or something else?

What kind of treatment (e.g., what kind of care provider and facility)

(If unclear) What was the goal of the treatment – what kinds of behaviors did it address?

Subsequent treatment

Age, Context, What kind

BMI Questions

Have you ever been hospitalized for an eating disorder? Number of times?

(If AN): what was your lowest weight for your height?

3. PERCEPTIONS OF EATING DISORDERS

How do you feel about having [E.D.]?

How do you explain it to other people, if you do?

Thinking about others who know you had [E.D.], what kinds of reactions have you gotten?

Are there reactions you don't like? What are they?

How would you ideally want someone to understand [E.D.]

All in all, do you feel like [E.D.] has been a problem for you?

What are the main ways it has been a problem?

Have you always felt like it was a problem?

Did you ever see your eating issues as something other than [E.D.]?

4. REACTIONS TO SPECIFIC IDEAS ABOUT EATING DISORDERS

Now I would like to ask for **your reaction** to a few different ways of thinking about [E.D.]

How do you react to the idea of [E.D.] as a

psychological problem

mental illness

brain disease

physical illness

choice

lifestyle

problem with our society or culture

So, besides the ways I listed (read back list), are there other ways you think make sense to look at eating disorders?

Thinking about all these ways of looking at eating disorders, how do you see [E.D.]? (How would you choose between them or combine them?)

Have your ideas about what [E.D.] is **changed over time**? How?

How about [AN/BN], would you describe that any differently from [E.D.]?

5. SPECIFIC CAUSES

I just want to go back to causal factors for a moment.

Are there any other **factors** you can think of that can help cause a person to develop [E.D.], even if they don't apply to you?

Are some **kinds of people** more likely or less likely to develop eating disorders? How does that work?

Why do more **women and girls** have eating disorders than men and boys?

Do some **situations, settings or environments** make people more likely or less likely to develop eating disorders? (Explain)

Now I want to shift gears and ask for your reaction to some more specific causal factors.

Some say there are *social or cultural* causes for [E.D.]. This would mean that something about our culture made you more likely to have [E.D.].

Why do you think that more women and girls develop [E.D.] compared to men and boys?

Some say there are *biological* causes for [E.D.]. This would mean that something about a person's body or brain could make them more likely to have [E.D.]

Have you heard this before?

If YES → What have you heard?

Some say there are *genetic* causes for [E.D.]. This would mean that something about a person's genes could make them more likely to have [E.D.]

Have you heard this before? (How or where did you hear about it?)

What have you heard?

The rest of the interview will be about your ideas or impressions of genetics in particular (best guesses, off the top of your head)

What is/was your reaction to the idea of a genetic causal factor?

Was your reaction more positive or more negative?

Does it seem relevant or not for you personally? Why/why not?

Now I have some questions about **how** genes could influence or help cause [E.D.]?

I just want your best guess, your imagination, something off the top of your head

Would everyone with those genes develop [E.D.]? (explain)

Does everyone who has [E.D.] have those genes? (explain)

What makes more sense to you - genes for [E.D.] specifically, or genes for something more general that in turn influences [E.D.]? (explain)

One idea is that genes affect temperament or personality, which in turn can make [E.D.] more likely. What do you think of this idea? What comes to mind? (kind of personality/temperament)

If there is a genetic cause, do you think other causes would still matter?

Thinking about the different causal factors, which is/are the most important? (Not consistently asked)

6. HYPOTHETICAL SCENARIO 1: MEDIA CAMPAIGN

Imagine there were a media campaign to publicize genetic causes of [E.D.] to the general public. So this would be something like posters or advertisements saying "Genes matter for [E.D.]" or "[E.D.] has genetic causes"

What effects do you think this would have?

Thinking specifically about people with [E.D.], what kinds of effects do you imagine?

Could it help people with [E.D.] in any way?

Could it have bad effects for people with [E.D.]?

7. HYPOTHETICAL SCENARIO 2: GENETIC TEST

Because both genes and environment play a role it is not likely that a genetic test could ever predict whether a person will have [E.D.]. BUT for a moment let's say you could get a genetic test to find out if your genes made you more likely to have [E.D.].

(Clarify: I'm talking about you, as you are right now: you have already had an eating disorder so I'm not talking about predicting one ahead of time. This is about whether you would like to know that you were genetically susceptible or not.)

Would you want to know? Why? Why not?

Which would you rather find out: that you had those genes or that you did not? Why?

Let's say you found out you had the genes.

What are the good and bad things about knowing this?

Would knowing you were genetically susceptible change how you think about ...?

...Your experience with [E.D.]

...What kind of treatment makes sense for you?

...Your recovery

...Family members who are genetically related to you?

...Children, raising them

How do you think family members would react to the idea that you had those genes?

Would it change how you talk about [E.D.] to other people?

(Would you be likely to mention/not mention your genetic results?)

Some have said that talking about the genetics of eating disorders helps make it less like the person's fault. What do you think about this?

IF EXTRA TIME:

Why do some men develop [E.D.]?

Do you see [E.D.] behavior in so-called normal people

8. CLOSING QUESTIONS

If you were giving advice to someone with your eating disorder about how to think about it, what advice would you give? Why?

Do you have any advice for scientists who are researching eating disorders? (what to focus on, what not) (Even though you are not yourself a researcher)

Do you have advice for therapists or others who try to help people with eating disorders?

Is there anything you expected me to ask that I didn't?

Were any questions surprising or uncomfortable?

Administer EDE-Q and give \$40 incentive

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