ABSTRACT

Lay Beliefs of Anxiety Etiology as a Cause of Perceived Anxiety Controllability: An Experimental Evaluation

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Perceived controllability over the symptoms of anxiety is a key determinant of anxiety severity and an important target in the treatment of clinically severe anxiety. Perceived controllability of anxiety symptoms appears to be shaped partially through verbal persuasion; however, little is known about the origins of these beliefs. Extant findings indicate that the perceived controllability of other mental health concerns can be caused by exposure to etiological explanations. Specifically, exposure to biological and genetic explanations causes lower perceived symptom controllability, whereas exposure to psychological and social explanations does not impact perceived mental health controllability in this way. Despite these findings, this causal relationship remains unexamined with regard to anxiety. The present study aimed to provide the first test of the potentially causal relationship between exposure to biogenetic and psychosocial etiological explanations and perceived anxiety controllability. Participants were randomized to view one of two presentations about the etiology of anxiety and completed a self-report measure of anxiety controllability. Any relationship between etiological beliefs and perceived anxiety controllability was impossible to determine due to failure of

the experimental manipulation to produce statistically significant differences between groups on a manipulation check. A model of naïve theory formation, perseverance, and change is used to compare methods of the current study with those of studies that effectively manipulated lay beliefs about mental health. Factors negatively impacting motivation for processing naïve theories and biases in processing of novel information are identified as potential causes of the manipulation failure in the present study. Future studies may successfully manipulate participants' beliefs by motivating naïve theory processing, establishing an environment conducive to naïve theory processing, and presenting content likely to overcome biases in information processing.

Lay Beliefs of Anxiety Etiology as a Cause of Perceived Anxiety Controllability:
An Experimental Evaluation

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CHAPTER ONE

Introduction

Anxiety is a naturally occurring emotional state that exists on a continuum from normative and adaptive responses to potential threats to impairing, clinically severe presentations (Kollman et al., 2006; Longley et al., 2010; Olatunji et al., 2010; Ruscio et. al, 2001; Silove et al., 2007; Weeks et al., 2009). On the adaptive side of this continuum, anxiety has been argued to support evolutionary fitness by orienting humans toward potential threats in their natural and social environments (Marks & Nesse, 1994; Öhman, 1993) or to potential failures to meet developmental goals critical to survival (Weems, 2008; Weems et al., 1998). At the other end of the continuum, anxiety may be highly impairing. Clinically severe presentations of anxiety – commonly termed anxiety disorders – impact around one-third of adults at some point in their lives (Kessler et al., 2012) and may contribute to significant impairment. Specifically, clinically severe anxiety is associated with lower overall quality of life (Olatunji et al., 2007), decreased work productivity (Kessler & Frank, 1997), negatively impacted interpersonal relationships (Aderka et al., 2012), and increased risk for mortality related to chronic disease (Pratt et al., 2016) and suicide (Voshaar et al., 2015). As reviewed below, the degree to which individuals perceive anxiety as controllable is a key determinant of anxiety symptom severity. Because controllability beliefs are so central to the experience of anxiety, the present study seeks to examine an underexplored aspect of anxiety controllability beliefs, namely how these beliefs may be shaped by exposure to certain

etiological explanations. The present study aims to contribute to the literature by providing the first known investigation of the potentially causal impact of exposure to etiological explanations of anxiety on anxiety controllability beliefs.

Perceived Anxiety Controllability

Low perceptions of control have been identified as a central feature of anxiety in numerous theoretical conceptualizations and empirical studies (e.g., Barlow, 2002; Chorpita & Barlow, 1998; Gallagher, Bentley, et al., 2014). Though conceptually distinct from one another, foundational concepts such as Rotter's (1954, 1966) locus of control, Seligman's (Abramson et al., 1978; Maier & Seligman, 1976) learned helplessness, and Bandura's (1982) self-efficacy have placed a similar emphasis the importance of perceptions of control, characterizing perceived control as a deciding factor in the degree to which an individual experiences distress. Building on this work, Barlow (2002) identified low perceived control as a central factor in the development of clinically severe anxiety, proposing that perceived control shapes individuals' expectancies of their ability to cope with anxiety. The central premise across these theories – that low perceived control is associated with clinically severe psychological distress – has been supported by numerous studies (Alloy et al., 1990; Gallagher, Naragon-Gainey, et al., 2014; Mineka & Kihlstrom, 1978); however, whereas this prior work conceptualized perceived control as a global sense of one's ability to control external events, situations, stressors, and associated anxiety symptoms, other theories of clinically severe anxiety, as discussed next, focus specifically on perceived control of the internal experiences of anxiety (Hofmann, 2007; Wells, 2009).

More precisely, both Hofmann (2007) and Wells (2009) described how low perceptions of control over the cognitive, emotional, and physical experience of anxiety itself are determinants of the severity of anxiety. Hofmann (2007) argued that low perceived control over anxiety symptoms engenders fears of social consequences (e.g., embarrassment). As a result of such fears, individuals putatively engage in behaviors intended to control the experience and outward expression of anxiety (e.g., staying silent to avoid revealing a trembling voice; holding one's hands in one's lap to avoid revealing that they are shaking). These avoidance behaviors are thought to maintain perceptions of low control over anxiety as they interfere with opportunities to increase perceptions of anxiety control (Hofmann, 2007). The role of low perceived anxiety control in Hofmann's (2007) model is supported by findings that fears of losing control or having a panic attack in a social situation were predictive of anxiety symptoms (Hofmann et al., 1995) and that perceived cost of social situations is determined by perceptions of anxiety control (Hofmann, 2005). Though narrowly focused on social evaluative anxiety, Hofmann's (2007) model is important to mention as an example supporting perceived control over anxiety symptoms as a determinant of anxiety severity and speaks to recognition in the field of anxiety research of the importance of perceived anxiety controllability.

This idea has received more investigation and empirical support from Wells's (2009) metacognitive model, which identifies low perceptions of control over mental health experiences as the basis for the development of clinically severe mental health concerns. Specifically within this model, it is proposed that individuals with particular beliefs about the uncontrollability and dangerousness of their thoughts (termed negative

metacognitive beliefs) become trapped in a cycle of repetitive negative thinking, which focuses the individual on threatening information and increases experiences of distress (Wells, 2009). Negative metacognitive beliefs are proposed to interfere with an individual's ability to regulate normative experiences with anxiety, such as worry, by contributing to the perception that experiences of anxiety are aberrant, uncontrollable, and signal threat (Wells, 2009). While Wells's (2009) model includes other factors that are proposed to maintain clinically severe anxiety once it emerges, negative metacognitive beliefs are identified as the central component of the model and the initiator of the cycle of clinically severe distress described above.

Wells's (2009) central premise regarding the role of perceived anxiety controllability has been supported by numerous empirical findings. In tests of the model, negative metacognitive beliefs including the uncontrollability of anxiety account for unique variance in anxiety symptom severity beyond other types of beliefs (e.g., beliefs about the self, world, and future) commonly implicated in the pathogenesis of anxiety symptom severity (Clark & Beck, 2010). In fact, within those studies, negative metacognitive beliefs are generally the only beliefs to evidence incremental explanatory power in relation to anxiety symptom severity supporting the specific importance of the uncontrollability of anxiety (Bailey & Wells, 2013; Barahmand, 2009; Davis & Valentiner, 2000; Fergus & Bardeen, 2017; Fergus & Wheless, 2018; Melli et al., 2016; Myers et al., 2009; Nordahl et al., 2017; Nordahl & Wells, 2017; Ryum et al., 2017). Moreover, studies have identified negative metacognitive beliefs, including those about the uncontrollability of anxiety, as the distinguishing factor between normative and

clinically severe anxiety (Cartwright-Hatton & Wells, 1997; Davis & Valentiner, 2000; Ruscio & Borkovec, 2004; Wells & Carter, 2001; Wells & Papageorgiou, 1998).

Elsewhere, in literature originating from the fields of social and personality psychology, findings demonstrate a causal link between perceptions of the controllability of anxiety and anxiety severity. Specifically, in one five-week longitudinal study, the degree of perceived controllability of anxiety as rated on a weekly basis was found to predict the amount of distress experienced in the subsequent week (Schroder, Kneeland, et al., 2019). Specifically, after controlling for potential confounds such as distress ratings from the previous week, distress increased as perceived control decreased (Schroder, Callahan, et al., 2019). Similarly, other studies have demonstrated that the extent to which anxiety controllability beliefs change over the course of anxiety treatment predicts the amount of anxiety symptom reduction that occurs as result of treatment. For example, in a study examining a cognitive-behavioral intervention, increases in perceived controllability of anxiety indirectly explained symptom improvement over the course of intervention (De Castella et al., 2015). Specifically within this trial, the intervention lead to significantly higher perceptions of anxiety controllability among the intervention participants compared to their waitlist counterparts, and these increases in perceived anxiety controllability explained pre- to post-intervention changes in symptom severity when baseline symptoms and other content-based beliefs were controlled (De Castella et al., 2015). Similarly, another study found that increases in perceived anxiety controllability accounted for intraindividual reductions in anxiety symptoms in a large sample of patients receiving treatment at an outpatient anxiety disorders clinic (Gallagher, Naragon-Gainey, et al., 2014). Results indicated that patients who initiated a

cognitive-behavioral intervention at the clinic demonstrated significant increases in perceived anxiety controllability and significant reductions in anxiety symptoms. Furthermore, on an intraindividual basis, increases in perceived anxiety controllability were responsible for the improvements observed in anxiety symptoms following treatment (Gallagher, Naragon-Gainey, et al., 2014). Together, these studies provide strong evidence that perceived anxiety controllability is a determinant of anxiety severity and therefore an important outcome for further study.

Further evidence for the importance of perceived anxiety controllability comes from its recognition as a treatment target. As noted in the above-mentioned studies, increases in perceptions of anxiety controllability led to reductions in anxiety symptoms within the context of treatment (De Castella et al., 2015; Gallagher, Naragon-Gainey, et al., 2014). Additionally, other scholars have found that low perceived controllability of psychological symptoms at the outset of treatment may negatively impact treatment efficacy. For example, baseline levels of perceived anxiety controllability were found to moderate reduction of anxiety symptoms over the course of exposure treatment (Valentiner et al., 2013). Specifically, while both groups experienced significant reductions in anxiety symptoms over the course of treatment, those participants with higher initial perceptions of anxiety controllability demonstrated significantly larger reductions in their reported symptoms (Valentiner et al., 2013). Furthermore, the average level of symptom severity reported at the end of treatment by the participants in the higher anxiety controllability group fell in a range considered to be non-clinically severe, whereas those who reported low perceived anxiety controllability at the outset continued to report clinically-severe anxiety symptoms at post-treatment (Valentiner et al., 2013).

Similarly, in a second study examining treatment outcomes following a partial hospitalization program, pre-treatment beliefs about the controllability of anxiety were found to predict anxiety symptoms at discharge (Schroder, Kneeland, et al., 2019). Specifically in this study, higher perceived anxiety controllability prior to the initiation of treatment predicted lower anxiety symptoms following a partial hospitalization treatment that utilized cognitive-behavioral interventions (Schroder, Kneeland, et al., 2019). When examined in light of the previously reviewed studies (De Castella et al., 2015; Gallagher, Naragon-Gainey, et al., 2014), these findings lend support to the idea that perceived anxiety controllability affects treatment outcomes and is therefore an important potential treatment target.

If so, some individuals may require treatment that focuses initially on increasing perceived anxiety controllability in order to receive benefit from treatment comparable to their peers who hold higher anxiety controllability beliefs. This conclusion aligns with recommendations from Wells (2009), who notes that beliefs about the uncontrollability of anxiety are an essential target for intervention. For example, Wells (2009) recommends utilizing exercises, such as worry and rumination postponement, to build self-efficacy around control of cognitive symptoms of anxiety while simultaneously reducing engagement in behaviors that maintain anxiety (i.e., worry and rumination). Through these exercises, patients are likely to learn that they are capable of altering their experience of anxiety through their actions and, thus, modify their beliefs that anxiety is uncontrollability. Similarly, patients might be instructed to attempt to lose control over their anxiety symptoms to produce the realization that uncontrollability surrounding anxiety symptoms is not possible. Such treatment techniques are part of Wells's (2009)

broader metacognitive therapy treatment package. Metacognitive therapy has been shown to produce significant decreases in negative metacognitive beliefs, including the uncontrollability of anxiety, and treatment gains have been maintained at six and twelve months following conclusion of treatment (Normann & Morina, 2018; van der Heiden et al., 2013; van der Heiden et al., 2012; Wells et al., 2010). In structuring treatment to first address patients' anxiety controllability beliefs, metacognitive therapy therefore fosters patients' belief in the conceptual basis within cognitive-behavioral therapy that one's actions impact one's affective experiences, directly intervening upon a crucial antecedent to any subsequent behavior change.

Origins of Anxiety Controllability Beliefs

Given the importance of perceived anxiety controllability to anxiety symptom severity and the treatment of clinically severe anxiety, it is important to understand the origins of anxiety controllability beliefs. Several authors have proposed that perceptions of control may be shaped through social and cultural transmission of information. For example, Bandura (1982) identified that belief in one's ability to successfully cope with distress may be shaped by verbal persuasion. Indeed, verbal persuasion has been shown to contribute to extinction of fear responses in anxiety-provoking situations indirectly through verbally induced increases in self-efficacy (Zlomuzica et al., 2015). A similar effect has also been found with regard to negative verbal persuasion (i.e., discouragement), which has been shown to decrease perceptions of self-efficacy to cope with anxiety (Brown et al., 2012). This information transfer regarding perceived controllability has also been argued to occur not only through direct persuasion but also through exposure to cultural beliefs and values. Wells (2005) argued that metacognitive

beliefs, such as negative beliefs about the controllability and danger associated with stress and worry, can be culturally transmitted. In addition, cultures differ with regard to common perceptions of where control is oriented, suggesting perceived control is likely shaped through social transmission via exposure to cultural beliefs, attitudes, and values systems (Cheng et al., 2013).

Consistent with this idea that controllability beliefs may be socially and culturally transmitted, controllability beliefs may be shaped by exposure to culturally promoted beliefs about mental health. In the absence of health expertise, lay persons nonetheless form beliefs about health conditions and associated health behaviors through exposure to health information available in popular media or from other people (Angermeyer & Matschinger, 1994; Chen & Mak, 2008; Giosan et al., 2001). These lay health beliefs can also be formed about mental health and have been shown to influence a wide variety of health-related outcomes including medication use (Spoont et al., 2005), cognitive appraisals of oneself (De Castella et al., 2014; Lilgendahl et al., 2013), and emotion regulation strategies (Kneeland et al., 2016; Schroder et al., 2015; Schroder et al., 2017). Lay beliefs about mental health include beliefs about the etiology, stability, behavioral consequences, and controllability of mental health conditions (Furnham & Telford, 2011). In particular, the etiological explanations that have been studied may be distinguished primarily based on the extent to which they emphasize biological factors including genetic factors (biogenetic explanations) or psychosocial factors (psychosocial explanations; Furnham & Telford, 2011).

According to authors who have evaluated these two specific explanations, biogenetic and psychosocial explanations are chosen for evaluation in comparison to one

another for multiple reasons. For example, the emerging prevalence of biogenetic explanations, tendency for biogenetic explanations to produce a particular thought pattern termed psychological essentialism, and the centrality of psychosocial beliefs within cognitive-behavioral models of mental health pathology and treatment are all cited reasons for evaluating these specific etiological explanations against one another (Deacon & Baird, 2009; Ganesan et al., 2019; Gershkovich et al., 2018; Lebowitz et al., 2014; Lee et al., 2016; Lüllmann et al., 2011). In addition, other authors have noted that while evidence-based psychotherapies based on the psychosocial model are available, heavy cultural adoption of biogenetic explanations societally and within the mental health field interferes with utilization of these treatments in favor of biogenetically based pharmacological interventions that have poor long-term outcomes (Deacon, 2013). As such, these factors are further reviewed in greater detail below.

In recent decades, biogenetic explanations of mental health etiology have become increasingly prevalent in both the academic and lay communities (O'Connor et al., 2012; Pescosolido et al., 2010; Schomerus et al., 2012) and have been held in contrast to biopsychosocial explanations (Anderson, 1998; Engel, 1977; Kaplan, 1990; Matarazzo,1980; Schwartz, 1982; Schwartz & Weiss, 1978). In the United States, several factors such as development of psychopharmacological treatments, direct-to-consumer marketing of these medications, completion of the Human Genome Project, and shifts in funding priorities toward genetic and neuroscience research have contributed to a cultural shift that emphasizes biogenetic explanations of mental health conditions over other types of explanations (Lebowitz & Appelbaum, 2019). The impact of this cultural shift has been explored primarily in literature examining the effects of biogenetic explanations on

general societal attitudes, mental health stigma, and clinician attitudes and practices with fewer studies focused on the impact of biogenetic explanations on individuals' perceptions of their own health (Lebowitz & Appelbaum, 2019). Biogenetic beliefs about mental health have been hypothesized to confer both positive and negative effects, as exemplified by Haslam and Kvaale's (2015) "mixed blessings" model. This model of the relationship between biogenetic explanations and mental health stigma proposes that endorsement of biogenetic causal beliefs is likely to reduce blame associated with having a mental health condition while simultaneously increasing prognostic pessimism, perceptions that people with mental health difficulties are dangerous, and desire for social distance from those with mental health conditions (Haslam & Kvaale, 2015). Indeed, biogenetic causal explanations contribute to both positive and negative effects (Kvaale et al., 2013), including reducing blame for mental health symptoms (Deacon & Baird, 2009) while also increasing prognostic pessimism about recovery from anxiety disorders following treatment (Lam & Salkovskis, 2007).

One possible reason biogenetic etiological explanations produce these effects is that they promote the adoption of essentialist beliefs by laypersons (Dar-Nimrod & Heine, 2011). Essentialism is the tendency to perceive and categorize entities according to observable characteristics that are presumed to originate from naturally occurring underlying "essences." These "essences" are thought to define the entities by directly imbuing them with the shared characteristics that make them recognizable as members of that category. Essentialist beliefs are also used to interpret the behaviors and personalities of people, a tendency termed "psychological essentialism" (Haslam et al., 2006). In essentialist thought about mental health conditions, a person may be categorized as

"mentally ill" or "having a mental illness" based on observations of behaviors or verbal expressions that are believed to indicate underlying mental health pathology. For example, when observing an individual exhibiting behavior that could be indicative of poor mental health (e.g., a person talking to himself on the street), the essentialist thinker may assume that the person possesses an underlying essence responsible for a mental health condition (e.g., schizophrenia) and may define that person by this essence (e.g., view the person as "schizophrenic" as opposed to viewing them as a person experiencing symptoms of schizophrenia). Individuals appear to use essentialist beliefs as cognitive heuristics to understand and classify the world around them, a tendency that appears to be present (albeit to varying degrees) across cultures (Norenzayan & Heine, 2005).

Several aspects of essentialist thought would suggest that biogenetic explanations of mental health may negatively impact perceived anxiety controllability. First, essentialist thought proposes that essences and their consequents (i.e., observable characteristics and categorical groupings of entities) are naturally occurring. The presumed "natural" origins of entities contribute to perceptions that entities are justified or that they "should" be as they "naturally" are (Dar-Nimrod & Heine, 2011). Essentialist perceptions of mental health conditions may therefore negatively impact motivation to alter the experience of mental health symptoms (Angermeyer & Matschinger, 1994; Baker & Menken, 2001). A second feature of essentialist thought that may negatively impact anxiety controllability beliefs is the presumed bi-directionality of the relationship between essences and entities' observable characteristics. Just as essences are presumed to cause observable characteristics of entities in essentialist thought, the observation of a characteristic is presumed to indicate the presence of an underlying essence that is

responsible for that characteristic (Dar-Nimrod & Heine, 2011). Bidirectionality of essentialist thought may be used to infer the presence of a mental health disorder by an individual who is experiencing ambiguous symptoms. For example, clinically severe anxiety that is focused on fear of physiological symptoms results when normative experiences of autonomic arousal are catastrophically misinterpreted by individuals as evidence that they may be "going crazy" or likely to "lose control" (Casey et al., 2004; Harvey et al., 1993), catastrophic misinterpretations that can be produced through social suggestion that individuals have low control over anxiety (Telch et al., 1996). Third, essentialist thought can exist without detailed understanding of the exact qualities, origins, or properties of a purported essence. Where the precise nature of the essence is not well understood, people may employ an "essence placeholder" (Medin & Ortony, 1989) to support their essentialist beliefs. In the case of mental health conditions, genes, supposed "imbalances" in neurotransmitter levels, and other biomedical factors may function as essence placeholders (Dar-Nimrod & Heine, 2011). Thus, a layperson need not have a detailed or accurate understanding of genes or other biological factors to form essentialist beliefs about mental health conditions based on these purported causes. Finally and most importantly, essences are presumed to be stable and immutable even while the observable characteristics that arise from the essence may be altered either directly or by environmental influences. Thus, psychological essentialist thought presumes that the potential for mental health difficulties is something constant and unchangeable within the individual despite changing environmental circumstances or treatment (Dar-Nimrod & Heine, 2011).

In contrast to biogenetic essentialist conceptualizations of mental health functioning, psychosocial explanations include psychological factors that may contribute to increased perceptions of the controllability of one's experiences. Within cognitive-behavioral models of clinically severe anxiety, which are largely psychosocial in nature, an individual's interpretations of his or her experiences and circumstances are believed to influence motivation to engage in particular behaviors that perpetuate anxiety experiences (e.g., Beck et al., 2005; Roemer et al., 2005; Wells 2009). Cognitive-behavioral conceptualizations of anxiety therefore offer clear routes by which the individual may act upon factors that are purported to maintain anxiety, which may contribute to greater perceptions of anxiety controllability. For example, as previously mentioned, worry and rumination postponement exercises are designed build perceived control over anxiety by demonstrating that intrusive anxious thoughts do not persist following disengagement from perseverative thinking styles (Wells, 2009). By placing a focus on how one's choice of actions impacts the symptoms one experiences, psychosocial etiological explanations imply that individuals can alter their experience of anxiety through strategic behavioral changes. It can therefore be hypothesized that exposure to psychosocial etiological explanations is likely to contribute to greater perceptions of anxiety controllability relative to exposure to biogenetic essentialist explanations.

In support of this hypothesis, an emerging literature has examined the impact of exposure to alternative explanations of mental health etiology on mental health beliefs. Within this literature, it had been demonstrated that exposure to biogenetic etiological explanations contributes to higher levels of mental health stigma (Crisafulli et al., 2010; Lincoln et al., 2007; Phelan, 2005) and increased prognostic pessimism including

increased perceptions of chronicity and duration of symptoms (Bennett et al., 2008; Lam & Salkovskis, 2007; Lam et al., 2005; Lebowitz et al., 2014; Phelan, 2005), and lowered perceptions of treatment effectiveness (Lincoln et al., 2007; Phelan et al., 2006; Schofield et al., 2018). Additionally, evidence demonstrates an association between agreement with biogenetic etiological explanations and lower perceptions of control over mental health symptoms. In a large-scale survey, Phelan et al. (2006) found that perceptions of mental health problems as genetic in origin were associated with lower perceptions of treatment effectiveness. Similarly, Deacon and Baird (2009) provided participants with two written etiological explanations for depression (i.e. a biogenetic "chemical imbalance" explanation and a biopsychosocial explanation) and asked participants to rate the credibility of each explanation and complete measures regarding perceptions of controllability and related constructs. The authors found that higher perceptions of credibility of the chemical imbalance model of depression were associated with lower perceptions of depression controllability, whereas higher perceptions of credibility of the biopsychosocial model were associated with higher perceptions that attitude and lifestyle changes would be helpful in addressing depression (Deacon & Baird, 2009). However, neither Phelan et al. (2006) nor Deacon and Baird (2009) experimentally manipulated etiological beliefs; therefore, these studies cannot speak to a causal relationship between etiological explanations and perceived mental health controllability.

Other studies, however, have demonstrated such causal relationships between biogenetic etiological explanations and lower perceived control. Lüllmann et al. (2011) demonstrated that being offered a psychological etiological explanation (in contrast to biological, biopsycho, or no causal explanations) increased perceptions of personal

control over psychotic symptoms. Likewise, Lebowitz and Appelbaum (2017) demonstrated that offering genetic versus non-genetic explanations for a fictionalized person's problematic alcohol use and gambling decreased respondents' perceptions about the person's agency to recover. Lebowitz et al. (2014) demonstrated that this pattern of results could be reversed through intervening on individuals' perceptions of mental health etiology. These authors utilized a sample of participants who had previously endorsed strong biogenetic beliefs about depression and provided a psychoeducation intervention designed to increase perceptions of depression malleability (Lebowitz et al., 2014). The authors found that participants' perceptions of positive prognosis following treatment increased, as did participants' beliefs in their ability to regulate their moods. Importantly, this effect was maintained six weeks following the psychoeducation intervention (Lebowitz et al., 2014).

Additionally, biogenetic and non-biogenetic etiological beliefs have been demonstrated to result in differences in health-related behaviors. Dar-Nimrod et al. (2014) conducted three studies in which they examined the impact of etiological explanations for excess weight on weight-related beliefs and behaviors. First, Dar-Nimrod et al. (2014) demonstrated that endorsement of biogenetic etiology for excess weight was associated with the perception that individuals experiencing excess weight cannot control their weight. Second, Dar-Nimrod et al. (2014) manipulated etiological beliefs by randomizing participants to received either a biogenetic or non-genetic physiological explanation of excess weight. Participants who received the non-genetic physiological explanation endorsed higher perceptions of the ability to control one's weight. Finally, participants were randomized to receive a biogenetic explanation,

psychosocial explanation, or no-explanation for excess weight before taking part in a food tasting task purportedly to evaluate the flavor of cookies. Participants who were randomized to receive the biogenetic explanation of excess weight ate significantly more than participants in the other conditions during this task (Dar-Nimrod et al., 2014). Together, these studies consistently demonstrate that exposure to alternative etiological explanations of mental health conditions causes differing perceptions of controllability surrounding health-related outcomes. Specifically, in contrast to psychosocial etiological explanations, exposure to biogenetic etiological explanations causes lower perceptions of control over health-related outcomes.

This relationship has also been demonstrated in studies in which individuals' perceptions of their own genetic risk for mental health difficulties were altered. Kemp et al. (2014) utilized an experimental manipulation in which participants with a reported history of depression provided saliva samples as part of a bogus "Rapid Depression Test" before being randomized to conditions in which they were told either that the test indicated the depressive symptoms they experienced were the result of an imbalance in serotonin or that the test indicated that the symptoms were not the result of a chemical imbalance. Participants led to believe a biological "imbalance" was responsible for their experiences with depression reported significantly lower perceptions of controllability and lower expectancies of personal ability to self-regulate one's mood. Similarly, Dar-Nimrod et al. (2013) obtained saliva samples from participants under the guise of testing them for genetic susceptibility to mental health disorders and randomized participants to receive information that they either had or did not have a genetic susceptibility to problematic alcohol use. Participants who received bogus information that they were at

risk for problematic alcohol use subsequently reported lower perceived control over alcohol consumption (Dar-Nimrod et al., 2013). These studies demonstrate that shaping individuals' beliefs about their personal genetic susceptibility for mental health difficulties can cause perceptions of mental health uncontrollability in a pattern that parallels the impact of exposure to differing explanations of mental health etiology on perceived mental health controllability.

Despite the wealth of evidence that differing etiological explanations impact mental health controllability beliefs, only one study to date has focused on the impact of etiological explanations specifically on perceptions of *anxiety* controllability. In that study, Gershkovich et al. (2018) presented participants who self-identified as experiencing obsessive-compulsive disorder (OCD) first with a biological explanation of their illness followed by a biopsychosocial explanation and subsequently asked participants to complete measures rating the credibility of the causal explanations presented and their perceptions of the prognosis and treatment of OCD. Higher endorsement of the biological explanation was associated with greater perceptions of OCD chronicity, need for long-term treatment, and the perception of a need for treatment with medication. Higher endorsement of the biopsychosocial model was associated with stronger expectancies that behavioral changes would help OCD, although this finding only trended toward significance. These effects were found controlling for severity of participants' OCD symptoms, demonstrating that differences in expectancies were not merely due to the severity of symptoms participants experienced. This study suggests an association between biogenetic etiological beliefs about anxiety and lower perceived control of anxiety symptoms; however, a causal link cannot be inferred from these results

due to the non-experimental study design. An experimental study design manipulating etiological beliefs is needed to examine a potentially causal relationship between etiological beliefs and anxiety controllability beliefs.

Present Study

Perceived controllability of anxiety symptoms is an important determinant of anxiety severity and an important target of anxiety treatment, yet its origins are not well understood. One potential source of anxiety controllability beliefs is exposure to differing explanations of anxiety etiology as evidenced by one study demonstrating an association between belief in biogenetic origins of anxiety and low perceived control over these symptoms (Gershkovich et al., 2018) and other studies demonstrating that perceptions of mental health controllability can be caused by exposure to differing explanations of the etiology of particular mental health concerns such as psychotic symptoms, excess weight, and problematic alcohol use (Crisafulli et al., 2010; Lüllmann et al., 2011). This area of inquiry is in its infancy and further studies are needed to replicate and extend exigent findings. Specifically, no study, to date, has examined the potentially causal impact of biogenetic and psychosocial etiological explanations on anxiety controllability beliefs.

The present study aimed to contribute to the literature by providing the first empirical evaluation of beliefs about the etiology of anxiety as a cause of perceived anxiety controllability. Specifically, this study examined the potentially causal impact of exposure to biogenetic and psychosocial explanations of anxiety etiology on perceptions of anxiety symptom controllability in an unselected sample of undergraduate students. In this way, the present study aimed to extend the findings of Gershkovich et al. (2018) by testing whether exposing individuals to biogenetic and psychosocial explanations of

anxiety etiology would *cause* differing levels of perceived anxiety controllability. Furthermore, given the strong associations between perceived controllability of anxiety and anxiety severity (Bailey & Wells, 2013; Barahmand, 2009; Davis & Valentiner, 2000; Fergus & Bardeen, 2017; Fergus & Wheless, 2018; Melli et al., 2016; Myers et al., 2009; Nordahl et al., 2017; Nordahl & Wells, 2017; Ryum et al., 2017; Schroder, Callahan, et al., 2019), the present study utilized a design that controlled for trait levels of anxiety in order to demonstrate that any causal relationship that exists between etiological beliefs and perceived anxiety controllability is not merely a byproduct of the general propensity to experience anxiety. Furthermore, by using an undergraduate sample, the present study aimed to explore potential origins of anxiety controllability beliefs within a population that is vulnerable to the development of clinically severe anxiety characterized by low perceptions of anxiety controllability. Specifically, clinically severe anxiety characterized by repetitive negative thinking and perceptions of anxiety uncontrollability has a median age of onset that follows the age of traditional undergraduate students (Kessler et al., 2012); therefore, it is instructive to explore how etiological explanations impact anxiety controllability beliefs in this population specifically as these beliefs may confer risk for later development of clinically severe anxiety. It was hypothesized that, controlling for the proneness to experience anxiety, exposure to a biogenetic etiological explanation of anxiety would cause lower perceived anxiety controllability relative to perceptions of anxiety controllability in those who were exposed to a psychosocial etiological explanation of anxiety.

If hypotheses were supported, the present study could contribute to the literature by extending our knowledge of clinically severe anxiety and related treatment efforts.

While other studies have demonstrated that etiological explanations impact mental health symptom controllability beliefs, it is important to expand this literature by testing this causal model within the context of anxiety given the proposed centrality of controllability beliefs to the etiology of clinically severe anxiety (Barlow, 2002; Chorpita & Barlow, 1998; Wells, 2009). Furthermore, given that anxiety controllability beliefs both impact and may be impacted by cognitive behavioral therapies for anxiety (De Castella et al., 2015; Gallagher, Naragon-Gainey et al., 2014; Meuret et al., 2010; Valentiner et al., 2013) and are an important treatment target in certain therapy approaches (e.g., metacognitive therapy; Wells, 2009), this extension of findings regarding a potentially causal relationship between etiological beliefs and anxiety controllability could contribute to therapeutic efforts to treat anxiety if study hypotheses are supported. The present study therefore aimed to contribute to better understanding of the impact of etiological explanations of anxiety on anxiety controllability beliefs, which could then be used to inform how clinicians present information about anxiety and its treatment. For example, clinicians could be encouraged to refrain from using statements that imply uncontrollability of anxiety when providing psychoeducation or explanations of the treatment model and instead aim to heighten perceptions of controllability by providing information regarding the potential for anxiety management through behavior change.

As previously noted, certain treatments view the enhancement of controllability beliefs as a necessary precursor to the initiation of other therapeutic activities targeting general symptoms (Wells, 2009). If the study hypotheses were supported, the present study would support this point and further emphasize the importance of considering patients' beliefs of the origins of symptoms, enhancing exigent literature that suggests

that matching treatments according to individuals' beliefs about the etiology of their symptoms produces superior outcomes (Vittengl et al., 2019). Additionally, the present study could contribute to the emerging literature on the broader societal impact of biogenetic and essentialist beliefs on mental health outcomes, bolstering bioethical arguments about the need for responsible dissemination of information about mental health. As numerous authors note, the rise of the biomedicalization and geneticization in both research and lay beliefs has wide-reaching implications for mental health consumers, mental health practitioners, researchers, and society as a whole (Árnason & Hjörleifsson, 2007; Deacon, 2013; Kong et al., 2017; Stein & Giordano, 2015). Popular media simplifies research findings almost by necessity; however, to the extent that this simplification results in misinterpretation or unintended extrapolation from findings, laypersons may adopt misinformed beliefs about themselves and others.

As previously noted, while availability of information about genetic contributions to mental health has been shown to reduce blame, it increases stigmatizing attitudes about the dangerousness of individuals with mental health difficulties and worsens perceptions of prognostic pessimism (Kvaale et al., 2013). Geneticization of mental health attitudes has also been argued to contribute to a perception of individuals as rational consumers of health information who are therefore tasked with the responsibility of managing their own recovery (Kong et al., 2017). Despite this perception, misinformation about the purported causes of mental health conditions abounds, as evidenced by the continued cultural success of the chemical imbalance explanation (Deacon, 2013), which negatively impacts treatment expectancies for efficacious psychotherapies (Kemp et al., 2014). Furthermore, the perception of a rational mental health consumer has been argued to

worsen mental health disparities by shifting the focus of intervention away from the social, environmental, and cultural factors that contribute to mental health difficulties, which disproportionately impact minority groups (Kong et al., 2017).

In summary, if the outlined hypotheses were supported, it would extend the current state of the literature by identifying a source of low perceived anxiety controllability, a proposed determinant of anxiety severity. If findings supported etiological explanations of anxiety as a source of anxiety controllability beliefs, the present study would suggest possible changes as to how mental health professionals provide psychoeducation about anxiety. Finally, if supported, the present study would contribute to the growing literature on the detrimental effects of psychological essentialism and call for potential ways mental health information is presented to stakeholders including patients, families, and the public at large.

CHAPTER TWO

Method

Research Design

Approval from the IRB committee of Baylor University was obtained prior to initiation of recruitment. Informed consent was obtained from all participants, and they were compensated with course credit for completion of the study activities. Data collection took place at a single timepoint, which required participants to complete various online, previously published questionnaires through the SONA system where they were directed to a link of online surveys. The online surveys were generated using Qualtrics research design software. Participants read an online consent form through the Qualtrics survey program and selected "I agree to participate in this study" to indicate their consent to participate.

Upon agreeing to participate in the study, participants were randomized to view one of two online presentations before completing complete various online, previously published questionnaires, a process that took approximately 60 minutes to complete. Previous studies demonstrate that etiological beliefs can be manipulated through an online platform (Gershkovich et al., 2018; Lüllmann et al., 2011). These presentations provided etiological explanations of anxiety ("Anxiety is Biologically Based," "Anxiety is Psychosocially Based") based on Gershkovich et al.'s (2018) "Biological (brain) explanation" and "Biopsychosocial explanation" statements on the origins of OCD. Language in the presentations specific to the clinical presentation of OCD was modified

to refer to anxiety generally and to distinguish between biogenetic and psychosocial explanations (see Appendix A and B). Participants then answered questionnaires on Qualtrics assessing demographic information, etiological beliefs, perceived anxiety controllability, and trait anxiety. All participants were allowed to discontinue participation at any time without penalty.

Participants

Participants were undergraduate students from Baylor University (n = 280), recruited from the Baylor University online subject pool system (SONA). Mean age and self-identification of gender and race/ethnicity are presented in Table 1. Prior to study recruitment, a power analysis for an ANCOVA was conducted using G*Power (Erdfelder et al., 1996) to determine the minimal detectable effect using the sample size of 279, an alpha of .05, and a power of .95 with 4 possible covariates (trait anxiety, age, gender, and ethnicity). Based on these assumptions, the analyses are powered to detect a small-to-medium effect (Cohen's d = 0.25), consistent with previous studies of experimental manipulations of etiological beliefs about mental health (e.g. Kvaale et al., 2013; Lebowitz & Appelbaum, 2017).

Measures

Etiological Beliefs

Etiological beliefs about mental illness were assessed with the Mental Illness Attribution Questionnaire (MIAQ), a measure of biological, social, and spiritual attributions for mental health difficulties (Knettel, 2019). The MIAQ consists of a vignette depicting an individual who experiences mental health difficulties followed by

55 items assessing purported biological, social, or spiritual causes of this condition. This vignette format is commonly used in measures of attitudes toward individuals with mental health difficulties (Cunningham et al., 1993; Luty et al., 2006; Phelan et al., 2006). The MIAQ has been validated with three alternate vignettes depicting depression, schizophrenia, and "alcoholism" (Knettel, 2019). On the MIAQ, respondents were asked to rate the degree to which they believe the causes are important reasons for the difficulties experienced by the individual on a 7-point Likert-style scale (1 = *This Cause is Not at All Important*, 7 = *This Cause is Very Important*). Mean scores were calculated for seven subscales measuring attributions related to Supernatural Forces, Social/Stress, Lifestyle, Physical Health, Substance Use, Personal Weakness, and Heredity/Biology.

Given that individuals can hold differing beliefs about the etiology of different mental health conditions (Furnham & Telford, 2012), it was determined that it would be useful in the present study to present only one vignette evocative of participants' etiological beliefs of anxiety. For this reason, the language of the vignette was modified to depict an individual with anxiety, and participants were asked to rate potential causes in response to this vignette. Specifically, the original vignettes states, "Harry is a 30-year-old single man with schizophrenia (/depression/alcoholism). Sometimes he hears voices and has strange beliefs that make him upset (/He often feels very unhappy and sometimes has thoughts about killing himself/He has been drinking heavily almost every day for the past 5 years). He lives alone in an apartment and works as a store clerk. He has been hospitalized because of his illness in the past" (Knettel, 2019). For the present study, this language was modified to state, "Harry is a 30-year-old single man with anxiety. He sometimes feels very nervous and often worries about many different things. He lives

alone in an apartment and works as a store clerk. He has been hospitalized because of his illness in the past." The language was modified in this way to describe worry, an anxiety symptom associated transdiagnostically with various presentations of clinically severe anxiety (Ehring & Watkins, 2008; McEvoy et al., 2013).

To test the effectiveness of the experimental manipulation on changes in etiological beliefs, a single item from the MIAQ assessing perceptions of anxiety as a biological or medical disorder ("It is a biological or medical disorder"; Knettel, 2019) was administered. This item was selected as a manipulation check because it was believed to be the most parsimonious means of assessing differences following the experimental manipulation between the groups with regard to endorsement of biological factors in anxiety etiology. Following evaluation of this manipulation check, exploratory analyses were conducted to examine participant beliefs in psychosocial causes of anxiety via the Social/Stress subscale. Internal consistencies of the Social/Stress subscale vary by vignette provided but are all within a range considered excellent (α = .93-.95). Given the limited published measures examining etiological beliefs about mental health, information about the convergent validity of the MIAQ is limited; however, authors evaluated convergent validity for the MIAQ by comparing scores on MIAQ subscales to scores on a measures of attributions of responsibility for mental health symptoms. In doing so, authors hypothesized that MIAQ subscales would differ with regard to the degree to which they depicted etiological explanations that were within or outside of a person's control. These ratings were then used to compare MIAQ subscales with scores on the previously validated responsibility scale. In this analysis, items on the Social/Stress scale were rated as depicting factors moderately within the control of the

individual and items on the Heredity/Biology scale were rated as outside of the control of the individual prior to analysis. Results of this analysis showed that the Social/Stress (combined vignette model β =-.16) and Heredity/Biology (combined vignette model β =-.48) subscales demonstrated convergent validity as significant predictors of scores on the similar measure evaluating responsibility for mental health symptoms (Knettel, 2019).

Anxiety Controllability

Anxiety controllability was assessed with the Theories of Anxiety Scale (TOA), a measure of beliefs about the controllability anxiety (Schroder et al., 2015). The TOA consists of four items assessing beliefs about mutability of anxiety. Respondents are asked to rate their agreement with statements on a 6-point Likert-style scale (1 = *Strongly Disagree*, 6 = *Strongly Agree*). Total scores are created by summing the items after reverse-scoring key items with higher scores indicating greater levels of controllability beliefs. The TOA has demonstrated high internal consistency (αs = .96-.97; Schroder, Callahan, et al., 2019; Schroder et al., 2015; 2016), strong one-week test-retest reliability (*r*s = .63-.72; Schroder, Callahan et al., 2019), and moderate five-week test-retest reliability (*r* = .53; Schroder, Callahan, et al., 2019). The TOA was administered as the primary outcome measure of the study. Internal consistency in the present study was found to be excellent, Cronbach's α = .92.

Trait Anxiety

Trait anxiety was assessed with the State-Trait Inventory for Cognitive and Somatic Anxiety (STICSA-Trait; Ree et al., 2008) to control for individual differences in trait anxiety, which could confound study findings. The STICSA-Trait is a measure of trait anxiety that is composed of 21 items assessing characteristic experiences of anxiety.

Respondents are asked to rate how characteristic it is of themselves to experience various anxiety symptoms on a 4-point Likert-style scale (1 = *Not at All*, 4 = *Very Much So*). The STICSA demonstrates high internal consistency (Cronbach's *α*s = .87-.90; Grös et al., 2007; Ree et al., 2008). The STICSA demonstrates strong convergent validity by association with a similar measure of trait anxiety (r = .66) and discriminant validity by showing a stronger association with a measure of anxiety symptom severity (r = .68) in comparison to a measure of depression symptom severity (r = .58; Grös et al., 2007). The STICSA was administered to control for the effects of trait anxiety on the dependent variable. Internal consistency in the present study was found to be excellent for the cognitive subscale, Cronbach's α = .900, and good for the somatic subscale, Cronbach's α = .90.

CHAPTER THREE

Results

All analyses were conducted using SPSS software. Before data analysis, data were cleaned to arrive at the current sample of participants. An original 427 entries were made in response to the online study. Entries of participants unable to provide consent based on age less than 18 years (4 entries) or missing data on age (92 entries) were excluded from analyses. Duplicate entries by individual participants as identified by Qualtrics ID number were then excluded (38 entries), preserving the first instance of participation as determined by survey completion timestamp. An additional 13 entries were excluded based on participants' failure of two attention checks embedded with the MIAQ. The result was a final sample of 280 participants. Descriptive statistics were then run on study variables. Chi-square analyses found that groups did not differ significantly with regard to participant gender or ethnicity, see Table 3.1. An independent *t*-test examining group differences on age was also not significant, indicating that experimental groups did not differ significantly based on age, see Table 1. Because no significant between-group differences were found on these variables, they were not entered as covariates in the analysis of the primary outcome measure.

Manipulation Check

After undergoing the experimental manipulation, groups did not differ significantly on the single item assessing perceptions of anxiety as a biological or

medical disorder ("It is a biological or medical disorder"; Knettel, 2019), see Table 3.1, suggesting exposure to the psychosocial video did not produce a significant effect on participant's perceptions of the biological origins of anxiety.

Main Outcome

An ANCOVA examining the effects of exposure to biological versus psychosocial explanations of anxiety etiology indicated that, consistent with study predictions, higher perceptions of anxiety controllability were associated with viewing the psychosocial etiological explanation compared with viewing the biological etiological explanation when controlling for cognitive and somatic trait anxiety, a small to medium effect, see Table 3.2. Cognitive trait anxiety was significant was a covariate, $F(1, 276) = 8.60$, $p = .004$, indicating that the association between etiological explanations of anxiety and perceptions of anxiety controllability was stronger for participants with higher trait cognitive anxiety, see Table 3.2.

Table 3.1. Participant Demographics and Manipulation Check

	Condition				
	Biological	Psychosocial			
	Mean (SD)		*t*	*df*	*p*
Age	18.83 (1.37)	19.01 (2.86)	0.65	278	.518
Manipulation Check	6.08 (1.04)	5.99 (1.18)	-0.71	278	.478
STICSA					
Cognitive	22.72 (7.41)	22.61 (8.11)	-0.12	278	.908
Somatic	18.93 (6.93)	19.20 (7.05)	0.33	278	.740
	%		X^2	*df*	*p*
Gender			0.23	1	.632
Male	25	27			
Female	110	118			
Race/Ethnicity			2.77	5	.735
Asian/Pacific Islander	16.8	14.0			
Black/African American	2.9	4.9			
Hispanic White	14.6	18.2			
Native American/Alaska Native	0.7	0			
Non-Hispanic White/Caucasian	59.1	58.0			
Not listed/More than one	5.8	4.9			

Note: Biological N = 137*, Psychosocial N* = 143.

Table 3.2. Group Differences in Perceived Anxiety Controllability, Controlling for Trait Cognitive and Somatic Anxiety

	Biological	Psychosocial		
	Mean (SD)		$F_{(1, 276)}$	*d*
Theories of Anxiety Scale	16.59 (4.58)	18.48 (4.31)	14.05*	0.45

*$p < .001$ (two-tailed)

Exploratory Analyses

Given the failure of the manipulation to produce statistically significant differences between groups, exploratory analyses were conducted to examine participants' endorsement of relative importance of biological and psychological factors in anxiety etiology following the manipulation and manipulation check and possible group differences in these ratings. After undergoing the experimental manipulation, participants across groups rated psychosocial causes of anxiety on the Social/Stress subscale of the MIAQ as moderately important based on a 7-point scale where ratings of 7 = "This cause is very important" and ratings of 1 = "This cause is not at all important,"

(see Table 3.3). Compared with these ratings of psychosocial causes, participants across groups rated biological causes of anxiety on the Heredity/Biology subscale as of significantly lower importance in the etiology of anxiety, see Table 3.3.

Experimental groups did not differ significantly on the Social/Stress subscale of the MIAQ, confirming that the experimental manipulation was ineffective in producing group differences in participants' beliefs about the psychosocial causes of anxiety, see Table 3. Experimental groups did differ significantly, however, on the Heredity/Biology subscale of the MIAQ, with a large effect in which participants in the biological condition endorsed significantly stronger beliefs in biological causes of anxiety as compared to participants in the psychological condition, see Table 3.3.

Table 3.3. Exploratory Analyses – Etiological Beliefs about Anxiety

Variable	Full Sample *Mean (SD)*		*t*	*df*	*p*	*d*
MIAQ			-7.64	279	< .001	1.29
Heredity/Biology	4.96 (1.29)					
Social/Stress	5.55 (0.99)					
	Condition					
	Biological	Psychosocial				
	Mean (SD)		*t*	*df*	*p*	*d*
MIAQ						
Heredity/Biology	5.30 (1.09)	4.64 (1.38)	-4.42	278	< .001	1.25
Social/Stress	5.60 (0.94)	5.51 (1.03)	-0.71	278	.478	

CHAPTER FOUR

Discussion

Perceptions of anxiety controllability are a key factor believed to contribute to anxiety severity, distinguishing clinically severe anxiety from normative experiences of anxiety (Bailey & Wells, 2013; Barahmand, 2009; Davis & Valentiner, 2000; Fergus & Bardeen, 2017; Fergus & Wheless, 2018; Melli et al., 2016; Myers et al., 2009; Nordahl et al., 2017; Nordahl & Wells, 2017; Ryum et al., 2017; Schroder, Callahan, et al., 2019). Given the importance of beliefs about the controllability of anxiety, it is important to understand where these beliefs originate. Research on lay beliefs about other mental health conditions suggests that perceptions of controllability of mental health symptoms may be caused by differing lay etiological models that alternatively emphasize biological or psychosocial causes of mental health conditions (Dar-Nimrod et al., 2013; Dar-Nimrod et al., 2014; Lebowitz & Appelbaum, 2017; Lebowitz et al., 2014; Lüllmann et al., 2011). In particular, previously published findings indicate that lay beliefs that identify biological causes of mental health conditions cause perceptions of mental health symptom controllability to be lower whereas lay beliefs in psychosocial origins of mental health conditions cause perceptions of symptom controllability to be higher (Lebowitz & Appelbaum, 2017; Lüllmann et al., 2011). No published study to date, however, has evaluated the possible causal link between biological and psychosocial etiological explanations of anxiety and anxiety controllability beliefs.

The present study sought to contribute to the literature by conducting the first study examining the possible causal impact of differing etiological explanations of anxiety on anxiety controllability using an experimental manipulation of beliefs about anxiety etiology. Any such impact of these explanations on perceived anxiety controllability was impossible to determine in the present study, however, due to the failure of the experimental manipulation to produce significant group differences in participants' etiological beliefs about anxiety. Specifically, participants who had been shown a psychosocial explanation of anxiety etiology did not differ from participants who had been shown a biological explanation of anxiety etiology in their ratings of perceived importance of biological causes of anxiety etiology on the manipulation check item ("It is a biological or medical disorder"). This pattern of results suggests that the manipulation was not effective in producing significant group differences in perceptions of the possible biological origins of anxiety, proposed to represent participants' beliefs in biological versus psychosocial etiological beliefs about anxiety. As a result, it cannot be determined in the present study whether holding biological versus psychosocial etiological beliefs about anxiety causes differences in perceptions of anxiety controllability.

Limitations with Manipulation Check

There are several potential limits with this manipulation check that make interpretation of these null results difficult. For example, there are limitations related to interpretation of group differences on a single item. Most pertinent to the present study is the issue of the single item standing as a proxy for participants' beliefs in larger etiological belief structures, namely biological and psychosocial etiological beliefs of

anxiety. The single item used as the manipulation check was selected based on previous literature on lay theories of mental health that suggests that beliefs about mental health etiology tend to focus on *either* the biological or psychosocial origins of a given condition (Angermeyer & Matschinger, 1994; Bennett et al., 2008; Dar-Nimrod et al., 2013; Dar-Nimrod et al., 2014; Furnham & Telford, 2011) and that belief in biological origins in particular is a differentiating factors in lay beliefs that is associated with key outcomes, such as lower perceptions of symptom control (Deacon & Baird, 2009; Lebowitz & Appelbaum, 2017; Lebowitz et al., 2014; Lüllmann et al., 2011). However, contrary to the implications of this literature, people can hold beliefs in multiple possible origins of a disorder simultaneously including beliefs in both biological and psychosocial causes. Exploratory analyses of participants' beliefs in the present study indicated that participants simultaneously held moderate to strong beliefs in the importance of both biological and psychosocial causes of anxiety following the manipulation. Thus, stronger endorsement of the "biological or medical" manipulation check item does not preclude similarly strong endorsement of psychosocial causes of anxiety and may therefore not be sufficient to determine whether the manipulation was effective in altering participants' beliefs about both biological and psychosocial etiologies.

A second problem with interpreting the null results of the manipulation check is that it cannot be determined whether the manipulation was ineffective or whether the manipulation was effective but administered to groups that were not equivalent in their beliefs at baseline. Due to the design of the present study wherein participants rated their beliefs in etiological explanations of anxiety at a single timepoint after viewing the manipulation, it cannot be stated definitively that the manipulation did not alter

participants' beliefs. It is possible that randomization failed to assign participants to experimental groups in an equivalent manner, distributing participants with preexisting beliefs in either the biological or psychosocial origins of anxiety evenly between experimental conditions. If participants randomized to the biological group held stronger psychosocial etiological beliefs prior to manipulation and vice versa, the manipulation could have effectively altered participants' beliefs in the expected direction while nonetheless producing null findings on the manipulation check. The present study, however, did not include measures of participants' preexisting etiological beliefs or of factors likely to have influenced those beliefs in favor of either biological or psychosocial explanations, such as exposure to academic coursework or previous encounters with mental health professionals providing psychoeducation on anxiety origins. In order to avoid this methodological problem in future studies, pretest measures could be administered to assess participants' preexisting beliefs about biological and psychosocial causes of anxiety and exposure to potential sources of information about anxiety etiology, and block randomization could be used to ensure that experimental groups contain equivalent numbers of participants endorsing particular preexisting etiological beliefs before they undergo the experimental manipulation. As the current data stands, however, little can be made of participants' endorsement of etiological beliefs of anxiety either on the manipulation check item or the broader biological and psychosocial scales of the MIAQ other than that the absence of pretest data makes it impossible to determine the causal impact of biological or psychosocial explanations of anxiety on perceived anxiety controllability.

Prior Manipulations

The possible failure of the experimental manipulation in this study raises questions around what is necessary to produce changes in etiological beliefs about anxiety, particularly in individuals who may hold preexisting beliefs in certain etiological explanations. Understanding what factors may contribute to alterations in etiological beliefs about anxiety may allow future studies to produce such changes in an experimental manipulation in order to examine any potential causal links between beliefs about anxiety etiology and outcomes of interest such as perceived anxiety controllability. Additionally, as previously noted, it may be helpful to understand how these beliefs may be altered to provide insight into how modification of patients' preexisting lay beliefs could be used to support the effectiveness of psychoeducation phases of psychotherapy for anxiety in which patients are frequently offered explanations for the origin and maintenance of their symptoms. Alternatively, it may be helpful to understand modification of lay beliefs about anxiety etiology to determine whether it may be more beneficial to patients to match treatments to patients' preexisting etiological beliefs, rather than attempting to modify these beliefs (Vittengl et al., 2019). Thus, how etiological beliefs about anxiety can be altered is a question that deserves further exploration and is therefore the primary focus of this discussion.

Before exploring theory and evidence regarding factors that impact the manipulation of beliefs about mental health etiologies, it may be useful to first identify other studies that have successfully manipulated such beliefs. As previously noted, numerous authors have utilized experimental manipulations exposing participants to differing etiological explanations of mental health conditions (Bennett et al., 2008;

Crisafulli et al., 2010; Deacon & Baird, 2009; Lam & Salkovskis, 2007; Lam et al., 2005; Lebowitz & Appelbaum, 2017; Lebowitz et al., 2014; Lincoln et al., 2007; Lüllmann et al., 2011; Phelan, 2005; Phelan et al., 2006; Schofield et al., 2018); however, upon closer examination, few of these studies employed what may be considered a true manipulation check measuring the impact of exposure to differing etiological explanations on beliefs about mental health etiology (Crisafulli et al., 2010; Lebowitz & Appelbaum, 2017; Lüllmann et al., 2011). Most studies evaluated the effectiveness of study manipulations by inquiring about the credibility or convincingness of the etiological explanations presented (Deacon & Baird, 2009; Lincoln et al., 2007; Phelan, 2005; Schofield et al., 2018) and several employed no manipulation check at all (Bennett et al., 2008; Lam & Salkovskis, 2007; Lam et al., 2005; Lebowitz et al., 2014; Phelan et al., 2006).

As a result, while some studies may speak to the relationship between etiological explanations that are perceived as credible and perceived controllability of mental health symptoms, only a small number of studies can speak to causation of perceived symptom controllability by etiological beliefs that have actually been experimentally manipulated. This distinction between participant's perceptions of credibility of manipulation content and actual manipulation of beliefs is important because an explanation may be perceived as credible without actually changing a person's beliefs, resulting in the failure of the experimental manipulation in question and the inability to speak to causation. Belief change requires more than perceived credibility of the message or source in question (Hoeken, 2001) and may be influenced by numerous factors including discrepancies between the informant's original beliefs and target message (Aronson et al., 1963; McGinnies, 1973; Tormala & Petty, 2004), congruence with targets' self-interests

(Walster et al., 1966), and time between manipulation and assessment of beliefs (Chung et al., 2008). In fact, proposed scientific explanations may be perceived as highly credible, but may not shift persons' actual beliefs (Impey et al., 2017; Lewandowsky et al., 2012; Thomm & Bromme, 2011; Zaboski & Therriault, 2020). Thus, for purposes of the present study testing etiological beliefs as a cause of perceived anxiety controllability, it is of interest to examine the methodologies of studies that successfully manipulated beliefs about mental health etiology as measured by respondents' ratings of proposed etiologies, not those who checked their manipulation with ratings of perceived credibility of the manipulations.

The absence in much of this literature of true manipulation checks measuring differences in beliefs about mental health etiology suggests that this literature may be less robust than previously thought. Without such checks, it cannot be definitively stated that belief in differing etiological explanations of mental health conditions – rather than some third variable such as genetic essentialist beliefs or implicit mindsets – is responsible for differences in perceived symptom controllability as claimed. Further complicating the question of what factors may contribute to manipulation of participants' preexisting beliefs, the lack of published studies with null findings – termed the "file-drawer problem" (Rosenthal, 1979) – may contribute to the absence of published studies that could provide examples of successful experimental manipulations of beliefs of mental health origins but which otherwise did not produce significant differences on outcome variables. The possibility of understanding what circumstances are necessary to effectively manipulate etiological beliefs about mental health – and anxiety in particular – is therefore limited by the limited small number of published studies actually evaluating

differences in etiological beliefs of mental health conditions following experimental manipulation of these beliefs.

In examining the three studies identified in this literature that do use such direct manipulation checks, additional limitations emerge regarding their generalizability to the present study. In particular, one of these studies by Lebowitz and Appelbaum (2017), used an experimental method that is notably different from the method used by Crisafulli et al. (2010), Lüllmann et al. (2011), or the present study. Unlike these studies which were focused on the impact of exposure to *differing* etiological explanations (e.g., biological, psychosocial), Lebowitz and Appelbaum (2017) were principally interested in the impact of a single genetic explanation in order to evaluate the impact of genetic essentialism on study outcomes. Lebowitz and Appelbaum (2017), therefore, employed a manipulation structure in which participants received explanations of mental illness that either stated that genes *were* a cause ("Charlie has his [drinking/gambling] problem because of his DNA") or *were not* a cause ("Charlie has a type of [drinking/gambling] problem that is NOT caused by his genes [DNA]"). In other words, participants were not exposed to alternative explanations of mental health etiology, and those in the non-biogenetic group were directly told that biogenetic causes were *not* involved. The manipulation check therefore was not endorsement of different etiological explanations but rather a simple evaluation of belief in a genetic contribution to the given mental illness. Lebowitz and Appelbaum (2017) therefore does not provide a test of the impact of exposure to differing etiological explanations on study outcomes and therefore does not offer clear evidence of how etiological beliefs may be manipulated to produce belief in psychosocial causes of mental illness. Therefore, only two studies to date in the

literature on perceived symptom controllability have performed an experimental manipulation of beliefs about mental health etiology and tested this manipulation after the fact using true manipulation checks evaluating beliefs about mental health etiology (Crisafulli et al., 2010; Lüllmann et al., 2011).

In Crisafulli et al. (2010), participants viewed one of three 5-minute videos describing the nature and etiology of anorexia. Videos began with a case vignette in which an actor portraying a character experiencing anorexia described her development the disorder. Participants were then randomized to view one of three segments in which a doctor character states that "much debate continues to surround the etiology of AN [anorexia] but that a great deal of evidence points to the role of (biological/ sociocultural/both biological and sociocultural) factors in the development of the disorder" (p. 761). The doctor then "elaborates the research evidence supporting the contributions of (biology/society/an interaction between biology and society) to the development of the disorder" (p. 761), presenting "nontechnical, educational language that was intended to reflect appropriate language for undergraduate students." Following these video presentations, the first author discussed the study with participants. Effects of the experimental manipulation were checked by assessing participants' ratings of biological, genetic, and psychosocial (society ideals) as causes of anorexia.

In Lüllmann et al. (2011), participants listed to an audio play that presented a case vignette of a person experiencing a first episode of psychosis. The vignette described the character's family history of psychosis and psychosocial stressors before depicting possible psychotic symptoms including paranoia and possible auditory hallucinations. Participants were then randomized to listen to an actor portraying a clinician present one

of three causal explanations for psychosis or a no-explanation control condition. The biological causal explanation described "chemical imbalance in the brain," "genetic disposition," and "structural abnormalities in the brain"; the psychological causal explanation described "negative experiences in the past," "present stressors," "emotional/behavioral reactions," and "cognitive processes"; and the combined causal explanation described "interactions between psychological and biological variables." The experimental manipulation was checked through administration of a measure of perceived causes of schizophrenia.

Crisafulli et al. (2010) and Lüllmann et al. (2011) successfully manipulated participants' lay theories about the origins of mental health conditions and demonstrated this by evaluating participants' endorsement of various possible etiologies for the mental health conditions studied. These authors used experimental manipulations developed for each of their respective studies, however, and did not cite research on the manipulation of lay theories in describing their methods. Furthermore, the literature on lay beliefs about mental health is largely agnostic regarding methods of belief manipulation or change and is focused instead on cognitive, emotional, and behavioral correlates of differing lay etiological beliefs. Reviews of the literature prior to design of the present study and after analyzing the results did not identify any studies reviewing methods of experimental manipulation of lay beliefs about health topics. Thus, limitations in the extant literature examining lay beliefs about mental health etiology make it difficult to determine how these beliefs may be experimentally manipulated.

Manipulation of Lay Theories Depends on Naïve Theory Processing

Earlier social cognition research based in attribution theory offers potential processes underlying the formation, maintenance, and change of lay beliefs and suggests how these beliefs may be manipulated for experimental purposes. Based on the earlier work of Heider (1958) which described the influence of "naïve theories" on beliefs about scientific phenomena and Anderson, Krull, and Weiner's (1996) model of explanation processes, Anderson and Lindsay (1998) proposed a model of naïve theory formation, perseverance, and change that describes the steps people undergo when considering an explanation for a particular phenomenon. Anderson and Lindsay's (1998) model describes the process whereby people generate and adapt their own naïve causal theories and, in particular, what factors contribute to the perseverance of preexisting naïve theories over belief change. According to the model, change in naïve theories – such as is the aim of experimental manipulations of lay theories – occurs through information processing that mirrors the scientific method. In this process, the individual encountering novel information, such as that which is presented in an experimental manipulation, identifies their preexisting naïve causal beliefs pertinent to the topic at hand, gathers and analyzes relevant data, and makes a determination to either maintain their preexisting naïve theory or alter their beliefs to account for the novel information. Whether processing is initiated, how information is processed, and what conclusions are accepted are impacted, however, by biases in cognition, typically resulting in preexisting naïve theories being maintained and making manipulation of beliefs difficult.

Thus, according to Anderson and Lindsay (1998), the manipulation of naïve theories requires overcoming cognitive biases that would inhibit initiation of naïve-theory

processing, the acceptance of novel information during processing, and the decision to supplant preexisting naïve theories with new beliefs incorporating novel information. According to Anderson and Lindsay (1998) whether these conditions are met will depend on the interaction of several factors including the individual's preexisting beliefs and cognitive biases, motivation, situational or environmental factors, and individual differences. The methods of studies that have successfully manipulated participants' beliefs (i.e., Crisafulli et al., 2010, and Lüllmann et al., 2011) may be contrasted with the methods of the present study to explicate the influence of each of these factors on the manipulation of naïve theories.

Naïve Theory Processing is Biased in Favor of Preexisting Naïve Theories

According to Anderson and Lindsay (1998), modification of beliefs depends on the interaction of novel content with the content of an individual's preexisting naïve theories in a process that is shaped by cognitive biases. Biases against belief change are inherent in naïve theory processing, according to the model, due to situational factors described below that make it more likely that people will maintain their preexisting beliefs rather than initiate naïve theory processing, which is more effortful, time consuming, and cognitively demanding. Even when naïve theory processing is initiated, biases in information processing favor maintenance of preexisting beliefs over the adoption of new beliefs. Specifically, Anderson and Lindsay (1998) note that the availability of preexisting naïve theories in memory shapes how individuals receive novel information and results in greater skepticism for information that is contradictory to preexisting beliefs. Novel information that is contradictory to preexisting beliefs tends to be perceived as less relevant or of poorer quality than data that is consistent with

preexisting beliefs. Furthermore, individuals tend to strengthen their own beliefs about a topic through mental rehearsal and elaboration of ideas both during and outside of naïve theory processing. When engaged in this elaborative cognitive style, individuals aim to test their beliefs by gathering evidence in support of them rather than by trying – and failing – to disprove them. Thus, individuals engaged in elaboration are likely to disregard or dismiss contradictory information, resulting in biased information processing that upholds preexisting beliefs over the adoption of new beliefs.

Due to these biases in information processing and tendency to disregard belief-inconsistent information, one factor that impacts the likelihood of belief manipulation is the content of novel information. Preexisting naïve theories are most likely to be manipulated by content that accounts for the particular content of the preexisting beliefs. Researchers aiming to manipulate naïve theories may therefore benefit from understanding the content of participants' preexisting naïve theories and developing manipulation content that is most likely to influence participants based on their beliefs. This possibility poses experimental design problems, however, due to the diversity of potential participants' preexisting beliefs. In a non-experimental situation, someone seeking to manipulate or change individuals' beliefs would likely be most effective by tailoring persuasive content to account for each individual's preexisting naïve theory. In an experimental paradigm, however, this would introduce unwanted variability and potential error into the experimental design. Thus, experimental manipulations aiming to modify participants' preexisting beliefs must present manipulation content that is standardized across participants but that is nonetheless capable of overcoming participants' biased processing to alter their diverse preexisting naïve theories.

Further complicating this matter, it is possible for individuals to hold preexisting beliefs in multiple causal explanations simultaneously. Naïve theories that individuals may hold prior to experimental manipulation are not arbitrary explanations but rather relatively complex causal knowledge structures, according to Anderson and Lindsay (1998), that are developed over time through an iterative process of formulation, testing, and reformulation. This conceptualization of naïve theories is consistent with literature on lay theories of mental health, where other authors have likewise identified that so-called naïve theories are not, in fact, so naïve, but rather consistent in many ways with expert opinion (Furnham & Telford, 2011; Kim & Ahn, 2002; Lee, 1997; Shaw, 2002). As previously noted, a potential flaw in the present study design appears to be the presumption that higher biological beliefs indicate lower psychosocial beliefs and vice versa when in fact participants rated both biological and psychosocial causes of anxiety as moderately- to highly important following the manipulation with little variability, supporting the idea that it is possible to hold beliefs in both factors simultaneously.

Given this complexity of content, preexisting naïve theories may be difficult to manipulate through simple one-time exposure to alternative explanations such as those used in the present study. Complexity in preexisting beliefs may make belief manipulation challenging if novel manipulation content fails to acknowledge individuals' preexisting "knowledge." To be effective, an experimental manipulation must therefore account for this complexity in participants' preexisting beliefs so that biases in information processing such as accessibility of preexisting beliefs that include both psychosocial and biological content do not interfere with participants' receptivity to novel information. One way to mitigate biased processing based on preexisting beliefs is

to present manipulation content using a two-sided refutational argument capturing common beliefs or misconceptions about manipulation content. This style of argumentation acknowledges two opposing sides of an issue while supporting one side and counterarguing the other. For example, rather than presenting an entirely psychosocial etiological explanation for anxiety that might not be convincing to participants who are aware of biological factors, an experimenter might present manipulation content that acknowledges that while some scientists have proposed that a biological chemical imbalance may be responsible for anxiety, no such imbalance has been demonstrated in the literature. After refuting possible preexisting beliefs, the manipulation may then proceed to discuss purported psychosocial causes of anxiety more effectively. Such two-sided refutational approaches have been shown to be more persuasive than either two-sided non-refutational arguments or one-sided arguments (Cornelis, Cauberghe, & De Pelsmacker, 2013; Cornelis, Heuvinck, & Majmunder, 2019; O'Keefe, 1999).

Crisafulli et al. (2010) and Lüllmann et al. (2011) did not include the texts of their manipulation content in their published reports; therefore, it cannot be determine how these studies may have used manipulation content to account for biases in participants' preexisting beliefs; however, it is clear that the present study presented information in a way that was unlikely to account for participants' preexisting beliefs, namely by providing one-sided arguments that did not acknowledge contradictory content that participants may have held within their preexisting beliefs. Due to the diversity of potential preexisting beliefs and the need to standardize experimental manipulations to reduce error, little can be done to ensure that experimental manipulations mitigate the

effects of each participant's idiosyncratic preexisting beliefs. Future studies may instead focus their efforts on mitigating the effect of common alternative explanations on processing of novel manipulation content.

Naïve Theory Processing Depends on Motivation

Anderson and Lindsay (1998) theorize that processing of naïve theories – and therefore naïve theory change or manipulation – proceeds only under conditions of sufficient motivation. According to the theory, when sufficient motivation is present, an individual will engage in processing of their preexisting naïve theories in light of novel information. When motivation for naïve theory processing is insufficient, however, the individual will fall back on their preexisting beliefs, rendering it impossible for any information they encounter in an experimental manipulation to alter their beliefs. Factors that influence motivation for naïve theory processing may be personal to the individual (as is discussed below in the section on individual differences) or general across most individuals. Here we discuss factors that influence motivation for naïve theory process across individuals by impacting initiation of naïve theory processing and engagement with novel information sufficient to manipulate preexisting beliefs.

The primary factor identified by Anderson and Lindsay (1998) that may motivate naïve theory processing is the perceived need to form a judgment or make a decision for which the naïve theory is relevant. This need may arise in formal decision-making contexts such as when determining the best applicant to hire for a job (e.g., activation of naïve theories about personality or productivity) or determining a candidate to vote for (e.g., activation of naïve theories about leadership or electability). In contexts where individuals are not engaged in a formal decision-making process, the need for judgment

or decision-making may still be present in routine information-processing but is less likely to be recognized by individuals. In these cases, individuals are likely to fall back on preexisting beliefs without engaging in the naïve-theory processing that may result in belief change. For this reason, studies aiming to manipulate naïve theories may benefit from motivating participants to engage in naïve theory processing by indicating they will need to use the information contained in the manipulation to make a decision later. Another way to foster motivation for naïve theory processing across individuals is by appealing to emotions that will engender empathy. When the need for decision making is not apparent or an issue is not seen as personally relevant, individuals may nonetheless be motivated to care enough about an issue to engage in naïve theory processing if they feel empathy for those affected by the issue. Empathy has been shown to enhance individual's receptivity to advertising messages (Escalas & Stern, 2003), predict persuasion (Shen, 2010; Shen, 2011), and facilitate attitude change (Shelton & Rogers, 1981). Empathy may also be particularly effective means by which to motivate processing of naïve theories about mental health given that difficult emotions and suffering often accompany mental health concerns.

Motivation for naïve theory processing was likely promoted by both Crisafulli et al. (2010) and Lüllmann et al.'s (2011) experimental designs, likely contributing to the effectiveness of these manipulations. First, the need for judgment or decision-making based on manipulation content was likely readily apparent to participants in both these studies. Lüllmann et al. (2011) required participants to complete pre-test measures of etiological beliefs about schizophrenia, effectively prompting them to engage in decision-making regarding "what causes they believed to be important in the development of

schizophrenia" and likely motivating continued processing of naïve theories as participants subsequently underwent the manipulation. Additionally, both Crisafulli et al. (2010) and Lüllmann et al. (2011) used case vignettes providing explication of a characters' background and their challenges with mental health symptoms prior to introducing the etiological explanations that comprised their manipulations. The vignette format likely fostered need for decision making and increased motivation for naïve-theory processing by prompting participants to consider why the characters were experiencing the symptoms described, priming them to be receptive to the etiological explanations presented afterward. Second, both studies contained elements likely to instill empathy in participants. The case vignette format likely promoted empathy in participants by humanizing the mental health conditions of study by showing how mental health symptoms may present in a person. Furthermore, both studies appealed to participants' emotions, likely further inducing empathy for the characters depicted and likely motivating participants for naïve theory processing Specifically, Crisafulli et al.'s (2010) vignette described the character's "suffering," "self-loathing," "desperate attempts" to appear normal, and feeling "more miserable than (she) had ever been in (her) entire life." Lüllmann et al. (2011)'s methodology repeatedly directed participants to imagine themselves as the central character described the manipulation and employed sounds of laughter accompanying the audio play to enhance participant's perceptions of the character's experience.

In contrast, the present study's methods appear insufficient to motivate naïve theory processing. First, any need for judgment or decision-making was likely unclear to participants. In this study, the prompting event for naïve-theory processing was a simple

instruction that participants were to watch informational presentations about the origins of anxiety. Participants were not told why these presentations were being shown or how they would be expected to utilize the information they contained; therefore, the need to form a judgment or make a decision regarding using this content would not have been apparent other than as a matter of intellectual disputation. Furthermore, the present study did not use a case vignette and was focused entirely from the outset on presentation of the etiological explanation thereby failing to prime participants to begin considering their own judgments about the origins of anxiety prior to exposure to the manipulation content. Additionally, the manipulation content was unlikely to instill empathy in participants. Information was presented in a matter-of-fact manner without appeals to emotion, references to distress or suffering that may accompany mental health symptoms, or the humanizing qualities of depicting symptoms through a character vignette, making it unlikely to generate empathy that might motivate participants to engage in naïve theory processing. It therefore appears likely that due to the design of the present study's manipulation, participants were insufficiently motivated for initiating naïve theory processing, which resulted in insufficient engagement with manipulation content, maintenance of preexisting beliefs, and the failure of the manipulation to produce group differences in etiological beliefs.

Naïve Theory Processing Depends on Situational and Environmental Factors

Situational and environmental factors also impact naïve theory processing and interact with motivation and bias to influence naïve theory processing. Anderson and Lindsay (1998) identify that naïve theory processing is performed only when sufficient time and cognitive resources are available. If resources are lacking, naïve theory

processing will be not be initiated or will be halted and preexisting naïve theories, which do not require these resources, will be used instead. Situational and environment factors that impact the amount of time an individual has to engage in naïve theory processing before needing to use a naïve theory or the amount of attention an individual can devote to naïve theory processing are therefore key determinates of the processing outcomes including whether beliefs are changed. Additionally, situational and environmental factors are likely to interact with motivation. Given sufficiently high motivation, individuals may persist in naïve theory processing despite a poor environment for processing, but in situations where motivation is low, individuals are unlikely to devote time or cognitive resources to naïve theory processing, particular when doing so would place demands on scarce resources.

A variety of situational and environmental factors may impact resources of time, cognition, and motivation necessary for processing. For example, how information is presented may influence individuals' receptivity to novel information during naïve theory processing. Information presented in written format which requires greater use of cognitive resources compared to information presented in audio format may present a barrier for reaching individuals who do not have sufficient motivation to expend the additional effort required by reading and retaining written information. Who delivers information may also impact motivation for initiating naïve theory processing as well as how receptive individuals are to novel information presented during processing. For example, purported experts may influence individuals to be more receptive to persuasive messages by reducing the burden of time and cognitive resources otherwise needed for assessing the credibility of a persuasive message. In addition to how information is

delivered, factors within the individual's environment may influence naïve theory processing. Processing is most likely to be successful when surrounding situational and environmental factors reduce demands on individuals' time or cognitive resources. Increased demands placed on cognitive resources by stimuli in the environment or within the individual (e.g. distracting thoughts, emotions, or sensations) may also negatively impact processing by making it more effortful, thereby decreasing motivation to initiate processing or biasing the individual against accepting information contradictory to preexisting beliefs. Finally, dose of novel information may influence an individual's receptivity to novel content. Information that is presented only once or twice during naïve theory processing may be disregarded, particularly if it conflicts with other information available, including individuals' preexisting beliefs. In contrast, information repeatedly presented over time may be more likely to influence an individuals' beliefs in part because naïve theory processing may continue to occur outside of discrete episodes of information exposure. Individuals may continue to engage in naïve-theory rehearsal and elaborative thinking between episodes of exposure to novel content, potentially increasing their receptivity to novel information at subsequent time points. In the present study, manipulation content was presented only once, limiting the dose of novel content available. Future studies may benefit from examining presenting novel information repeatedly over a period of time may increase the likelihood of naïve theory processing and belief manipulation.

Crisafulli et al. (2010) and Lüllmann et al.'s (2011) methods appear to have managed situational and environmental factors to support naïve theory processing in a manner superior to the present study. With regard to resources of time, Lüllmann et al.

(2011) utilized a longer experimental manipulation, potentially allowing for further processing of naïve theories. In contrast, both the present study and Crisafulli et al. (2010) utilized experimental manipulations that were relatively brief (approximately 5 minutes in duration). Crisafulli et al.'s (2010) manipulation, however, was likely superior to the present study's with regard to allowance of cognitive capacity. Unlike the present study or Lüllmann et al. (2011) where participants completed their participation online, Crisafulli et al. (2010) provided their manipulation content in a classroom setting, where participants viewed the manipulation in groups overseen by the principal investigator and where they were provided with the opportunity to ask the principal investigator questions following the manipulation. This setting may have contributed to fewer distractions from the informational material and greater social pressure and motivation to attend to and process video content. In the present study, no such measures existed, and participants were free via the online administration of the study to complete the study at any time and in any environment.

Furthermore, the factor of limited motivation already highlighted may have interacted with this variable of cognitive capacity to further negatively affect processing of participants' naïve theories. Those who were less motivated to attend to the manipulation presentation may have been less likely to provide their full cognitive capacity to the task of viewing the manipulation videos, particularly in environments where other potentially more motivating demands on attention were present. Additionally, Crisafulli et al. (2010) and Lüllmann et al (2011) used purported experts to present the causal explanations. Use of a professional presenter may have supported the success of these experimental manipulations by enhancing motivation for naïve theory

processing, lending credibility to the content presented, and decreasing demand on cognitive resources that otherwise would be devoted to assessing the quality of evidence presented. In contrast, the present study presented its explanations by voiceover without referencing the expertise of the presenter, potentially requiring participants to exert more effort and devote more cognitive resources to judging the credibility of the claims and processing novel information in light of their preexisting naïve theories. Together, with a potentially distracting environment in which participants may have participated in the present study, situational factors likely worked against the potential of the manipulation materials to alter participants' beliefs.

Naïve Theory Processing Depends on Individual Differences

In addition to factors that impact naïve theory processing and manipulation across participants, individual difference factors within participants may contribute to greater likelihood of successfully manipulating some participants' beliefs more than others. Factors such as need for cognition may motivate some individuals to be more likely to initiate naïve theory processing or to engage in naïve theory processing more often, particularly in informal processing contexts such as the circumstances of the present study (Anderson & Lindsay, 1998). Additionally, naïve theory content on particular topics may be more applicable to some individuals than others, contributing to stronger motivation for processing. In the case of the present study, a particularly important individual difference factor is trait anxiety. For participants higher in trait anxiety, the topic of anxiety is likely particularly salient. These participants may therefore have experienced higher motivation for naïve theory processing upon receiving the prompt that they were to watch a video about anxiety origins. In contrast, those lower in trait anxiety

for whom anxiety origins may appear less salient, may have been less motivated for naïve theory processing by this instruction.

Individual differences may also impact whether individuals are receptive to the naïve content they encounter and by extension whether they are willing to accept modification of their preexisting theories. If the conclusions suggested by novel information encountered during naïve theory processing are undesirable, the individual may disregard this information in favor of maintenance of their preexisting theory. For example, biological explanatory models of mental health may be more desirable to individuals as they are associated with less self-blame for mental health difficulties (Deacon & Baird, 2013; Fisher & Farina, 1979) whereas psychosocial models of mental illness are associated with increased self-stigmatization (Deacon & Baird, 2013; Lee, Farrell, McKibbin, & Deacon, 2016). Thus, participants may be inclined to reject novel etiological explanations that emphasize psychosocial etiological factors as the implications of this explanation are undesirable. In order to account for individual difference factors that may impact manipulation of beliefs, studies may benefit from identifying such factors and controlling for their potential effects on the outcome of interest.

Both Crisafulli et al. (2010) and Lüllmann et al. (2011) attempted to control for individual differences by controlling for participants' familiarity with the mental health conditions of interest in each of their studies. In the present study, the association between etiological beliefs of anxiety and perceived anxiety controllability was stronger depending on level of trait cognitive anxiety. Given that the experimental manipulation appears to have been ineffective based on the manipulation check, it is difficult to

interpret whether this finding bears any relevance; however, future studies on the topic may benefit from controlling for potentially relevant individual differences such as this to examine whether these factors impact naïve theory processing and manipulation of beliefs. Given the variability of how individual difference factors may impact naïve theory processing and manipulation of beliefs, it may not be possible to modify experimental manipulations to satisfactorily account for these factors. Rather, researchers may opt to merely control for anticipated individual difference factors when evaluating study outcomes.

Methods for Effective Manipulation of Naïve Theories

Given seemingly pervasive biases against naïve theory processing and change, one may question whether it is productive to attempt to manipulate beliefs to study their impact on variables of interest such as perceived anxiety controllability. From a methodological perspective, it is necessary to experimentally manipulate etiological beliefs in order to evaluate potential causal links between etiological beliefs and outcomes; however, given Anderson and Lindsay's (1998) elaboration of the multiple factors contributing to persistence of naïve theories, it is important to note that this process is complex, requiring consideration of preexisting beliefs, biases against belief change, motivational factors, situational and environmental factors, and individual differences. Study methodologies aiming to manipulate participants' beliefs must therefore accommodate multiple barriers to belief change by developing experimental methods that will sufficiently motivate processing of participants' preexisting naïve theories while mitigating bias in naïve theory processing and belief consolidation.

One means to achieve this would be to simply replicate the methodology of those who were successful in manipulating lay beliefs about mental health (Crisafulli et al., 2010; Lüllmann et al. 2011). This replication may be accomplished either by directly utilizing their manipulations modified for the disorder of interest (e.g., anxiety) or by employing the conditions common between these studies that likely contributed to manipulation success such as a case vignette, empathy-inducing appeals to emotion, expert presenters, and careful control of potential individual differences. Alternatively, researchers may use other methods to produce successful manipulation of participants' preexisting naïve theories. Motivation for naïve theory processing could be increased, for example, by prompting participants to pay attention to manipulation content by instructing them that they will need to utilize what they learn to make an informed decision (e.g., about what treatments a person should use to manage anxiety or whether more resources should be devoted to identifying and treating pathological anxiety in school children). Biases within processing may be mitigated by employing two-sided refutational arguments within manipulation content to provide counterarguments to participants' preexisting beliefs. Barriers to adopting ego-dystonic beliefs could be mitigated by asking participants to write a short passage providing a rationale in support of these beliefs.

Researchers designing potential future study methodologies in this area may alternatively move away from the comparison of larger etiological belief systems to a more fine-grained approach, examining what factors within these belief systems are most salient to the outcome of interest. For example, genetic essentialist beliefs have been identified as a key factor causing the associations between biological causal explanations

and perceptions of mental health symptom control (Lebowitz & Appelbaum, 2017). Alternatively, research on mindset theory suggests that implicit mindsets promoted by etiological explanations may be responsible for associations between belief systems and mental health outcomes (Schroder, Moran, Donnellan, & Moser, 2014, Zimmermann, Hmaidan, Preiser, & Papa, 2020). Thus, it may not be the biological, psychological, or biopsychosocial knowledge belief systems as wholes that contribute to differences in key outcomes such as anxiety controllability. Rather, underlying factors within these belief systems may be of principal interest and most relevant as a target of intervention. Studies identifying, manipulating, and evaluating the effects of these underlying factors may be both more ecologically valid as well as more productive toward understanding the relationships between belief systems and outcomes such as anxiety controllability.

Additional Study Limitations

In addition to the limitations related to manipulation of beliefs described above, this study was limited in other ways. First, characteristics of the sample utilized may limit generalization of results to other populations. The present sample was composed of participants within the undergraduate psychology study pool at the university where the study was conducted. This sample was selected in part due to the relevance of clinically significant anxiety to individuals in the typical age range of university students based on this being an age range wherein clinically significant anxiety commonly emerges (de Lijster et al., 2017); however, findings may not generalize to individuals of other age groups. For example, based on the iterative process of naïve theory processing, younger individuals may hold less crystalized preexisting naïve theories of anxiety origins, potentially allowing their beliefs to be more easily manipulated. Older individuals of

different generations may also differ in their preexisting beliefs from current study participants, depending on generationally-specific cultural beliefs about mental health. Thus, further study is needed to evaluate potential causal links between etiological beliefs and perceived anxiety controllability in samples of individuals of different ages. Additionally, the preexisting naïve theories about mental health held by participants in the present study may not generalize to other samples who differ in cultural background.

As previously noted, lay beliefs about mental health are culturally derived and culturally specific (Angermeyer & Matschinger, 1994; Chen & Mak, 2008; Giosan, Glovsky, & Haslam, 2001). Important differences emerge in lay beliefs about mental health etiology across cultures, particularly between collectivist and individualist cultures (Altweck, Marshall, Ferenczi, & Lefringhausen, 2015; Chen & Mak, 2008), and some authors have proposed that cultural adoption of particular etiological beliefs may in fact occur in response to susceptibility to mental health concerns in certain populations (Chiao & Blizinsky, 2010). The primary variable in the present study that may reflect the cultural backgrounds of participants was race/ethnicity, which found that a majority of the sample identified as non-Hispanic White. This variable provides little information, however, about participants' culturally specific beliefs about mental health. Other cultural factors that could provide more insight into culturally specific beliefs about mental health such as national origin, acculturation status, endorsement of collectivist/individualist beliefs, or religious beliefs were not assessed in the present study. Therefore, potentially salient features of the cultural background of participants in the sample is unknown, making it difficult to know how findings may generalize to other populations.

Finally, with regard to the characteristics of the present sample, study findings may also be limited based on the gender distribution of the sample. Participants in the present study overwhelmingly identified as female with a smaller portion of participants identifying as male and no participants identifying as another gender. The lack of non-binary or genderqueer-identified individuals in the study is a significant limitation, particularly given that non-binary and genderqueer individuals experience anxiety at a significantly higher rate than binary-identified individuals of any gender (Thorne et al., 2019). Furthermore, the predominance of female-identified people in the sample presents an additional limitation as female-identified people report clinically significant symptoms of anxiety at a higher rate compared to male-identified people across various presentations of anxiety (McLean, Asnaani, Litz, & Hofmann, 2011). While the present study's large proportion of female-identified participants is a strength with regard to the saliency of anxiety to this population, the generalizability of study results to male-identified and non-binary or genderqueer individuals is limited. Thus, the characteristics of sample of the present study's sample may critically limit generalization across populations.

Another limitation of the present study is that it did not utilize an integrated biopsychosocial condition in addition to the biological and psychosocial conditions like Crisafulli et al. (2010) and Lüllmann et al. (2011) did. As previously noted, the decision to exclude a biopsychosocial condition was based on the concern about evaluating causation if there were overlap in manipulation content between conditions. As a result, Gershkovich et al.'s (2018) manipulation content upon which the present study's manipulations were based was edited to create a script for a purely psychosocial rather

than biopsychosocial condition. However, when including biological and psychosocial conditions in addition to a biopsychosocial condition, it is possible to test the potential interactive effects of the biological and psychosocial etiological beliefs on study outcomes while also testing causation. While the apparent failure of the experimental manipulation makes the results of the study moot on this point, future studies may benefit from inclusion of a biopsychosocial condition. Without the use of the condition, study results cannot speak to the potential role of lay beliefs in the dominant expert theory of anxiety (i.e., the biopsychosocial model, impact perceived anxiety controllability). The inclusion of a biopsychosocial condition would therefore allow future studies to draw more ecologically valid conclusions by comparing the impact of the dominant expert theory to theories that lay persons may hold but which are understood by psychological experts to be incomplete.

A final key limitation of the present study was that data was collected during the Fall of 2020 during the COVID-19 pandemic, an historic event that makes generalization of study results to other historic contexts difficult. The COVID-19 pandemic caused significant disruption in individuals' daily lives and contributed to heightened symptoms of depression and anxiety and experiences of stress in the general public across populations (Salari et al., 2020), impacting not only those diagnosed with COVID-19 or who personally knew someone with the virus but also large portions of the population as a whole (Taylor, Landry, Paluszek, Fergus, McKay, & Asmundson, 2020). As undergraduates at Baylor University, participants in the present study were tasked with additional demands in their daily lives related to the pandemic that may also have contributed to stress and anxiety during the period of data collection including adjustment

to virtual learning and requirements to respond to routine screenings for the SARS-CoV-2 virus. In addition to requiring adjustments in daily routines, the COVID-19 pandemic contributed to heightened stress for many individuals (Taylor et al., 2020). It is unknown to what degree participants in the present study were impacted by COVID-19 either through contracting the virus themselves, having family members or loved ones who contracted the virus, or experiencing COVID-19 stress; however, a risk factor associated with higher COVID stress syndrome features was female gender, an identity that a majority of study participants endorsed (Taylor et al. 2010). Furthermore, this syndrome and experiences either directly or indirectly with the virus are particularly pertinent to the topic of study as individuals experiencing pre-morbid anxiety-related disorders have been shown to demonstrate higher COVID-19-related stress (Asmundson et al., 2020; Taylor et al., 2020). The convergence of historical events, possible exposure to COVID-19 either directly or indirectly, interactive effects of premorbid anxiety and COVID stress syndrome, and the present study's focus on clinically significant anxiety in particular likely limits generalizability of study results.

On the one hand, the pandemic contributing to greater stress and anxiety in the general public may have increased the pertinence of anxiety for some participants in the present study, which may have in turn contributed to study engagement; however, given the prevalence of heightened anxiety during this period of time, participants may likewise have experienced the anxiety-focused content of the present study as particularly aversive, potentially contributing to avoidant coping and disengagement from study materials. Additionally, knowledge of the biological nature of the pandemic as resulting from viral transmission may have biased participants toward attunement to biological

factors in general, potentially impacting awareness of biological factors not only in COVID-19 disease transmission but also in other health conditions as well including mental health conditions like anxiety. Thus, it is difficult to know how the context of the pandemic may have impacted study results; however, the historical context of the pandemic limits generalization of study results to other historical contexts.

CHAPTER FIVE

Conclusions

It is important to understand the origins of perceptions of anxiety controllability, which have been shown to be a key determinant of anxiety symptoms severity and which therefore is a possible distinguishing factor between normative experiences of anxiety and clinically severe anxiety. A potential source of perceived anxiety controllability is the naïve explanations people hold about anxiety origins that may be culturally transmitted. Biological and psychosocial etiological explanations in particular are two primary etiological explanations that have been identified in the literature on lay beliefs about mental health. The present study aimed to experimentally manipulate participants' beliefs about the origins of anxiety in order to evaluate potential causal links between biological or psychosocial etiological explanations of anxiety and perceptions of anxiety controllability. In the present study, however, any such links could not be evaluated due to the apparent failure of the manipulation to produce significant group differences on a manipulation check assessing ratings of anxiety as a "biological or medical disorder."

Failure to manipulate beliefs in the present study may be attributed to a variety of factors that inhibit the processing of naïve theories and result in the maintenance of preexisting beliefs over modification or manipulation of beliefs. Biases within information processing, the tendency to uphold preexisting beliefs, lack of motivation for naïve theory processing, situational and environmental factors, and individual difference factors may be responsible for the apparent failure of the manipulation in the present

study. Future studies aiming to evaluate possible causal links between etiological beliefs and perceptions of anxiety controllability must overcome these barriers to belief change in order to successfully manipulate participants' beliefs. Knowledge of possible causal links between certain etiological beliefs and low perceived anxiety controllability would suggest that it may be important to assess patients' etiological beliefs prior to initiating treatment for clinically severe anxiety. A causal relationship between biological etiological beliefs and lower perceived anxiety controllability would also suggest that interventions focused on patients' beliefs about the biological origins of anxiety may be a key early treatment target in order to enhance patient's perceptions of anxiety controllability and facilitate readiness for other cognitive-behavioral interventions targeting symptom interference.

APPENDICES

APPENDIX A

Etiological Explanations - Gershkovich et al. (2018)

Biological (Brain) Explanation

Research suggests that OCD is caused by problems in the brain. Specifically, data from brain imaging studies show that in individuals with OCD there are problems in communication between the front part of the brain (which involves planning and control over one's actions) and deeper brain structures (which involve emotions such as fear and anxiety). These brain structures use neurotransmitters (basically, chemical messengers) and data suggest that OCD involves an imbalance in these brain chemicals (particularly one called serotonin). Medications that affect serotonin (serotonin reuptake inhibitors, or SRIs) work to correct this chemical imbalance and restore normal brain function.

Biopsychosocial Explanation

There are a number of reasons why a person might develop OCD, including having a family history of OCD, which may contribute both biological/genetic and environmental aspects. Stressful life experiences, like the death of a loved one, may also play a role. Individuals with OCD may begin having high levels of anxiety and over time develop problematic patterns of relying on rituals (compulsions) as a way to manage their anxiety. Some experts have suggested that specific “thinking mistakes” occur in individuals with OCD, which may cause them to believe that terrible things will happen if they do not do their rituals. Over time OCD can worsen as the person learns to rely on

rituals more and more. It is probably a combination of biological, environmental, and psychological factors that causes OCD.

APPENDIX B

Etiological Explanations – Present study

Biological (Brain) Explanation

Research suggests that anxiety is caused by problems in the brain. Specifically, data from brain imaging studies show that in individuals with anxiety there are problems in communication between the front part of the brain (which involves planning and control over one's actions) and deeper brain structures (which involve emotions such as fear and anxiety). These brain structures use neurotransmitters (basically, chemical messengers) and data suggest that anxiety involves an imbalance in these brain chemicals (particularly one called serotonin). Medications that affect serotonin (serotonin reuptake inhibitors, or SRIs) work to correct this chemical imbalance and restore normal brain function.

Psychosocial Explanation

There are a number of reasons why a person might develop an anxiety disorder, including having a family history of anxiety, which may influence how parents attempt to manage their children's experiences. For example, research shows that parents who are overly controlling of their children's behavior can contribute to more anxiety in their children. Stressful life experiences, like the death of a loved one, may also play a role. Individuals with anxiety disorder may begin having high levels of anxiety and over time develop problematic patterns of relying on certain behaviors as a way to manage their

anxiety. These behaviors include doing things like avoiding anxiety-provoking situations like social events, repeatedly asking friends or family for reassurance, or using alcohol or drugs to reduce feelings of anxiety. Therapists often refer to these behaviors as “safety behaviors” because people with anxiety use them to lessen anxiety and feel safer. Some experts have suggested that specific “thinking mistakes” occur in individuals with anxiety, which may cause them to believe that terrible things will happen if they do not use safety behaviors to manage their anxiety. Over time anxiety can worsen as the person learns to rely on safety behaviors more and more. It is probably a combination of early childhood experiences, environmental, and psychological factors that causes anxiety.

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