Wesleyan ♦ University

‘SOMETHING THAT PEOPLE CANNOT CONTROL, SOMETHING THAT PEOPLE CAN OVERCOME’: PARADOXES IN DISCOURSE ABOUT DEPRESSION

By

Ethan David Hoffman

Faculty Advisor: Dr. Jill G. Morawski

A Dissertation submitted to the Faculty of Wesleyan University in partial fulfillment of the requirements for the Degree of Master of Arts in Psychology

Middletown, Connecticut **April, 2015**

Table of Contents

Acknowledgements

I cannot begin to express my gratitude to Jill Morawski, not just for her mentorship on this thesis but for all she has taught me in the two and a half years since I entered her lab. When I first came into Jill's lab, I was uncertain about my direction as a student in psychology. Since then, she has shown me how to reflect critically about the discipline and subject matter of psychology, to work collaboratively, and to never forget first questions and first principles. From her decision to forgo attending the 2013 APA conference in Honolulu (where she was to be honored with a lifetime achievement award) and to instead accompany N. Reed Myerberg and me to a small conference on the Milgram experiments in northern Ontario, to her invaluable mentorship throughout the master's program at Wesleyan, Jill has done so much for me. I am honored to have had the opportunity to study and work with her.

I would also be remiss not to thank Chuck Sanislow for his generous advice throughout this thesis, for opening my eyes to the world of clinical psychology, and for his work blinding my dataset. This thesis would simply not have been possible without his help and patience.

Thank you to Steve Woolley at the Institute of Living, exemplar of methodological precision, relentless drive and persistence, persistence, persistence.

A huge thanks to Dr. Woo-Kyoung Ahn and her doctoral student Matthew Lebowitz at Yale University's Thinking Lab for their outstanding scholarship and for agreeing to let me borrow their stimuli for this project.

I am also extremely indebted to the other students I have worked with in the Morawski Lab and elsewhere. Specifically, I would like to thank N. Reed Myerberg, for helping to introduce me to the wild west of critical theory, for the many long days of monastically combing the Milgram archives, and for his companionship from Paris to Bracebridge; and Sean Winnik, for always being there to bounce ideas and emotions against, for helping me drive across the country, and for inspiring me with his polymath abilities and interests.

I would like to extend my thanks to the ever-reliable Geneva Jonathan and Kristen Lynch for coding the data of this thesis with systematic sensitivity and dedication.

A resounding thank you to the Psychology Department at Wesleyan University for curricular and financial support over the last several years. The orientation of this thesis owes in large part to the interdisciplinary environment that the department and university have fostered and nourished.

To the residents and frequent guests of 49 and 80 Home Avenue, the WST (2010), Maybeck friends, the Prairie Schooner Crew, and Laëtitia Krisel—thank you for always being there for me with your love and support.

Lastly, but certainly not least, I want to give thanks to my family: to my father, for sharing his passions, showing me how to find beauty in the world, and inspiring me with his memory; to Olivia, you are my younger sister but I learn so much more from you than you could possibly imagine, especially from your compassion and your ability to persevere through any challenge; and to my mother, for teaching me how to be strong and believing in me even when I did not.

0. Abstract

Recent studies have demonstrated that explanations of depression as a biological illness lead individuals with and without depression to be more pessimistic about recovery and to feel less in control of their mood. However, this research has not examined the effects of biological explanations of depression in the discursive and social context in which talk of health and illness travels. The present study compares the effects of two psychoeducational interventions about the biology of depression, one deterministic and one malleable. Participants were exposed to one of the two interventions or were shown no intervention and were then asked to produce an open account of the cause and recovery from depression. Quantitative content analysis of open responses indicated that, relative to control, the deterministic biological explanation led to increased prognostic pessimism and decreased optimism while the malleable biological explanation increased pessimism without decreasing optimism. Qualitative content analysis reveals inconsistencies in participants' responses whereby the depressed person is represented as both in control of their recovery and not responsible for their descent into depression. This analysis also suggests differences between participants who view sadness and depression as existing on a spectrum and those who view sadness and depression as categorically distinct entities. The findings are discussed in light of the social and moral dimensions of health and illness accounts.

1. Introduction: Biomedicine and Psychoeducation in the Decades of the Brain

1990 was a landmark year in the world of mental health public policy. In that year, President George H. W. Bush proclaimed two major initiatives related to mental health care and research. In July, President Bush declared the 1990s the Decade of the Brain, drawing attention to "a new era of discovery" in brain research—research which promised improved treatments for depressive disorders, among other mental illnesses, and which evidenced the United States' "determination to conquer brain disease" (Executive Office of the President, 1990a). Several months later, President Bush declared National Mental Illness Awareness Week (1990b), which called for greater recognition of mental disorders and the stigma faced by those who suffer from them. The proclamation again pointed to brain research as promising an eventual cure to mental disorders.

Continuing policy interests in the brain sciences and biomedical approaches to mental disorders were by no means confined to the 1990s, as can be seen with the 2013 launch of President Barack Obama's BRAIN Initiative, a multi-billion dollar funding project with the aim of advancing technologies to map the human brain and helping "researchers uncover the mysteries of brain disorders, such as Alzheimer's and Parkinson's diseases, depression, and traumatic brain injury" (White House, n.d.). Also in 2013, the National Institute of Mental Health (NIMH) announced that it was shifting gears away from the Diagnostic and Statistical Manual of Mental Disorders (DSM) and would instead use its new Research Domain Criteria (RDoC) as a guide

for research and funding. Although some have emphasized this new framework's potential for facilitating "explanatory pluralism" (Sanislow et al., 2010, p. 633), according to Thomas Insel, the director of the NIMH, the RDoC rests on the assumption that "mental disorders are biological disorders involving brain circuits that implicate specific domains of cognition, emotion, or behavior" (Insel, 2013; see also Insel & Quirion, 2005). What is interesting about such recent statements is not so much their scientific validity (whether or not mental disorders *really are* biological disorders), and not so much whether such statements provide a good framework to wise funding decisions, but the extent to which they signal the broad based acceptance of biomedical thinking in the scientific community. In rhetorically placing the frontier of mental health research at the neurobiological level, these initiatives are indicative of our "fascination with all things brain, a fixation that extends far beyond the spheres of science and medicine and infiltrates virtually every corner of daily existence" (Thornton, p. 2).

In addition to the growing "saturation" (Thornton, 2011, pp. 114-5) of brain discourse in the public sphere and the increasing institutional support for biomedical research in psychiatry, the proclamation of Mental Illness Awareness Week and the inception of Anxiety and Depression Awareness Week in 1994 (Freedom From Fear, n.d.) suggest a growing concern in government as well as in civil society about the stigmatization of people suffering from mental disorders. In its public policy statement on stigma and discrimination, the National Alliance on Mental Illnesses (NAMI) states that:

> Mental illness is essentially biological in nature. Mental illness affects behavior and behavior can affect mental illness—but mental illnesses are not behavioral. The term "behavioral health" obscures and hinders effective treatment of co-occurring disorders. Also, because behavior is perceived as a matter of choice ("good" or "bad" behavior), the very term "behavioral health" can add to the stigma and discrimination endured by people living with a mental illness. (National Alliance on Mental Illness, 2014)

The inclusion of a statement about the etiology of mental disorders within an account that otherwise describes the consequences of mental disorders is revealing. This subsumption suggests that an explanation of mental health does not consist of meaningfully separable appraisals of *cause*, *severity*, *duration*, or *social outcomes*, but instead must be understood as a narrative whole. This inclusion also exemplifies the intertwining of representations of the etiology of mental illness with questions of agency, and suggests that certain explanations of mental disorders might lend themselves to or come packaged along with a particular understanding of what it means to be a person. Such close linkages between personhood and biomedicalization are not a new observation. Within science studies and related fields, the impact of biomedical discourse on contemporary personhood has garnered significant scholarly attention in recent years (Martin, 2010; Ortega, 2009; Rose, 2003; Thornton, 2011; Vidal, 2009).

Biological explanations, according to NAMI, are supposed to be less stigmatizing because—apparently unlike psychological explanations—they do not implicate the individual's agency in the etiology of mental disorders. And while recent research has suggested that biomedical explanations might reduce feelings of guilt or responsibility amongst patients with depression (Deacon & Baird, 2009;

Goldstein & Rosselli, 2003; Mehta & Farina, 1997; Phelan et al., 2002); these biological understandings might also have some unintended "side effects" (Haslam, 2011; Kvaale et al., 2013): greater pessimism about recovery, increased hopelessness and reduced feelings of agency (Kemp et al., 2014; Lebowitz et al., 2013). Greater pessimism about recovery has been documented in numerous correlational studies (Brown et al., 2007; Lebowtiz et al., 2013; Rüsch et al., 2010) as well as in several recent experiments in health psychology (Deacon & Baird; Kemp et al., 2014; Lam & Salkovskis, 2007; Lebowitz et al., 2013). However, this recent experimental literature has largely neglected the aforementioned scholarship in science studies, and the task of relating these negative "side effects" to changing forms of personhood remains largely unaccomplished.

In an era of continuing commitment to the biological paradigm, it is crucial to ascertain how the scientific stories we tell about mental health fare once they are disseminated in the public sphere, and to establish the kind of life they live. This project moves beyond the question of how such biomedical explanations effect treatment decisions and attitudes, to examine how these explanations affect accounts of depression and personhood more broadly conceived. To this end, the present study elaborates on the aforementioned experimental research on biological explanations of depression (Lebowitz et al., 2013) and ties this research to discursive and dynamic theories of personhood. Methodologically, this entails looking at the effects of different psychoeducational strategies using both qualitative and quantitative methods. Through content analysis of accounts of depression under various

experimental conditions, this study aims to validate extant findings about the effects of biomedical conceptions of depression, as well as to describe and more fully explore the shape and dynamic nature of accounts about depression. It is believed that a more nuanced description of how people account for depression must precede any attempt to enrich or inform those accounts.

2. Literature Review

2.0 Overview of Chapter Subsections

The present chapter ties together several threads of literature on health and illness representations as they relate to the topic of psychoeducation about depression. This review argues that failing to consider accounts of depression in both their proximal and distal social contexts has theoretically hampered recent experimental studies by occluding the variety, flexibility, and multiplicity of depression accounts, as well as obscuring potential transformations in personhood that take place in such accounts. Better understanding of the dynamics of depression accounts might help explain why biomedical explanations have negative effects on expectations of recovery and feelings of agency. However, consideration of the discursive and socio-historical aspects of health and illness accounts necessitates a shift away from traditional survey methods of research towards textual analysis.

In addition to a theoretical discussion of health and illness accounts and the dynamics of public understandings of mental health, the review previews key categories to be used in this study's analysis: biogenic and other etiologies of depression, prognosis or outlook towards depression, treatment preferences, strategies of discourse, and shifting forms of personhood. While such categories are, in the end, not separate aspects of accounting but rather connected parts of a narrative whole, examining them separately allows the present study to be put into dialogue with earlier experimental studies on the effects of biomedical explanations of depression.

This review of the literature on explanations of health, illness, and depression specifically is divided into four sections. The first section presents a brief overview of *what* the biomedical explanation of depression is and gives evidence that its endorsement is increasing amongst experts and members of the public alike. That such understandings have become increasingly biological suggests the importance of investigating the effects of biomedical explanations of depression. The next section gives a brief theoretical discussion of this study's aims and assumptions, followed by a critical comparison of traditional cognitive models of health and illness *representations* with alternative, more socially attuned conceptions that provide a more nuanced description of health and illness *accounts*. In order to situate contemporary biomedical discourse within a broader socio-cultural milieu, the chapter's third section highlights research and theory about how knowledge travels between the public and the scientific community, specifically psychology and related disciplines The final section returns to research that has looked specifically at the impact that biomedical explanations of depression have on behaviors and public attitudes related to depression, foregrounding the design of the present study. A consideration of these recent studies in light of scholarship on talk about health and illness indicates that research on mental health education and stigma reduction needs to move beyond an examination of beliefs about depression as static representations to a more process-oriented and context-sensitive investigation of how members of the public negotiate variegated discourses of depression and what social and moral goals they accomplish in giving an account of depression.

2.0.1 Note on Terminology

This study considers not only the understandings that the public holds about health, illness, and specifically depression, but also scholarly understandings of those public understandings. Psychologists, sociologists, and others have forwarded a dizzying array of labels for these public understandings: beliefs, attitudes, explanations, conceptualizations, representations, accounts, narratives, models and lay-theories, to name just a few. This situation can make for quite a terminological mess. Although it is necessary to create a distinction between the understandings held by the public and the scholarly understandings-of-those-understandings in order to elucidate how knowledge travels between science and society, forcing a sharp separation in lay versus expert terminology would erroneously suggest that these understandings live in different worlds from each other. They do not. As Richards (2002) has underscored, professional psychological language does not arise out of nowhere. Rather, professional psychological language borrows, reconceives and assimilates extant everyday psychological knowledge that is at once prior to psychology (after all, the discipline of modern psychology is little more than 150 years old) and, conversely, is molded and reconfigured by the psychology that seeks to describe it. This dynamic conceptualization of psychology-as-scientific-discipline and psychology-as-object-of-study is discussed further below in Section Three of the present chapter (pp. 22-27).

2.1 A Definition of the Biomedical Model

The biomedical view of depression, known interchangeably as the "biological model" (Cowen, 2008) or the "neurochemical imbalance model" (France et al., 2007), refers to a model of depression as a fundamentally physical disease. While by no means the only model circulated among scholars and practitioners, it is the dominant understanding of depression employed by contemporary clinical researchers (Deacon & Baird, 2013). Engel (1977) has described the biomedical model as an illness-model that operates on the presupposition that all psychiatric disorders are rooted in somatic or biological abnormality. Social, psychological, or behavioral factors are deprioritized or outright neglected under the biomedical model; moreover, when these non-biological factors are mentioned, they are explained as the product of physiological deviance (Engel, p. 130). Writing more than 30 years later, Thornton has noted that increasingly, even beyond psychiatry, explanations of human difference, behavior, and health must rhetorically "'pass through' the brain" to achieve a significant truth effect (Thornton, 2011, p. 126).

The biomedical model of depression offers an explanatory framework for depression that views biological abnormality as the root cause of mood disturbance. A 2011 educational publication by the NIMH exemplifies this framework. While paying lip service to psychosocial causes and risk factors and briefly mentioning psychotherapeutic treatments, the resounding message is that depressive illnesses are disorders of the brain (National Institute of Mental Health, p. 6). The depressed brain is thought to have a neurochemical imbalance and to "look different" (p. 6) than the

normal brain. When psychosocial factors are brought into the discussion, they are brought in as "triggers" that unmask an underlying biological vulnerability. While the loss of a loved one, workplace stress and trauma are considered in the etiology of depression as stressors, they are ascribed a less fundamental role than the biological diathesis.

The aforementioned NIMH publication's discussion of treatment options for depression further reinforces the primacy of biology over the psychosocial. Nearly 1600 words are devoted to antidepressant medications and other biomedical interventions like electroconvulsive therapy or vagus nerve stimulation, with the section on psychotherapy consisting of 200 words—half of which reviews combinations of medication and psychotherapy. Cognitive behavioral therapy is suggested to help restructure the negative thought patterns that are "*contributing* to the depression" or "making it *worse*" (National Institute of Mental Health, p. 16, emphasis added). Just as the death of a parent or spouse is described as a trigger but not as a primary cause, therapy is characterized as treating *contributing* factors and not primary factors. Only once in the entire document of 25 pages is a psychosocial factor mentioned as a possible "cause" of depression (p. 16). In summary, while the NIMH educational booklet does mention the social and psychological, their inclusion is overarchingly contextualized through a biological framework that they must rhetorically 'pass through.'

Given the predominance of the biomedical model of depression in contemporary scientific and medical discourse, what are its alternatives? Alternatives

can be found, for instance, in the history of Western psychiatry and clinical psychology. Psychoanalytic or psychodynamic accounts of depression or melancholia (Freud, 1917) emphasize the causal role of life events or patterns of attachment (p. 250; see also Abraham, 1927). Other more sociologically oriented thinkers such as Simmel (1903) or Szasz (1974) have proposed that mental suffering of all sorts has a sociogenesis; that is, that mental disorders result from social and political injustices and inequalities. Still others with a more cognitive orientation have pointed to repetitive negative thinking patterns as the root of depression or anxiety (Beck, 1963). Indeed, while biomedical conceptions are predominant among experts, their views are by no means homogenous (Ahn et al., 2009). Such variegated and diverse views evident *within* the discipline of psychology on the nature of mental disorders are but a microcosm of the intellectual variety that exists in the public sphere. Explanations for mental disorders vary widely across languages (Beeman, 1985), cultures (Jorm et al., 2005; Karasz, 2005; Keyes, 1985; Ryder et al., 2008), generations (Farrer et al., 2008; Jorm et al., 2006; Lawrence et al., 2006), genders (Emsilie et al. 2006; Schreiber & Hartrick, 2002), and ethnic and racial groups (Carpenter-Song et al. 2010; Schnittker et al., 2000). Alternatives to the biomedical model can also be found in contemporary self-help discourse. Recent books like *Happiness is a Choice* (Minirth & Meier, 2013) or *Emotional Discipline* (Manz, 2003) have suggested that depression can be avoided by adopting healthy mental habits and possessing cognitive rigor, a position echoing the historical association of sinfulness with acedia, a depression-like illness (Jackson, 1981).

Amidst this diversity, it appears unlikely that biomedical models operate autonomously from other modes of explanation. For instance, although the Western public attributes a chemical imbalance as a cause of depression more frequently than any other cause or factor (France et al., 2007), empirical research into lay and expert understandings of depression has indicated that the view is not unanimous and might represent only a slight plurality (Ahn et al., 2006). As noted in the previous discussion about the NIMH's brochure on depression, expert views on depression *do* at the very least pay lip service to an integrationist or pluralistic model of depression. It might be the case that, as with the professional eclecticism, biomedical conceptualizations of depression exist alongside or are embedded within other understandings of depression amongst members of the public (O'Connor & Joffe, 2013). Instead, as Thornton (2011) has observed, the role that neurobiology plays in public discourse is multiple, and can be mobilized to support a variety of (at times oppositional) arguments and conclusions. While the brain can be understood as determining or producing the mind, notions of a plastic brain also facilitate an inversion of that causality (pp. 57-62). The ability of brain rhetoric to discursively switch between these two causalities "is not a problematic contradiction… but rather a source of rhetorical power" (p. 62). A key shortcoming of much health psychology research on representations of depression has been the neglect of this flexibility and variation, and how such contradictions present discursive benefits to those who speak about depression. It remains an open question whether the public is able to mobilize notions of a plastic brain when presented with the biomedical model of depression, or

whether such a model, as Lebowitz et al.'s (2013) research suggests, generally results in a diminishment of perceived agency or reduced sense of control over the disorder.

Despite alternative models and vocal critics, endorsement of the biomedical explanation of depression has been steadily growing among laypersons. Using data from the General Social Survey, researchers have found that the American public's endorsement of neurobiology as a cause of depression increased roughly 13% from 1996 to 2006 (Pescosolido et al., 2010). Whereas in 1996 78% of respondents attributed depression to "the normal ups and downs of life," the most commonly cited cause for depression at that time, by 2006 the percentage of respondents endorsing "ups and downs" as a cause for depression had fallen to 67%, with a "neurochemical imbalance" instead gaining the highest level of endorsement at 80% (pp. 1322-4). Endorsement of biomedical explanations might be particularly pronounced for patients. In a meta-analysis of research on patients' explanations for depression, Thwaites et al. (2004) found that people affected by depression tend to endorse biological causes for depression more strongly than those without experience of depression. Another meta-analysis of surveys throughout Western countries revealed a 19.2% increase in endorsement of genes as a cause of depression from 1990 to 2006 (Schomerus et al., 2012, p. 443). These results show that regardless of multiplicity and variation, the public in both the United States and elsewhere in the Western world has increasingly come to see depression as biological. A central question, then, is to determine how exactly this biological account gets mobilized in discourse and

whether it accommodates other kinds of etiologies about or treatment models for depression.

2.2 Theorizing Accounts of Health and Illness

As noted in the preceding section, the history of psychiatry and clinical psychology has been marked by continued debate about the etiology of depression and mental illness more generally. These debates seek to get at the truth of mental disorders, to find out 'what is really happening.' Instead, following Thornton (2011), it is crucial to focus *not* on the validity of various explanations of mental health but on the way we *speak* about mental health. It is typically assumed that the way we construct an explanation or understanding of mental health "is real, in the sense that it has force in our lives" (Thornton, p. 34). This is not to claim that experiences of mental health and illness are wholly constructed by our discourse about them, but rather to recognize how those experiences are shaped, modified, and reinterpreted in light of this discourse. This perspective—that the way we speak about mental health will affect our attitudes, behaviors and social interactions—allows one to bracket the question of *what* is the scientifically valid way to speak about depression and to turn, instead, to the question of *how* people conceive of and speak about depression. The importance of discourse is further underscored by the findings that explanations alone can exert powerful changes in prognostic expectations and feelings of control (Deacon & Baird, 2009; Lam & Salkovskis, 2007; Lebowitz et al., 2013).

Attention to the ways in which understandings of illness impact attitudes and actions inherits much from research on health beliefs and cognitive representations of health and illness. Researchers utilizing Rosenstock and Becker's Health Beliefs Model (Becker, 1974; Rosenstock et al., 1966) and later similar theoretical frameworks such as Leventhal's Common Sense Model (1980) have sought to ascertain how individuals' beliefs about illness and treatments affect their decisions and actions with regards to prevention, identification, and treatment of illness. According to such cognitive theories of illness representations, questions of what symptoms should warrant concern, when to see a doctor, and whether to ask for medications depend upon what we know and believe about particular illnesses—like how long an illness is supposed to last and how dangerous an illness is.

Although numerous systems for dimensionalizing illness representations have been proposed (notably those coming from an attribution theory framework), Leventhal's Common Sense Model has seen the greatest usage across the literature. Instruments based off of the common-sense model, such as the Illness Perception Questionnaire (Weinman et al., 1996) or the Implicit Models of Illness Questionnaire (Turk et al., 1986), abound in studies of the behavioral and attitudinal effects of illness representations. Leventhal et al. (2003) claimed that these representations consist of five content domains: (1) *identity*, the name of the illness and what symptoms it is supposed to present with; (2) *timeline*, how long the illness is expected to last and whether it persists chronically, cyclically, or only acutely; (3) *consequences*, in terms of how the illness is perceived to disable the individual or

impact the individual's social status; (4) *cause*, or what is believed to have triggered the illness and; (5) *control*, or what actions are perceived to mitigate or cure the illness and how effective those actions are expected to be (p. 49). Leventhal proposed that these beliefs are called upon either when a somatic cue suggests that something is wrong with our body or in reaction to an external suggestion of risk for illness conveyed by the media or by health professionals, or by observation of someone else falling ill (p. 47). When the bodily cue or social suggestion reaches a threshold where fear becomes intolerable, it produces the need to reduce the danger of illness or otherwise manage the fear of illness through an action plan or coping mechanism that accords with one's beliefs about the illness (Rosenstock et al., 1966, Leventhal et al., 2003, p. 48).

According to these cognitive theories, plans of action are governed by individuals'' understandings of what particular treatments and preventions are available and appropriate for the given illness, and how beneficial or costly a particular course of action will be. Assumptions about the cause of a given illness are expected to lead individuals to seek treatment that is theoretically congruent; that is, treatment that is consistent with the patient's etiological beliefs. Conversely, practical experience with treatment is also thought to influence beliefs about cause and treatment. For instance, Leykin et al. (2007) found that patients who had already successfully undergone treatment with cognitive therapy were less likely to endorse antidepressant medication as an efficacious treatment, demonstrating that patients

tend to reject explanations that are discordant with whatever treatment has worked for them.

The Health Beliefs Model and the Common Sense Model both emphasize the influence of health representations on our motivations and strategies for achieving and maintaining health. Moreover, insofar as Leventhal's identity domain consists of assigning an illness label to a set of actual physical experiences and symptoms, health representations influence not only action but also subjectivity more broadly. Cognitive models of health representations acknowledge that the experience of being sick is not just the physiological result of disease but also the psychological result of appraising of the body as ill. These cognitive theories provide "details of the building blocks of lay representations—the immediate characteristics, the consequences and causes" of illness (Murray, 1990, p. 89). Synthesizing these frameworks, Murray has contended that what these cognitive theories have in common is their detailed description of the dimensions and properties of illness beliefs and their acknowledgment that these dimensions and properties might not correspond to the actual disease itself.

Starting with the development of the Health Beliefs Model, from the 1950s through to the present, a key concern of cognitive models of health beliefs has been to determine "why and under what conditions people take action to prevent, detect and diagnose disease," a determination that might eventually enable researchers and health workers to "to persuade people to modify their health practices" (Rosenstock, 1966, p. 94). A related focus has been on individuals' inability to make rational health

decisions, even in the face of sufficient information about risk and preventative measures (Rosenstock, 1966, p. 114-115; Joffe, 2003). From the 1960s onward, health psychologists have insisted that we do *not* think about health simply in terms of rational risk-reduction. Instead, health psychologists have been concerned with cognitive shortcomings as "barriers to action" (Rosenstock, pp. 99-100) and how to persuasively overcome these barriers—as exemplified by a strong emphasis on fear and other emotions as motivators for healthy behavior (Dillard & Nabi, 2006; Soames, 1988) or on the effective framing of health information (Gallagher & Updegraff, 2012; O'Keefe & Jensen, 2009; Rothman & Salovey, 1997). In the effort to surmount these barriers, these cognitive theories of health representations have adhered to a deficit model of public understanding.

Wynne (1993) proposed the term *deficit model* to describe and critique a view of laypersons' knowledge as deficient and subjective in comparison to scientists' full and objective understanding. Following Wynne, Joffe (2002) argued that cognitive theories of health representation, despite attending to the material and affective dimensions of health representations, perpetuate:

> The downgrading of emotive, value-based aspects of human thinking, by privileging rational structures of the mind. The explicit low status accorded to the emotional underpinnings of 'perception' belies the apparent openness to moving away from conceptualising the ideal response to risk as rational information. (p. 152)

Thus, although cognitive theories like Leventhal's Common Sense Model (2003) attend to, and indeed focus on the material and affective bases of health representations, they represent these bases as *subtractions from* and not as *additions*

to a purely rational representation of health. In research employing these cognitive theories, affective, and social contexts are examined, but only insofar as they detract from a rational or objective understanding of health and illness.

Researchers interested in the social context of representations of illness have identified other limitations to the dominant cognitive theories. Murray (1990) claimed that cognitive theories of illness representation are static and do not provide a detailed understanding of health and illness representations' location within a socially and historically contingent context (p. 89) or their "dynamic and unfolding qualities" (p. 65). Flick (2000) has noted that beyond neglecting the socio-cultural reasons why certain representations differ from one individual to another, these cognitive theories fail to consider how culture-wide themes of discourse about health and illness more broadly are socio-culturally situated. One alternative to these static and deficit model conceptualizations of health and illness representations is Moscovici's social representations theory (1988).

Social representations theory views representations of health, among other domains, as not existing solely in the individual but also in the collective (Moscovici, 1988, pp. 243-4). Action taken to address an externally perceived health threat or internal physiological cue results from a process that is as much emotional, symbolic, and social as it is informational and 'rational' (Joffe, 2003). Health representations are not only social in the sense that they have social origins in science, medicine, the media, and other disseminators of health information—a view already endorsed by Leventhal (2003) and Rosenstock (1966); in short, it is not simply that health

representations *come from* the social. Health representations are also radically social in the sense that they *reflect* society. When presented with a novel or unfamiliar concept or perception, "we begin by anchoring it to an existing social representation. The whole entity acquires an everyday meaning in the process" (Moscovici, 1988, p. 235). Such processes are evidenced in Thornton's observation that contemporary brain rhetoric resonates strongly with the logic of the neoliberal economy: by conceiving of the psyche in terms of quantifiable neurochemical events and processes, mental life becomes something that can be calculated, assessed, and rationalized much like an investment or banking decision (p. 113). The similarity between neurobiological paradigms and economic paradigms illustrates the extent to which society and accounts of health are "interdependent" and reflect each other (p. 36).

There is one further sense in which health representations are social: health representations are social in the sense that they are *relational*, that is, always spoken by a person to another person in a particular social situation. According to Radley and Billig (1996), the study of health representations is not so much the study of an object lying deep within a person's reservoir of memory and perceptions but the study of the activity of representing or accounting (pp. 223-4). They noted:

> People do not merely have health beliefs, as they might have eggs carried in a shopping basket. They also construct their state of health as part of their ongoing identity in relation to others, as something vital to the conduct of everyday life. (p. 221)

Considered as a relational process between two social beings, health and illness discourse is not simply the expression of what a person believes and holds to be true

about health or illness. Beyond making claims about the scientific or medical truth of health and illness, through health and illness accounts, individuals also negotiate their position as "more or less 'fit' participants in the activities of in the social world" (p. 221). Furthermore, a new line of inquiry is opened up by conceiving of health and illness accounts as the dynamic and transient activity of engaging with one's own body and with the world (both medical and non-medical objects, experts and non-experts). When the full relational, unfolding, and social nature of these accounts is acknowledged, the question moves from 'how do health beliefs affect individuals' actions?' to 'what do individuals accomplish in accounting for health and illness the way they do?'

2.3 The Etiology of Etiologies

Radley and Billig's (1996) approach to illness provides a promising model for empirically investigating how health and illness accounts are locally situated and arise in particular contexts in relation to specific and immediate social demands. In short, their work lays the foundation for a bottom-up approach to health and illness accounts. However, Radley and Billig's model alone does not give a full picture of how culture-wide modes of explanation, biomedical or otherwise, are common amongst broad swaths of the public, and how this public (and more specific constituencies of patients) reflexively relates to the knowledge that describes their health and illness. Just as individual instances of accounting for mental health and illness are more than the manifestation of a static set of intra or interpersonal beliefs,

more general modes of discourse about mental health are not simply the received wisdom of clinical psychology and psychiatry but the dynamic interplay of knowledges, experts, and laypersons alike.

Scholars in the public understanding of science have sought to ascertain how laypersons come to understand scientific information. Similar to cognitive models of health and illness representations, early 'science literacy' models of the public understanding of science stressed a top-down dissemination process and attributed "a knowledge deficit to an insufficiently literate public" (Bauer et al., 2007). Critics of this deficit model have noted that it leaves no room for negative attitudes towards science or the contestation of textbook facts, and additionally that it precludes the co-existence of superstitious beliefs alongside acceptance of scientific knowledge (Bauer et al., p. 82). Responding to these critiques, contemporary approaches to the public understanding of science have attended to the pre-existence of local knowledge of matters about which the scientific community wishes to educate the public (Brossard & Lewenstein, 2010). Such a shift in frameworks parallels a turn away from static cognitive models of health beliefs—where the public's knowledge of medicine is measured against scientific truth—and towards a contextualized account which acknowledges both the interaction of novel information with *a priori* ideas and experiences concerning health as well as the way in which these ideas and experiences are anchored in extant and socially distributed conceptual frameworks. This model also shares with historical psychology and science studies, among other fields, an emphasis on the dynamic interactions between laypersons, scientific

institutions, and scientific ideas.

Scholars within science studies and historical psychology have sought to articulate how knowledge in the psychological sciences emerges out of the social world and also necessarily alters that social world. More broadly, these dynamic historical models "challenge the conventional boundaries between social reality and accounts of social reality, scientific knowing and popular knowing, knowledge and culture, and the real and nominal" (Morawski, 2001, p. 409). Hacking's (2007) notion of the "looping effects of human kinds," Rose's (2003) account of neurochemical selfhood, and Thornton's (2011) analysis of brain culture all give instructive models for how patient groups who are themselves the objects of empirical research are effected by the accounts generated by that research, and, in turn, effect the shape of future research.

Hacking's (2007) model of the looping effect of human kinds proposes that the lived experiences of groups of people classified and studied by the human sciences are not antecedent to the research on those groups. Hacking has claimed that only after science identifies a previously uncategorized group of people for study does that identity become a way for that group to experience themselves in the world (p. 304). Through the routine processes of counting, quantification, norming, medicalization, and biologization, science is not only engaged in learning about people but also partakes in "making up people" (p. 305). The dynamism does not stop there—these new ways of being a person are not static identities but are "moving targets," continually changing in relation to what experts, institutions, society, and the

individuals who are themselves classified know about the classification. In the case of depression, the lived experience and the moral situation of the depressed patient is not what it was before biomedical inquiry—such inquiry illuminates previously unknown biological correlates of depression, and in so doing, relieves depressed individuals from personal responsibility for their depression (Hacking, p. 310).

Rose (2003) has provided a similar account of the interface between biomedicalization and personhood (one dimension in the aforementioned dynamic process) that is focused particularly on how shifting public understandings of depression relate to broader changes in personhood. Drawing on the discourse of psychopharmaceutical advertisements, Rose has proposed that the late 20th century development of a diverse psychopharmacopeia has led the public not just to view mental illness and medicine in different ways than they had previously, but "to understand our minds and selves in terms of our brains and bodies" (p. 46). At the same time that modern neuropharmacology has contributed to the "serotonin hypothesis of depression" (Read: biomedical model) (Rose, p. 47), the late 20th century has witnessed a transformation of personhood whereby we no longer view ourselves, at the core, as beings inhabiting a deep and unconscious psychological space. Instead, Rose has observed, this depth psychology of the self has come to be supplemented by a "somatic individuality" (p. 54).

Thornton (2011), among others (Martin, 2010; Ortega, 2009; Vidal, 2009), has speculated that the rise of neurochemical selfhood and the biomedicalization of depression lies in the promise that individuals can monitor, control, and optimize their

bodies not just *away* from pathology, but also *towards* an ideal of functioning. Problems of living, hopes for self-improvement, and aspirations for self-knowledge are increasingly understood by the subject in terms of projects fundamentally involving the body (Thornton, p. 12); witness the emergence and popularity of mind-body connection discourse, yoga, and brain training regimens, alongside the explosion of the psychopharmaceutical industry with its market-driven emphasis on developing medications that target particularly *chronic* illnesses (Rose, 2003, p. 54). Health, in short, has become an unending project in need of continuous effort and renewal (Thornton, p. 18)**.** In relation to the growing 'healthism' of our society, the brain and neurobiological explanations provide a rhetorically useful way of understanding ourselves. Particularly so in the context of mental health: the brain provides both justification for why one is suffering and a location to intervene in that suffering. The brain can both provide moral relief from feelings of responsibility about being depressed and suggest actionable measures to remedy depression.

Dynamic theories of biomedicalization, neurochemical selfhood, and brain culture connect the biomedical model of depression with broader changes in the forms of personhood prevailing in contemporary society. Thornton (2011) and Rose (2003) have drawn attention to the broad saturation of Western culture with biomedical discourse, and Hacking's (2007) looping theory gives a formal structure for how these discursive developments travel between the science of depression, institutions of healthcare, and depressed patients themselves. In short, such dynamic theories can be seen as supplying the top-down complement to Radley and Billig's

(1996) bottom-up picture of health and illness accounts. In addition to emphasizing the social life of accounts about depression, the relational, discursive, and dynamic theories reviewed above suggest that much more is at stake in accounting for depression than perceptions of prognosis or feelings of control over mood. At stake is one's personhood itself.

2.4 The Effects of Biomedical Explanations

Recent experimental and survey research has provided some tentative support to the claim that the politics of personhood and agency are deeply intertwined with explanations, representations, and accounts of health and illness. Just as pre-treatment expectations about the efficacy of antidepressants have been demonstrated to influence the power of the placebo effect (Krell et al., 2004), beliefs about depression have been shown to effect treatment outcomes. Several studies have determined that having an expectation of shorter illness duration at baseline improves middle-term treatment outcomes (Glattacker et al., 2013; Lynch et al., 2015), although another study has failed to find a significant effect (Dunlop et al, 2011). Patients' etiologies of depression have also been established to be significant predictors of treatment preferences. For example, Meyer & Garcia-Roberts (2007) concluded that endorsement of a biological etiology is correlated with motivation to receive medication, whereas endorsement of childhood as a cause of depression is associated with motivation to pursue psychodynamic therapy. Other research has demonstrated that giving patients a treatment they prefer leads to improved treatment outcomes (Lin

et al., 2005). This effect is supported by a recent meta-analysis that determined that, in addition to experiencing better outcomes, patients who are given preferred treatment are half as likely to drop out of treatment as those not matched by preference (Swift & Callahan, 2009). This research demonstrates that treatment outcomes can be influenced not just by prognostic optimism but also by the overall coherence of patients' beliefs about etiology with the treatment that they receive.

Research specifically addressing the effects of *biomedical* explanations on patients and nonpatients alike has indicated that biomedical explanations impact, among others, two key psychological variables: perceived agency and prognosis perception. Prognosis perception refers to patients' expectations for the course of illness, specifically the perceived duration and progression of the illness with or without various treatments and not to the patient's perceived ability to *affect* the course of illness, which is a matter of perceived agency or control. In studies of populations that are both depression-symptomatic and non-symptomatic, pessimism about recovery has been shown to correlate to endorsement of biological etiologies (Brown et al., 2007; Lebowitz, 2014).

Correlational studies have examined the relationship between biomedical illness beliefs and perceived agency, a variable corresponding with the controllability dimension of Leventhal's Common Sense Model (2003). In one sense, perceived agency can be conceived of as having to do with how much blame a depressed person deserves for their condition. Mehta and Farina (1997) found that individuals whose depression was explained as biological were viewed as being less blame-worthy for

poor task performance than those whose depression was explained through a sociogenic framework. However, these studies demonstrating a reduction in blameworthiness have failed to replicate (Kemp et al. 2014), and population studies have established that although endorsement for biomedical etiologies of depression and other mental disorders is growing, ascriptions of responsibility remain unaffected (Angermeyer et al., 2011; Pescosolido et al., 2010).

Recent *experimental* studies have also examined the effects of biomedical explanations of depression on perceived agency and prognosis perception, and have demonstrated that biological explanations' deleterious effects stem causally from a biological etiology of depression. In one experimental study, Lebowitz et al. (2013) compared the effects of watching a psychoeducational video that described depression as a biological illness and a video that described the biology leading to depression as malleable and non-determinative. Among participants with symptoms of depression, the video that framed depression as determined by biology elicited considerable pessimism about the possibility of recovery and the duration of illness compared to those participants who watched the video describing depression as arising from complex and malleable factors. In another experiment, Kemp et al. (2014) used a bogus saliva test to persuade current or former patients with depression that their depression was the result of a chemical imbalance, and concluded that biological etiologies both increase pessimism about the duration of illness and fail to reduce feelings of guilt about being depressed.

This recent scholarship—theoretical and empirical—has greatly advanced our understanding of how certain causal explanations for depression affect health behavior and other beliefs about depression. However, these empirical and theoretical developments can benefit from integration; that is, in order to understand the effects of the psychoeducational interventions, we must first ascertain the fully social and relational context in which those interventions take place. Fully accounting for the social life of accounts about depression requires an approach that is informed simultaneously by immediate, proximal social demands such as those demonstrated by Lebowitz et al. (2013) as well as larger more distal social structure. What is needed, then, is a synthesis. Radley and Billig's (1996) discursive approach, with its attention to the relational demands that give rise to health and illness representations *in situ,* complements Thornton (2011), Hacking's (2007), and others' historical dynamic models, with their attention to how larger systems of power and control structure representations of health and illness. Such a theoretical synthesis understands accounts of depression as more than simply dimensionalized responses to a questionnaire that reveal an 'underlying' belief about depression.

3. Goals of Present Study

The present study aims to elaborate on the aforementioned experimental research by Lebowitz et al. (2013) by looking more closely at the discursive effects of different psychoeducational strategies. It attends closely to the accounts of personhood and representations of depression's etiology and prognosis that are engendered by psychoeducational strategies that frame depression using either deterministic or plastic brain discourses. This study aims to use a quantitative content analysis and qualitative exploration of accounts of depression to triangulate extant findings about the effects of neurobiological conceptions of depression, as well as to describe more fully the shape and dynamic nature of accounts about depression.

Because the present study borrows many elements of design from Lebowitz et al.'s (2013) study of psychoeducation about depression, their study's procedure is described before proceeding into a discussion of how and why the present study differs. Lebowitz et al. sought to experimentally investigate the negative effects of a deterministic biological explanation for depression and to evaluate an intervention to alleviate these negative effects. To assess the effects of these two ways of explaining the biology of depression, the researchers asked a pool of online participants to watch one of two psychoeducational videos: a video that presented a deterministic explanation of depression's biogenesis, and one that gave a more qualified and malleable picture of depression's etiology. A third control group did not watch any video. All participants were then asked to respond to ordinal scales about how long

they expected their depressed mood[1] to last, how much control they felt over their mood, and how guilty they felt about their mood, to a 0-100% scale about how likely they thought their depression was to remit, as well as to a modified version of the Beck Hopeless Scale.

Lebowitz et al.'s (2013) results largely replicated earlier research on biomedical explanations of depression (Deacon & Baird, 2009; Lam & Salkovskis, 2007) finding that, compared to the malleable video, the video depicting a deterministic account of depression's biogenesis led to increased prognostic pessimism and failed to improve a sense of agency in relation to depression. Nevertheless, these effects deserve close examination, given both the mixed findings about feelings of guilt (see p. 28) and potentially problematic assumptions embedded in Lebowtiz et al.'s design.

To take seriously a theoretical understanding of the dynamic, socio-historical and rhetorical nature of health and illness accounts raises the question of the conceptual validity of ordinal scales measuring beliefs about prognosis pessimism, hopelessness, and so on. If health beliefs are not simply held like "eggs carried in a basket" (Radley & Billig, 1996, p. 223) but instead are unfolding processes, experimental participants must be asked to engage in those processes. It follows then that, in asking participants to respond about decontextualized and discrete dimensions of beliefs about depression, Lebowitz et al. (2013) did not fully capture the whole and

---

[1] Participants scoring higher than 16 on the CESD-R severity scale for depression had all questions phrased to be about their own depression, while those scoring below the cutoff

situated nature of illness and health accounts. An attention to context is paramount if we hope to come to a better understanding of *why* certain explanations of depression engender negative attitudinal effects because a lack of context forecloses on discursive claims of agency or worthiness of the self that accompany health and illness talk from entering into the researcher's frame of reference. An investigation of accounts of depression, simply put, needs to leave room for personhood to reveal itself. Given the high priority that negotiations of selfhood take in health and illness accounts (Charmaz, 1999; Kokanovic, 2013; Martin, 2010; Radley & Billig, 1996; Ridge et al., 2011; Riessman, 2003) to understand why biological explanations of depression lead to pessimism, stigma or hopelessness, the researcher must frame inquiry at the level at which self-making does (or at least can) take place; that is, at the discursive level. In their discursive form, perceptions of prognosis or control constitute less a set of discrete beliefs and more a series of different steps in a dance. To do full justice to this complex choreography and improvisation, the present study extends Lebowitz et al.'s belief survey items with an open response.

Re-approaching the effects of Lebowitz et al.'s (2013) videos by soliciting open-responses from participants provides an opportunity for validating their findings and for further exploration. Participants' responses will be coded for the kinds of etiology of depression being claimed, the recommended types of treatments, indications of a particular source of knowledge, as well as prognostic outlook. Furthermore, open-response data analyzed both for thematic content frequencies and for qualitative structure allows for the triangulation of Lebowitz et al.'s quantitative

survey research. The present study aims to achieve several forms of triangulation (Denzin, 2012) by examining multiple methodological perspectives, sources of data and theoretical assumption. Such triangulation functions both as a strategy of validation and as a strategy for gaining deeper understanding about the ambiguity and at times multiple meanings of accounts of depression. In addition to triangulation, such a qualitative approach to data collection will also allow the dynamic, discursive and paradoxical nature of those accounts to emerge, presenting an opportunity to explore possible mechanisms behind the negative effects of biological explanations of depression.

4. Hypotheses

4.0 Analyses by Condition

The following hypotheses predict the effects of the psychoeducational videos on the frequency and variety of themes present in the open response, coded at the level of the sentence. These hypotheses are intended to replicate findings from Lebowitz et al. (2013) as well as to test several novel predictions. These novel predictions include the expectation that participants exposed to the biological illness video will defer more to expert sources of knowledge, and give a less varied account of the etiology and treatment of depression; the opposite is predicted for the control group. Given Thornton's (2011) observations of the expansiveness of brain discourse, it is reasonable to assume that biological etiologies will appear to be sufficient for making an explanation about depression. Furthermore, because such biological etiologies are generally more technical than psychosocial explanations, it is reasonable to assume that participants will hedge their responses by saying that what they know comes from expert authorities.

H1a: *Responses from the biological illness condition will have a higher frequency of sentences claiming a biogenic etiology of depression, a biomedical treatment for depression, and a negative or ambiguous outlook for depression; a lower frequency of sentences claiming a positive outlook for depression; and a higher frequency of*

sentences claiming that knowledge of depression was learned from expert sources than responses from either the control or malleable conditions.

H1b: *Responses from the biological illness condition will have a smaller variety of etiologies and treatment preferences for depression than responses from either the malleable or control conditions.*

H2: *Responses from the control condition will have a higher frequency of sentences claiming that knowledge of depression was learned from non-expert sources (self or others) than responses from either the malleable or biological illness conditions.*

H3: *Responses from the malleable condition will have a higher frequency of sentences claiming a positive outlook towards depression than responses from either the control or biological illness condition.*

4.1 Analyses by Mental Health History and Video Appraisals

In addition to comparisons across experimental groups, analyses will examine the extent to which past experiences with mental health care affect the frequency of sentences claiming particular treatment preferences and etiologies for depression. It is assumed, following Leykin (2007), that these treatment preferences and explanations will be consistent with participants' past experiences.

H4a: *Responses from participants with a history of taking psychiatric medication will contain a higher frequency of sentences claiming a biogenic etiology for depression*

or a biomedical treatment for depression than responses from participant with no history of taking psychiatric medications.

H4b: *Responses from participants with a history of non-biomedical treatment (psychotherapy or lifestyle change) will contain a higher frequency of sentences claiming a non-biomedical etiology for depression than responses from participants without a history of psychotherapy.*

The following hypothesis predicts that low levels of agreement with stimuli videos will cause participants to draw on non-expert knowledge in reaction to the expert knowledge presented in the videos.

H5: *In both the malleable and biological illness conditions, being less convinced by and disagreeing with the videos will predict higher frequencies of sentences claiming that knowledge of depression was learned from non-expert sources (self or others).*

4.2 Goals of Exploratory Coding

In addition to testing the above hypotheses, open responses will be explored for several discursive elements. Of particular interest are discourses of the self: negotiations of agency (Rose, 2003; Thornton 2011) as well as what Martin (2010) terms "self-making." Also of focus will be whether and how multiple explanations of depression travel together and/or contradict each other.

5. Methods

5.0 Participants

Sixty-seven undergraduate students (thirty-one females) at a small university in New England participated in the study as part of the research participation requirement for their introductory psychology class. Participants had the option of either participating in the study or writing a short essay as part of their course requirement. Two participants were excluded from analyses because they failed to answer the open-ended prompt. No participants actively declined participation in the study either during or after consenting. The final sample consisted of sixty-five participants (thirty-one females) with a mean age of 19.1 (*SD* = 1.67). In total, 73.9% of participants identified as White, 15.4% as East Asian, 13.9% as Chicano, 6.2% as Black, 3.1% as South Asian, and 3.1% as Native American. 13.9% of participants identified with more than one ethnic or racial category. This demographic sample approximates the diversity of the university from which it was drawn.

Of the final sample, 22 participants (33.9%) stated that they either had been or were currently receiving psychotherapy, 16 (24.6%) stated that they had been told by a mental health professional that they suffered from an emotional, mental or psychiatric disorder, and 15 (23.1%) stated that they had taken a medication for an emotional, mental or psychiatric disorder. Two participants reported taking medication despite reporting that they had never been diagnosed with a mental disorder.

5.1 Procedure

Upon arriving in the laboratory, participants were greeted by the experimenter and asked to give informed consent. The informed consent document can be found in Appendix D. After consenting, the experimenter left the room, leaving each participant to complete the study on a computer with no other persons present in the room. All survey questions and experimental stimuli were hosted on a Qualtrics web survey. Participants were first asked questions related to age, gender, and ethnic demographics. Participants were then randomly sorted into three separate conditions: the biological illness condition, the malleable condition, and the control condition.

Participants in the biological illness condition (n = 22) were shown one of the psychoeducational videos designed by Lebowitz et al. (2013). The biological illness video explained that depression is a brain disorder, focusing on a large role played by genetics in determining one's vulnerability to depression. The biological illness video also emphasized research showing that depressed individuals have brain abnormalities. Lastly, the biological illness video explained that while medications might help treat depression, depression is likely to return if medication is discontinued.

Participants randomly assigned to the malleable condition (n = 21) watched another video designed for the Lebowitz et al. (2013) study. The video from the malleable condition focused on the ambiguity of biomedical research about depression, noting that there is no biological test for depression. The malleable video

explained that genetics alone cannot determine depression, and, gesturing to research in epigenetics, described the ways that behaviors and environmental factors can turn genes for depression "on or off." Lastly, the video focused on both medications and psychotherapeutic techniques that can help reshape depressed individuals' brains. Both the biological illness and the malleable videos were approximately six minutes in duration, and were careful to focus on biological precipitants for depression and not on environmental causes, which Deacon and Baird (2009) noted can allay the negative effects of biomedical explanations. The scripts for both videos are presented in Appendix B.

Participants watching either the biological illness video or the malleable video were asked a series of questions in the form of three five-level Likert items. These questions prompted participants to report how clear they found the video, how convincing they found the video, and the extent to which they agreed with the video. These questions were included to encourage participants both to think critically about the videos and to consider how their own views of depression might differ from those expressed by the videos, instead of simply recapitulating the contents of the video when answering the proceeding open-response question. Also with the aim of discouraging overt parroting of the videos, these questions were intended to allow for a pause in time between watching the videos and the open response portion of the study. Participants in the control condition (n = 22) were not shown any video, nor asked any questions about their reaction to the video.

After the questions, participants were asked to answer in more than three hundred words an open-ended question: “Why do you believe people become sad, blue or depressed? Do you believe people can recover from these feelings, and if so, how?” Throughout all procedures, care was taken to ensure that depression was worded as “sadness, blueness or depression,” to avoid forcing participants to consider depression as a medical condition. Participants also were instructed to consider their answer in terms of the average depressed person, to avoid soliciting potentially ethically problematic data that might indicate a risk of suicide. Upon finishing the open-ended response, participants were asked whether they had ever been diagnosed with a mental disorder, and whether they had ever received psychopharmacological or psychotherapeutic treatments. Participants were then prompted to alert the experimenter that they had completed the study, and were fully debriefed by the experimenter (see Appendix E for the debriefing script). From consenting to debriefing, the amount of time required for participation was approximately 35 minutes, and less for participants in the control condition.

5.2 Coding Procedure

An independent rater with no knowledge of the study’s hypotheses or design coded the open-response data using an *a priori* coding scheme. Coding units were sized at the level of sentence in order to allow for frequency analyses (Babbie, 2001, p. 365). The codebook is included in Appendix A. The reliability of the coding was assessed by having a second rater code a subsample (n = 20) of the open-response

data and then comparing both raters' coding. In order to facilitate the coders' independence, an independent team of researchers redacted all mentions of the word "video" from all responses, making as few deletions as possible while maintaining the flow of responses. These mentions of the videos were then re-inserted for the exploratory, open coding described below (pp. 43-44). Coding was completed using Atlas.TI software for qualitative research, and results of the coding were transferred to SPSS for statistical analysis.

Although all of the superordinate categories listed in the initial codebook contained more than two subordinate categories, these subordinate categories were collapsed into binaries in order to facilitate analyses (Krippendorff, 2004, p. 430). Within each superordinate coding category, such as etiology, we reduced the number of subordinate categories, such as sociocultural etiology, to two codes—for example, biogenic etiology and non-biogenic etiology. Specifically, codes measuring a kind of etiology other than the biogenic etiology code were collapsed into a 'non-biogenic etiology' code; all codes measuring a treatment preference other than 'biomedical treatment preference' were collapsed into a 'non-biomedical treatment preference' code; and the 'self-knowledge source' and 'non-expert knowledge source' codes were collapsed to form a larger 'non-expert knowledge source' code. The distinctions between the coding categories that were collapsed were insignificant for the purposes hypothesis testing because these collapsed categories were not mentioned in the hypotheses, and moreover, were defined in opposition to those stated in the hypotheses. Thus, the collapsing of categories allowed the analysis to assess both the

explicitly stated hypotheses and their corollary inversions. Furthermore, because the code 'negative outlook to recovery' occurred with very low frequency in the subsample, it was collapsed with the 'ambiguous outlook to recovery' code to form a larger 'ambiguous/negative outlook to recovery' code. This last simplification was deemed non-problematic because both codes capture a degree of pessimism about recovery: both ambiguous and negative outlook codes indicate that complete recovery is impossible, even while they do differ in signaling the possibility of *remission.* Furthermore, these outlook codes were predicted to exhibit similar changes across conditions.

After initial coding with the *a priori* coding scheme, the primary investigator submitted the open-response data to a second round of coding using the grounded theory principles of open coding specified by Strauss and Corbin (1990). Theoretical sensitivity was derived from a review of the literature discussed above (pp. 8-31), with particular sensitivity to participants' talk of agency accompanied particular etiological or prognostic claims (Thornton, 2011). The open coding attended in particular to instances in which participants mobilized similar language—such as the metaphor of 'masking the root cause of depression'—to distinct explanatory ends. The qualitative analysis was also guided by the previously made claims that talk about depression is marked by self-making (Martin, 2010) and self-justificatory gestures (Radley and Billig, 1996). Finally, open coding was informed by the results of the quantitative content analysis (pp. 45-60).This exploratory coding was conducted in two stages: the first conducted blind to condition, and the second with

access to participants' condition and responses to other questions. The results of this open, exploratory coding are presented in Section 8 (pp. 61-79)—below the quantitative content analysis results and discussion sections—and are meant to suggest further theoretical exploration.

6. Results of Quantitative Content Analysis

6.0 Approach to Analyses

Pearson consistency coefficients and intra-class correlation coefficients (ICC) were calculated for each code, correlating the frequency of sentences coded by two raters in a subsample (n = 20) of open responses in order to assess interrater reliability. Descriptive statistics for each code were examined to assess the distribution of the data. Levene's test (1960) was then run on each coding category to determine if the code's distribution violated homogeneity of variance. A one-way ANOVA was performed to examine the effect of condition on all coding categories not violating homogeneity of variance, and Welch's test of equality of means assessed the effect of condition on those coding categories that did violate homogeneity of variance. For hypothesis testing, planned comparisons were calculated pairwise, with one tail, and assuming equal variance where indicated by the Levene test. One-tailed testing was deemed acceptable because the hypotheses were stated directionally. Post hoc testing was computed for coding categories with an F or *Welch's F* score with significance near or below an alpha of 0.05. Tukey's HSD was used for post-hoc testing of codes not violating homogeneity of variance, and Games Howell's test was used for codes that did violate homogeneity of variance. Two-tailed independent samples t-tests were run to examine the effect of mental health history on coding frequencies. Finally, median tests were calculated to determine the effect of video appraisal on coding frequencies.

6.1 Interrater Reliability

Pearson's consistency correlations and ICCs were used to assess interrater reliability within a subsample of responses (n = 20) coded by two raters because they measure the consistency of raters' coding. Instead of measuring precise matches between two codes, these calculations assess the consistency of two raters' coding: if one rater coded a *relatively* high frequency of sentences for a certain coding category, did the other rater also code a *relatively* high frequency of sentences claiming that category? For example, the first rater might apply 10 instances code A to Response 1, and 6 instances of code A to Response 2, while the second rater might apply 5 instances of code A to Response 1 and 3 instances of code A to Response 2. In this case, the raters exhibit high levels of interrater consistency as measured by Pearson's *r,* despite the second rater coding only half as frequently as the first rater.

The two raters reached a Pearson's *r* ranging from 0.49-1.00 (see Table 1). Only one code, non-biomedical treatment preference, fell below a consistency correlation of r = 0.70, a standard threshold for inter-rater consistency (Stemler, 2004). The ICCs varied from 0.59-1.00 (see Table 1). Again, non-biomedical treatment preference deviated significantly below the ICC's for the other codes.

6.2 Analyses by Condition

Levene's statistic was calculated for each coding category and indicated that the non-biogenic etiology, expert knowledge source, non-expert knowledge source, and ambiguous/negative outlook to depression codes significantly violated

Table 1. ***Pearson consistency coefficients and intra-class correlation coefficients (average measures) for interrater reliability within subsample (n = 20)***

Code	$f_{\text{rater 1}}$	$f_{\text{rater 2}}$	Agreements	Disagreements	Pearson's *r*	ICC
Biogenic Etiology	33	35	30	8	0.955	0.974
Non-Biogenic Etiology	70	105	56	63	0.720	0.837
Biomedicine Preference	21	23	18	8	0.789	0.869
Non-Biomedicine Preference	38	69	35	37	0.486	0.590
Expert Knowledge Source	6	4	4	2	1.000	1.000
Non-Expert Knowledge Source	121	126	113	21	0.911	0.954
Positive Outlook to Recovery	35	35	25	20	0.799	0.888
Ambiguous Outlook to Recovery	38	28	24	18	0.937	0.951
Total	362	425	304	179		

homogeneity of variance. The main effect of condition on code frequencies was assessed using a one-way analysis of variance (ANOVA) for those categories not violating homogeneity of variance: biogenic etiology, biomedical treatment preference, non-biomedical treatment preference, positive outlook to recovery, variety of explanations, and variety of treatment preferences. The ANOVA (see Table 2) and Welch's test of equality of means (see Table 3) obtained a significant omnibus effect of condition on the frequency of sentences containing a biogenic etiology for depression (p = .002), a non-biogenic etiology for depression (p = .024), and an ambiguous/negative outlook to depression (p = .023). Effects of condition approaching significance were found on the variety of etiologies for depression given within a response (p = .054), as well as on the frequency of sentences containing a positive outlook towards recovery (p = .175), a biomedical treatment preference (p = .149), and a non-biomedical treatment preference (p = .185). No significant effect of condition was found on the variety of treatment preferences given within a response, or on the frequency of sentences claiming that knowledge was learned from an expert or non-expert (self or other) source.

6.2.1 Effect of Condition on Prognostic Accounting

A one-way Welch's test of equality of means examined the effect of condition on the frequency of sentences claiming an ambiguous/negative outlook to recovery, and indicated a significant effect, *Welch's* $F(2, 36.4) = 4.170$, p = .023 (see Figure 1). Pairwise comparisons for planned contrasts found the frequency of sentences

Table 2. ***One-way ANOVA examining main effect of condition*** **(C)** ***on sentence frequencies***

	Biological Illness (C_{BI})		Malleable (C_M)		Control (C_C)		One-way ANOVA,	Planned Comparisons, One-Tailed	
	Mean	SD	Mean	SD	Mean	SD	*df* = 2, 64	Contrast Groups	*p* value
Biogenic Etiology	2.773	1.998	1.238	1.375	1.045	1.618	$F = 6.883^{**}$	$C_{BI} > C_C$ **	.001
							$p = .002$	$C_{BI} > C_M$ **	.002
Biomedicine Preference	1.591	1.532	1.095	1.136	0.864	0.990	$F = 1.965$	$C_{BI} > C_C$ *	.029
							$p = .149$	$C_{BI} > C_M$	.098
Non-Biomedicine Preference	2.000	1.718	2.810	1.806	1.955	1.527	$F = 1.736$		
							$p = .185$		
Positive Outlook to Recovery	1.455	1.654	2.429	2.521	2.364	1.364	$F = 1.792$	$C_{BI} < C_C$	.059
							$p = .175$	$C_{BI} < C_M$ *	.049
								$C_M > C_C$	.506
Variety of Etiologies	2.636	0.954	3.143	0.964	2.455	0.912	$F = 3.058$	$C_{BI} < C_C$	.213
							$p = .054$	$C_{BI} < C_M$ *	.042
Variety of Treatment Preferences	1.909	1.065	2.238	0.700	1.864	1.086	$F = 0.949$	$C_{BI} < C_C$	.439
							$p = .393$	$C_{BI} < C_M$	.135

* Indicates that results are statistically significant with $p < .05$, ** indicates that results are statistically significant with $p < .005$

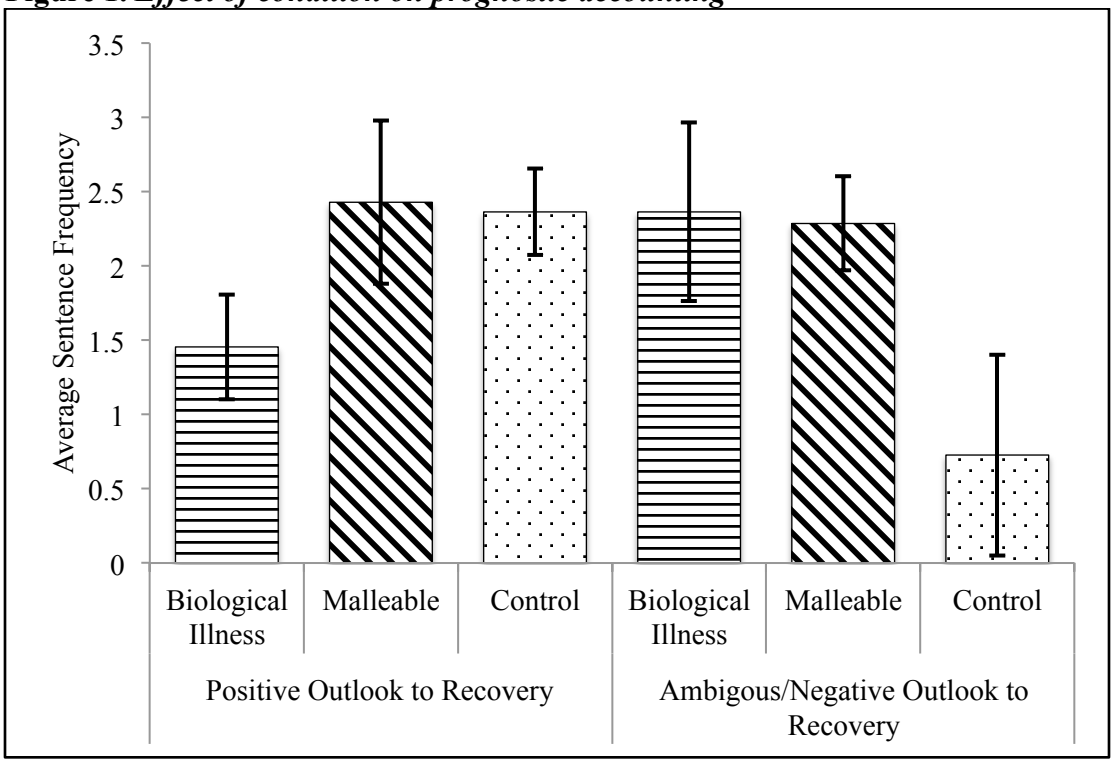
Table 3. ***Welch's test of equality of means examining main effect of condition*** **(C)** ***on sentence frequencies***

	Biological Illness (C_{BI})		Malleable (C_M)		Control (C_C)		Welch's Test of Equality of Means	Planned Comparisons, One-Tailed	
	Mean	SD	Mean	SD	Mean	SD		Contrast Groups	*p* value
Non-Biogenic Etiology	3.045	1.838	5.143	2.816	3.773	1.478	$F = 4.106^{*}$ $df = 2, 38.8$ $p = .024$		
Expert Knowledge Source	0.455	0.912	0.238	0.539	0.182	0.501	$F = 0.745$ $df = 2, 39.7$ $p = .481$	$C_{BI} > C_C$ $C_{BI} > C_M$	.114 .174
Non-Expert Knowledge Source	6.682	3.564	6.095	2.587	7.636	5.215	$F = 0.794$ $df = 2, 39.2$ $p = .459$	$C_{BI} < C_C$ $C_M < C_C$	.242 .113
Ambiguous/ Negative Outlook to Recovery	2.364	2.821	2.286	3.101	0.727	1.486	$F = 4.170^{*}$ $df = 2, 36.4$ $p = .023$	$C_{BI} > C_C^{*}$ $C_{BI} > C_M$	.019 .462

* Indicates that results are statistically significant with $p < .05$, ** indicates that results are statistically significant with $p < .005$

claiming an ambiguous/negative outlook was higher in the biological illness condition (M = 2.36, SD = 2.82) than in the control condition (M = 0.73, SD = 1.49), t(31.8) = 2.407, p = .011, and failed to find a significant difference between the biological illness and malleable conditions (M = 2.29, SD =3.10). Post-hoc testing showed that the malleable condition led to an increased frequency of sentences claiming an ambiguous/negative outlook to depression compared to control (p = .121). The one-way ANOVA failed to find a significant mean difference across conditions in terms of the frequency of sentences claiming a positive prognostic outlook F(2, 62) = 1.792, p = .175. Nevertheless, planned contrasts showed that the biological illness condition lowered the frequency of sentences claiming a positive outlook to depression.

Figure 1. ***Effect of condition on prognostic accounting***

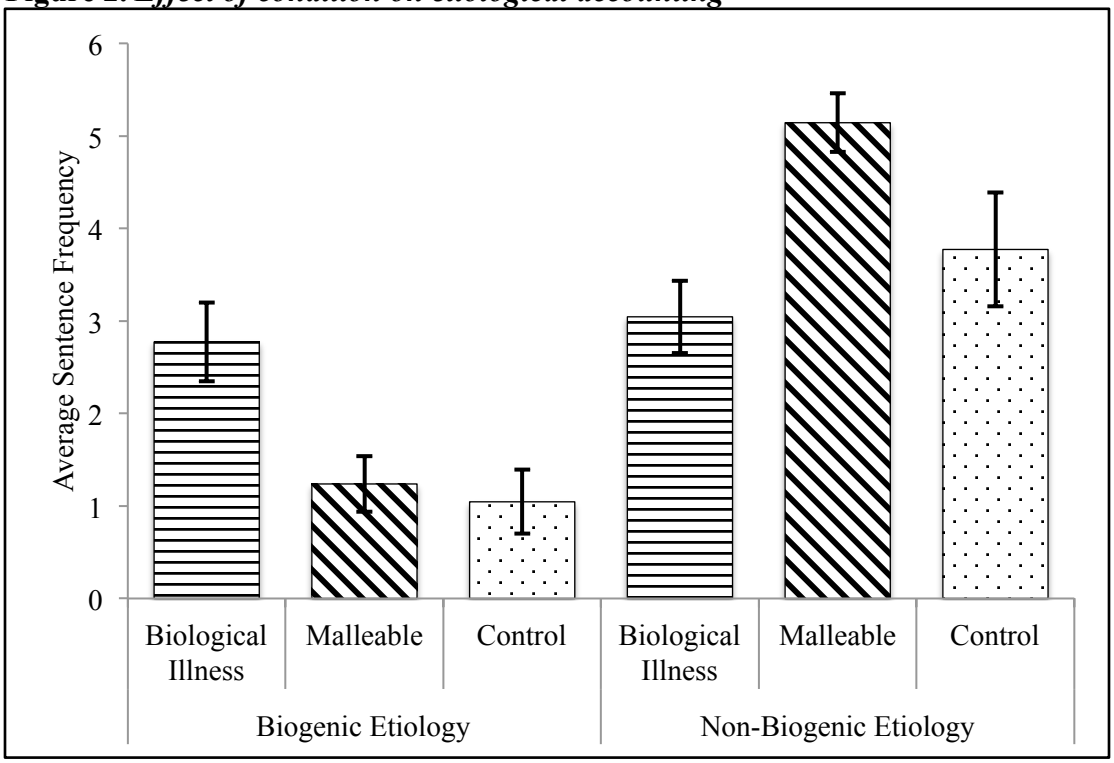


(M = 1.45, SD = 1.65) below the control condition (M = 2.36, SD = 1.36) with an effect approaching significance, $t(62) = 1.587$, $p = .059$. These results suggest that both the biological illness video and the malleable video led participants to express more ambiguity or negativity about the prospect of recovery, while only the biological illness video inhibited the expression of positivity.

6.2.2 Effect of Condition on Etiological Accounting

As predicted, the frequency of sentences claiming a biogenic etiology for depression differed significantly across conditions, $F(2, 64) = 6.883$, $p = .002$, as did the frequency of sentences claiming a non-biogenic etiology, *Welch's* $F(2, 38.8) = 4.106$, $p = .024$ (see Figure 2). Planned contrasts found that biogenic etiologies were claimed more frequently in the biological illness condition (M = 2.77, SD = 2.00) than in the control condition (M = 1.05, SD = 1.62), $t(62) = 3.394$, $p = .0005$, or malleable condition (M = 1.24, SD = 1.38), $t(62) = 2.980$, $p = .002$. Tukey's HSD revealed that the malleable condition was found to have a significantly higher frequency of sentences claiming a non-biogenic etiology, (M = 5.14, SD = 2.82) than the biological illness condition (M = 3.05, SD = 1.838), $p = .005$. The malleable condition was also higher than the control condition in terms of sentences claiming a non-biogenic etiology (M = 3.77, SD = 1.478), with an effect approaching significance, $p = .092$. These results show that respondents tended to echo the model of depression expressed in the video that they watched. They also suggest that the malleable video led participants to think more about the non-biological causes of depression than they would have without any psychoeducational intervention.

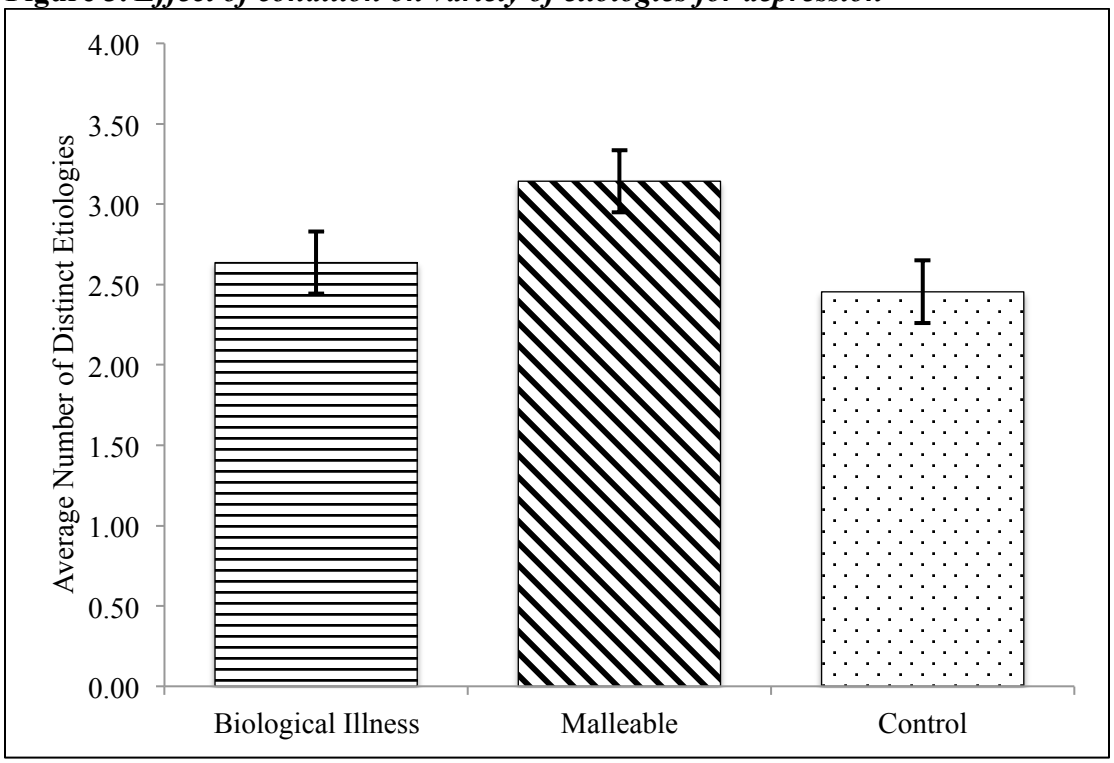
Figure 2. ***Effect of condition on etiological accounting***



6.2.3 Effect of Condition on Stated Treatment Preferences

The omnibus ANOVA found no effect of condition on the frequency of sentences claiming biomedical treatment preferences and non-biomedical treatment preferences. Planned contrasts indicated that responses in the biological illness condition had a significantly higher frequency of sentences claiming biomedical treatment preferences (M = 1.59, SD = 1.53) than the control condition (M = 0.86, SD = 0.99), $t(62) = 1.941$, $p = .029$, while frequencies differed between the biological illness and malleable conditions (M = 1.10, SD =1.14) with an effect approaching significance, $t(62) = 1.308$, $p = .098$. These results give some evidence that the

Figure 3. ***Effect of condition on variety of etiologies for depression***



biological illness video induces people to express more interest in antidepressants or other biomedical treatments for depression.

6.2.4 Effect of Condition on Variety of Etiologies

The variety of etiologies given within a response was determined by scoring the number of distinct etiology for depression codes present in each response. This scoring was based on the non-simplified set of five codes for etiology (see pp. 42-43 and Appendix A). For example, if a response contained a sentence claiming a biogenic etiology, another sentence claiming both a socio-cultural and a personal history etiology, and a third sentence again claiming a biogenic etiology and a failure of agency etiology, the response was given a score of 4. The one-way ANOVA

revealed an omnibus effect of condition on the variety of etiologies approaching significance, $F(2, 62) = 3.058$, $p = .054$ (see Figure 3). Planned comparisons demonstrated, as predicted, that responses from the biological illness condition contained a smaller variety of etiologies ($M = 2.64$, $SD = 0.95$) than the malleable condition ($M = 3.14$, $SD = 0.96$), $t(62) = 1.761$, $p = .042$. The variety of etiologies did not differ significantly between the control condition ($M = 2.45$, $SD = 0.91$) and biological illness condition. Post-hoc exploration revealed that the malleable condition, unexpectedly, contained an almost significantly greater variety of etiologies than the control condition, $p = .051$. These results also find that the malleable video led participants to consider different causes for depression than they would have at baseline, whereas the biological illness video did not lead participants to negotiate additional etiologies.

6.3 Analyses by Mental Health History

Two one-tailed independent samples *t*-test was carried out to determine the effect of having a history of psychiatric medication ($n = 15$) or psychotherapy ($n = 22$) on etiological accounting and the expression of treatment preferences. Against the hypotheses, both *t*-tests failed to find a significant effect of either form of prior treatment (both p's $> .05$).

Exploration of the data did reveal one effect of mental health history on outlook statements. An independent samples *t*-test indicated that respondents who had taken medication in the past gave significantly fewer statements claiming a positive

outlook for depression than those with no history of medication, $t(63) = 2.580$, $p = .012$, two-tailed. Although a lack of optimism could be interpreted as a symptom of depression itself among these former patients, no such relationship emerged between positive outlook and history of being diagnosed with a mental disorder—which suggests that this lack of positivity results only from having taken medication. However, it should be noted that it was not ascertained whether or not these respondents took medication for depression or for another mental disorder.

6.3 Analyses by Appraisals of the Videos

All participants in the biological illness and malleable conditions were assessed for how their appraisal of the video effected the content of their open-response. Specifically, participants' ranking of the video's convincingness and their self-reported level of agreement with the video was expected to influence the frequency of sentences claiming knowledge of depression was learned from either an expert or non-expert source. A median test found participants who rated the videos as more convincing were more likely to include in their open response sentences claiming their knowledge was learned from an expert source than participants who found the video less convincing, $x^2(4) = 8.381$, $p = .039$, but no effect was found on the frequency of sentences claiming a non-expert knowledge source. Rated level of agreement had no significant effect on knowledge source. Finally, it appeared that the videos did not differ significantly from each other in terms of clarity, convincingness, or agreement.

7. Discussion of Quantitative Content Analysis

This study examined how the content of accounts of depression varies after exposure to different explanations of depression's biology. As predicted, when participants were exposed to the biological illness video, their etiological accounting was marked by more frequent mentions of biogenesis. Similarly, when respondents watched the malleable video, they were more likely to give a non-biogenic etiology. These results could be interpreted as a sign that participants internalized the narratives with which they were presented. It might be that participants learned something from the video, a lesson that persists either in the short or long-term—although the study's design does not permit assessment of such temporal effects.

Another interpretation of the effect of condition on etiological accounting focuses on demand characteristics: the possibility that participants echoed the etiology presented in their stimulus not because they were persuaded by the video, but because they wanted to give the experimenter the 'correct' response to the prompt. Although the study design attempted to reassure participants that we were interested only in their subjective understanding of depression by asking for "as open an account" as possible (see Appendix D), it is reasonable to consider that participants might have felt pressured to give the 'scientific' answer or to use a 'scientific' style of writing and thinking. However, such interpretations that focus solely on demand characteristics and repetition of the video scripts are complicated on at least one account: the malleable video gave no more non-biogenic explanations for depression

than did the biological illness video, describing instead the ways that *biology* can be influenced and shaped by external factors. Therefore, if respondents were repeating what they learned in the malleable video, then they were at the very least elaborating beyond what was presented in the video script. This elaboration suggests that more is at play that mere repetition.

The two video interventions' effects on participants' prognostic accounting were largely consistent with the observations of Lebowitz et al. (2013). As Lebowitz et al. noted, amongst non-symptomatic individuals, the malleable condition did not cause a significant increase in prognostic pessimism relative to the control condition, whereas the biological illness video did engender greater pessimism (pp. 521-2). Our results align with earlier research linking biological explanations to prognostic pessimism (Deacon & Baird, 2009; Lam & Salkovskis, 2007), but present a slightly more qualified view. In the present study, although the biological illness video decreased the frequency with which participants made *optimistic* statements relative to control—an effect not exhibited by the malleable video—both videos led to an increase in ambiguous or pessimistic statements about recovery compared to control. This finding counters Lebowitz et al.'s observations that the malleable video produced no negative effects in terms of prognosis perception (p. 523-4). Levels of standard error in the present study call for conservative interpretation, yet it seems fair to suggest that although biological explanations—malleable *or* deterministic—lead people to consider the difficulty of recovery, deterministic accounts are particularly corrosive to optimism. Simply put, the malleable video led participants to

speak more pluralistically—to give both a pessimistic *and* an optimistic outlook within the same accounts—whereas the biological illness video caused participants to describe the course of depression with a more uniform pessimism.

A distinction between uniformity versus multiplicity is also clearly evident in differences across conditions in the variety of distinct etiologies given within a response. The respondents who watched the malleable video mobilized more distinct etiologies for depression than did those in both the control and biological illness conditions. It appears that the malleable video's explanation of depression's biology invites in complementary non-biogenic etiologies of depression, while the biological illness video's explanation of depression's biology tends to displace non-biogenic etiologies. Still, it is important to note that these differences are not pronounced, and that participants across all conditions used a wide variety of etiologies, ones that were at times seemingly inconsistent with one another. The consistency and inconsistency of accounts about depression will be revisited in the following chapter.

Some of the more surprising results of this study include null relationships between past experiences of mental health treatment and etiological accounting as well as treatment preferences. Based on earlier research (Iselin & Addis, 2003; Meyer & Garcia-Roberts, 2007) it was expected that having taken medication in the past for a mental disorder would likely increase the number of sentences claiming a biogenic etiology or biomedical treatment preference, and for a similar pattern to emerge with regards to participants with a past experience of psychotherapy. The only effect of mental health history was on prognostic accounting: a history of taking medication

predicts fewer statements of optimism. This effect of personal history converges with the above observation that the biological illness video decreased the frequency of statements giving an optimistic outlook to depression. Such a convergence suggests that both exposure to the determinist explanation and enactment of that explanation as a consumer of psychopharmaceuticals reduces optimism.

The quantitative content analysis is a valuable component of triangulating Lebowitz et al.'s findings in that it provides continued support for their results while at the same time qualifying some of their conclusions. Our data provide further evidence for Lebowitz et al.'s claim that the biological illness video leads to prognostic pessimism. The results do not, however, replicate the finding that the malleable video led to increased prognostic optimism, with the malleable and control conditions having a similar level of optimism. Also contrary to Lebowitz et al.'s findings, the malleable video led to more statements of ambiguity or negativity about prognosis. Finally, the malleable video led participants to speak about a greater variety of distinct causes for depression than did either the biological illness video or the control condition. These analyses suggest directions for exploration in open coding. Particularly, the finding that the variety of etiologies differed across conditions suggests that attention should be paid to the ways that certain modes of discourse can be adapted to different etiologies of depression, and to the extent to which distinct explanations of depression and conceptions of agency coexist and are juxtaposed against each other within participants' accounts of depression.

8. Findings and Discussion of Qualitative Analysis

8.0 Qualitative Findings

Before beginning open coding, it was assumed that the most salient variations among responses would consist in differences in content, that is, distinctions between biological and non-biological explanations. While there were some key divergences separating biological and non-biological explanations, during initial work on open coding it became evident that there were notable *similarities* between those who emphasized biology and those who did not. One notable similarity was the usage of metaphors like 'masking the root' cause of depression. These metaphors were made into central coding categories.

Conversely, conspicuous *differences* in narrative continuity were manifest across responses, leading to the creation of narrative continuity and discontinuity as central analytic categories. Some participants had little trouble maintaining narrative continuity while shifting between discourses of biological determinism and malleability, a potentially paradoxical movement in terms of how these modes of discourse configure the individual's agency as, alternatively, determined or subject to free will. Nevertheless, the first group of participants, 'the lumpers,' were able to travel harmoniously between two stances: between a deterministic view of depression's cause and a malleable view of recovery in which the individual's will comes to the fore. Other participants, however, did not navigate between or integrate languages of determinism or malleability. Instead, this second group of participants,

'the splitters,' made definitive demarcations between feelings of sadness and depression as an illness. Such distinctions, similar to the differences between the two stances discussed above, were also accompanied by differences in the configuration of agency. Furthermore, 'the splitters' also tended to depict depression as vague, incomprehensible, or 'without reason', and contrastingly represented sadness as understandable, commonsensical, and as triggered by a definite cause. In coding responses by both the 'splitters' and the 'lumpers,' configurations of agency as well as language of comprehensibility and reasonableness emerged as coding categories.

8.1 'Masked' Analogies

While the biological illness video and malleable video might seem to portray entirely distinct understandings of how the brain relates to depression, these accounts contain some illustrative similarities. One such similarity is the metaphor of 'masking the root,' or of palliative treatment obscuring the underlying cause. Evoking a Freudian topology, many participants stressed that certain causes were more fundamental than others. In broader culture, this metaphor is most familiarly applied to antidepressants as 'masking' the symptoms of depression but not fully treating the condition. As Participant 1 articulated, "Drugs might make the person feel better but it is only a temporary fix and does not help the person deal with the root cause of the depression." Biology is, by this view, less essential or less fundamental than psychological or sociological causes of depression.

While eschewing biology, Participant 1's account accords with the narratives given by pharmaceutical companies, which underscore the necessity of a chronic medication regime. The very fact that medication appears only as a "temporary fix" serves to strengthen the notion of medicating as a treatment act needing constant renewal. Participant 31 stated this tension more explicitly:

> It is highly unlikely that a person can take anti-depressants for a short period of time and then be free of depression thereafter. Most patients must stay on these types of medications for an extended period of time to achieve benefits. Once a person stops taking medication for depression, symptoms will most likely continue. Thus, medications to treat it are a short-term fix for a long-term problem. (biological illness condition)

It is as if, even at the same time that the brain is deprioritized explicitly, the brain is implicitly reprioritized. This reprioritization occurs in the sense that—although not fundamental—neurobiology still requires the individual's continual attention and care. Medications are conceived of as short-term fixes or 'masks' that are nevertheless appropriate for a long-term problem or 'root' cause.

The 'masking the root' metaphor is not only put to work in justifying the consumption of medication and underscoring its shortcomings. Rather, this metaphor can also be used to explain why therapy as well is not sufficient for treating depression. Just as Participant 1 in the biological illness condition claimed medications to be insufficient because they treat only symptoms and not 'the root cause,' several participants' narratives described how psychotherapy 'masks' a problem that, at its 'root,' is fundamentally biological. According to this mobilization of the 'masking the root' metaphor, therapy is understood to provide skills for coping

with depression, but still fails to target the ontologically true (and biological) depression:

> In my opinion, people with depression may be able to mask their symptoms through change in lifestyle, exercise, medication or therapy, but the predisposition to depression will always be inside of them to arise at times when they least except it. Mental illness can never be truly eradicated, people just learn to cope and live with it. (Participant 2, malleable condition)

Indeed, the 'masking the root' metaphor is also reflected in the scripts of the video stimuli. Where the biological illness video explains:

> Some scientists believe that psychotherapy—the process of talking to a professional therapist—is beneficial mainly because it helps patients make sure they take their medication regularly. Psychotherapy may also help depressed individuals *cope* with their symptoms and *endure* the negative effects of their disorder. (Emphasis added)

This video's explanation presents therapy as purely aimed at the downstream symptoms and effects of depression instead of at its root. The malleable video upends this metaphor: "For instance, medications are available that can help reduce or control the symptoms of depression, which can make it easier to learn the kinds of skills that allow people to be in control of their mood." Once more, what is expressed here is the familiar view that medications provide enough palliative relief to begin the actual work of learning how to control one's mood, yet these medications fail to address the 'root' psychological dysfunction presumed by this view to be fundamental to depression.

What participants' employment of the 'masking the root' metaphor reveals is that people can use similar and even identical modes of discourse to understand health and illness *even while* those modes signify distinct etiologies, prognoses, and

treatment preferences. Such varied use of the 'masking the root' metaphor exemplifies the rhetorical adaptability that Thornton (2011) has observed in discourse about the brain. She has noted how the brain can pivot between agency and determinism. Similarly, the 'masking the root' metaphor demonstrates that while the *content* of participants' responses differed in terms of etiologies and treatment preferences, those responses might nevertheless be similar in terms of their *structure*. However, the differences between those participants who emphasized the biology of depression and those who focused on psychosocial causes, while illuminating, did not appear as salient as structural differences in the narrative continuity of responses. Although some responses appeared to portray a sharp gap between sadness and depression, other responses depicted a continuous movement between sadness and depression.

8.2 Discordant Harmonies

As indicated by the quantitative content analysis, participants explained depression using a variety of etiologies. The large number of responses containing more than one etiology is testimony to the explanatory multiplicity of health and illness accounts. Such multiplicity is not unique to the participants' responses: it is also evident in the scripts of both psychoeducational videos. Consistent with the NIMH brochure on depression (see pp. 10-11), the biological illness video gives extreme explanatory priority to the biological. For example, the video's script claims that environmental causes of depression are the result of genetic dispositions, and

those dispositions, in turn, lead people into situations that produce depression (see Appendix B). By explicitly incorporating neurobiological terminology, these explanations gain a significant truth effect (Thornton, 2011; see also Weisberg, 2008). However, our data indicate that when such neurobiological discourse stands alone, it is *not* enough to produce a truth effect. Rather, our findings show that even though biological explanations for depression often rhetorically pass *through* the brain or genetics, it might be the case that these biological explanations are not as compelling unless they also figuratively pass *out* of the brain and appertain to non-biological, psychosocial factors. Indeed, just one of the sixty-five responses included *only* a biogenic etiology of depression.

In all experimental conditions, and often even within a single response, etiologies were varied: participants gave an average of 2.7 distinct etiologies (*SD* = 0.973) out of the 5 possible etiologies for which we coded. This pattern was further evidenced by an almost stereotypical opening sentence:

> I believe there can be multiple reasons behind depression. (Participant 1, biological illness condition)
>
> I believe that there is a huge variety of triggers that can bring about feelings of sadness or depression. (Participant 4, malleable condition)
>
> I think depression and, or [*SIC*] sadness can come from a variety of different things. (Participant 10, control condition)
>
> People tend to express feelings of depression or become depressed for any numer [*SIC*] of reasons. (Participant 12, biological illness condition)

Nearly identical opening statements were made by Participants 14, 27, 28, 30, 35, 36, 37, 40, 43, 49, 52, 53, and 58, with even more participants who provided a pluralistic

view of depression further into the body of their response. Such statements were made by participants in every condition, and the pervasive multiplicity of etiologies evident within participants' responses indicates that public understandings of depression do not consist of simple and singular causal models.

Beyond assembling varied etiologies, participants also connected etiologies to treatments in unexpected and, at times, seemingly paradoxical ways. The quantitative content analysis determined that there was no significant difference in the treatment preferences given across conditions, indicating that treatment preferences are not as linked to particular explanations of depression as might be initially assumed. Descriptions of a biological etiology were not necessarily paired with descriptions of a biological cure, and, similarly, a depression claimed to be caused by past trauma often was seen as amenable to treatment with antidepressants. Such variation evidences Thornton's claim that there is "something of a paradox" in the way that brain discourse is deployed to support both determinism and agency (p. 4). In particular, participants' responses frequently moved from biogenic causes to psychosocial cures, a pattern exemplified by Participant 44:

> People become depressed primarily because of their genetic makeup. An individual is far more likely to exhibit symptoms of depression if there is a history of depression in his or her family, even if they not [*SIC*] exposed to any significant traumatic experiences as a child nor suffer a catastrophe in adulthood that might naturally induce a depressed state…. I firmly believe a person can overcome feelings of sadness and even severe depression simply by experiencing support from those around them…. There is also something to be said about therapy and prescription medicine, though I believe over time being surrounded by loved ones who value and cherish you is the most effective medicine.

Participant 44, a respondent from the biological illness condition, began his statement by making a clear etiological claim about depression: it is biological. Although such an etiology might on the surface seem like a basis for ruling out the efficacy of any treatment that does *not* attempt to intervene in that biology, he explicitly stated that "being surrounded by loved ones" is superior to "prescription medicine." While appearing paradoxical or inconsistent, the frequency with which participants made such reversals indicates that moving from "genetic makeup" to "loved ones" serves a definite self-making function. This shift functions to assuage the responsibility of the depressed person for becoming depressed *and* asserts their capacity for recovery.

Many other participants echoed the movement between the biological and the psychosocial. For Participant 32, also from the biological illness condition, the transition from biological causes to psychosocial cures was accomplished within a single sentence. He explained, "Depression can be caused by weak gene's [*SIC*], but is recovered from by strong bonds." This movement can also be seen more fully in the paragraph immediately prior to the above sentence:

> When a person finds something bad happening to them, with no way to resolve the situation, forcing them to "just deal" with it, people become depressed as the only emotional response they are able to give is one of sadness, or depression. When I was in the military overseas, my comrades would del [*SIC*] with depression regularly in different ways. It was because bad things would happen, and we just had to push on and deal with it. However, there [*SIC*] camaraderie and the moving on was what helped the most. Sometimes dealing with something is just acceptance, putting it into context and moving on from it, rationalizing chaos and uncontrollable things as just "shitty."

'Dealing with it' is a refrain repeated three times in this response, and yet its meaning is by no means singular. In the first instance the respondent sees 'just dealing' as

precipitating depression: 'just dealing' is an undesirable and 'forced' condition. In the second instance, 'just dealing' is positioned as an unavoidable fact, something that Participant 32 'just had' to do. Finally, in the last sentence, the inescapable need to 'deal' is reconfigured as the way out of depression. In sum, this response demonstrates a shift from a view of depression where there is very little choice or control, where it is the "only emotional response" one can give, to the very assertion of one's agency or autonomy.

These 'lumper' participants were able to move seamlessly from descriptions of the depressed person as out of control of their experience to assertions of free will and control. In general, the movement from non-agency to agency and uncontrollability to controllability parallels the shift from biological to psychosocial explanations. As Thornton (2011) suggests, talk of determinism and agency each carry rhetorical costs and benefits (p. 157). Determinism naturalizes experiences one might otherwise want to change, but excuses one from responsibility for those experiences. Agency allows for the possibility of intervening in those experiences, however it also simultaneously places potentially onerous and "unremitting obligations to accumulate more health…and enjoy more happiness" (p. 157). Our data suggest that the opposition between these two stances and their implied view of agency is by no means incommensurable, for the 'lumper' participants in this study appear to have been quite capable of having their cake and eating it too.

In talking about depression, shifting from the biological to the psychosocial pays enormous dividends. Such a shift in discourse accomplishes at once the guilt-

relief of biological explanations while simultaneously asserting agency and control—a win-win discursive feat that Participant 26 (biological illness condition) most clearly illustrated. He wrote, "I do believe that depression is something that people cannot control, but I also believe that it something that people can overcome." The ability of some participants to travel so elegantly from seemingly incommensurable frameworks might explain why the malleable video did not lead to a reduction in positive statements as the biological illness video did. Since the malleable video led participants to state a greater variety of etiologies for depression, it gave participants the vocabulary they needed to both assuage guilt and assert control. As Participant 7 (control condition) illustrated, an expanded and pluralistic vocabulary can also lead to greater sympathy for people with depression. She reasoned, "There are oftentimes so many factors that can play into its causation that it becomes more understandable why there are many people that suffer for so long." That a pluralistic understanding of depression might enable greater acceptance of people suffering from depression makes sense in light of the extant theories of essentialism used to explain the negative effects of biological explanations (Dar-Nimrod & Heine, 2011; Haslam, 2011)—it is simply much harder to maintain that a mental illness has an *immutable*, *fundamental discrete*, *homogenous* and *natural* essence when it has a variegated and indeterminate causal origin. Among 'the lumpers,' such pluralism might also facilitate a certain open-mindedness with regards to depressed individuals: 'even while I might not understand why, you nonetheless have legitimate reasons for being depressed.'

8.3 Binaries of Depression

Despite its clear rhetorical benefits, the varied combinations of etiologies and treatments described above were by no means the only kinds of multiplicity evident in participants' accounts. Just as there were responses that mentioned the multiple *causes and treatments* for depression, there were an equal (and largely separate) number of responses that described in detail multiple kinds of *depressions*. Such responses were exemplified by another archetypal opening sentence:

> Theres [*SIC*] a difference between sad, blue, and depressed. (Participant 11, biological illness condition)
>
> I usually distinguish between sadness and depression because I feel they are different. (Participant 16, control condition)
>
> Sadness, blueness and depression all mean different things to me. (Participant 48, control condition)
>
> As a person with depression, I tend to differentiate between feelings of depression and feelings of sadness. (Participant 63, biological illness condition)

Participants 3, 28, 42, 54 and 60 also opened their responses with a similar phrasing, and still more 'splitters' reflected on the boundaries between sadness and depression, and on the different kinds of depression. Unlike the 'lumpers,' these 'splitters' did not claim these different mental states to be continuous with one another.

The distinction between depression and sadness often included a parallel differentiation in etiologies: while sadness was deemed circumstantial, depression was depicted as biological. As Participant 15, from the control condition, explained:

> A person suffering from mild to severe depression has an underlying biological basis as to their 'feelings'. Some malfunction is causing the individual to experience an intensified state of being sad, and no matter what

> that individual might do to try to make themselves happy again they remain unable to do so. An average sad person has experienced some sort of event or stress that is causing them to feel upset. These people almost always overcome the event and their sadness to return to a baseline happy [*SIC*] or state of indifference.

Here, as in other responses among the 'splitters,' depression and sadness are depicted as so distinct that they appear categorically different. One is a biological disorder, and the other is "an appropriate response to a tragic/ traumatic event" (Participant 16, control condition). One is normal, the other is not. Such explanations contain some of the elements of essentialist discourse (Dar-Nimrod & Heine, 2011), in that depression appears here to be *fundamental*, *immutable*, *homogenous* and *discrete*. However, such explanations also depart from the common understanding of essentialist thinking in several significant ways. By our participants' accounts, the particularly salient qualities of depression run orthogonal to Haslam (2011) and Dar Nimrod and Heine's set of defining characteristics for essentialist categories. Depression is not described as particularly *natural*, one of the defining elements of an essentialist category (Dar-Nimrod & Heine, p. 2), and is instead contrasted with the "*natural*" quality of sadness. As Participant 6 wrote, "Depression or sadness due to an emotional strain, I feel [*SIC*] is a bit different than the psychological disorder of depression. I think it is extremely *natural* to feel sad when you have lost someone you love" (Participant 6, control condition, emphasis added). Participant 6's response further evidences a broad pattern of demarcating depression from "*natural*" feelings of sadness that occur within a concrete context. Furthermore, depression was described by many participants in a more technical and vague language than was sadness: depression is a

"psychological disorder," or results from an "underlying biological basis" or "*some* malfunction" (Participant 6, emphasis added). Rather than appearing to have a "specific etiology," (Dar-Nimrod & Heine, p. 3; see also Meehl, 1977) depression, as depicted by Participant 6 and others, does not have a concrete or *specified* etiology. Instead, depression appears to emerge from the *unspecified* and mystified black box of biology.

Also evident in Participant 15's response is a distinction between agency and control that often parallels the sadness/depression binary. Although depressed people remain "unable… to make themselves happy" (Participant 15), those who are sad can actively "overcome." Rose (2003), following Ehrenberg (1998), notes that the twenty first century's emphasis on "continual incitements to action, to choice, to self realization and self improvement" (p. 54) configure the opposites of agency as *pathological*. Depression, with its symptoms of low energy and low productivity, thus becomes the "exemplary" pathology of the postmodern experience. Rose observes that the rise of neurochemical selfhood and neurobiological discourse does not simply entail "blurring the borders between normality and pathology," (p. 58) as seen among the 'lumper' participants who describe depression and sadness as continuous with one another. Rather, neurochemical selfhood, in its most realized form and as evidenced in Participant 15's response, does not position psychiatric medications as promising a restoration to a "fixed norm of civilized conduct through a once off program of normalization. Rather, they oblige the individual to engage in constant risk management" (pp. 58-59). In demarcating sadness from depression, Participant 15

exemplifies a tension between the agentic and healthist logic of continually trying to treat depression and the deterministic trap of remaining "unable to do so." It is almost as though even if a person with depression could return to a seemingly 'normal' mental state, by Participant 15's view, this 'normality' would be in constant threat of lapsing back into pathology.

The division of sadness from depression and agency from non-agency also accompanied a distinction between the *comprehensibility* of sadness versus the *incomprehensibility* of depression. In Participant 15's response, the insertion of quotation marks around the word "feeling" is particularly significant in that it signals that what depressed people are experiencing as feelings are, in fact, not feelings—at least not feelings in the normal sense of the word. Instead, the quotation marks serve to further obscure the depressed experience. Furthermore, just as the distinction between sadness and depression accompanies a difference between the commonplace and the incomprehensible, certain *types* of depression were viewed as more understandable than others. For example, Participant 2, from the malleable condition, wrote that "while the onset of some types of depression is due to trauma, death, pain or loss, other types of depression come paired with no quantifiable 'reason' for being depressed." Similarly, Participant 38 (biological illness condition) attested that while there is one kind of depression caused by environmental circumstances, other people, "who become depressed with no apparent reason, prove that our genes ultimately cause depression." Even among those 'splitters' who did not endorse a clean cut

between depression and sadness, a distinguishing feature of 'pure depression' was its unknowable or impenetrable etiology. As Participant 3 (control condition) articulated:

> Sadness I'm sure can lead to depression but I believe that a person can become depressed without having been sad previously. I think that someone can just sort of wake up depressed one morning and that this can persist for a long time.

This unknowable quality of depression at once "prove[s]" (Participant 38) the distinctiveness of depression as a separate kind of experience and gives depression its power. As Participant 16 explained, "A sad person can easily tell what is going on with their life and seek to improve it. Meanwhile a depressed person has a rough journey ahead of them to recover." Indeed, a common theme throughout the responses was the following notion: that not being able to give a specific etiology for depression makes the experience of depression more difficult. Paradoxically, this obscured depression, without namable psychosocial causes, appears more evidently material or more biologically *real*.

For many participants, especially those endorsing a distinction between depression and sadness, the first step towards recovery from depression is often the diagnosis of its etiology. Participant 47, from the control condition, illustrated this notion of sense-making as integral to recovery:

> Another thing that can make us feel "sad" is the feeling that we can't identify what feels wrong inside of us… The key to "solving" depression is not merely a list of quick-fix solutions, but a true analysis of the source of the catalysts and then a path towards improvement and alleviation.

Many participants echoed this psychoanalytic logic. The road to recovery is, according to this line of thought, a road towards the discovery of depression's

etiology. By making a wider variety of etiologies plausible and opening up space to navigate through biological, psychological, *and* social factors, the malleable video influenced participants to give more positive statements. The same pluralizing mechanism might be behind the findings of increased optimism and feelings of agency in previous studies that used the malleable video (Lebowitz et al., 2013).

When biology is the only etiology that holds sufficient truth value for a participant, the road to discovering the specific etiology of one's depression appears to be much more challenging. Participant 62 (biological condition) challenges the sense-making capacity of biological discourse:

> I find that during my 3 year period of depression in high school, a lot of it was triggered by the media and how I felt I was supposed to look, act, think and feel about myself. I did not fit the standards that were constantly being asked of me and of all women. I especially suffered from body dis-morphia [*SIC*] and developed a serious eating disorder and practiced self harm. And now, looking into my family history and thinking about my own diagnosis of depression, it is odd for me to think that I am not genetically prone to have depression because neither my parents or my grandparents ever suffered from sever [*SIC*] or even moderate depression. I understand that people get sad, really really sad, and I am sure that many people in my family have suffered from deep sadness, but no one was ever diagnosed with depression. I guess that it is possible that my great grand parents or even my grandparents could have had depression but just didn't have the recourses [*SIC*] to be treated or to pay any type of attention to it… I have thoroughly asked my parents about their possibile [*SIC*] history with depression and neither of them felt that they ever suffered from it.

The power *and* insufficiency of the biological explanation is evident here. Despite there being available reasons behind her depression—that she felt unable to live up to unhealthy standards of body image—the respondent continued to search for a biological explanation. It is almost as if the psychosocial causes did not hold sufficient explanatory power for her. Furthermore, because of her clear-cut distinction

between sadness and depression, she actively struggled to relate her own depression to the "deep sadness" experienced by others in her family.

8.4 Summary of Qualitative Analysis

Beyond the ways that such categorical thinking limits the potential for harmonious discord—the ability to pluralistically travel between paradoxical stances of controllability/uncontrollability, comprehensibility/incomprehensibility and biology/psychosociality—it is particularly striking which participants did *not* make categorical cuts between mental states. Of all the participants in the malleable condition, only Participant 19 made a categorical distinction between "depression" and "unhappiness." Instead, respondents from the malleable condition tended to express opinions more in line with Participant 40, who wrote that she views "sadness as a gradient, with depression on one end and lighter emotions on the other." Sadness/depression are not so different that one cannot move from one to the other. Nevertheless, the binary remains. The movement from sadness to depression is still accompanied by a transition from controllability and agency to uncontrollability and determination. When one moves from sadness into depression, it is like crossing over a tipping point, or a point when "depression truly sinks in" (Participant 59, malleable condition). After that reversal, a sense of meaningful etiology and control vanishes, or, as Participant 7 wrote, "I feel that it makes it such [*SIC*] harder to emerge from the feelings of sadness because the affected person doesn't understand why they are feeling the way they are. This sense of feeling out of control then becomes an issue

and the person can easily spiral downwards from there." Yet, by conceiving depression/sadness as a spectrum and not as two distinct kinds, the 'lumpers' could articulate a more confident path to recovery by rhetorically travelling in and out of determinism and agency. Perhaps more than anything else, the crucial difference between participants had less to do with the kind of etiologies they gave and more with whether or not they were able to rhetorically reconcile distinct etiologies.

The similarity of metaphors and languages across responses bears witness to Thornton's observation of the rhetorical adaptability of neurobiological discourse. The variegated application of the 'masking the root metaphor,' which does not in and of itself resonate with biology, demonstrates that not only brain discourse, but mental health discourse more broadly, can be used in contrary and at times paradoxical ways.

Furthermore, 'the lumpers'' paradoxical shifting from biological causes to psychosocial cures suggests that talk of depression is more than a recitation of facts but a negotiation of the depressed person's position as a worthy participant in the social world (Radley & Billig, 1996, p. 221). When respondents shifted away from biology they did so just as biological discourse ceased to assuage the responsibility of the depressed person, and moved towards psychosocial explanations at the very moment that biological determinism would have otherwise left the depressed person without the agency to affect a change in their depression. Among those 'splitter' participants who did reconcile sadness and depression or determinism and agency, the category of depression appears particularly problematic. These 'splitters' were left with a view of depression that was considerably more pessimistic than that of their

'lumper' counterparts: a view of depression as discrete from sadness, as determined by biology, and, crucially, as *incomprehensible* and 'without reason.' This incomprehensible quality, in particular, might account for deterministic biological explanations' negative effects observed both in this study and earlier studies (Deacon & Baird, 2009; Lebowitz et al. 2013), and supplements claims that such negative effects result from essentialist thinking (Haslam, 2011; Dar-Nimrod & Heine, 2011).

9. Conclusion

9.0 Limitations of Present Study and Directions for Future Research

This study provides further support for Lebowitz et al.'s (2013) finding that biomedical explanations for depression produce unwanted effects in terms of prognostic accounting. The finding that the biological video leads to more statements of prognostic pessimism and fewer statements of prognostic optimism challenges the received wisdom of anti-stigma campaigns, which has posited that disseminating the science of disease suffices to destigmatize the illness. The study finds only modest support for the previously documented effects of the malleable explanation of depression's biology (Lebowitz et al.): participants who viewed the malleable video did not differ from the control condition's participants in terms of prognostic optimism, and expressed more prognostic pessimism than control participants. However, the quantitative content analysis of participants' responses demonstrates that the malleable video leads to an increase in the variety of distinct etiologies employed in accounting for depression. Moreover, the qualitative analysis suggests that participants who watched the malleable video found it easier to navigate between a deterministic discourse of biological depression and a discourse of sadness as controllable. Such a pluralistic flexibility and rhetorical integration of sadness and depression might combat an 'othering' or mystification of depression. Indeed, it might be that the negative effects of the biological explanation are due not just to the way that biological explanations facilitate an essentializing mode of cognition (Dar-

Nimrod & Heine, 2011; Haslam, 2011; Lebowitz et al., 2013), but rather the way that they separate depressed experiences from common and relatable experiences.

While the findings of this study have important implications for educating the public about mental health, it is not without several notable limitations that should be addressed in conducting future research. In terms of sampling, the study draws from a participant pool of college students in an introductory psychology course who were required to take part in several research studies for course credit. Because of the blurring between research and education in this experimental encounter, the role of the researcher might be less like that of the distanced observer, engaged interviewer, or compassionate clinician, and instead might appear more similar to an evaluating educator. Given the need in a classroom environment to be 'correct' by the educator's standards and not by one's own standards, the study design, with its resemblance to an essay exam, might have elicited responses that reveal more about what students believe *their professors* think about depression than about what students *themselves* say about depression in other contexts. Future research should examine different populations' accounts of depression, as well as accounts of depression that take place in other contexts, including patient-therapist or patient-psychiatrist encounters, peer-to-peer discussions, and parent-child conversations. Particularly crucial is the project of understanding how biomedical discourse about depression changes as one begins to identify as a patient with depression, or as one ceases to identify with patient status through the course of recovery.

As the codebook for the quantitative content analysis specified that categories were to be coded for *manifest* meaning and not *latent* meaning, the quantitative results should be interpreted conservatively. Sentence frequencies do not necessarily indicate what respondents truly think. It is possible for a participant to speak at length about the biology of depression while nevertheless *not* viewing it to be as important in causing depression as the death of a loved one. Counting sentences allows for the quantification of themes across conditions and a more 'objective' mode of content analysis, yet the analysis could have easily assessed responses for the mere presence of a theme, or employed an ordinal scale for the endorsement of a certain theme. Indeed, future studies might use such approaches in quantifying open-response data.

This study's content analysis is also limited by the decision to create coding categories *a priori,* and not through engagement with the data. Although creating coding categories without knowledge of the content or structure of the data allows for hypothesis testing and controls experimenter bias, this procedure has a trade-off in terms of theoretical sensitivity, and the *a priori* emphasis on biological explanations may have detracted from an examination of other kinds of explanations. Future studies might fruitfully examine how *non-biological* explanations of depression focusing on psychological or socio-cultural etiologies compare to the two explanations of depression's biology used in this study. Although conveying the emerging biomedical science of depression to patients and the public is certainly important, emphasizing biology at the expense of psychosocial factors might not be

the best approach for reducing stigma, increasing optimism about recovery, or improving a sense of agency over mood.

Future efforts also might shift attention away from explanations of depression itself and instead examine how individuals describe individual treatment options like therapy or medication. Furthermore, since talk of treatment sometimes departs dramatically from etiological accounting in terms of how agency is negotiated, research might examine more of the ways that seemingly divergent views of the self are made to fit together within an illness explanation. Additionally, future studies might take a more applied perspective by moving away from discourse and instead examining how these two psychoeducational interventions affect treatment outcomes and decisions. Glattacker et al.'s (2013) finding that shorter expectations of illness duration lead to improved treatment outcomes and Lebowtiz et al.'s (2013) observation that the malleable video causes expectations of shorter illness duration, considered together, have powerful implications. It might be that simply giving an explanation of depression to patients, in a way that respects agency without blaming the patient, is an important and efficacious step on the road to recovery. To address the effects of explanations on recovery, a project might track patients' progress and explanations of depression longitudinally after exposing them—and possibly their families—to a psychoeducational intervention, in order to come to a better understanding of the placebo and nocebo effects of psychoeducation.

9.1 Call to Action: Implications for Anti-Stigma Interventions

Recent calls for interventions designed to reduce stigma against people with depression and other mental illness groups have highlighted strategies of emphasizing the importance of gene-environment interactions or otherwise underscoring the plasticity and malleability of neurobiology (Dar-Nimrod & Heine, 2011, p. 14; Lebowitz et al., 2012). Such recent calls have contested earlier campaigns that claimed that simply educating the public about the biology of depression (see pp. 3-5) should be sufficient to reduce stigma. Instead, recent champions of malleable explanations have added a crucial nuance by insisting that there are *different kinds* of biological explanations. Moreover, they have documented how deterministic explanations tend to actually increase aspects of stigma against mental illness groups.

Instead of educating the public about the plastic biology of depression, our data indicate an even more significant break with earlier anti-stigma campaigns. Our participants' responses indicate that understandings of depression appear particularly pessimistic about control and prognosis when depression is considered as determined by biology, and as distinct, vague and incomprehensible compared to commonplace emotionality. Attempts to reduce stigma against people with depression, then, should not aim to merely convey information about depression to the public. Instead of aiming for education, anti-stigma campaigns might strive to help the public reflect on what they already know. The 'splitters'' pessimistic conceptualization of depression as incomprehensible might be remedied by helping them to integrate their understanding of depression with more quotidian experiences of sadness, to

contemplate their own experiences of moving between uncontrollable and controllable emotions, and to consider the multitude of reasons that have caused them or people they know to experience sadness or depression. A project encouraging reflection and the integration of scientific stories and commonsensical understandings neither negates emerging biomedical sciences nor invalidates the public's complex, nuanced, and dynamical understandings of depression.

References

Abraham, K. (1927). Melancholia and obsessional neurosis. In Douglas Bryan and Alix Strachey (Trans.), *Selected papers of Karl Abraham M.D*. (422-432). London: Hogarth.

Ahn, W. K., Flanagan, E. H., Marsh, J. K., & Sanislow, C. A. (2006). Beliefs about essences and the reality of mental disorders. *Psychological Science*, 17(9), 759-766.

Ahn, W. K., Proctor, C. C., & Flanagan, E. H. (2009). Mental health clinicians' beliefs about the biological, psychological, and environmental bases of mental disorders. *Cognitive Science*, *33*(2), 147-182.

Angermeyer, M. C., Holzinger, A., Carta, M. G., & Schomerus, G. (2011). Biogenetic explanations and public acceptance of mental illness: systematic review of population studies. *The British Journal of Psychiatry*, *199*(5), 367-372.

Babbie, E., & Mouton, J. (2001). *The practice of social science research*. Belmont, CA: Wadsworth.

Bauer, M. W., Allum, N., & Miller, S. (2007). What can we learn from 25 years of PUS survey research? Liberating and expanding the agenda. *Public Understanding of Science*, 16(1), 79-95.

Beck, A. T. (1963). Thinking and depression: I. Idiosyncratic content and cognitive distortions. *Archives of General Psychiatry*, *9*(4), 324-333.

Becker, M. H. (1974). The health belief model and personal health behavior (Vol. 2, No. 4). *Slack*.

Beeman, W. O. (1985). Dimensions of dysphoria: the view from linguistic anthropology. *Culture and depression*: *Studies in the anthropology and cross-cultural psychiatry of affect and disorder*. A. Kleinman and B. Good, eds, 216-243. Oakland, CA: University of California Press.

Brossard, D., & Lewenstein, B. V. (2009). A critical appraisal of models of public understanding of science. *Communicating science: New agendas in communication,* 11-39. New York: Taylor & Francis.

Brown, C., Battista, D. R., Sereika, S. M., Bruehlman, R. D., Dunbar-Jacob, J., & Thase, M. E. (2007). Primary care patients' personal illness models for depression: relationship to coping behavior and functional disability. *General Hospital Psychiatry*, *29*(6), 492-500.

Carpenter-Song, E., Chu, E., Drake, R. E., Ritsema, M., Smith, B., & Alverson, H. (2010). Ethno-cultural variations in the experience and meaning of mental illness and treatment: Implications for access and utilization. *Transcultural Psychiatry*, 47(2), 224-251.

Charmaz, K. (1999). Stories of suffering: Subjective tales and research narratives. *Qualitative Health Research*, *9*(3), 362-382.

Cowen, P. J. (2008). Serotonin and depression: pathophysiological mechanism or marketing myth? *Trends in Pharmacological Sciences*, 29(9), 433-436.

Dar-Nimrod, I., & Heine, S. J. (2011). Genetic essentialism: On the deceptive determinism of DNA. *Psychological Bulletin*, *137*(5), 800.

Deacon, B. J., & Baird, G. L. (2009). The chemical imbalance explanation of depression: reducing blame at what cost? *Journal of Social and Clinical Psychology*, 28(4), 415-435.

Denzin, N. K. (2012). Triangulation 2.0. *Journal of Mixed Methods Research*, *6*(2), 80-88.

Dillard, J. P., & Nabi, R. L. (2006). The persuasive influence of emotion in cancer prevention and detection messages. *Journal of Communication*, 56(s1), S123-S139.

Dunlop, B. W., Kelley, M. E., Mletzko, T. C., Velasquez, C. M., Craighead, W. E., & Mayberg, H. S. (2012). Depression beliefs, treatment preference, and outcomes in a randomized trial for major depressive disorder. *Journal of Psychiatric Research,* 46(3), 375-381.

Ehrenberg, A. (1998). *La fatigue d'être soi: Dépression et société*. Odile Jacob: Paris [For Trans see: Ehrenberg, A. (2009). *Weariness of the Self: Diagnosing the history of depression in the contemporary age*. Montreal: McGill-Queen's Press.]

Engel, G. L. (1977). The need for a new medical model: a challenge for biomedicine. *Science*, 196(4286), 129-136.

Executive Office of the President of the United States (1990a). Proclamation No 6158. Decade of the Brain, 1990-1999.

Executive Office of the President of the United States (1990b). Proclamation No 6196. Mental Health Awareness Week, 1990

Farrer, L., Leach, L., Griffiths, K. M., Christensen, H., & Jorm, A. F. (2008). Age differences in mental health literacy. *BMC Public Health*, 8(1), 125.

Flick, U. (2000). Qualitative inquiries into social representations of health. *Journal of Health Psychology,* 5(3), 315-324.

France, C. M., Lysaker, P. H., & Robinson, R. P. (2007). The" chemical imbalance" explanation for depression: Origins, lay endorsement, and clinical implications. *Professional Psychology: Research and Practice*, 38(4), 411.

Freedom From Fear. (n.d.). National Anxiety and Depression Awareness Week. Retrieved January 30, 2015, from http://www.freedomfromfear.org/NationalAnxietyandDepressionAwarenessWeek.en.html

Freud, S. (1917). Mourning and Melancholia. *The standard edition of the complete psychological works of Sigmund Freud, volume XIV (1914-1916): On the history of the psycho-analytic movement, papers on metapsychology and other works*, 237-258. London: Hogarth.

Gallagher, K. M., & Updegraff, J. A. (2012). Health message framing effects on attitudes, intentions, and behavior: a meta-analytic review. *Annals of Behavioral Medicine*, 43(1), 101-116

Glattacker, M., Heyduck, K., & Meffert, C. (2013). Illness beliefs and treatment beliefs as predictors of short and middle term outcome in depression. *Journal of Health Psychology,* 18(1), 139-152

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

Hacking, I. (2007). Kinds of people: Moving targets. *Proceedings of the British Academy*, Vol. 151, p. 285, Oxford University Press Inc: Oxford.

Haslam, N. (2011). Genetic essentialism, neuroessentialism, and stigma: commentary on Dar-Nimrod and Heine (2011). *Psychological Bulletin*, *137*(5), 819-824.

Insel, T. (2013, April 1). Transforming Diagnosis. *Director's Blog.* Retrieved April 20, 2015, from http://www.nimh.nih.gov/about/director/2013/transforming-diagnosis.shtml

Insel, T. R., & Quirion, R. (2005). Psychiatry as a clinical neuroscience discipline. *JAMA*, 294(17), 2221-2224.

Jackson, S. W. (1980). Acedia the sin and its relationship to sorrow and melancholia in medieval times. *Bulletin of the History of Medicine,* 55(2), 172-181.

Joffe, H. (2002). Social representations and health psychology. *Social Science Information*, *41*(4), 559-580.

Joffe, H. (2003). Risk: From perception to social representation. *British Journal of Social Psychology*, 42(1), 55-73.

Jorm, A. F., Kelly, C. M., Wright, A., Parslow, R. A., Harris, M. G., & McGorry, P. D. (2006). Belief in dealing with depression alone: Results from community surveys of adolescents and adults. *Journal of Affective Disorders*, 96(1), 59-65.

Jorm, A. F., Nakane, Y., Christensen, H., Yoshioka, K., Griffiths, K. M., & Wata, Y. (2005). Public beliefs about treatment and outcome of mental disorders: a comparison of Australia and Japan. *BMC Medicine*, 3(1), 12.

Karasz, A. (2005). Cultural differences in conceptual models of depression. *Social Science & Medicine*, 60(7), 1625-1635.

Kemp, J. J., Lickel, J. J., & Deacon, B. J. (2014). Effects of a chemical imbalance causal explanation on individuals' perceptions of their depressive symptoms. *Behaviour Research and Therapy,* 56, 47-52.

Keyes, C. F. (1985). The interpretive basis of depression. *Culture and depression: Studies in the anthropology and cross-cultural psychiatry of affect and disorder*. A. Kleinman and B. Good, eds, 153-175. Oakland, CA: University of California Press.

Kokanovic, R., Butler, E., Halilovich, H., Palmer, V., Griffiths, F., Dowrick, C., & Gunn, J. (2013). Maps, models, and narratives: The ways people talk about depression. *Qualitative Health Research*, *23*(1), 114-125.

Krell, H. V., Leuchter, A. F., Morgan, M., Cook, I. A., & Abrams, M. (2004). Subject expectations of treatment effectiveness and outcome of treatment with an experimental antidepressant. *Journal of Clinical Psychiatry*.

Krippendorff, K. (2004) "Reliability in content analysis: Some common misconceptions and recommendations." *Human Communication Research*. Vol. 30, pp. 411-433.

Kvaale EP, Haslam N, Gottdiener WH (2013) The 'side effects' of medicalization: A

meta-analytic review of how biogenetic explanations affect stigma. *Clinical Psychology Review*, 33(6):782–794.

Lam, D. C., & Salkovskis, P. M. (2007). An experimental investigation of the impact of biological and psychological causal explanations on anxious and depressed patients' perception of a person with panic disorder. *Behavior Research and Therapy*, *45*(2), 405-411.

Lawrence, V., Murray, J., Banerjee, S., Turner, S., Sangha, K., Byng, R., ... & Macdonald, A. (2006). Concepts and causation of depression: A cross-cultural study of the beliefs of older adults. *The Gerontologist*, 46(1), 23-32.

Lebowitz, M. S. (2014). Biological conceptualizations of mental disorders among affected individuals: A review of correlates and consequences. *Clinical Psychology: Science and Practice*, 21(1), 67-83.

Lebowitz, M. S., & Ahn, W. K. (2012). Combining Biomedical Accounts of Mental Disorders With Treatability Information to Reduce Mental Illness Stigma. *Psychiatric Services*, *63*(5), 496-499.

Lebowitz, M. S., Ahn, W.K, & Nolen-Hoeksema, S. (2013). Fixable or fate? Perceptions of the biology of depression. *Journal of Consulting and Clinical Psychology,* 81, 518.

Levene, H. (1960). Robust tests for equality of variances. *Contributions to probability and statistics: Essays in honor of Harold Hotelling*, 2, 278-292. Stanford, CA: Stanford University Press.

Leventhal, H., Brissette, I., & Leventhal, E. A. (2003). The common-sense model of self-regulation of health and illness. *The self-regulation of health and illness behaviour,* 1, 42-65. New York: Psychology Press

Leventhal, H., Meyer, D., & Nerenz, D. (1980). The common sense representation of illness danger. *Contributions to Medical Psychology,* 2, 7-30.

Leykin, Y., DeRubeis, R. J., Shelton, R. C., & Amsterdam, J. D. (2007). Changes in patients' beliefs about the causes of their depression following successful treatment. *Cognitive Therapy and Research*, 31(4), 437-449.

Lin, P., Campbell, D. G., Chaney, E. F., Liu, C. F., Heagerty, P., Felker, B. L., & Hedrick, S. C. (2005). The influence of patient preference on depression treatment in primary care. *Annals of Behavioral Medicine*, 30(2), 164-173.

Lynch, J., Moore, M., Moss-Morris, R., & Kendrick, T. (2015). Do patients′ illness beliefs predict depression measures at six months in primary care; a longitudinal study. *Journal of Affective Disorders*, *174*, 665-671.

Manz, C. (2003). *Emotional discipline: The power to choose how you feel; 5 life changing steps to feeling better every day.* San Francisco, CA: Berrett-Koehler Publishers.

Martin, E. (2010). Self-making and the brain. *Subjectivity*, *3*(4), 366-381.

Meehl, P. E. (1977). Specific etiology and other forms of strong influence: Some quantitative meanings. *Journal of Medicine and Philosophy*, *2*(1), 33-53.

Mehta, S., & Farina, A. (1997). Is being "sick" really better? Effect of the disease view of mental disorder on stigma. *Journal of Social and Clinical Psychology*, *16*(4), 405-419.

Meyer, B., & Garcia-Roberts, L. (2007). Congruence between reasons for depression and motivations for specific interventions. *Psychology and Psychotherapy: Theory, Research and Practice*, 80(4), 525-542.

Minirth, F., & Meier, P. (2013). *Happiness is a choice: New ways to enhance joy and meaning in your life*. Grand Rapids, MI: Baker Books.

Morawski, J. G. (2001). The dynamics of uncertainty. *History and Theory*, *40*(3), 401-418.

Moscovici, S. (1988). Notes towards a description of social representations. *European Journal of Social Psychology,* 18(3), 211-250.

Murray, M., Bennett, P., & Weinman, J. (1990). Lay representations of illness. *Current developments in health psychology*, 63-92.

National Alliance on Mental Illness (2014). Public policy platform of NAMI: The National Alliance on Mental Illness. [Brochure]. Arlington, VA

National Institute of Mental Health. (2011). Depression. United States Department of Health and Human Services: Washington, DC

O'Keefe, D. J., & Jensen, J. D. (2009). The Relative Persuasiveness of Gain-Framed and Loss-Framed Messages for Encouraging Disease Detection Behaviors: A Meta-Analytic Review. *Journal of Communication*, 59(2), 296-316.

O'Connor, C., & Joffe, H. (2013). How has neuroscience affected lay understandings of personhood? A review of the evidence. *Public Understanding of Science*, 22(3), 254-268.

Ortega, F. (2009). The cerebral subject and the challenge of neurodiversity. *BioSocieties*, 4(4), 425-445.

Pescosolido, B. A., Martin, J. K., Long, J. S., Medina, T. R., Phelan, J. C., & Link, B. G. (2010). "A disease like any other"? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *American Journal of Psychiatry*, 167(11), 1321-1330.

Phelan, J. C., Cruz-Rojas, R., & Reiff, M. (2002). Genes and stigma: The connection between perceived genetic etiology and attitudes and beliefs about mental illness. *Psychiatric Rehabilitation Skills*, *6*(2), 159-185.

Radley, A., & Billig, M. (1996). Accounts of health and illness: Dilemmas and representations. *Sociology of Health & Illness*, *18*(2), 220-240.

Richards, G. (2002). The Psychology of Psychology A Historically Grounded Sketch. *Theory & Psychology*, *12*(1), 7-36.

Ridge, D., Emslie, C., & White, A. (2011). Understanding how men experience, express and cope with mental distress: where next?. *Sociology of Health & Illness*, *33*(1), 145-159.

Riessman, C. K. (2003). Performing identities in illness narrative: Masculinity and multiple sclerosis. *Qualitative Research*, *3*(1), 5-33.

Rose, N. (1999). *Powers of freedom: Reframing political thought*. New York: Cambridge University Press.

Rose, N. (2003). Neurochemical selves. *Society*, 41(1), 46-59.

Rosenstock, I. M. (1966). Why people use health services. *The Milbank Memorial Fund Quarterly*, 94-127.

Rothman, A. J., & Salovey, P. (1997). Shaping perceptions to motivate healthy behavior: the role of message framing. *Psychological Bulletin*, 121(1), 3.

Rüsch, N., Todd, A. R., Bodenhausen, G. V., & Corrigan, P. W. (2010). Biogenetic models of psychopathology, implicit guilt, and mental illness stigma. *Psychiatry Research*, *179*(3), 328-332.

Ryder, A. G., Yang, J., Zhu, X., Yao, S., Yi, J., Heine, S. J., & Bagby, R. M. (2008). The cultural shaping of depression: somatic symptoms in China, psychological symptoms in North America? *Journal of Abnormal Psychology*, 117(2), 300.

Sanislow, C. A., Pine, D. S., Quinn, K. J., Kozak, M. J., Garvey, M. A., Heinssen, R. K., ... & Cuthbert, B. N. (2010). Developing constructs for psychopathology research: research domain criteria. *Journal of Abnormal Psychology*, *119*(4), 631.

Schnittker, J., Freese, J., & Powell, B. (2000). Nature, nurture, neither, nor: Black-White differences in beliefs about the causes and appropriate treatment of mental illness. *Social Forces*, 78(3), 1101-1132.

Schomerus, G., Schwahn, C., Holzinger, A., Corrigan, P. W., Grabe, H. J., Carta, M. G., & Angermeyer, M. C. (2012). Evolution of public attitudes about mental illness: a systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, 125(6), 440-452.

Schreiber, R., & Hartrick, G. (2002). Keeping it together: How women use the biomedical explanatory model to manage the stigma of depression. *Issues in Mental Health Nursing*, 23(2), 91-105.

Simmel, G "The Metropolis and Mental Life" (1903). Gary Bridge and Sophie Watson, eds. *The Blackwell city reader*. Oxford and Malden, MA: Wiley-Blackwell, 2002.

Soames Job, R. F. (1988). Effective and ineffective use of fear in health promotion campaigns. *American Journal of Public Health*, 78(2), 163-167.

Stemler, S. E. (2004). A comparison of consensus, consistency, and measurement approaches to estimating interrater reliability.

Strauss, A., & Corbin, J. M. (1990). *Basics of qualitative research: Grounded theory procedures and techniques.* Boston: Sage Publications, Inc.

Swift, J. K., & Callahan, J. L. (2009). The impact of client treatment preferences on outcome: a meta-analysis. *Journal of Clinical Psychology*, 65(4), 368-381.

Szasz, T. S. (1974). *The myth of mental illness: Foundations of a theory of personal conduct.* New York: HarperPerennial.

The White House (n.d.). The BRAIN Initiative. (Retrieved January 21, 2015, from http://www.whitehouse.gov/BRAIN

Thornton, D. J. (2011). *Brain culture: Neuroscience and popular media*. New Brunswick, NJ: Rutgers University Press.

Thwaites, R., Dagnan, D., Huey, D., & Addis, M. E. (2004). The Reasons for Depression Questionnaire (RFD): UK Standardization for clinical and non-

clinical populations. *Psychology and Psychotherapy: Theory, Research and Practice*, 77(3), 363-374.

Turk, D. C., Rudy, T. E., & Salovey, P. (1986). Implicit models of illness. *Journal of Behavioral Medicine*, 9(5), 453-474.

Vidal, F. (2009). Brainhood, anthropological figure of modernity. *History of the Human Sciences*, 22(1), 5-36.

Weinman, J., Petrie, K. J., Moss-Morris, R., & Horne, R. (1996). The illness perception questionnaire: a new method for assessing the cognitive representation of illness. *Psychology and Health*, 11(3), 431-445.

Weisberg, D. S., Keil, F. C., Goodstein, J., Rawson, E., & Gray, J. R. (2008). The seductive allure of neuroscience explanations. *Journal of Cognitive Neuroscience*, *20*(3), 470-477.

Wynne, B. (1993). Public uptake of science: a case for institutional reflexivity. *Public Understanding of Science,* 2(4), 321-337.

Appendices

Appendix A: Codebook

- Category: Explanation (E)
  - Biomedical Explanation (BE)
    - *Depression as result of any biological cause including genetics/heredity[2], neurochemical imbalances, brain disease, and traumatic brain injury, as well as bioenvironmental causes such as viruses and toxins, etc.…*
  - Sociocultural Explanation (SCE)
    - *Depression as result of adverse socio-cultural conditions, political disempowerment, discrimination, poverty, unemployment, sane reaction to insane circumstances, etc.…*
  - Failure of Agency Explanation (FAE)
    - *Depression as result of failure to reign in negative thought patterns, lack of strength, unwillingness to take care of self, lack of responsibility, self-inflicted punishment for moral failure, etc.…*
  - Personal History Explanation (PHE)
    - *Depression as result of traumatic event resurfacing as depression or longstanding attachment difficulties, the death of a loved one, marriage difficulties, estrangement from friends, etc.…*
  - Other Explanation (OE)
    - *Code if none of the above*

- Category: Treatment Preference (P)
  - Biomedicine Preference (BMP)
    - *Recommendation or positive mention of medications as well as electro-shock therapy, trans-cranial magnetic stimulation, vagus nerve stimulation, neurosurgery etc.…*
  - Psychotherapy Preference (PP)
    - *Recommendation or positive mention of any form of talk-based therapy, behavioral therapy, CBT, group therapy, etc.…*
  - Lifestyle Change Preference (LP)
    - *Recommendation or positive mention of changing daily routine, exercise, finding new friends, getting a job, etc.…*
  - Other Treatment Preference (OP)
    - *Code if recommendation for treatment that is not listed above*

---

[2] Heredity is meant in the *genetic* sense of the word, and not in the risk for depression that might be passed down because of the way parents *nurture* a child.

- Category: Knowledge Source (S)
    - Self Knowledge Source (SKS)
        - *Code when the response claims that knowledge was learned from within. This includes when such knowledge comes from a personal experience of one's own depression, or when it states "I know" without claiming any other source.,*
    - Expert Knowledge Source (EKS)
        - *Code when the response claims knowledge was learned from an expert source (scientific authority/publication, professor, education, science media, doctors, therapists, etc.…)*
    - Non-Expert Knowledge Source (NEKS)
        - *Code when the response claims knowledge was learned by observing another's depression or being told by a non-expert other about depression*

- Category: Outlook Towards Recovery (O)
    - Full Recovery Possible From Symptoms and Condition (OO)
        - *Full recovery possible*
    - Recovery Not Possible (PO)
        - *Can never fully recover (symptoms may improve, but not completely)*
    - Remission Possible From Symptoms but not Condition (AO)
        - *Ex: Full recovery possible, but medication must be continued forever (symptoms may completely improve, but the underlying condition is still present)*

- Other Codes
    - Negation (N)
        - *Code any sentence that contains a negation. Examples: "I don't think that depression is caused by interpersonal relationships," "Recovery is not possible" or "I'm not sure."*

Rules for Coding

- The size of each coding unit ("quote" in Atlas.TI) is one sentence
- Any code can be applied to as many sentences as are applicable in each p-doc
- Any given sentence can contain as many codes as are applicable

Appendix B: Transcripts of Stimuli Videos

"Biological Illness" video narration:

Many people who have symptoms of depression, or know someone who does, wonder what causes these kinds of problems. Mood problems, such as depression, run in families. Genetics are a large part of what puts a person at risk for becoming depressed. For example, immediate family members of a depressed person have a significantly higher risk of becoming depressed than would an average person, and having two parents with a history of depression more than doubles a person's risk of becoming depressed. This is because genetics play such a large part in determining whether someone becomes depressed or not.

Genes are like manufacturing instructions for building our brains and bodies. As you may know, genes determine how we look, and what kinds of traits we have. Likewise, certain genes create chemical imbalances in the brain, which are known as major causes of depression. These genes have even been called "depression genes." Furthermore, genes determine whether or not people become depressed in response to stressful events. For example, not everybody who gets mistreated as a child becomes depressed later in life; it is the person's genetic makeup that determines whether that person will become depressed in response to the abuse. Our genes can even influence what environments we end up in, so some people may find themselves in depressing circumstances because of their genes.

In recent years, neuroscience has shown us that depression is truly a brain disorder. Brain imaging has shown that there are real differences between the brains of depressed people and the brains of non-depressed people. Depressed people have abnormalities in critical areas of the brain. The area that is involved in emotional reactions to the environment is over-active; this explains why many depressed people over-react to stressful situations in their lives. At the same time, the brain area that is involved in solving problems is under-active, making it difficult for depressed individuals to think clearly or act effectively to solve their problems. The following clip will explain a little bit about the biology of depression.

[Clip from http://www.thevisualmd.com/health_centers/neurological_health/depression/what_is_depression_video]

As you may know, antidepressants are sometimes given to people with depression. The benefits of these medications, however, tend to be only temporary; scientific studies have shown that depression frequently comes back when people stop taking their medication. Once the medication wears off, there is no longer anything stopping the person's genes from causing chemical imbalances in the brain, so brain chemistry often returns to the way it was before the person started taking the medication.

Therefore, it is important for depressed people to continue taking their medication consistently, much like people with diabetes, high cholesterol, high blood pressure or other chronic illnesses must take their medication every day. Some scientists believe that psychotherapy—the process of talking to a professional therapist—is beneficial mainly because it helps patients make sure they take their medication regularly. Psychotherapy may also help depressed individuals cope with their symptoms and endure the negative effects of their disorder. There are also self-help books that can be used for this purpose. Sometimes, when other treatments are not working, doctors will try electroconvulsive therapy – sometimes called electric shock therapy -- to treat depression. This treatment causes a seizure in the brain, and while scientists do not fully understand how it works, it is known to benefit some very depressed individuals. Although no treatment for depression has a 100% success rate, there are several options that professionals can use when treating a person with this disease.

"Malleable" video narration:

Many people who have symptoms of depression, or know someone who does, wonder what causes these kinds of problems. The truth is, whether or not a person becomes depressed depends upon a wide variety of factors. Genetics alone can never make someone depressed. In fact, even among people who have an immediate family member with depression, a large majority do not become depressed themselves. Even if a person has a genetically identical twin with depression, most of the time that person will not become depressed.

There are many reasons why genes are not the deciding factor in depression. For example, even if a person has depression-related genes, these genes may not be active. Like a light switch, genes can be turned on or off. Research has shown that lifestyle factors like diet, exercise, and levels of stress will affect whether or not genes related to depression will actually be turned on. You could think of genes like the heating system in your house, while other factors act like the thermostat. The heating system is always there, but it is not always active—the settings on the thermostat determine whether the heat will be on or not. Similarly, the genes we are born with are always there, but this does not mean all of them will always be active or "turned on." The following clip, from the University of Utah's Genetic Science Learning Center, will explain a little bit about how genes get turned on and off.

[Clip from video at http://learn.genetics.utah.edu/content/epigenetics/twins]

Some people have heard that depression is caused by a "chemical imbalance" in the brain. In reality though, biological tests of brain activity or brain chemicals cannot even be used to diagnose depression. What's more, scientists have found that the brain is constantly changing because of the experiences and environments we choose. The brain can be compared to a muscle: it grows and changes according to how it is used or "exercised." One way to "exercise" the brain is through learning, which can

strengthen or change the activity of cells in the brain. For a depressed person, it can be very helpful to learn new ways of thinking or interacting with others, sometimes with help from a professional therapist, or the kinds of self-help books that are widely available.

Brain-imaging studies have looked at changes in the brains of people whose depression improved after learning and practicing these kinds of skills and have found something remarkable. These people's patterns of brain activity were found to look more like those of people without depression. That is, their brain activity changed for the better, and because of what they had learned, their depression improved.

There are many pathways out of depression, and there are many things that can help people along these paths. For instance, medications are available that can help reduce or control the symptoms of depression, which can make it easier to learn the kinds of skills that allow people to be in control of their mood. Such skills will stay with a person for a long time—just like learning to ride a bicycle, and many people find that learning them can help keep depression away. Aerobic exercise and exposure to sunlight have also been shown to change brain chemistry and activity in a way that helps with feelings of depression. Whatever might be causing a person's depression, there are many types of support available to help deal with it.

Appendix C: Study Protocol

Welcome to our study! When you are ready to proceed, and the experimenter has left the room, please press the arrows below to begin. If you need any help you can ask the experimenter, who will be right outside to assist you.

Which of the following best characterizes your ethnicity or ethnic background? (Select all that apply)

- ❑ Non-Hispanic White, Caucasian or European American
- ❑ Black, Afro-Caribbean or African American
- ❑ Latino, Chicano or Hispanic American
- ❑ East Asian or Asian American
- ❑ South Asian, Indian or Pakistani American
- ❑ Middle Eastern or Arab American
- ❑ Native American or Alaskan Native
- ❑ Pacific Islander or Hawaiian American
- ❑ Other/Unknown (Please Specify): _____

Which of the following best characterizes your sex (that is, your biological sex and not necessarily the gender you identify)?

- ❍ Female
- ❍ Intersex
- ❍ Male
- ❍ Other (Please Specify): _____

What is your age?

Our lab is very interested in the ways that scientific knowledge about depression is communicated, both between scientists, from scientists to patients, and amongst the general population. We are going to ask you to watch a video depicting a scientific understanding of what feelings of sadness, blueness or depression signify. After you are done watching this video, we will ask you to write an essay, where you will be given an opportunity to express your own understanding of these feelings.

[Video presented]

Please rank the clarity of how this video explained feelings of sadness, blueness or depression:
- ❍ Very Clear
- ❍ Somewhat Clear
- ❍ Neither Clear Nor Unclear
- ❍ Somewhat Unclear
- ❍ Very Unclear

Please rank how convincing you thought this video was:
- ❍ Very Convincing
- ❍ Somewhat Convincing
- ❍ Neither Convincing nor Unconvincing
- ❍ Somewhat Unconvincing
- ❍ Very Unconvincing

Please rank how much you agreed with what this video said about feelings of sadness, blueness or depression:
- ❍ Strongly Agreed
- ❍ Somewhat Agreed
- ❍ Neither Agreed nor Disagreed
- ❍ Somewhat Disagreed
- ❍ Strongly Disagreed

We'd like to ask you to write a 300 word or longer account of feelings of sadness, blueness or depression. We are interested in getting as open an account from you as possible. Please write about your understanding of the average depressed person.

Why do you believe people become sad, blue or depressed? Do you believe people can recover from these feelings, and if so, how?

[Participant given text box for response entry]

Has a psychologist, doctor, therapist or other health professional ever told you that you have an emotional, psychiatric or mental disorder, or treated you for an emotional, psychiatric or mental disorder?
- ❍ Yes
- ❍ No

Have you ever been in psychotherapy?
- ❍ Yes
- ❍ No

Have you ever taken medication for an emotional, psychiatric, or mental disorder?
- ❍ Yes
- ❍ No

Thank you for your participation. The experimenter will now debrief you.

Appendix D: Informed Consent Form

Informed Consent for Research Participation

Student Investigator: Ethan Hoffman
Department of Psychology
Wesleyan University, Middletown, CT
510-292-9671

Purpose

We are conducting a research study to examine the current landscape of common knowledge about feelings and thoughts associated with mental health and distress.

Procedures

A member of the research team will review this form with you and answer any questions you may have. If you choose to participate you will be asked to sign this form. You will be given a copy of this form, which you can take with you.

For this study you will be asked some general questions about yourself and background, and you will also asked to write your opinion about matters related to mental health. You may be asked to watch a video pertaining to mental health. Last, we will ask you some questions about your health history. After you have completed the procedures, the researcher will describe the goals of the study and give you a chance to ask any questions.

Your participation will require 1 hour to participate. You will receive no monetary compensation for your participation, but will receive credit for participation for your PSYC 105 course.

Risks and Benefits

Although it is rare, on some occasions participants may experience discomfort when answering questions about emotions or mental health experiences. This study will not benefit you personally, but we hope that our results will add to public knowledge about how people think about and understand mental health and distress.

Confidentiality

All of your responses will be maintained in strict confidence in accordance with applicable state and federal laws. However, private identifiable information about you may only be used or disclosed for purposes of these research projects as described in this study's authorization form. Only the researchers involved in this

study and those responsible for research oversight will have access to the information you provide. Your responses will be numbered and the code linking your number with your name will be stored in a separate locked file cabinet which only the research team will have access to. Any identifying information will not be revealed in any presentation of these data.

Voluntary Participation

Participation in this study is completely voluntary. You are free to decline to participate, to stop participating at any time for any reason. If you do so, you will still receive course credit. You may also decline to answer any individual question without penalty or loss or course credit.

Questions

If you have any questions about this study, you may contact the student investigator Ethan Hoffman (510-292-9671), or the study's faculty supervisor, Jill Morawski (860-685-2344, Judd Hall 317). If you would like to talk with someone other than the researchers to discuss problems or concerns, or to discuss your rights as a research participant, you may contact Andrea Patalano, Chair of the Psychology Department (860-685-2310) or Jennifer Rose, Chair of the University IRB (860-685-2406).

Agreement to Participate

I am at least 18 years of age. I have read the above information, have had the opportunity to have any questions about this study answered and agree to participate in this study. I consent to participating in the study.

_____ _____

(Printed Name) (date)

_____

(Signature)

_____ _____

(Person Obtaining Participants Signature) (date)

_____

(Signature)

Appendix E: Debriefing Script

The experiment is now over.

For those of you who watched a video, we were interested in how the videos that some of you saw effected the way you wrote about depression in your essay. Some of you watched a video that described depression as determined or caused by neurochemical differences or genetics. However, others of you watched a video that stated that neurochemical differences or genetics do not cause depression by themselves, but lead to depression through a complex interaction with the environment, cognitive styles and "nurture." Furthermore, some of you were in a control group, and did not watch any video or participate in what we called the "first study". These videos were both experimental in nature and were not meant to reflect the current cutting-edge of scientific knowledge about depression.

Previous research has shown that when patients are experimentally led to view depression as a biological disease and as neurochemically or genetically determined, they are more likely to be pessimistic about their prospects for recovery and to feel less control over their condition than people who are led to believe that depression arises from a complex interaction of biology and environment. Furthermore, people who are led to understand depression as biologically-determined are more likely to put faith into psychiatric medication, while viewing psychotherapy as less important for recovery than either experimental groups primed with an interactionist model of depression or control groups.

It was very important to us that you or any potential subjects not know about our hypothesis beforehand, because this knowledge might bias our results. We hope that you understand why we needed to mislead you a little bit. We also hope that you understand the absolute need to keep this experience confidential until the study finishes data collection. If future subjects know about our experiment ahead of time, about our hypothesis, or that the videos are not really psychoeducational videos, this knowledge could make data we collect from them potentially meaningless. Please exercise the utmost responsibility and discretion and do not discuss the experiment with any other students or any friends until the study has concluded in April of 2015, at which point you will receive an email from the research team informing you that our data collection has ended. If you need to discuss this study or your rights as a research participant with someone outside of the research team, you may contact Andrea Patalano, Chair of the Psychology Department (860-685-2310) or Jennifer Rose, Chair of the University IRB (860-685-2406).

Since the subject matter of this study may be upsetting to some subjects, we would like to know if we can help in any way.

Do you feel upset after completing this experiment?

☐ Yes

☐ No

Would you like one of the members of the research team to contact you about this study?

☐ Yes

☐ No

There are also services available on campus to assist with mental health and well-being. You can reach Wesleyan's Counseling and Psychological Services by contacting any of the following numbers or email addresses:

Counseling and Psychological Services (CAPS): 860-685-2910
CAPS Crisis Appointment: 860-685-3143
CAPS After-Hours On-Call: 860-685-2910
CAPS email: counseling@wesleyan.edu

4.) We expect that this study will lead to more studies of a similar nature in the future. Would you be willing to participate in another study like this one in the future? Please indicate whether you would be willing for a researcher to contact you in the future with more participation opportunities

☐ Yes, feel free to contact me in the future

☐ No

5.) To learn more about this work, we suggest the following readings:

Lebowitz, Matthew S. "Biological Conceptualizations of Mental Disorders among Affected Individuals: A Review of Correlates and Consequences." *Clinical Psychology: Science and Practice* 21.1 (2014): 67-83. Print.

Haslam, N., Ban, L., & Kaufmann, L. (2007). Lay conceptions of mental disorder: The folk psychiatry model. *Australian Psychologist*, *42*(2), 129-137.

Kuyken, Willem, et al. "Causal Beliefs About Depression in Depressed Patients, Clinical Psychologists and Lay Persons." *British Journal of Medical Psychology* 65.3 (1992): 257-68. Print.

If you have more questions about this research after reading these sources, you may also contact the student investigator of the study at edhoffman@wesleyan.edu, or the student's faculty mentor, Jill Morawski, at jmorawski@wesleyan.edu.

Thank you for your participation!