THE CHRONIC DISEASE CONCEPT OF ADDICTION: HELPFUL OR HARMFUL?

by

Thomas K. Wiens

B.A., The University of British Columbia, 2012

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF ARTS

in

THE FACULTY OF GRADUATE AND POSTDOCTORAL STUDIES

(Psychology)

THE UNIVERSITY OF BRITISH COLUMBIA

(Vancouver)

August 2014

© Thomas K. Wiens, 2014

Abstract

In contemporary culture, socially deviant behavior is increasingly being conceptualized as the result of a disease. This, perhaps, is most salient in regards to addiction. The chronic disease model of alcoholism has its roots in early assumptions that have recently been discredited or at least challenged. For example, it has been found that the majority of alcoholics permanently overcome their addiction, without treatment, and within a few years. This thesis employs an experimental method to examine whether telling individuals with a mild to moderate alcohol addiction that they have a chronic brain disease influences their perceptions of addiction-related agency as well as their feelings of shame and stigma. Participants, recruited online, were randomly assigned to internalize statements promoting (a) a disease model of addiction, (b) a psychosocial model, or (c) a neutral control condition; they then completed several indices of agency in relation to drinking, as well as measures of perceived and internalized stigma and state shame. Participants who internalized the disease model of addiction tended to have weaker perceptions of drinking self-efficacy, whereas internalizing psychosocial model beliefs tended to induce a stronger internal locus of control and weaker entitization of addiction. Both the disease and the psychosocial conditions increased, in equal amounts, both feelings of stigma and shame relative to the control condition. The relationships between various demographic, attitudinal, and trait measures, as well as indices of agency, stigma, and shame, were assessed. The implications of these findings are discussed in terms of the benefits and costs of each of these models of addiction.

Preface

This thesis is original, unpublished, and independent work by the author, Thomas K. Wiens. The research reported here was approved by the UBC Behavioural Research Ethics Board [certificate #H13-02041].

Table of Contents

Abstract.....	ii
Preface.....	iii
Table of Contents	iv
List of Tables	vi
List of Abbreviations	vii
Acknowledgements	ix
Chapter 1: Introduction	1
The Disease Concept of Addiction	1
Abandoning Traditional Misconceptions: An Empirical View of Alcoholism	15
DSM-5: Expanding the Definition of Addiction	19
Reconsidering the Disease Concept.....	22
Research Design.....	37
Chapter 2: Method.....	40
Participants.....	40
Procedure	50
Wave 2 and 3 Response Rates	53
Measures	53
Power Analysis	66
Chapter 3: Results.....	68
Overview.....	68
Main Analyses of the Experimental Intervention	70
Exploratory Analyses using the ABSQ Items.....	80
Correlational Analyses.....	82
Chapter 4: Discussion	91
Manipulating Addiction Beliefs: The Impressionable Nature of Addiction Beliefs	92
Typical Addiction Beliefs Among Mild to Moderate Alcoholics	93
The Influence of Addiction Beliefs on Perceptions of Agency	94
Typical Perceptions of Addiction-Related Agency Among Mild to Moderate Alcoholics	95
The Influence of Addiction Beliefs on Perceptions of Stigma and Shame.....	96
Agency and Shame and Stigma	100
The Perceived Importance of Health Professionals: Examining Locus of Control	101

Perceptions of Drinking Self-Efficacy: Contributions of Disease and Psychosocial Models	103
Addiction Entitization: Examining Perceptions of Chronicity and Immutability	104
The Disease and Psychosocial Models of Addiction: Benefits and Costs.....	105
References	108
Appendix: Manipulation	126
Psychosocial Model Condition	126
Disease Model Condition.....	128
Neutral Condition.....	130

List of Tables

Table 1: Wave 1 Sample Demographics ($N = 1894$)	44
Table 2: Comparisons of Demographic Data Across Addiction Groups.....	46
Table 3: Demographics of Participants Meeting Study Inclusion Criteria ($N = 159$)	48
Table 4: DSM-5 Alcohol Use Disorder Diagnostic Symptoms.....	55
Table 5: Psychosocial Model Beliefs (ABS) Main Effect Follow-up Analyses for Condition	72
Table 6: Disease Model Beliefs (ABS) Simple Main Effects Follow-up Analyses for Condition at Both Waves.....	73
Table 7: Locus of Control (MHLC) Simple Main Effects Follow-up Analyses for Condition at Wave 2	75
Table 8: Drinking Self-Efficacy (CDESES) Main Effect Follow-up Analyses for Condition	76
Table 9: Addiction Entitization (AAES) Simple Main Effects Follow-up Analyses for Condition at Wave 2	77
Table 10: Stigma (SSMIS) Aware Subscale Simple Main Effects Follow-up Analyses for Condition at Wave 2	78
Table 11: Shame (SSGS) Main Effect Follow-up Analyses for Condition	80
Table 12: ABSQ Exploratory Comparisons Between the Disease and Psychosocial Conditions.....	81
Table 13: Correlations Between Demographics and Disease and Psychosocial Addiction Beliefs ($N = 28$)	83
Table 14: Correlations Between Addiction Beliefs, Attitudinal Beliefs, and Personality Traits .	84
Table 15: Intercorrelations Between Indices of Agency, Stigma, and Shame.....	86
Table 16: Correlations Between Addiction Beliefs, Attitudinal Beliefs, and Personality Traits .	87
Table 17: Correlations Between Alcohol Use, Addiction Severity and Agency, Stigma, and Shame	90

List of Abbreviations

AA: Alcoholics Anonymous

ABS: Alcohol Belief Scale

ABSQ: Addiction Belief Summary Questions

AAES: Alcohol Addiction Entitization Scale

ANOVA: Analysis of variance

APA: American Psychiatric Association

ASAM: American Society of Addiction Medicine

BACQ: Brief Approach/Avoidance Coping Questionnaire

CDSSES: Controlled Drinking Self-Efficacy Scale

DSM-4-TR: Diagnostic and Statistical Manual of Mental Disorders 4 (Text Revision)

DSM-5: Diagnostic and Statistical Manual of Mental Disorders 5

FAD-Plus: Free Will and Scientific Determinism Plus

MHLC: Multidimensional Health Locus of Control

MTurk: Mechanical Turk

NIAAA: National Institutes on Alcohol Abuse and Alcoholism

NIDA: National Institute on Drug Abuse

NIH: National Institutes of Health

NIMH: National Institute of Mental Health

OCD: obsessive-compulsive disorder

PDD: Perceived Devaluation–Discrimination Scale

SSAGA II: Semi-Structured Assessment for the Genetics of Alcoholism II

SAMHSA: Substance Abuse and Mental Health Services Administration

SSGS: State Shame and Guilt Scale

SSMIS: Self-Stigma of Mental Illness Scale

Acknowledgements

I am particularly thankful for my extremely caring research supervisor, Dr. Larry Walker, who went well above and beyond the call of duty to make this thesis a successful and enjoyable project.

I am grateful to the members of my supervisory committee, Drs. David Klonsky, Steve Heine, and Anita DeLongis, for their time and support.

I thank my research assistant, Shereen Chao, who did a great job with the technical details of creating the online survey.

I want to give special thanks to my lovely wife, Trilby, for her endless encouragement.

I would also like to express my appreciation to both the UBC Department of Psychology and the Social Sciences and Humanities Research Council for their fellowship support.

CHAPTER 1: INTRODUCTION

In contemporary culture, socially deviant behavior is increasingly being conceptualized as the result of a disease (Conrad, 1992; Szasz, 2007). This medicalized explanation of behavior is especially prominent in regards to substance addiction. Within the modern conceptualization, people who consume problematic amounts of alcohol or drugs are thought to do so as the result of having a particular illness, namely the disease of addiction (Leshner, 1997). This has become known as the chronic disease concept of addiction.

This conceptualization of addiction first came to popularity in the middle of the 20th century with Jellinek's (1946) famous study and subsequent influential book, *The Disease Concept of Alcoholism* (1960). This work was thought to provide the scientific support that fortified the views and frameworks already employed in the emerging Alcoholics Anonymous (AA) movement (Fingarette, 1988, 1990). The growth of the disease conception of addiction has paralleled the explosion of AA groups, which now make up the vast majority of alcohol treatment options with over 60,000 groups in North America and 100,000 groups worldwide (AA, 2012). The disease concept of addiction is a core tenet of AA and has been fundamental to its apparent success (Maxwell, 1984).

The Disease Concept of Addiction

The Widespread Contemporary Influence of the Disease Concept

While the disease concept of addiction has its nascence in AA groups, the philosophy has become firmly entrenched in the broader addiction treatment culture. It is the model employed by the National Institutes of Health (NIH), which have two divisions concerning addiction, one specifically concerned with alcohol addiction, the National Institute on Alcohol Abuse and Alcoholism (NIAAA), and another dealing with drug and alcohol addiction, the National

Institute on Drug Abuse (NIDA). These organizations represent the largest alcohol research agencies in North America and describe alcoholism as a chronic medical disease: “Addiction is defined as a chronic, relapsing brain disease that is characterized by compulsive drug seeking and use” (NIDA, 2012, “What is Drug Addiction?” para. 1) and “a disease [that] is typically considered chronic, meaning that it lasts a person’s lifetime” (NIAAA, n.d.-a, “Alcohol Use Disorders,” para. 5). This conception is similarly reflected in the recent broader paradigm shift of the parent organization, the National Institute of Mental Health (NIMH); in a recent update its director, Thomas Insel, asserted that our conception of mental illness needs to shift, from that of “mental or behavioral disorder” to that of “brain disorder” (NIMH, 2013, “Mental Disorders as Brain Disorders,” para. 3). Within this biological model, addiction is considered to be the result of a malfunctioning brain—a sick brain—characterized by impaired decision-making capabilities. Similar views are held by other major addiction organizations, such as the American Society of Addiction Medicine (ASAM) which defines an addiction as a “primary, chronic disease of brain reward, motivation, memory, and related circuitry” (ASAM, 2011, “Definition of Addiction,” para. 1). Furthermore, even the recent U.S. presidential administration’s drug policy has affirmed that “decades of scientific study show that addiction is a disease of the brain” (Executive Office, 2013, p. 27).

The disease model also represents the dominant orientation among addiction health professionals in North America. The most recent survey on the matter asked family physicians and psychiatrists the extent to which they judged alcoholism to be a disease (Lawrence, Rasinski, Yoon, & Curlin, 2013). The majority of family physicians (92%) and psychiatrists (96%) agreed that addiction was either “somewhat” or “a lot” a disease (pp. 257-258). Yet, the majority (78% of family physicians; 86% of psychiatrists) also endorsed the psychological notion that

alcoholism is a “response to psychological woundedness” and a minority (25% of family physicians, 14% of psychiatrists) also considered alcoholism “a result of moral failings” (pp. 257-258). A survey of a broader scope of health professionals (viz., psychologists, physicians, counselors, social workers, nurses, program and case managers) found that 85% agreed that “addiction is really a disease” (Forman, Bovasso & Woody, 2001, p. 4). Another survey conducted with doctoral-level addiction specialists found that about 64% employed the disease model to conceptualize addiction treatment (Morgenstern & McCrady, 1992). One reason for this relatively lower endorsement of the disease model is that the sample included a sizeable portion of experts with a behavioral orientation, none of whom endorsed notions of the disease model. However, all of the other theoretical traditions surveyed (e.g., psychodynamic, existential, family systems, eclectic) had experts who endorsed the model.

The conceptualization of addiction is not merely an esoteric triviality limited to the realm of professionals. Rather, this distinction—that addiction is a biological disease, rather than a psychologically or socially driven problem—is being promulgated to mental health workers, students, and the lay public (Fingarette, 1988; Heyman, 2009). This widespread popularization of the notion occurs through scientific textbooks and journal articles, government-supplied bulletins, presentations provided to schools, community groups, and the public, as well as the media (Heyman, 2009).

For instance, an article in a leading scientific journal by a former director of NIDA described addiction as a chronic and relapsing brain disease just like other diseases afflicting the brain, such as Alzheimer’s, stroke, and schizophrenia (Leshner, 1997). A specialized section of NIDA geared toward younger adolescents (aged 11-15) states that “NIDA science shows that addiction is a disease, just as cancer and asthma are diseases” in which “free will has been

cruelly hijacked, and the desire to seek and use drugs is beyond their control” (NIDA, 2013, “Fear of Stigma,” para. 4). Other articles have grouped addiction alongside other biological diseases such as hypertension, diabetes, asthma, arthritis, Alzheimer’s, schizophrenia, cancer, and even epilepsy (e.g., Leshner, 1997; Mack, Franklin, & Frances, 2003; McLellan, Lewis, O’Brien, & Kleber, 2000; Milam & Ketcham, 1981; O’Brien & McLellan, 1996).

The same messages fueled by national institutions are being communicated to the public. For example, NIDA developed a community presentation, titled *Addiction: It’s a Brain Disease Beyond a Reasonable Doubt*, to educate the public on how “scientific advances have revolutionized our understanding of drug abuse and addiction, which is now recognized as a chronic relapsing brain disease” for which the “inability to stop is the essence of addiction, like riding in a car with no brakes” (NIDA, n.d., p. 1). Other initiatives to promote awareness regarding the disease essence of substance addiction include the Home Box Office documentary and its companion book, *Addiction: Why Can’t They Just Stop?* (Hoffman & Froemke, 2007).

The Disease Concept Philosophy

The disease philosophy conceives of addiction as a fundamentally biological malfunction that causes compulsive substance use (Leshner, 1997). The concept of disease—a physiological abnormality—is the philosophical foundation upon which treatment principles are built (Cook, 1988). This means that terms such as “illness,” “disease,” and “powerlessness over alcohol” are commonplace within such recovery settings (Cook, 1988; Laundergan, 1982; Maxwell, 1984). In fact, such terms are routinely used to inaugurate new members (Maxwell, 1984). Traditional belief has held that the chemical dependence embodies the disease entity (i.e., physiological abnormality), which is thought to undermine a person’s volition and lead to a loss of control over substance use (Hill, 1985; Milam & Ketcham, 1981).

It is important to make a distinction between the disease model of addiction and the notions of genetic vulnerability and acquired physiological dependence. The latter have been well-established as risk factors for both developing (genetic vulnerability) and maintaining addiction (due to physiological dependence; Pickens & Svikis, 1988); however, they do not imply, as disease proponents argue, that addicts are “powerless” or completely “unable” to control their drinking behavior (especially those with a less severe dependency). The disease conceptualization, on the other hand, is a theory that posits the disease of addiction to be an internal and stable entity in which individual effort to break free from its thralls is largely ineffective (Morgenstern & McCrady, 1992).

The disease model has undergone somewhat of a resurgence within the last few decades with technological improvements in brain imaging. This is the reason for the renewed widespread promulgation that “science” has established addiction as a brain disease (e.g., NIDA, 2013). For instance, the director of NIDA from 1994 until 2001 stated in an influential review paper “that addiction is tied to changes in the brain structure and function is what makes it, fundamentally, a disease” (Leshner, 1997, p. 46). His successor, Glen Hanson, also stated that three decades of research demonstrate that “once a person becomes addicted, the drug has literally changed his or her brain” and “therefore we say it is a brain disease” (Hanson, Leshner, & Tai, 2002, p. 69). Similarly and more recently, NIDA (2010) has stated that addiction is considered a “brain disease because drugs change the brain—they change its structure and how it works” (“What is Drug Addiction?” para. 1). The same website cites brain imaging studies stating that drugs alter brain regions involved in decision making, which impairs an addict’s ability to abstain and leads to continued involuntary and chronic substance use (NIDA, 2010).

Professionals working within this paradigm heavily promulgate (sometimes dogmatically so) that the disease is incurable and will afflict the alcoholic for the remainder of his/her life (Fingarette, 1988; Milam & Ketcham, 1981; Schaler, 2000). Hence, treatment efforts focus on restraining and putting to remission the effects of the disease (Maxwell, 1984; Milam & Ketcham, 1981). Subsequently, treatment staff are highly concerned with promoting addicts' identification with the role of a sick patient and aligning their conception of their problem as the result of a disease (Morgenstern & McCrady, 1992). Indeed, accepting one's fate as unable to engender change on one's own efforts is an emphasized treatment goal in the disease model programs (Laudergan, 1982; Morgenstern & McCrady, 1992). Reluctance to do so is often considered to reflect a state of "denial" which is thought to be "a symptom of the disease" (Schaler, 2000, p. 43). While accepting personal responsibility for recovery is encouraged (Morgenstern & McCrady, 1992; Wallace, 1996), putting the disease to remission is thought to necessitate treatment (i.e., treatment principles and/or medication); self-management, on the other hand, remains of comparably peripheral concern (Morgenstern & McCrady, 1992; Schaler, 2000).

Criticisms of the Disease Concept

Even though the disease concept of addiction currently predominates, it has not gone unchallenged. Criticisms tend to cluster around two main objections. The first is that the disease model does not have empirical support and that it actually belies scientific data (e.g., Chiauuzzi & Liljegren, 1993; Fingarette, 1988, 1990). These critics argue that the model is a reified social construction, arising from societal and ideological trends. The second criticism concerns the potential harmfulness of promoting such a conceptualization of addiction (e.g., Douglas, 1986; Fingarette, 1990). These concerns revolve around the manner in which the disease conception of

addiction is thought to undermine addicts' sense of agency and motivation for effortful involvement in combating their addiction. While this latter criticism will be discussed in a later section on the utility of the disease conception, the former criticism on the veridicality of the model will now be addressed.

Critics of the disease model of addiction have pointed out that empirical findings fail to support and even point away from the notion of a chronic physiological disease of addiction. These critics argue that (a) the heterogeneity of signs/symptoms and patterns of behavior among alcoholics fails to account for one particular disease symptomology and etiology (Fingarette, 1988; Jones, Gill, & Ray, 2012; Schaler, 2000); (b) the fact that medical diseases normally require pharmacological treatment, which is generally not recommended for alcohol addiction (Morgenstern & McCrady, 1992; Ogborne, Wild, Braun, & Newton-Taylor, 1998); (c) biological factors are but one relatively less influential factor in a slew of other personal (e.g., values, gender) and social factors (e.g., culture, religion) influencing alcohol addiction (Butcher, Mineka, Hooley, Taylor, & Antony, 2010; Fingarette, 1990; Peele, 1989); (d) chemical dependence is more aptly described as an intermittent impairment of the will that is also influenced by environmental context rather than the loss-of-control effects exaggerated by the media, government, and medical community (Davies, 1992; Edwards & Gross, 1976; Fingarette, 1988, 1990); and (e) the majority of people meeting criteria for alcohol dependence resolve their addiction without the help of treatment (e.g., Cunningham, Lin, Ross, & Walsh, 2000; Dawson et al., 2005; Sobell, Cunningham, & Sobell, 1996) and do not continually relapse back into cycles of alcohol dependence in the fashion described by disease model advocates (Dawson et al., 2005; Dawson, Goldstein, & Grant, 2007; de Bruijn, van den Brink, de Graaf, & Vollebergh, 2006; Willenbring, 2010).

All of this is to say that, in spite of the widespread acceptance of the disease concept, many have vehemently argued that it belies scientific findings. Yet, as has been discussed above, proponents reject the aforementioned data and confidently assert that science (referring to brain imaging studies) has demonstrated the disease nature of addiction that “hijack[s]” the brain’s decision-making abilities (e.g., NIDA, 2013, “Fear of Stigma,” para. 4). While the disease model came to dominance before such technological sophistication (Levine, 1978), neuroscience has brought a renewed confidence to the disease model of addiction.

Has Not Neuroscience Shown Addiction is a Brain Disease?

The role of brain imaging technology has played an instrumental role in understanding mental illness (NIMH, 2013). When addiction organizations and researchers state that science has demonstrated that addiction is a chronic compulsive disease, they are referring to the neuroscientific data on the structural and functional brain changes resulting from alcohol consumption (e.g., ASAM, 2011; NIMH, 2013). Such studies have demonstrated that drug addicts have physical changes that occur in the brain regions associated with decision making and judgment, learning and memory, and behavior control (Fowler, Volkow, Kassed, & Chang, 2007); scientists believe this is what “alter[s] the way the brain works, and ... help[s] explain the compulsive and destructive behaviors of addiction” (NIDA, 2010, “Is Continued Drug Abuse a Voluntary Behavior?” para. 1).

One leading critic who has recently objected to the claim that addiction is a chronic and compulsive brain disease is research psychologist Gene Heyman. A lecturer on addiction at Harvard and researcher at Boston’s McLeans’ Hospital—a neuroscience and psychiatric research powerhouse—Heyman is intimately familiar with the subject. In his book, *Addiction: A Disorder of Choice* (2009), he rejects the claim that substance-induced brain changes are

indicative of a chronic and compulsive condition: That “drugs change the brain is a logical necessity as well as experimental fact. Drugs change behavior, mood, and thought; the brain is the organ of behavior, mood, and thought; thus drugs change the brain” (p. 95). He continues to explain that genetic predispositions or changes to the brain would only distinguish addiction as involuntary behavior if such biological influences were not associated with voluntary behaviors. But that is certainly not the case.

In terms of genetic predisposition, Heyman (2009) argues that many voluntary behaviors or beliefs are influenced by heredity. One example concerns religious observance, a voluntarily chosen activity, which has been shown to have significant genetic influence (Heyman, 2009). In one study examining this issue, researchers tried to assess the genetic contributions to religiosity by comparing the correlations between siblings of monozygotic twin dyads reared apart with the correlations between siblings of dizygotic twin dyads reared apart (Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990). Because each of the twin pairs was separated very early in life (mean separation at 0.24 years), higher correlations in religiosity between siblings in monozygotic twin dyads than between siblings in dizygotic twin dyads would suggest a genetic influence. In order to ensure differences reflected genetic similarities, rather than environmental similarities, researchers also assessed for biases in environment placement.

The study confirmed the role of genetic influences and found strong correlations between the siblings of monozygotic twin dyads’ religious values ($r = .55$), religious interests ($r = .39$), and religious fundamentalism ($r = .55$); the siblings of dizygotic twin dyads, on the other hand, shared no such relation ($r_s = -.08, .04$, and $-.22$, respectively); biases in environmental placement were not a relevant factor (Waller et al., 1990). These researchers concluded that approximately 50% of the variance of a person’s religious preferences is explained by genetic factors. Thus,

religious observance is considered to provide an example of voluntary behavior that also has a strong genetic influence (Heyman, 2009). This study represents but one of many that has demonstrated the heritability of voluntarily chosen beliefs and attitudes.

A similar line of reasoning needs to be applied when considering the claim that substance-induced brain changes lead to compulsive behavior (Heyman, 2009). If brain plasticity is associated with voluntary behavior, then addicts' brains are not necessarily chronically impaired. This is clearly supported in one example concerning the treatment of obsessive-compulsive disorder (OCD). Heyman (2009) discusses how OCD patients who learned to ignore their obsessions were able to not only reduce the amount of intrusive thoughts (and subsequent obsessive rituals) but also to reduce the neural activity in obsession-related brain regions to the point that their brains displayed similar patterns to the brains of OCD-free comparison subjects (Schwartz, 1998). Indeed, similar findings can be found in "hundreds of studies documenting that changes in voluntary activities are associated with changes in the brain" (Heyman, 2009, p. 96). Therefore, Heyman argues that neuroscience does not indicate the extent to which behavior is voluntary or the addict is "in control" of his/her behavior. Rather the brain imaging techniques offer a mere description of the brain activity that occurs as individuals interact with their environment. To understand the extent to which people can control a behavior, their behavior and the motivations behind their behavior, not brain activity, need to be analyzed.

Understanding the natural course of alcohol addiction is best accomplished through longitudinal studies (Heyman, 2009). The largest longitudinal study on alcohol addiction to date, the NIAAA's National Epidemiological Study on Alcohol and Related Conditions (2001-2005), has demonstrated that 72% of alcoholics—people diagnosed with alcohol dependence—gain

control of their drinking, without the help of professional treatment, within a few years ($M = 3.7$ years; Dawson et al., 2005; Hasin, Stinson, Ogburn, & Grant; 2007); contrary to the chronic disease conceptualization, this majority of alcoholics does not have a chronic addiction and does not continually relapse (Dawson et al., 2007; Willenbring, 2010). Similar results were found across the other three large representative national studies that have been conducted: The Epidemiologic Catchment Area Study (1980-1984) and the two National Comorbidity Surveys (1990-1992 and 2001-2002). After examining all of these data Heyman (2009) concludes: “The four largest, most methodologically rigorous studies of psychiatric disorders and their correlates fail to support the claim [that addiction is a chronic disorder]” (p. 74). Yet, for some reason, authors of clinical texts and journal articles routinely ignore these findings (Heyman, 2009). Moreover, the reasons people give for successfully quitting their drinking are common reasons that influence people’s everyday decisions: financial obligations, risk of harm, and concern over maintaining familial relationships (Heyman, 2009).

My focus here is not to debate the veracity of the chronic disease concept but rather to concretely demonstrate that there are many empirical reasons for which numerous researchers reject the model. The question then arises, if the model has minimal scientific basis, why has it gained such prominence in the last century? The answer: Societal conditions were ripe. As one critic put it, “the idea that alcoholism is a disease has always been a political and moral notion with no scientific basis” (Fingarette, 1990, p. 48). The societal factors that gave rise to the disease concept of addiction will now be discussed.

The Historical Emergence of the Disease Concept

The historical emergence of the disease model of alcohol addiction has been thoroughly investigated by the sociologist Harry Levine (1978), and an overview, relying on his analysis,

will now be given. The disease concept of addiction was introduced and slowly began its ascent to popularity in North America in the late 1700s. Before this, Americans were well known for their heavy consumption of alcohol. While some objected to drunkenness on moral grounds (e.g., the Puritans), heavy drinking was considered to be normal and it was thought that “drunkards” autonomously chose to pursue their greatest pleasure, a life filled with drunkenness; in the traditional psyche, the notion of an unwanted addiction to alcohol was absent. It was not until the early 1800s that the freedom of an alcoholic’s will was called into question.

Spearheaded by medical professionals, the conceptualization of the alcoholic’s will shifted to one characterized by impairment rather than freedom. Benjamin Rush was the first to articulate a new paradigm of addiction in which alcohol addiction was portrayed as a disease that was caused by consuming liquor, characterized by a loss of control and treated by nothing other than total abstinence. This paradigm became the foundation of the Temperance Movement and popularized the disease nature of alcoholism, namely that its victims were stripped of their autonomy and no longer had control of their drinking.

The disease model came to even greater prominence in the 1930s and 1940s with the development of AA and Jellinek’s work at the Yale Center of Alcohol Studies (Levine, 1978). With the alleged empirical backing of Jellinek’s (1946) study of the phases of alcoholism AA rapidly increased in popularity (Fingarette, 1990; Levine, 1978). Jellinek’s (1946) study provided empirical evidence of the sequential progression of alcoholism that starts with initial experiences of intoxication, follows several stages (e.g., blackouts, weekend drunks, daytime drunks), and eventually leads to a complete loss of control over drinking. The last stage is said to occur when the alcoholic hits his/her “lowest point” (Jellinek, 1946, p. 46), which is personally defined and can vary from being incarcerated to disgracing one’s family. This work

seemed to provide empirical support for the message AA had already been promulgating. Yet, critics point out that this study consisted of questionnaires developed by, and conducted on, 100 self-selected AA members (Fingarette, 1990). Indeed, Jellinek (1946) humbly expressed these limitations of his work. Nonetheless, this hopeless portrait of the inevitable stages of the diseased alcoholic was now given a “scientific foundation” that has been promulgated by AA advocates and has played a key role in its enormous popularity (Fingarette, 1990).

The broader societal trends of the 20th century also facilitated the shift to a medicalized model of addiction. Within the established Enlightenment worldview, a medicalization of mental illness and deviant behavior in general was taking place; thus, the societal conditions were ripe for the entrance of the disease concept of addiction (Foucault, 1965; Rothman, 1971). Within the amenable social conditions, physicians such as Benjamin Rush were able to lay the paradigmatic groundwork after which temperance and AA advocates provided the initiative and Jellinek (1946, 1960) produced the alleged scientific credibility. Thus, a new model of alcoholism was born (Levine, 1978; Schaler, 2000). Tracking the historical emergence of the disease model of addiction, Levine (1978) concluded his article:

The invention of the concept of addiction, or the discovery of the phenomenon of addiction ... can be best understood not as an independent medical or scientific discovery, but as part of a transformation in social thought grounded in fundamental changes in social life—in the structure of society. (pp. 165-166)

Indeed, many consider the disease concept of addiction a reified social construction, employed not as a result of medical evidence but rather as a social convenience (e.g., Chiauzzi & Liljegren, 1993; Douglas, 1986; Fingarette, 1988; Lester, 1989; Levine, 1978; Schaler, 2000; Szasz, 2007). The question therefore arises, how useful is such a conceptualization of addiction?

The Utility of the Disease Concept

The utility of the disease model of addiction is a contentious issue. In the past, the issue has been so contentious that Harvard Medical School's *Mental Health Review* featured a debate on the matter in which the views of two prominent and opposing voices (Vaillant and Fingarette) in the field were discussed (Grinspoon & Bakalar, 1990; cited in Martin, 1999). Critics of the disease model, such as Fingarette, assert that telling alcoholics they have a disease is problematic and harmful as it strips them of their sense of agency over their own behavior, removes the onus of personal responsibility for one's behavior, and fosters a sense of learned helplessness (Chiauzzi & Liljegren, 1993; Fingarette, 1988). More recently, others have similarly castigated the manner in which modern science (and medical professionals) has "promoted the view that people are not free to choose or control their actions" (Vohs & Baumeister, 2009, p. 231). These authors continue, stating that as soon as someone misbehaves or acts irresponsibly, "science stands by them ready to supply supportive rationalizations [for their misdemeanors]" (Vohs & Baumeister, 2009, p. 232). The force of this critique is aptly summed up by one critic—and ex-alcoholic—who asserted that if addicts could not actually control their behavior (as the modern disease model suggests) then "AA would be useless [and] nothing short of death could stop [the alcoholic] from drinking" (Carr, 2012, "How I Stopped Drowning in Drink," para. 7). The notions of agency and responsibility are central to this side of the argument; they also constitute the chief concern of this thesis and will be further discussed later.

From the other perspective, the chronic disease model of addiction is considered to be an important advancement in addiction therapy (Maxwell, 1984; Milam & Ketcham, 1981). It is lauded as the key to removing the blame and stigma commonly associated with the traditional morality-based model of addiction, by moving its conceptualization of the problem from the

moral realm into the medical one (Maxwell, 1984). They assert that a medical conceptualization of addiction brings freedom from the traditional moral models in which alcoholics were depicted as weak-willed, sinful, criminal, and morally flawed (Gordis, 1995; Jones & Helrich, 1972). From this perspective it is thought that telling someone their problematic behavior is due to a chronic disease frees them from blame, shame, guilt, and stigma (Milam & Ketcham, 1981); as a former director of the NIAAA commenting on this progress put it, the “disease concept ... has helped remove the stigma from a chronic disorder that is no more inherently immoral than diabetes or heart disease” (Gordis, 1995, p. 5). The notions of stigma and blame are central to this side of the argument; they constitute a secondary investigation of this thesis and will be discussed later.

The contentious debate over the appropriateness of the disease concept of addiction has been going on for well over a century (Levine, 1978). Yet, there are two recent developments in the field of addiction that have brought this debate to the fore and call into question the validity and usefulness of defining addiction as a chronic disease: (a) the recent longitudinal data that has renewed the understanding of the course of alcoholism and (b) the re-defining of addiction in the most recent DSM-5 (American Psychiatric Association [APA], 2013a).

Abandoning Traditional Misconceptions: An Empirical View of Alcoholism

The modern understanding of the course of alcoholism has been that of a chronic, relapsing addiction that begins with harmless recreational drinking and ends with the addict losing everything: job, house, family, and friends (e.g., Jellinek, 1960; Milam & Ketcham, 1981). Research developments in the last decade, however, have demonstrated that such dire beliefs of the lifelong alcoholic are unfounded (Dawson et al., 2007; Moss, Chen, & Yi, 2007; NIAAA, 2010; Willenbring, 2010).

The largest epidemiological study on alcohol-related disorders to date has challenged the stereotype that anyone and everyone caught in the grips of alcohol dependence follows a fateful destiny (Moss et al., 2007). This NIAAA-funded study followed over 43,000 participants for 3 years, providing the first nationally representative data on the remission rates of alcoholism (Dawson et al., 2007; NIAAA, 2010). The study found that the majority of alcoholics, about 72% of people carrying an alcohol-dependence diagnosis (based on DSM 4-TR), only had one episode of dependence that lasted, on average, 3.7 years (Hasin et al., 2007; Willenbring, 2010). Of these, about half developed dependence by 19-20 years of age and almost all fully recovered by the age of 25. The other half or so of this group tended to develop a mild to moderate dependence in their mid-30s and overcame the addiction within a few years. Thus, for this majority of alcoholics, alcohol addiction was not a chronic disease. Furthermore, the majority (75%) of alcoholics (i.e., with a diagnosis of alcohol dependence) overcame their addiction without the help of treatment (Dawson et al., 2005).

It is precisely because these alcoholics typically do not seek treatment that their profiles have been overlooked in addiction research. In the previously discussed NIAAA study, only 16% of those meeting criteria for substance dependence were currently in treatment (Stinson et al., 2005) and only 26% had ever received any treatment (Dawson et al., 2005); among those with a current alcohol abuse diagnosis, only about 3% had received treatment (Hasin et al., 2007). Moreover, this study broadly defined treatment to include support from religious leaders, self-help and community groups, employee programs, and various others (Stinson et al., 2005).

Another large national survey of psychiatric disorders has demonstrated that only about 30% of people with a substance use disorder even mention this to a health specialist, let alone seek treatment for it (Anthony & Helzer, 1991). Yet, those with a more severe chronic

dependency, on the other hand, were much more likely to seek treatment (66%) than those with a shorter length of dependency (9%; Moss et al., 2007). Therefore, the traditional clinical picture of alcoholism has been based on only a small fraction—the most severely afflicted—of those meeting diagnostic criteria for alcohol dependence (Moss et al., 2007; Willenbring, 2010).

This minority of severely afflicted alcoholics make up roughly 28% of all alcoholics (Hasin et al., 2007; Willenbring, 2010). The prevalence of such chronic alcoholics is even less when considered within the broader population of problem drinkers—those considered at-risk of an alcohol use disorder—who vastly outnumber chronically dependent alcoholics at a rate of 21:1 (Hasin et al., 2007; Willenbring, 2010). Thus, it is only for a small minority that addiction tends to be a chronic and relapsing disorder. Yet, it is this small minority that has shaped the image of the clinical alcoholic as a “falling-down booze-hound: an older person, usually male, staggering down the street and clutching a brown paper bag. A pathetic image, hopeless and depraved” (Knapp, 1999, pp. 12-13).

Studies on alcohol dependence have typically been confined to this minority of severely afflicted individuals (Heyman, 2009; Willenbring, 2010). This has biased the findings of hundreds of studies, including the foundational study of alcoholism by Jellinek (1946), and has led to the prevalent stereotypic conception of the lifelong alcoholic (Willenbring, 2010). These most severely impaired addicts are the ones who are most likely to seek treatment, the least likely to recover, and the most likely to endure a chronic course of addiction (Heyman, 2009; Willenbring, 2010). This is thought to be because of a well-known bias in the medical field, called Berkson’s bias (Berkson, 1946; Heyman, 2009; Maric et al., 2004). This refers to a type of sampling bias, in which patients in treatment are more likely to suffer from additional mental or medical disorders that are independent of the disorder in question, because their more intense

suffering motivates them to seek treatment. Therefore, their substance addiction is more severe, but this is due to its interaction with other independent illnesses, not solely from alcohol addiction (Heyman, 2009; Willenbring, 2010). Indeed one goal behind NIAAA's recent mammoth epidemiological study on the general population was to correct the "serious problem" of relying purely on clinical samples that do not accurately represent the continuum of alcohol disorders within the population of substance abusers (Grant, Stinson, Dawson, Chou, Ruan et al., 2004, p. 361).

Epidemiological studies on the broader population have found that, out of everyone who meets diagnostic criteria for an alcohol use disorder, those in treatment settings are significantly more likely to have other non-substance-related psychiatric diagnoses (Grant, Stinson, Dawson, Chou, Dufour et al., 2004; Hasin et al., 2007; Stinson et al., 2005). For example, while 41% of those seeking treatment for a substance use disorder had an independent mood disorder and 33% had an independent anxiety disorder, only around half as many (19% and 17%, respectively) of people with a substance use disorder in the general population had such comorbid disorders. Those who seek treatment are also much more likely to be addicted to other substances, such as cannabis, cocaine, or methamphetamine (Stinson et al., 2005). Additionally, those in treatment settings are much more likely to have a lot of other risk factors that influence their addiction, such as growing up in chaotic family circumstances, coming from families with several generations of alcohol dependence, exhibiting behavioral problems early in life and developing antisocial personality disorder as adults, and starting to drink at a very early age (early to mid-teens; Moss et al., 2007; Willenbring, 2010).

Even though this group of severely ill individuals represents the minority of alcoholics and a small fraction of all people with an alcohol addiction, it is their profiles that have led to the

disease concept of addiction. In his review, Heyman sums this up: “Although their [the severe minority of alcoholics] histories differ from those of most addicts, their biographies are the ones that have informed clinical texts and expert opinion on the nature of addiction” (2009, p. 85).

DSM-5: Expanding the Definition of Addiction

The recent changing definition of substance disorders in the DSM-5 (APA, 2013a) is also an important development in considering the disease concept of addiction. Until the release of the DSM-5, the distinction between an alcoholic—someone with a physiological dependency and thus the disease of addiction—and a non-dependent alcohol abuser was made in order to determine who was afflicted with the disease (e.g., Jellinek, 1960; Milam & Ketcham, 1981). This was reflected in the different DSM-4-TR diagnoses of Alcohol Abuse and Alcohol Dependence (APA, 2000). However, the revisions of the recent DSM-5 have eliminated these diagnostic distinctions, combining the two disorders, and relegating any information of dependence to a specifier of the diagnosis. With the revisions a diagnosis of alcohol dependence has changed from requiring 3 out of 7 possible symptoms (DSM-4) to only 2 out of 11 symptoms (DSM-5; Wakefield, 2013). This is conceptually important, as it changes the definition of alcoholic from someone physiologically dependent on alcohol to someone with maladaptive substance use. One reason for this redefinition was to reflect the severity of alcohol abuse among some non-physiologically dependent individuals, sometimes comparable to those diagnosed with alcohol dependence (APA, 2013b; Jones et al., 2012). As a result, alcoholics will no longer be classified by the presence of signs of dependence, but rather by the extent to which alcohol abuse is harming their life.

This redefining of alcohol addiction has been met with much controversy (Martin, Steinley, Verges, & Sher, 2011; Mewton, Slade, McBride, Grove, & Teesson; Wakefield, 2013).

A critical review by Martin et al. (2011) describes the manner in which this new definition of addiction is overly lenient and will pathologize normal or mildly impairing behavior. They point out that several of the 11 symptoms are not accurate indicators of pathology: tolerance to alcohol, which commonly develops without consequence and is particularly normative among adolescents and young adults; substance use in hazardous situations, which is often reflective of carelessness rather than addiction; and the two symptoms indicating lack of control, which are frequently misunderstood or inappropriately endorsed for other social reasons (e.g., conforming to social norms). Indeed, they have demonstrated that this new classification scheme diagnoses those with the moderate consumption of an average of 3.79 ($SE = 0.09$) standard drinks per drinking day. Furthermore, only a minority of those diagnosed (28%) engaged in binge drinking (five or more drinks) at least once a week (Martin et al., 2011). These authors conclude their critique by stating that “a 2/11 threshold means that many diagnosed cases may have mild levels of substance involvement and no meaningful pathology” (p. 2008).

Others have conducted epidemiological estimates to demonstrate this increase in prevalence. For instance, Mewton et al. (2011) investigated epidemiological data from the 1997 Australian National Survey of Mental Health and Well-Being ($N = 7,746$) and found that diagnoses would increase from 6.0% (1.9% alcohol abuse and 4.1% alcohol dependence) to 9.7% (alcohol use disorder), which effectively diagnoses a tenth of the population with mild to severe alcoholism. Others have argued that the previous alcohol abuse diagnosis itself was problematic, over-pathologizing normal behavior, stating that alcohol dependence is a more valid measure of substance problems (Martin et al., 2011). Employing this logic, these authors found that the prevalence of alcohol dependence would increase from 5.0% (DSM-4) to 12.4% (DSM-5; Martin et al., 2011). These estimates point to the weighty influence of these changes, which

suggest that this expanded definition of addiction could include up to several million more North Americans than before.

This redefining of addiction is most controversial concerning college students and has sparked a public outcry. For example, in one article titled “DSM-5 Could Categorize 40% of College Students as Alcoholics,” TIME magazine decries the effects of this change (Szalavitz, 2012). A main concern is over-labeling and stigmatizing individuals, particularly college students, with mild, temporary, and intermittent alcohol-related problems (Frances, 2010). With the new definition, most¹ (if not all) of the 38% of college students who qualify for an alcohol abuse or dependence diagnosis based on DSM-4-TR (Knight et al., 2002) will now be labeled as alcoholics (viz., alcohol use disorders) and encouraged to “see themselves as having a chronic, relapsing disease that requires a lifetime of attendance at 12-step meetings” (NIAAA, 2010). Such a disease label is suggested to be both misleading and harmful (Frances, 2010).

Up until this point I have tried to contrast some of the facts surrounding addiction from popularly held beliefs. The goal in this is to illustrate that the disease concept of addiction is not an empirically supported fact, but rather a construct rooted in social and ideological assumptions. This is not to say that its role as a social construct has not been important; it has been instrumental in shifting the concept of the addict from a careless perpetrator to a more humane view of a person who is in need of help (e.g., Milam & Ketcham, 1981). Yet, the question remains whether the disease concept of addiction may have unintended harmful consequences and if it still remains a useful construct; this is especially so for the majority who have a

---

¹ The exact number meeting DSM-5 criteria cannot be calculated given that the criteria for substance use disorder requires the endorsement of one more symptom than the DSM-4-TR diagnosis of alcohol abuse and one less than for alcohol dependence, and also needs to include the new symptom regarding craving as part of the overall criteria.

relatively less severe and non-chronic alcohol use disorder. Proponents of the disease model argue that it is vitally needed to reduce the blame and stigma associated with alcohol use disorders (e.g., Milam & Ketcham, 1981; NIDA, 2013). However, these long-held beliefs, that a chronic disease conceptualization will remove blame and reduce stigma, reflect mere conjecture, which may seem logical, but some have argued it to be “unverified” and “actually contradicted by some research” (Mehta & Farina, 1997, p. 407). Some of these empirical “contradictions” will now be discussed.

Reconsidering the Disease Concept

One main goal of biological conceptualizations of mental disorders is to reduce the blame and stigma associated with personal responsibility for acquiring the disorder (Corrigan & Watson, 2004). The traditional assumption has been that, by conceiving of alcoholism as resulting from a biological abnormality and thus something largely outside of a person’s control, it absolves the addict of responsibility for the disorder (e.g., Milam & Ketcham, 1981; NIDA, 2013). This is an important function given that alcohol addiction is an especially stigmatized disorder, more so than other mental disorders (e.g., Corrigan, Kuwabara, & O’Shaughnessy, 2009; Schomerus, Lucht et al., 2011). Therefore, the main question to examine is not whether alcoholics are a stigmatized group but, rather, what are the aspects that are exacerbating and those that are mitigating this stigma.

The question of what minimizes the stigmatization of alcoholism has become a prevalent issue of debate. Many have argued that the disease model is the golden ticket to fighting stigma (e.g., NIDA, 2013; Vaillant, 1995). Others have questioned this logic, arguing that labeling drinkers with a chronic, relapsing disease of the brain is demeaning and harmful (e.g., Fingarette, 1988; Douglas, 1986). Given that the disease model of addiction has been heavily popularized

(e.g., Heyman, 2009), the changes in public attitudes can be examined to get a sense of its influence.

When considering the General Social Survey dataset, for the 10-year span from 1996 to 2006, researchers found that disease model popularization efforts had succeeded in increasing the amount of people attributing alcohol addiction to a biological cause from 58% to 68% (Pescosolido, Martin, Long, Medina, Phelan, & Link, 2010). While this did change people's attitudes regarding the alcoholic's need for medical treatment and prescription medication, it did not reduce attributions of blame; in fact, the proportion of people attributing the cause of alcohol addiction to "bad character" also rose, perhaps somewhat paradoxically, from 49% (1996) to 65% (2006). Moreover, the stigma facing alcoholics remained high and unchanged: The majority of people expressed an unwillingness to work closely (74%) or to socialize (54%) with someone who had an addiction to alcohol; alcoholics were also considered to be violent and dangerous (67%) and people did not want them marrying into their family (79%). Another large public survey found that many (48%) view those with an alcohol addiction as a danger to society (Substance Abuse and Mental Health Services Administration [SAMHSA], 2008).

One reason for the perseverance of a strong sigma, despite the majority of the public having adopted a disease model, may be that the modern conception of an alcoholic is that of a hopeless, chronically relapsing addict. For instance, in one study when participants were asked to describe an "alcoholic," the majority (73%) described him/her as a "skid row habitué" (Dean & Poremba, 1983, p. 748). The majority of this sample held to a disease view of alcoholism and their conception of an alcoholic was that of a homeless and lifelong addict, commonly described as "unshaven," "physically dirty," and "unkempt" (Dean & Poremba, 1983, p. 748). Negative associations with the disease label (in relation to alcoholism) have likewise been shown to

subconsciously permeate people's attitudes. Researchers using an implicit associations test to ascertain people's subconscious feelings towards the label found that people had a significant negative bias towards the "disease" label (Hayes, 2002). This negative bias was found to be as severe as the negative biases associated with other stigmatized terms in the study, such as "addict," "alcoholic," "drug addiction," "relapse," and "drunk."

A second possible reason that the disease concept may be stigmatizing is that a biological cause is often thought to imply unpredictability (Jorm & Oh, 2009). When mental illness is considered to be a result of a biological cause, the afflicted are more likely to be viewed as not being in control of their behavior (Jorm & Oh, 2009; Read & Law, 1999). This tends to increase public perceptions of unpredictability and dangerousness (Read & Law, 1999). Yet, when mental illnesses are conceptualized as arising from a psychosocial cause, people are less fearful of those with psychiatric conditions (Read & Law, 1999). This is because, within a psychosocial conception, the symptoms of mental illness seem to be better understood as relatively normal reactions to environmental stressors that are beyond one's control; in contrast, the causal link of mental illness arising from a brain abnormality seems less clear and is more likely to be perceived as erratic and therefore frequently construed as abnormal and dangerous (Lam, Salkovskis, & Warwick, 2005; Read & Law, 1999).

A third possible reason for the stigma associated with the chronic brain disease model is the perception that it is unchangeable (Phelan, 2005). Corrigan and Watson (2004) point out that, in the effort to reduce stigma among the mentally ill, many focus on reducing the blame associated with the onset of the disorder, yet the stigma related to the perception of the offset of the disorder needs to also be considered. While emphasizing the brain disease etiology of the disorder may reduce blame regarding the onset of alcohol dependence (Mehta & Farina, 1997),

this conception can increase stigma associated with the offset of the disorder (Corrigan & Watson, 2004). In one interesting example of this, participants were found to verbalize less blame towards a former psychiatric patient who was described as having had a past biological disease (compared to one described with a past psychosocial disorder), yet they also treated them harsher (viz., administered harsher shocks in a learning task; Mehta & Farina, 1997). This study, at the very least, demonstrated that consciously expressed blame does not equate to the actual blame-related discrimination that face those with mental illness. Moreover, the authors speculate that people, convinced they should not blame the sick for failing to measure up, may also insidiously begin to believe that people with brain diseases are “like children,” incompetent of living independently and therefore needing to be “treated firmly” (Mehta & Farina, 1997, p. 416). This type of thinking, however well-intentioned, is the stepping stone to paternalism.

This concern is arguably most problematic in regards to alcohol-addicted college students. Given that up to 38% of college students qualify for an alcohol use disorder (Knight et al., 2002), there is concern as to the harmful effects that a chronic brain disease label may have (e.g., Frances, 2010). It seems that this might be problematic in at least two regards. First, it sets up the false expectation that alcohol addiction is a chronic disease, characterized by uncontrollable alcohol use. The state of the matter is that (a) 72% of alcoholics who drink during their college-aged years only go on to have one lifetime episode of dependence, which tends to last less than 4 years (Hasin et al., 2007); (b) almost all of those who start drinking and develop a dependence in their college years (18-24)² permanently overcome this addiction within a few

---

² It is those individuals who start drinking in their early and mid-teens and continue throughout their college years who tend to develop more chronic forms of alcohol addiction (Moss et al., 2007).

years (Hasin et al., 2007; Moss et al., 2007); (c) the majority in this age group (73%) strongly believe alcohol addiction can be overcome; and (d) the majority (77%) also believe that a full recovery is possible (SAMHSA, 2008). Given these facts, many argue it is unwise, perhaps even unethical, to undermine their strong sense of responsibility and capability to control drinking with a chronic disease label—which does not even reflect reality (e.g., Chiauuzzi & Liljegren, 1993; Fingarette, 1988; Heyman, 2009; Schaler, 2000). Since most college-aged adults think they can and do overcome alcohol addiction, it seems reasonable to think that promulgating notions of a chronic and compulsive disease, which “hijacks” self-control, will only serve to build false maladaptive expectations and weaken an already strong sense of drinking self-efficacy.

A second, and opposite problem, with promulgating the disease model to college-aged adults, is that many will fail to identify with such a severe profile and overlook their need for help (Knight et al., 2002; Shaw, Cartwright, Spratley, & Harwin, 1978). Only a small minority of college students (1.7% of those with alcohol abuse and 6.2% with alcohol dependence) seek help during these especially heavy drinking years (Knight et al., 2002). This failure to seek treatment may stem from not wanting to identify with a stigmatized chronic disease or, alternatively, their very real alcohol-related problems may go unrecognized, as they do not match the severe, chronic alcoholic profile. Indeed, only a quarter of alcohol-dependent college students consider themselves a “heavy or problem drinker” and only about half consider their drinking to be a problem, while almost no students with alcohol abuse problems endorsed either of these notions (4.3% and 16.4%, respectively; Knight et al., 2002). Regardless of the reason that help-seeking is hindered, the result remains the same: The serious alcohol abuse problem, in which over 50% college-aged adults engage in binge drinking (Tsai, Ford, Li, Pearson, & Zhao,

2010) and up to 38% have an alcohol use disorder (Knight et al., 2002), will continue to go largely unresolved. Of course, there are similar concerns regarding the vast majority (72%) of mild to moderate alcoholics, for whom alcohol addiction does not represent a chronic disease (Dawson et al., 2005; Hasin et al., 2007; Willenbring, 2010).

Up until now I have provided an empirical case against the appropriateness of the chronic brain disease conceptualization of addiction. This is a long-standing debate that has its roots in a time when relatively little was known regarding the typical course of alcohol addiction (Levine, 1978). In recent decades, however, psychological science has made much progress in understanding the typical course of alcoholism. It is now time to follow the advice of Mark Willenbring (2010), past NIAAA director of treatment and recovery, to move beyond early models of treatment which relied on “anecdotal findings and assumptions” and have now “been proven wrong, [or] at best incomplete” (p. 55); only then, will we be able to build an understanding of alcohol addiction that is “based firmly on evidence” (p. 56).

Therefore, it seems that the traditional debate concerning the disease concept of addiction has resurfaced with renewed vigor as empirical findings have demonstrated that: (a) the understanding of the typical alcoholic has changed from a severely dependent skid-row alcoholic to a mildly or moderately dependent high-functioning alcoholic (e.g., Willenbring, 2010); (b) the traditional belief that physiological withdrawal effects, resulting from dependence, play the strongest role in maintaining addiction has been rejected (Leshner, 1997); (c) the majority of physiologically dependent alcoholics are able to quit drinking within a few years ($M = 3.7$ years; Dawson et al., 2005; Hasin et al., 2007); (d) the majority of alcoholics (72%) successfully reduce or stop drinking without the help of any professional treatment or lay support group (Dawson et al., 2005; Willenbring, 2010); (e) the recent redefining and expanding of the definition of alcohol

addiction to include non-dependent alcohol abusers (DSM-5; APA, 2013a); and (f) the empirical evidence that calls into question the long-held assumption that a chronic disease conceptualization minimizes stigma (e.g., Jorm & Oh, 2009).

Given that the disease concept of addiction has been traditionally employed to reduce stigma for those severely dependent—perhaps those for whom alcohol addiction does represent a form of chronic, relapsing disease—and given the fact that we now know that such alcoholics represent the minority (25%) of alcohol dependent individuals (Hasin et al., 2007), the question arises: How does the disease concept affect the self-conceptions of the majority (72%) of people with alcohol dependence who do not have a chronic dependency? Given their relative lack of a severe and chronic addiction, might the disease label have harmful consequences? Does the disease label threaten their sense of agency that is so instrumental in helping this majority of less severe alcoholics to quit or reduce their drinking?

This thesis seeks to address these questions by evaluating the consequences of promulgating the disease model of addiction. This will be done by examining the impact of the disease model on mild to moderate alcoholics' sense of agency and personal control over their substance consumption. Several such indices that have been put forth as instrumental to addiction recovery are: (a) personal autonomy, (b) locus of control, (c) coping style, (d) self-efficacy, and (e) entity or incremental views of the disease. The literature regarding the influence of these factors will now be discussed.

Overcoming Addiction: The Role of Agency and Related Factors

The question of human free will, let alone free will for a substance-dependent individual, is a weighty and much debated matter. While theorizing concerning free will is typically dealt with in the realm of philosophy, it is within psychology that we must turn to answer the

empirical question: How does one's conceptualization of their substance addiction impact their personal sense of agency over the problem? Critics of the medicalization of alcohol addiction have argued that the disease model threatens a person's sense of agency. They assert that telling alcoholics that their behavior is a direct result of an entity outside of themselves, namely a disease that is out of their control, removes any sense of personal responsibility, negatively affects attributions and future expectations, and promotes learned helplessness (e.g., see Chiauuzzi & Liljegren, 1993, for a review; Cook, 1988; Fingarette, 1988; Szasz, 2007; Zeldman, Ryan, & Fiscella, 2004). While there has been a wealth of theorizing and speculation, such claims have not received any direct empirical investigation. Surprisingly, given such a controversy, no study has yet, to my knowledge, tested the causal effects of the disease label of addiction. Yet, there is a large literature that discusses the importance of a personal sense of agency in human behavior.

One form of agency supposed to be vital to treatment success is to take personal responsibility for developing the addiction. The importance of this is reflected in professional opinions regarding the therapeutic mechanisms of change, as well as in empirical findings among alcoholics. In terms of the former, the majority of addiction professionals endorses the importance of taking responsibility for one's addiction. For instance, the large survey that was discussed earlier (Morgenstern & McCrady, 1992), declaring the prevalence of the disease model in contemporary treatment settings, also reviewed professionals' opinions regarding the most effective therapeutic mechanisms implicated in treatment success. In order to accomplish this, the authors of this survey first reviewed and identified 35 treatment processes or therapeutic mechanisms described across the addiction treatment literature. They then had doctoral-level professionals rank the importance of each of these treatment processes. It was found that professionals did not attribute much importance to the therapeutic factor of having addicts accept

their addiction as a disease (it ranked 28th of 40 therapeutic factors), whereas taking responsibility for change was rated as the overall most important factor for positive change. Similarly, other therapeutic mechanisms that ranked highly concerned agentic notions emphasizing the person's active participation in overcoming addiction, such as: preparing to deal with relapse, boosting confidence in ability to control drinking behavior, and developing effective coping mechanisms (Morgenstern & McCrady, 1992). Similar opinions regarding the primary importance of taking responsibility for change in drinking behavior and weaker concern over accepting notions of disease and powerlessness over alcohol have also been found among Canadian mental health professionals (Ogborne et al., 1998).

In terms of empirical investigation among alcoholics, one study found that alcoholic inpatients who took personal responsibility for their addiction by attributing its cause to personal factors tended to have better treatment success as well as improved psychological functioning (Kingree, Sullivan, & Thompson, 1999). On the other hand, those in this study who attributed their addiction to external (social) causes were less likely to complete the treatment program and more likely to be depressed. Such uncontrollable social attributions of responsibility would seem to parallel those inherent in the disease concept of addiction in which responsibility is attributed to an external cause (the disease) that is beyond one's control. Indeed, the influence of adopting personal responsibility (i.e., having an internal locus of control) for one's behavior is a strong and well-established determinant of behavior across many contexts (e.g., Wallston & Wallston, 1978). In a similar vein, it has been found that active coping (i.e., taking responsibility for one's substance dependence problem) as opposed to avoidance coping (i.e., failing to take responsibility) positively predicted better drinking outcomes 2 and 8 years post-treatment (Moos, Finney, & Cronkite, 1990). Indeed, the most commonly self-claimed factor of successfully

overcoming an addiction is taking responsibility and exercising self-control (Maisto, O'Farrell, Connors, McKay, & Pelcovits, 1988).

Another agentic factor related to overcoming alcohol addictions is a sense of internal motivation. One unpublished study found that internal motivation, governed by a personal desire for recovery (as opposed to rewards or punishment), was the best predictor of treatment adherence and abstinence (Wilde, Cunningham, Roberts, & Ryan, 2004; cited in Zeldman et al., 2004). Similar findings have emerged among smokers (Williams, Gagne, Ryan, & Deci, 2002) and individuals who are morbidly obese (Williams, Grow, Freedman, Ryan, & Deci, 1996). Indeed, when reflecting on which factors were most influential in helping them remain abstinent 30 months post-treatment, individuals with alcohol addiction identified personal motivation to change as the most important factor and treatments such as AA as considerably less important (i.e., ranked 15th out of 30; McKay, Maisto, & O'Farrell, 1996). Furthermore, another study found that just being in an environment that promotes internal forms of motivation increases addicts' treatment success (Zeldman et al., 2004).

Does the Disease Concept Promote or Hinder a Sense of Personal Agency?

The importance of a sense of personal agency in overcoming addiction is evident, yet it remains uncertain as to whether the disease model hinders or promotes such feelings of autonomy. One exploratory study attempting to address this question employed the entity–incremental conceptual framework to study the addiction-related self-conceptions of heroin addicts (Zeldman et al., 2004). This scheme, originally applied to conceptions of intelligence, distinguishes between the entity view of intelligence, in which the individual views intelligence as largely stable and unchangeable with effort, compared to the incremental view, in which

intelligence is seen as malleable and can be increased with effort (Blackwell, Trzesniewski, & Dweck, 2007).

In their research, Zeldman et al. (2004) extended this scheme to compare addicts whose self-conceptions tended to resemble either an entity view of their addiction, namely a stable disease entity that could not be changed with effort, or an incremental view of addiction, namely a personological variable that could be influenced by effort. They compared heroin addicts receiving methadone treatment along with supportive group sessions and individual counseling at an outpatient clinic. It was theorized that holding an incremental view would be more adaptive, as it spurs motivation and effort while protecting from learned helplessness in a manner comparable to other domains of behavior (e.g., intelligence).

The extent to which these heroin addicts held an entity or incremental view of their addiction was assessed using an addiction-specific version of the entity–incremental framework, and was then correlated with various treatment outcome measures. An entity view was associated with attendance at support group or individual counseling sessions ($r = -.28, p < .05$), treatment progress (as rated by staff; $r = -.24, p < .05$), and a lower likelihood of relapse ($r = -.23, p < .05$). While it was expected that an incremental, rather than entity, view would prove to be adaptive, the authors theorize that holding an entity view seemed to prompt participants to the need for treatment, which was subsequently related to improved attendance and outcomes. Yet, these relationships are tenuous. After controlling for two other significant predictors of outcome success (employment status and obligatory/voluntary referral), only attendance remained significantly ($p = .05$) associated with an entity view. Interestingly, individuals who were unable to maintain employment and/or whose treatment enrolment was obligatory (mandated by the

courts or social services), both predictors of relapse ($r = -.31$ and $.24$; $p < .01$ and $< .05$, respectively), were more likely to hold an entity (disease) view of their addiction.

It also needs to be emphasized that this was a sample of individuals with a long-term addiction to heroin—a highly addictive substance—for which addiction may possibly be labeled a chronic relapsing disease. The severity of addiction of these heroin users is vastly different than the majority of people addicted to alcohol (and perhaps even early-stage heroin addicts): Participants, on average, were middle-aged ($M = 41$ years) and unemployed (78% unemployed), had been using the illicit drug for 21.4 years, been in treatment for over 1.5 years, and had already tried quitting with, on average, 3.6 other addiction treatments. This is a profile that might resemble the minority (9%) of severely dependent long-term alcoholics (Moss et al., 2007) but is much more chronic and severe than the vast majority of people with an alcohol use disorder. Furthermore, a core aspect of treatment involved daily medical intervention (viz., methadone injections), something that is more reflective of a disease treatment paradigm and which is generally not recommended for the majority of people with alcohol addiction (Zeldman et al., 2004). These authors noted the exploratory nature of their study and emphasized the need to replicate and extend their work to other areas of addiction.

To date, this exploratory work on heroin addiction remains the closest examination of the disease concept's influence on addicts' self-conceptions and subsequent substance use behaviors. Another line of research that can shed some light on this issue concerns the importance of having a free will perception of one's own actions.

The Consequences of Removing a Perception in Free Will

One of the ways the disease model of addiction is thought to influence addicts' sense of autonomy is by undermining their belief in free will. To test the consequences of this,

experimental studies have been designed to examine the impact of manipulating a person's perceived free will. Research into the general views on free will demonstrates that people typically hold a free will view of the world, in which they feel they are in control of their actions and freely choose their behavior (Vohs & Baumeister, 2009).

This line of research has developed methods to manipulate a person's sense of free will by having them read research articles or statements that portray either free will or deterministic perspectives of the world (Vohs & Baumeister, 2009). Several studies have shown that leading people to have a more deterministic view of the world does, in fact, negatively influence their behavior. For example, it can make people lazier, as people led to believe they were not in control of their behavior exhibited poorer objectively measured work performance (Stillman et al., 2010). It also loosens individuals' moral restraints: Experimentally manipulating individuals to believe they are not in control of their behavior can make people more aggressive toward innocent others, less helpful toward needy strangers (Baumeister, Masicampo, & DeWall, 2009), and more likely to cheat (Vohs & Schooler, 2008). Likewise, promoting a deterministic view of behavior causes people to loosen their attribution of blame and punishment towards serious criminals, such as convicted child molesters and rapists (Carey & Paulhus, 2013).

In addition to being more lax in their judgments on misbehavior, people who themselves misbehave also gravitate to a deterministic view of their behavior. For instance, when participants were asked to reflect upon past wrongdoings, they preferred to hold a deterministic view of this past behavior (Vohs & Baumeister, 2009). These authors state that a deterministic view of one's own inappropriate behavior reduces feelings of guilt and culpability; in other words, when individuals do something wrong they want to be told that they did not have a choice in the matter and that it was not their fault. Put another way, "disbelieving in free will provides

an excuse for one's own misbehavior" (Carey & Paulhus, 2013, p. 132). Thus, conceptions that downplay the role of free will (such as the disease concept) may seem quite attractive to addicts (Vohs & Baumeister, 2009).

Disease concept proponents argue that absolving the alcoholic of personal responsibility is essential to reducing blame and harmful feelings of guilt. Yet, the uncomfortable feelings of guilt may represent boon as well as bane. For instance, university students with more intense feelings of guilt over a past shameful behavior were more likely to learn from it ($\beta = .40, p < .001$; Stillman & Baumeister, 2010). Of particular note is that the extent to which they learned from their past was dependent upon whether they were led to believe that they were or were not responsible for (i.e., in control of) their behavior. Only those led to internalize their personal responsibility for the misbehavior were able to translate their negative emotion into learning a "great deal" from the incident ($M = 6.8$ on a 7-point Likert scale; Stillman & Baumeister, 2010, p. 954); comparably less was gained among those led to believe their behavior was out of their control ($M = 3.8$).

A follow-up study went beyond assessing learning to seeing if the combination of feelings of guilt and a sense of personal responsibility would promote behavior change (Stillman & Baumeister, 2010). They found that participants who were made to feel guilty about their role in socially irresponsible behavior (i.e., environmentally destructive behavior) and were led to internalize a sense of personal responsibility for these behaviors had a greater intention to act and rectify the situation (i.e., volunteer). However, no such intention occurred when individuals were led to believe they were not responsible for (i.e., could not control) their behavior. Thus, both a sense of personal responsibility for one's behavior and the accompanying feelings of guilt

were found to be essential to learning from past irresponsible behavior and mustering up an intention to change.

In summarizing their work on the importance of maintaining a belief in free will and its particular relation to addiction, Vohs and Baumeister (2009) recommend a model of addiction that does not undermine a person's tendency to believe in their free will. Yet, the question remains: Does promoting a disease conception of addiction reduce a person's disposition to believe in the freedom of his/her actions? Does it reduce a sense of personal agency?

To date, the link between the disease concept of addiction and disbelief in free will and personal agency has not been directly examined. While much theorizing and related work has brought the matter to the fore, this remains an empirical question that demands experimental insight. It seems that disease-model proponents rely heavily on assumptions and intuition, rather than science. They argue it reduces stigma and increases hopefulness, although such presuppositions have not been empirically demonstrated; in fact, as has been discussed in the literature review, there is evidence to suggest this might be misguided. This is disconcerting, and particularly so, given that their theories and assumptions are often promulgated in the name of "science" (e.g., NIDA, 2013). Critics of the disease model, on the other hand, also rely on unsupported assumptions, theorizing that the disease model undermines a sense of agency and personal responsibility. Neither of these claims, that the disease model improves stigmatism or undermines agency, have, to my knowledge, received sufficient direct empirical backing.

To properly address this question, an experimental study is needed. A randomized experimental design will allow me to elucidate the causal relationship between belief in the disease model and notions of personal agency and stigma. Being able to determine the causal relationship between these notions is vital to understanding the effects of popularizing a disease

conception of addiction. Employing a randomized experimental design is the only sure way to navigate through all the related, but entangling, confounding variables in these complex relationships. It is the best way to adequately answer the question at hand: How, exactly, does the disease concept of addiction affect the important notions of agency and stigma? While the primary analyses of this study center around experimental methods, secondary correlational analyses were also used to elucidate relationships between addiction beliefs and various factors, such as demographics, personality traits, various attitudinal beliefs, and addiction severity.

Research Design

Within this experimental study, belief in the disease model of addiction is the independent variable that was manipulated by randomly assigning participants to one of three conditions, in which they read and (hopefully) internalized statements that (a) promoted the disease model, (b) promoted an alternate psychosocial model,³ or (c) were geographical facts (a neutral intervention). This experimental manipulation is an adaptation of Velten's (1968) manipulation, which has been used with much success in the belief-in-free-will studies (e.g., Vohs & Baumeister, 2009). The goal of such a manipulation was to simulate the manner in which individuals become convinced (as a result of hearing claims made by addiction authority figures) that addiction is either a chronic disease or a psychosocial problem. In an effort to do so, the statements were taken from two authoritative texts which expound the arguments promoted by theorists from each side of the debate (Milam & Ketcham, 1981, for the disease model; Fingarette, 1988, for the psychosocial model).

---

³ For various theories on the reasons people pursue destructive drinking habits, see Fingarette (1988), Heyman (2009), and Szasz (2007).

Following reflection upon these statements participants completed a measure of disease and psychosocial model addiction beliefs, which served as a manipulation check. Participants then completed several measures of agency: (a) personal autonomy (i.e., belief in one's own free will), (b) locus of control, (c) coping style, (d) self-efficacy, and (e) entity or incremental view of addiction. Assessing the effects of belief in the disease model of addiction on personal agency was the chief goal of this thesis. A secondary goal was to assess the extent to which the disease model reduces feelings of shame and stigma. To accomplish this, participants completed measures assessing these constructs.

The main hypothesis of this study is that a manipulated belief in the disease model will be associated with a reduced sense of personal agency. This would consist of those led to believe in the disease model having lower scores on self-reported (a) belief in free will, (b) internal locus of control, (c) drinking-specific self-efficacy, (d) approach coping, and (e) a higher entitization of addiction. Likewise, it is hypothesized that manipulating a belief in the psychosocial model will be associated with a stronger sense of agency, as assessed by these same five measures of agency. It is important to note that three of these measures (viz., internal locus of control, drinking-specific self-efficacy, and entitization of addiction) tap facets of agency that directly concern addiction-specific behavioral beliefs; on the other hand, the remaining two measures (viz., belief in free will and approach coping) tap facets of agency that are broad in scope and concern general behavioral beliefs (see the Measures section for example items from each of these scales).

In terms of shame and stigma, my hypothesis is less clear. While disease proponents put forth compelling theories to suspect the disease model reduces stigma, the empirical work on the matter has failed to assuredly bear out these intuitions and suggests it may actually be harmful

(Jorm & Oh, 2009). Yet, it is also apparent that, at this point in time, the majority of the public does not endorse a purely disease explanation of addiction and thus the benefits of a disease model may be hidden (Pescosolido et al., 2010). Thus, I am charting murky waters, which need to be explored, but are difficult to predict with any certainty.

CHAPTER 2: METHOD

Participants

A community sample of people with alcohol use disorders was recruited. A community sample, rather than a clinical sample, was recruited for several reasons. The first reason concerns societal implications. Surveys have shown that the majority (75%) of individuals who have an alcohol use disorder do not receive treatment and will not be found in a treatment setting (Dawson et al., 2005). Yet, it is this population with less severe addictions who constitute the majority of people with an alcohol addiction (Willenbring, 2010); as a result, this population poses the larger alcohol-related burden to society, costing substantially more than the less numerous amount of people with severe alcohol dependence (e.g., due to drunk driving, missed work, domestic violence; Institute of Medicine, 1990; Moore & Gerstein, 1981). Therefore, it is imperative to see how this proportion of people with mild to moderate alcohol use disorders is influenced by societal promulgation of the disease concept of addiction.

The second reason for studying a community, rather than clinical, sample of people with alcohol use disorders is conceptual in nature. Given that the disease paradigm is currently the predominant North American model espoused in addiction treatment programs, the majority of those currently receiving treatment will have been taught (and possibly indoctrinated) to think of their addiction as a chronic disease. (Indeed, for about 25% of those with alcohol dependence, this may be an apt description [Willenbring, 2010]). For the purposes of this study (on mild to moderate addiction), however, those who have been taught by a professional espousing the disease model (or any other model) are more likely to be resistant to our manipulation of belief in/against the disease concept. It was thought that those who have received little or no professional contact for their alcohol addiction may possess a relatively neutral view of their

addiction, which may be more amenable to our manipulation of belief in the disease concept of addiction.

Participants were recruited from an online survey-based website, administered by Amazon.com and called Mechanical Turk (MTurk; for the quality of such data see Buhrmester, Kwang, & Gosling, 2011). A recent study has demonstrated that MTurk provides overall high quality data even among clinical and subclinical populations (Shapiro, Chandler, & Mueller, 2013). In terms of alcohol use problems, previous research has indicated that a large proportion (37%) of MTurkers screened positive for possible alcohol abuse or subclinical alcohol abuse and about 9% reported being heavy drinkers (females drinking more than 7 and males more than 14 drinks per week; Shapiro et al., 2013). Furthermore, only a small minority (4%) reported ever seeking treatment. It was also found that MTurkers are comfortable, even more so than in in-person interviews, in disclosing clinically significant information online, possibly because it ensures them anonymity (they do not provide identifying information when they sign up for an account, but are rather identified with an ID number; see Paolacci, Chandler, & Ipeirotis, 2010). Therefore, this provides an ideal means by which to reach a sample of people who have a non-chronic alcohol addiction and have not been exposed to addiction treatment.

Participants aged 18 years and older were recruited from the United States. Based on best-practice recommendations (Shapiro et al., 2013), a preliminary screener (Wave 1) was used to ensure a high-quality sample. In this screener, participants completed a short demographic measure to assess if they meet inclusion criteria for the study: (a) meeting criteria for a DSM-5 mild or moderate alcohol use disorder and (b) never having received alcohol-related treatment. The first criterion was assessed using the Modified Semi-Structured Assessment for the Genetics of Alcoholism II (SSAGA II), which is a structured diagnostic measure that accords with DSM-5

diagnoses. The DSM-5 defines a mild alcohol use disorder as endorsing two or three of the specified 11 symptoms and a moderate alcohol use disorder as endorsing four or five symptoms. The population of interest to this study was individuals with a non-severe alcohol use disorder and, therefore, we do not distinguish between mild and moderate severities in our study. The second criterion was assessed by one question that queries participants' previous engagement in any form of alcohol-related treatment (e.g., professional talk therapy, self-help groups, medication). Only participants with no type of alcohol-related treatment experience were included in the study.

Participants received a \$0.30 honorarium for filling out this screener, taking up to 15 minutes. Those who met inclusion criteria were contacted to see if they were interested in participating in a two-part study (Waves 2 and 3). Those who participated in Waves 2 (about 45 minutes) and 3 (about 30 minutes) received a \$2.00 honorarium (per wave), which is a pay rate sufficiently higher than average (\$1.38 per hour; see Horton & Chilton, 2010) to maximize the response rate. About 2 weeks after completing Wave 2, participants were contacted for their involvement in Wave 3. Those who participated in Wave 3 completed most of the same measures as in Wave 2 to assess the extent to which the changes induced by the manipulation were sustained over time. As another precaution to maximize the quality of the data, quality control items were included at random parts in each of the surveys (two items in Wave 1 and three items in each of Wave 2 and Wave 3). Each of these items stated that "this is a quality control item" and asked participants to demonstrate their active involvement by choosing a particular multiple-choice response. Only participants who answered all of these quality control items correctly were included in the analyses.

A total of 1934 participants started the Wave 1 questionnaire. Participants were first asked several demographic questions after which they were asked whether they presently consume alcohol on a regular basis or had done so in the past. Participants who had not regularly consumed alcohol at any point in their life were directed to the end of the survey, bypassing the remainder of the survey which consisted of the SSAGA II diagnostic measure.

The Wave 1 survey included two quality control items. The first was located at the end of the demographics section, right before the questions assessing alcohol consumption; the second quality control item was located at the end of the SSAGA II diagnostic measure. Three participants were excluded for failing the first quality control item. The second quality control item was only answered by participants who completed the SSAGA II (i.e., those who reported present or past regular alcohol consumption). Participants who reported not ever having consumed alcohol regularly skipped the second quality control item as a result of bypassing the SSAGA II diagnostic assessment. Of those who answered the second quality control item, one participant answered the item incorrectly and was excluded. Furthermore, of those commencing the SSAGA II diagnostic assessment, 36 participants (2% of the sample) failed to complete the survey in its entirety and were also excluded. This resulted in a sample of 1894 participants who met quality control standards. See Table 1 for sample demographics.

Table 1

Wave 1 Sample Demographics (N = 1894)

Variable	<i>M</i>	<i>SD</i>
Number of DSM-5 alcohol use disorder symptoms	0.87	2.44
Age	31.81	10.38
Political Orientation	-0.83	1.57
Variable	<i>n</i>	%
Political Orientation		
Liberal (-3, -2, -1)	1118	59%
Neutral (0)	411	22%
Conservative (+3, +2, +1)	365	19%
Religion		
A religious person	579	31%
Not a religious person	826	44%
An atheist	489	26%
Sex		
Male	1084	57%
Female	810	43%
Ethnicity		
African American/Black	133	7%
Asian American/Asian	203	11%
Caucasian/White	1423	75%
Hispanic/Latino	97	5%
Native American	10	1%
Other	28	2%
Marital Status		
Cohabiting or married	734	39%
Divorced	99	5%
Single, never married	1043	55%
Widowed	18	1%
Education		
Some high school	21	1%
High school diploma	186	10%
Some college/university	840	44%
Bachelor degree	688	36%

Grad/professional degree	159	8%
Income		
Less than \$20,000	769	41%
\$20,000 - \$39,999	523	28%
\$40,000 - \$59,999	320	17%
\$60,000 - \$79,999	166	9%
\$80,000 or more	116	6%
Health ^a		
Mental illness diagnosis by a professional	576	30%
Received therapy for mental illness	448	24%
Non-psychiatric chronic illness/physical disability	219	12%
Most common type of alcoholic beverage(s) consumed ^a		
Beer/cider or equivalent	1090	58%
Distilled/hard alcohol	1365	72%
Wine	656	35%
Liqueur/cocktail	479	25%

---

^aCategories are not mutually exclusive.

Participant scores were then evaluated for study inclusion criteria. The first criterion assessed whether participants met alcohol addiction criteria for a mild to moderate alcohol use disorder. Participants were first asked whether they had ever consumed alcohol and whether this constituted a regular pattern of consumption. A total of 759 (40%) participants had not regularly consumed alcohol at any point in their life and a subset of these, 148 (8%) participants, reported never consuming any alcohol in their lifetime. In total, 1133 participants had regularly consumed alcohol and completed the entirety of the Wave 1 questionnaire. Their SSAGA II scores were then examined to determine which participants met the first inclusion criterion of a mild to moderate alcohol use disorder.

Of the initial sample of 1894 participants who met quality control standards, 177 (9%) met this first criterion of a mild to moderate alcohol use disorder. Of those who did not meet this criterion, 1624 (86%) did not qualify for an alcohol use disorder and 93 (5%) qualified for a

severe alcohol use disorder (see Table 2 for a comparison of demographic information between each of these three groups).

Table 2

Comparisons of Demographic Data Across Addiction Groups

	Alcohol Use Disorder Symptoms		
	No	Mild or Moderate	Severe
Variable	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
Number of DSM-5 alcohol use disorder symptoms	0.12 (0.32)	3.01 (1.10)	10.00 (3.94)
Age	32.00 (10.65)	30.41 (8.45)	31.15 (8.62)
Political Orientation	-0.79 (1.58)	-1.06 (1.56)	-1.03 (1.35)
Variable	<i>n (%)</i>	<i>n (%)</i>	<i>n (%)</i>
Political Orientation			
Liberal (-3, -2, -1)	938 (58%)	117 (66%)	61 (68%)
Neutral (0)	364 (22%)	29 (16%)	18 (19%)
Conservative (+3, +2, +1)	322 (20%)	31 (17%)	12 (13%)
Religion			
A religious person	523 (32%)	38 (22%)	18 (19%)
Not a religious person	704 (43%)	78 (44%)	44 (47%)
An atheist	397 (24%)	61 (35%)	31 (33%)
Sex			
Male	912 (56%)	121 (68%)	51 (55%)
Female	712 (44%)	56 (32%)	42 (45%)
Ethnicity			
African American/Black	118 (7%)	10 (6%)	5 (5%)
Asian American/Asian	179 (11%)	18 (10%)	6 (7%)
Caucasian/White	1209 (74%)	136 (77%)	78 (84%)
Hispanic/Latino	82 (5%)	11 (6%)	4 (4%)
Native American	10 (1%)	0 (0%)	0 (0%)
Other	26 (2%)	2 (1%)	0 (0%)

Marital Status

Cohabiting or married	649 (40%)	52 (29%)	33 (36%)
Divorced	79 (5%)	12 (7%)	8 (9%)
Single, never married	881 (54%)	111 (63%)	51 (55%)
Widowed	15 (1%)	2 (1%)	1 (1%)

Education

Some high school	16 (1%)	3 (2%)	2 (2%)
High school diploma	157 (10%)	16 (9%)	13 (14%)
Some college/university	705 (43%)	82 (46%)	53 (57%)
Bachelor degree	605 (37%)	61 (35%)	22 (24%)
Grad/professional degree	141 (9%)	15 (9%)	3 (3%)

Employment

Currently unemployed	285 (18%)	27 (15%)	19 (20%)
Employed Part-time	299 (18%)	24 (14%)	25 (27%)
Employed Full-time	673 (41%)	84 (48%)	32 (34%)
Student	338 (21%)	41 (23%)	16 (17%)
Retired	29 (2%)	1 (1%)	1 (1%)

Income

Less than \$20,000	645 (40%)	77 (44%)	47 (50%)
\$20,000 - \$39,999	445 (27%)	52 (29%)	26 (28%)
\$40,000 - \$59,999	280 (17%)	30 (17%)	10 (11%)
\$60,000 - \$79,999	150 (9%)	8 (5%)	8 (9%)
\$80,000 or more	104 (6%)	10 (6%)	2 (2%)

Health^a

Mental illness diagnosis by a professional	452 (28%)	67 (38%)	57 (61%)
Received therapy for mental illness	348 (21%)	54 (31%)	46 (50%)
Non-psychiatric chronic illness/disability	180 (11%)	24 (14%)	15 (16%)

Most common type of alcoholic beverage(s) consumed^a

Beer/cider or equivalent	878 (54%)	137 (77%)	75 (81%)
Distilled/hard alcohol	400 (25%)	85 (48%)	44 (47%)
Wine	568 (35%)	58 (33%)	30 (32%)
Liqueur/cocktail	404 (25%)	50 (28%)	25 (27%)

Note. 0-1 symptoms = no alcohol use disorder; 2-5 symptoms = mild to moderate alcohol use disorder; 6 or more symptoms = severe alcohol use disorder.

^aCategories are not mutually exclusive.

The second inclusion criterion assessed participants' addiction-specific treatment history. Participants were only included if they were not or had not engaged in alcohol-specific talk-related therapies or medical treatment. Of the initial sample of 1894 participants who met quality control standards, 128 (7%) engaged in alcohol-specific talk-related therapies (e.g., A.A., psychotherapy) and 26 (1%) received alcohol-specific medical treatment. These participants were not included.

In total, 159 (8%) participants met both of our inclusion criteria. See Table 3 for demographic information.

Table 3

Demographics of Participants Meeting Study Inclusion Criteria (N = 159)

Variable	<i>M</i>	<i>SD</i>
Number of DSM-5 alcohol use disorder symptoms	3.02	1.11
Age	29.84	8.14
Political Orientation	-1.63	1.05
Variable	<i>n</i>	%
Political Orientation		
Liberal (-3, -2, -1)	107	67%
Neutral (0)	26	16%
Conservative (+3, +2, +1)	26	16%
Religion		
A religious person	34	21%
Not a religious person	57	36%
An atheist	68	43%
Sex		
Male	106	67%
Female	53	33%
Ethnicity		
African American/Black	9	6%

Asian American/Asian	14	9%
Caucasian/White	125	79%
Hispanic/Latino	9	6%
Native American	0	0%
Other	2	1%
Marital Status		
Cohabiting or married	49	31%
Divorced	10	6%
Single, never married	99	62%
Widowed	1	1%
Education		
Some high school	3	2%
High school diploma	14	9%
Some college/university	75	47%
Bachelor degree	54	34%
Grad/professional degree	13	8%
Employment		
Currently unemployed	24	15%
Employed Part-time	20	13%
Employed Full-time	74	47%
Student	40	25%
Retired	1	1%
Income		
Less than \$20,000	70	44%
\$20,000 - \$39,999	46	29%
\$40,000 - \$59,999	27	17%
\$60,000 - \$79,999	7	4%
\$80,000 or more	9	6%
Health ^a		
Mental illness diagnosis by a professional	57	36%
Received therapy for mental illness	45	28%
Non-psychiatric chronic illness/physical disability	20	13%
Most common type of alcoholic beverage(s) consumed ^a		
Beer/cider or equivalent	121	76%
Distilled/hard alcohol	78	49%
Wine	52	33%
Liqueur/cocktail	46	29%

---

^aCategories are not mutually exclusive.

Procedure

An ad describing the study (Wave 1) as a “psychology study on people’s beverage preferences” was posted on Mechanical Turk. Given the importance of minimizing demand characteristics on MTurk (e.g., Shapiro et al., 2013), this advertisement was sufficiently vague as to not reveal much regarding the purpose of the study, while still attracting respondents who consume at least some alcohol. The study was advertised as a “short survey on beverage preferences, will take 15 minutes or less.”

Upon signing up for Wave 1 of the study, participants were given a link that directed them to the start of the screener. Participants filled out a short measure assessing demographics and an assessment of alcohol use disorders (SSAGA II; Bucholz et al., 1994). Upon completion of this measure, participants were provided with a link to NIAAA’s support and treatment website (NIAAA, n.d.-b) which provides the appropriate information to access alcohol-related help across the United States, and were thanked and paid for their participation. Those participants who met the study’s inclusion criteria were contacted within 8 weeks of the first study to participate in Wave 2. These participants were identified by their MTurk ID number and then contacted by MTurk for a prompt to join the Wave 2 study.

Those who agreed to participate in Wave 2 were given a prompt that included the study title and description which enabled them to sign up through MTurk. Participants were then provided with a link that directed them to the start of the experiment. All participants first completed three preliminary scales (satisfaction with life, belief in a just world, and personality traits) that have been shown to correlate with factors that might influence belief in a disease model of addiction. Belief in a just world has been found to be related to belief in free will (Carey & Paulhus, 2013), whereas personality traits (Bogg & Roberts, 2004) as well as

satisfaction with life (Koivumaa-Honkanen et al., 2012) have been found to be associated with level of alcohol use.

Upon completion of these initial measures, participants were randomly assigned to one of three conditions in which they read and (hopefully) internalized statements that either (a) promoted the disease model of addiction (disease condition), (b) promoted the psychosocial model (psychosocial condition), or (c) were geographical facts (neutral condition). These statements are provided in the Appendix. This manipulation was based on the procedure presented by Velten (1968), in which participants read and reflected on 15 statements, one statement per page. Since the study involves online participation, participants were also told at the beginning of the manipulation, that at the end they would be asked to answer a few (open-ended) questions regarding the statements they just read. The reason for this was threefold: (a) to ensure they maintained engagement, (b) to ensure that the participants understood the statements, and (c) to serve as a manipulation check. Only participants who correctly answered at least three of the four questions, including Question 4, were included in the analyses (questions and answers are provided in the Appendix).

This procedure has been successfully used several times in a comparable manner to experimentally induce change in a person's belief in free will (e.g., Baumeister et al., 2009; Stillman & Baumeister, 2010; Vohs & Baumeister, 2009).⁴ While the purpose of these studies was to investigate the effects of decreasing belief in free will, the purpose of the present study was to manipulate participants' belief in the disease model of addiction. Following the

---

⁴ This specific manipulation has not yet been conducted online; however, other comparable types of experimental manipulation (viz., the Prisoner's Dilemma, priming, and framing experiments) have been shown to work just as effectively on MTurk as they do in the lab (Horton, Rand, & Zeckhauser, 2011).

manipulation participants completed a first set of questionnaires that included the Alcohol Belief Scale (ABS; Schaler, 2000) and a series of Addiction Belief Summary Questions (ABSQ), which assessed the extent to which participants in each of the groups endorsed and identified with various alcohol addiction beliefs. The former (i.e., ABS) served as the main measure of addiction beliefs as well as the manipulation check, whereas the latter (ABSQ) was used for exploratory purposes. The ABS provides an assessment of belief in both the disease and the psychosocial models of addiction, while the ABSQ provides a broader assessment of other related alcohol addiction beliefs (viz., types of causes, extent of self-control, reasons for consumption, and identification with the addiction). Additionally, the ABSQ provides an in-depth assessment regarding possible terminological or semantic differences that might confound results. These two indices of belief in the disease and psychosocial models were presented in a randomized order.

After reading through the 15 statements and completing the ABS and the ABSQ, participants then completed a second set of questionnaires. This second set included multiple questionnaires that were presented in a randomized order to prevent any order effects. While the order of these varied between participants, each participant filled out all of the measures listed above (belief in free will, locus of control, coping style, drinking self-efficacy, entitization of addiction, and alcohol-addiction related feelings of perceived general as well as internalized stigma and state shame). Upon completing these measures participants were provided with the same NIAAA support and treatment website (NIAAA, n.d.-b) as in Wave 1, and were thanked and paid for their participation.

All the participants from Wave 2 were contacted (by MTurk) about 2 weeks later and sent a prompt to participate in Wave 3 of the study. For Wave 3, participants completed all of

the same measures (again in randomized order) that they did in Wave 2; the two surveys were the same with the exception of the experimental manipulation and manipulation check questions, which are exclusive to Wave 2. The purpose of Wave 3 was to assess the extent to which all the changes induced by the experimental manipulation in Wave 2 would be sustained a couple weeks later.

Wave 2 and 3 Response Rates

The 159 participants who completed the Wave 1 measures and met inclusion criteria were contacted about 8 weeks after their completion of Wave 1 and prompted to participate in Wave 2. Of the 159 participants who were prompted, 104 completed Wave 2 (response rate = 65%). Of these participants, three failed a quality control item and nine failed the manipulation check, leaving a Wave 2 sample of 92 participants (i.e., disease: $n = 34$; psychosocial: $n = 30$; control: $n = 28$). Participants were then prompted again 2 weeks later to participate in Wave 3. Of the 104 respondents who completed Wave 2, 91 completed Wave 3 (response rate = 88%). Of the Wave 3 participants, 10 failed a quality control item, leaving a Wave 3 sample size of 81 participants. Minor differences in sample sizes among each condition (i.e., disease: $n = 29$; psychosocial: $n = 28$; control: $n = 24$) resulted from uneven response rates across conditions as well as uneven discarding of data that failed to meet quality control measures.

Measures

The reliability analyses of the SSAGA II were based on 105 participants who completed two trials of the instrument. The reliability analyses of the rest of the measures were calculated on the 91 participants from Wave 2 (viz., the Addiction Belief Scale, the Addiction Belief Summary Questions, the Satisfaction with Life Scale, the Global Belief in a Just World Scale, the Ten-Item Personality Inventory, the Free Will and Scientific Determinism Plus Scale, the

Multidimensional Health Locus of Control Scale, the Brief Approach/Avoidance Coping Questionnaire, the Controlled Drinking Self-Efficacy Scale, the Alcohol Addiction Entitization Scale, the Perceived Devaluation–Discrimination Scale, the Self-Stigma of Mental Illness Scale, and the State Shame and Guilt Scale).

Demographic Measures. Demographic items included participant age, sex, ethnicity, place of residence, marital status, education, employment status, income, treatment status, political orientation, religiosity, and types of alcoholic beverages most often consumed. Treatment status consists of five categorical questions (viz., *yes/no*) that assess participation in any past or present alcohol- or non-alcohol-related treatment, as well as lifetime mental and physical illness diagnostic history (as indicated by a professional). Political orientation was assessed using one item (i.e., “Describe your political orientation”) rated on a 7-point scale ranging from -3 (*strongly liberal*) to 3 (*strongly conservative*; Frimer, Biesanz, Walker, & MacKinlay, 2013). Religiosity was measured using a single item (i.e., “Independently of whether you attend religious services or not, would you say you are ...”), which has three possible responses: “a religious person,” “not a religious person,” or “an atheist.” This question has been frequently used in large scale surveys to assess religiosity (World Values Survey, 2011).

Modified Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA II). Items from the SSAGA II⁵ (Bucholz et al., 1994; Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999) were used to assess alcohol use disorders (based on DSM-5) in Wave 1. While the SSAGA II is a structured diagnostic interview assessment, its items have been used

---

⁵ The SSAGA II has been provided by the Collaborative Study on the Genetics of Alcoholism, supported by NIH Grant U10AA08401 from the NIAAA.

before as a self-report questionnaire to assess the prevalence of alcohol use disorders (e.g., Knight et al., 2002). Furthermore, self-reports of alcohol use are generally considered to be quite reliable and compare well to in-person interviews (Babor, Kranzler, & Lauerman, 1989; Winters, Stinchfield, Henly, & Schwartz, 1990). As a diagnostic assessment, the original SSAGA II has demonstrated strong test-retest reliability for assessing alcohol abuse and dependence ($\kappa = .84$; Bucholz et al., 1994) and good sensitivity (92%) and specificity (72%; Hesselbrock et al., 1999). These same authors found that the SSAGA II has adequate concordance with the Schedule for Clinical Assessment in Neuropsychiatry ($\kappa = .63$) in assessing alcohol dependence. While the original SSAGA II assessed substance use disorders according to the DSM-3, the second edition of the measure was updated to assess diagnostic criteria for the DSM-4. The items used from the SSAGA II were those that reflect the current DSM-5 alcohol use diagnostic criteria. Thus, the items assessing the criterion of alcohol-related legal problems were omitted and items assessing the additional DSM-5 criterion of alcohol-related craving were added (APA, 2013a; see Table 4 for the DSM-5 diagnostic criteria for alcohol use disorder).

Table 4

DSM-5 Alcohol Use Disorder Diagnostic Symptoms

A problematic pattern of alcohol use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. Alcohol is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control alcohol use.
3. A great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects.
4. Craving, or a strong desire or urge to use alcohol.
5. Recurrent alcohol use resulting in a failure to fulfill major role obligations at work,

school, or home.

6. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.
7. Important social, occupational, or recreational activities are given up or reduced because of alcohol use.
8. Recurrent alcohol use in situations in which it is physically hazardous.
9. Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol.
10. Tolerance, as defined by either of the following:
  - i. A need for markedly increased amount of alcohol to achieve intoxication or desired effect.
  - ii. A markedly diminished effect with continued use of the same amount of alcohol.
11. Withdrawal, as manifested by either of the following:
  - i. The characteristic withdrawal syndrome for alcohol.
  - ii. Alcohol (or a closely related substance, such as a benzodiazepine) is taken to relieve or avoid withdrawal symptoms.

Note. Diagnostic criteria were adapted from the DSM-5 (APA, 2013a).

Additionally, the endorsement of at least two symptoms will be required to meet the criteria for a DSM-5 alcohol use disorder. A mild alcohol use disorder is defined as endorsing two or three alcohol use symptoms. A moderate alcohol use disorder is defined as endorsing four or five alcohol use symptoms. A severe alcohol use disorder is defined as endorsing six or more alcohol use symptoms. In order to qualify for a current alcohol use disorder these symptoms need to have occurred within the last 12-month period. This study examines individuals with a current mild to moderate alcohol use disorder and therefore inclusion criteria (described in the Participants section above) requires participants to endorse between two and five alcohol use disorder symptoms within the last 12-month period.

In the present study, the test-retest reliability of the SSAGA II was assessed by a subset of participants ($n = 105$) who completed the assessment twice.⁶ This occurred as the result of a programming error in which some participants were unintentionally allowed to participate in the Wave 1 study twice. These data were then used to assess the test-retest reliability of the SSAGA II. Participants completed the Wave 1 study for the second time, on average, about 3 weeks ($SD = 1.97$) later. Test-retest reliability was assessed using both Cohen's kappa and intraclass correlation. Reliability using Cohen's kappa was poor ($\kappa = .31$). This low reliability can be explained, in part, by the low base-rate of participants with a mild to moderate alcohol use disorder. For instance, the first SSAGA trial found only 16 participants (15%) who qualified for a mild to moderate alcohol use disorder. It is for this reason that intraclass correlation was also used to assess reliability. Intraclass correlation was used to assess the stability of the SSAGA II in diagnosing the total number of alcohol use symptoms across the two points in time. Reliability was found to be adequate with an intraclass correlation of .80.

Satisfaction with Life Scale. Satisfaction with life was measured using the 5-item Satisfaction with Life Scale (Diener, Emmons, Larsen, & Griffin, 1985) on which items are rated using a 7-point Likert scale ranging from 1 (*strongly disagree*) to 7 (*strongly agree*). Higher scores demonstrate greater satisfaction with life. It has also been shown to correlate with other measures of subjective well-being (Diener et al., 1985). An example item is "In most ways my life is close to my ideal." This scale has good internal consistency ($\alpha = .87$) and test-retest

---

⁶ A total of six participants who completed the Wave 1 questionnaire twice also completed Wave 2 and 3. Of these, one was in the disease condition, two were in the psychosocial condition, and three were in the control condition. Independent t -tests found no significant differences between these individuals and their respective condition means in terms of their ABS scores ($0.16 < ts > 1.82$, $.08 < ps > .87$). Their data was therefore not excluded from Wave 2 and 3.

reliability ($r = .82$; Diener et al., 1985). In the present study, internal consistency was found to be $\alpha = .92$.

Global Belief in a Just World Scale. Belief in a just world was assessed using Lipkus's (1991) 7-item Global Belief in a Just World Scale on which items are rated on a 6-point Likert scale, ranging from 1 (*strong disagreement*) to 6 (*strong agreement*). Higher scores demonstrate a stronger belief in a just world. An example item is "I feel that people get what they are entitled to have." This scale has demonstrated good reliability ($\alpha = .83$; Lipkus, 1991). This scale has been shown to significantly correlate with other measures of belief in a just world (Lipkus, Dalbert, & Siegler, 1996). In the present study, internal consistency was found to be $\alpha = .91$.

Ten-Item Personality Inventory. The Big Five personality traits were measured using the Ten-Item Personality Inventory (Gosling, Rentfrow, & Swann, 2003) on which participants rate how well each trait describes them on a 7-point Likert scale ranging from 1 (*disagree strongly*) to 7 (*agree strongly*). Higher scores demonstrate stronger agreement with the personality statement. An example item is "I see myself as extraverted, enthusiastic." Each of the five traits has a subscale that consists of two items that are summed. This scale has demonstrated sufficient test-retest reliability ($r = .72$) as well as strong correlations with each of the five personality factors in the 44-item Big-Five Inventory ($r_s = .65-.87$; Gosling et al., 2003). In the present study, internal consistency for all the subscales was found to be between $\alpha = .56$ (agreeableness) and $\alpha = .85$ (neuroticism).

Addiction Belief Scale (ABS). The ABS was developed to assess the strength of belief in the disease model of addiction (Schaler, 1995, 2000). It consists of nine items designed to assess the strength of belief in the disease model of addiction and nine reverse-score items designed to assess belief in the psychosocial model of addiction. Using a 5-point Likert scale,

participants rate each item from -2 (*strongly disagree*) to +2 (*strongly agree*), with higher scores indicating stronger belief in the disease model and lower scores indicating stronger belief in the psychosocial model. The ABS is a highly face-valid questionnaire that includes items such as “Addicts cannot control themselves when they drink or take drugs” and “Addiction has more to do with the environment people live in than the drugs they are addicted to.” The overall scale has strong reliability with an $\alpha = .91$ (Schaler, 1995).

This measure was slightly modified to replace the term “addict,” which has negative connotations of severe addiction, to “people with an alcohol addiction.” While the DSM-5 uses the term “alcohol use disorder” rather than “addiction,” the latter is a term commonly used in the public sphere (e.g., NIDA, 2012, “What is Drug Addiction?” para. 1). For the purposes of this study, the scale was divided into two 9-item subscales, one characterizing each model of addiction. This was done by using the nine items tapping belief in the disease model of addiction as one subscale and using the nine reverse-score items tapping belief in the psychosocial model of addiction as another subscale. These two subscales, rather than the whole scale, were used as independent measures of the disease and psychosocial models because past surveys (e.g., Lawrence et al., 2013; Pescosolido et al., 2010) have indicated that people often do not think of these models as diametrically opposite, but rather often endorse a combination of both. In the present study, internal consistency was found to be $\alpha = .77$ for the psychosocial subscale and $\alpha = .85$ for the disease subscale.

Addiction Belief Summary Questions (ABSQ). Thirteen items, rated on a 7-point scale, from -3 (*strongly disagree*) to +3 (*strongly agree*), were used to assess participants’ views on eight issues; the extent to which participants: (a) view alcohol addiction as resulting from a brain disease, (b) view alcohol addiction as resulting from social or psychological causes, (c)

view alcohol addiction as resulting from a weak will or lack of self-control, (d) consider someone addicted to alcohol to be in control of their drinking, (e) identify with having an alcohol problem and/or disease of addiction themselves, (f) consider their alcohol problem/disease of addiction to be a severe problem, (g) expect to overcome their current (or if currently not addicted, then a potential future) alcohol problem and/or disease of addiction, and (h) identify with a need to seek treatment. A multiple-choice question also assessed the reason(s) participants engaged in heavy drinking. These items served to explore participants' beliefs on these various issues that are central to one's conceptualization of addiction. The ABSQ includes four similar sets of questions which are identical, save referring to addiction in either medical (viz., disease) or non-medical (viz., alcohol problem). These semantic differences were included to assess for potential confounds arising from differences in terminology.

Free Will and Scientific Determinism Plus (FAD-Plus). The FAD-Plus is an updated version of the FAD scale which assesses lay people's beliefs in free will. It consists of 27 items that tap four different views of the world: scientific determinism, fatalistic determinism, randomness, and free will (Carey & Paulhus, 2013; Paulhus & Carey, 2011). The first three are related facets of belief in determinism and the latter, a measure of belief in free will. Participants respond on a 5-point Likert scale ranging from 1 (*totally disagree*) to 5 (*totally agree*). For the current study, the three determinism subscales were reverse-scored, summed along with the free will subscale, and then averaged to form one overall score (with a possible range between 1 and 5), in which higher scores represent a stronger belief and lower scores represent a weaker belief in free will. The FAD-Plus measures broad non-alcohol-specific beliefs of the world. An example item is "People's biological makeup determines their talents and personality." The FAD-Plus has been shown to have adequate reliability for each of the four subscales, with α s of

.69 to .82 (Paulhus & Carey, 2011). It has also demonstrated similar psychometric properties in an online MTurk sample (Paulhus & Carey, 2011). In the present study, internal consistency for the overall scale was found to be $\alpha = .68$ and between $\alpha = .65$ (scientific determinism subscale) and $\alpha = .86$ (fatalistic determinism subscale) for each of the individual subscales. Of most concern in relation to its validity was whether it represented a distinct construct from the related construct of locus of control. It has been found that there are only moderate relations between the related FAD-Plus and locus of control scores ($.16 < r_s < .49$), and that belief in free will is clearly a distinct construct that encompasses broader dimensions than locus of control (Paulhus & Carey, 2011). The present study found minimal to moderate relations between the related FAD-Plus and locus of control subscale scores ($.02 < r_s < .41$).

Multidimensional Health Locus of Control (MHLC–Form C). The MHLC is a measure of locus of control specifically related to health contexts (Wallston, Stein, & Smith, 1994). This scale consists of 18 items that tap four determinants (subscales) of behavior: internal, chance, and powerful others (further broken into doctors and other people). Using a 6-point Likert scale participants rate the extent to which they 1 (*strongly disagree*) to 6 (*strongly agree*) with each statement. Higher scores indicate stronger agreement with the determinant of behavior tapped by that particular subscale. Form C is a slightly modified version of the original health-related locus of control scale that allows the researcher to tailor its measure to a specific health or medical problem. Within each item is included the statement “my condition,” which is supposed to be replaced by the specific health/medical problem in question (Wallston et al., 1994). For example, the item “I am directly responsible for [my condition] getting better or worse” was modified for this study to be “I am directly responsible for my alcohol use getting better or worse.” The scale has good reliability with α s ranging between .70 (for the 3-item

subscales of “doctors” and “others”) to .87 (for the 6-item “internal” subscale) for each of the subscales (Wallston et al., 1994). The scale has also demonstrated adequate construct validity, correlating moderately with related subscales of the more established MHLC–Form B ($.38 < r_s < .65$; Wallston et al., 1994). For the current study, the chance and powerful others subscales were reverse-scored, summed with the internal subscale, and then averaged to form one overall score (with a range between 1 and 6), in which higher scores indicate a stronger, and lower scores indicate a weaker, internal locus of control. In the present study, internal consistency for the overall scale was found to be $\alpha = .82$ and between $\alpha = .61$ (others subscale) and $\alpha = .83$ (internal subscale) for each of the individual subscales.

Brief Approach/Avoidance Coping Questionnaire (BACQ). The BACQ is a 12-item measure designed to give a short and broad assessment of one’s coping style in response to general life problems and illnesses (Finset, Steine, Haugli, Steen, & Laerum, 2002). The scale includes two sub-categories, one assessing an approach orientation and the other an avoidance orientation. Participants respond on a 5-point Likert scale ranging from 1 (*disagree completely*) to 5 (*agree completely*). Example items include “I like to talk with a few chosen people when things get too much for me” and “I make an active effort to find a solution to my problems.” In the current study, the avoidance subscale was reverse-scored, summed with the approach subscale, and then averaged to form one overall score (ranging between 1 and 5), in which higher scores represent a stronger approach coping style and lower scores represent an avoidance coping style. The BACQ has been shown to have satisfactory psychometric properties. The overall scale has a reliability coefficient of $\alpha = .68$ and it has fairly strong correlations with related measures, such as the Seeking Emotional Social Support ($r = .55$) and Active Coping ($r =$

.47) subscales of the COPE Scale (Finset et al., 2002). In the present study, internal consistency for the overall scale was $\alpha = .70$.

Controlled Drinking Self-Efficacy Scale (CDESES). The CDESES (Sitharthan, Job, Kavanagh, Sitharthan, & Hough, 2003) is a measure of people's confidence that they will be able to control the extent of their drinking in various situations. It is ideal for this study in that it provides a measure of controlled drinking (as opposed to abstinence), which is more common among those with less severe alcohol use disorders. The CDESES is based on the Problem Drinking Self-Efficacy Scale. It consists of 20 items that are rated on a 11-point scale from 0% (*not at all confident*) to 100% (*very confident*) in 10% increments, and asks participants how confident they are in their ability to control their drinking in various settings over the course of the next 6 months, such as, "How confident are you that you can stop drinking alcohol at least three days a week?" The scale has both strong internal consistency ($\alpha = .95$) and 2-week test-retest reliability ($r = .90$; Sitharthan et al., 2003). In the present study, internal consistency was found to be $\alpha = .89$. The CDESES has also demonstrated significant predictive validity, predicting alcohol consumption a year after entering treatment ($r = -.39$; Sitharthan et al., 2003). The CDESES is scored by taking the average of the 20 items, thus providing a total score between 0% and 100%.

Alcohol Addiction Entitization Scale (AAES). The AAES is my adaptation of Dweck, Chi-yue, and Ying-yi's (1995) entity–incremental conceptualization of intelligence to the disease model of addiction. Within this conceptualization, an entity view of addiction is thought to resemble the disease-model paradigm, in which alcohol addiction is something that is viewed as a result of a disease and unable to be improved with effort. On the other hand, the incremental conceptualization embodies a non-disease model in which alcohol addiction can be influenced by

willful effort. I modified items from Dweck and colleagues' (1995) measure to apply them to "alcohol use/addiction." Six questions were developed which participants rate on a 6-point Likert rating scale, ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). Items include "You can learn new strategies, but you can't really overcome your alcohol use/addiction" and "No matter who you are, you can change your alcohol use/addiction a lot." After accounting for reverse-scored items, total scores are calculated, with larger scores representing a stronger belief in the entity view of addiction. A very similar scale was used by Zeldman et al. (2004)⁷ who reported good internal consistency ($\alpha = .86$). In the present study, internal consistency was found to be $\alpha = .77$. While this is a brief measure with relatively little psychometric validation, it represents one current popularly used model (of intelligence), which also holds potential as a model for conceptualizations of alcohol addiction.

Perceived Devaluation–Discrimination Scale (PDD). An alcohol-adapted version of the PDD (Link, 1987) was used to assess how belief in the disease model affects stigma towards people with a past history of alcohol addiction. The PDD consists of 12 items that were slightly modified (replacing "mental patient" with "someone with an alcohol addiction") to assess perceived stigma of alcohol addiction. Items are rated on a 6-point Likert scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). Higher scores are associated with a decreased perception of stigma. It taps expectations of devaluation and discrimination by asking how "most other people" would think and act toward those with an alcohol addiction. An example item is, "Most people would willingly accept someone with a former alcohol addiction as a close friend." This scale has been widely used (e.g., Smith, Dawson, Goldstein, & Grant, 2010) and

---

⁷ Unfortunately, I could not obtain Zeldman et al.'s measure as they no longer had it on file.

has strong test-retest reliability ($\alpha = .82$, Smith et al., 2010; ICC = .93, Ruan et al., 2008), and content and face validity (Luoma, O'Hair, Kohlenberg, Hayes, & Fletcher, 2010). In the present study, internal consistency was found to be $\alpha = .88$. It has also demonstrated good construct reliability in measuring perceived alcohol stigma in samples of alcohol consumers (Glass, Kristjansson, & Bucholz, 2013).

Self-Stigma of Mental Illness Scale (SSMIS). The SSMIS (Corrigan, Watson, & Barr, 2006) assesses the extent to which those with mental illness have internalized stigma. The SSMIS was slightly modified to assess the stigma associated with those who have an “alcohol addiction” rather than those with a more general “mental illness.” While the PDD assesses the extent of stigma associated with a past history of an alcohol addiction, the SSMIS assesses stigma associated with a current alcohol addiction. The SSMIS consists of four subscales that are each 10 items and assess increasingly deeper and more damaging internalizations of stigma. The subscales assess the extent to which people are (a) aware of, (b) agree with, (c) apply, and (d) harmed by stigma. Example items are: “I think the public believes most persons with an alcohol addiction cannot be trusted” (aware subscale), “I think most persons with an alcohol addiction are to blame for their problems” (agree subscale), “because I have an alcohol addiction I cannot be trusted” (apply subscale), and “I currently respect myself less because I am dangerous” (harm subscale). Items are rated on a 9-point Likert scale, from 1 (*I strongly disagree*) to 9 (*I strongly agree*). In the current study, items were summed across the four subscales and then averaged to yield one overall score (ranging between 1 and 9), in which higher scores indicate stronger feelings of stigma. Each of the four subscales has good reliability ($\alpha = .72-.91$) and adequate test-retest reliability ($\kappa = .68-.82$; Corrigan et al., 2006). In the present study, internal consistency for the scale as a whole was found to be $\alpha = .91$ and between

$\alpha = .86$ (agree subscale) and $\alpha = .93$ (aware subscale) for each of the individual subscales. The scale has also demonstrated construct validity, being significantly correlated to other related constructs such as self-efficacy, self-esteem, and depression (Corrigan et al., 2006).

State Shame and Guilt Scale (SSGS). The SSGS assesses in-the-moment (state) feelings of guilt, shame, and pride (Marschall, Sanftner, & Tangney, 1994). Only the shame subscale of the SSGS was used for the current study. It consists of five items that are brief phenomenological descriptions of shame (e.g., “I want to sink into the floor and disappear”) and are rated on a 5-point Likert scale, from 1 (*not feeling this way at all*) to 5 (*feeling this way very strongly*; Tangney & Dearing, 2002). Higher scores indicate a greater sense of shame. The SSGS has good psychometric properties, with good reliability for the shame subscale ($\alpha = .89$; Tracy, Robbins, & Tangney, 2007) and good discriminative validity, being able to distinguish between those who had and had not been induced to feel shame (Tangney & Dearing, 2002). In the present study, internal consistency for the shame subscale was found to be $\alpha = .90$.

Power Analysis

Previous research using this type of Velten-style (1968) manipulation has found that it produces differences in belief in free will (on the original FAD scale) with effect sizes of up to $|d| = 1.2$ and 1.6 (Experiments 1 & 2, respectively; Vohs & Schooler, 2008). Other research has found that this same manipulation produced differences in group mean scores to the question “my actions are due to genetics and my past experiences, and nothing more” of $|d| = .24$ and $.48$ (Experiments 1 & 2, respectively; Stillman & Baumeister, 2010). This manipulation has also been found to produce fairly large group differences in response to a measure of helpfulness ($|d| = .69$; Baumeister et al., 2009).

Since our manipulation is intended to produce changes in belief in the disease model of addiction rather than belief in free will, we can expect to have a weaker effect than the large effect sizes reported ($|d| = 1.2$ and 1.6) by Vohs and Schooler (2008). Yet these previous studies do demonstrate the effectiveness of this type of manipulation. On the other hand, we suspect that mean differences between conditions will have a somewhat larger effect size than those ($|d| = .24$ and $.48$) found by Stillman and Baumeister (2010) who simply assessed the effects of their manipulation with one question (rather than a full scale). The most realistic effect size that we can expect is likely the one reported by Baumeister et al. (2009; $|d| = .69$) as this represents the group mean differences, caused by the manipulation, on a conceptually different measure (helpfulness measure). Therefore, we feel that, given these previous findings, a conservative estimate of the likely effect size produced by our manipulation is $|d| = .50$. With this moderate effect size a sample of $n = 130$ is needed to have a roughly 80% chance of detecting an effect, with a two-tailed hypothesis at $p = .05$.

CHAPTER 3: RESULTS

Overview

The primary purpose of this study was to experimentally assess the different effects of adopting a chronic disease compared to a psychosocial conceptualization of alcohol addiction. Primary analyses employed mixed-model repeated measures ANOVA to assess differences between the three experimental conditions (disease, psychosocial, and control) in terms of personal notions of agency, shame, and stigma. These data were collected at two time-points, at Wave 2 immediately following the intervention and then again, about 2 weeks later, at Wave 3.

Repeated measures ANOVA analyses were first used as a manipulation check, to assess the effects of the experimental manipulation on addiction beliefs (viz., the ABS disease and psychosocial subscales). The main repeated measures ANOVA analyses assessed the influence of experimental condition on five measures of agency (viz., autonomy, FAD-Plus; locus of control, MHLC; entitization of addiction, AAES; coping style, BACQ; and self-efficacy, CDESES) and three measures of stigma and shame (viz., stigma, SSMIS and PDD; shame, SSGS shame subscale). It was hypothesized that those led to internalize a disease model of addiction would experience a decreased sense of agency, whereas those who internalized a psychosocial view would experience an enhanced sense of agency. No hypotheses were made regarding the relationship between manipulated addiction beliefs and feelings of stigma and shame.

Exploratory analyses comparing group differences were also conducted on the ABSQ. Secondary analyses employed bivariate correlational analyses to elucidate the relationships between addiction beliefs and various personality, attitudinal, and demographic variables. It was hypothesized that addiction beliefs might be related to demographic variables such as education and religiosity; to personality traits such as conscientiousness and neuroticism; and to attitudinal

beliefs such as belief in a just world and satisfaction with life. Bivariate correlations were also conducted between these aforementioned variables and alcohol use (viz., number of alcoholic drinks consumed in the past week) and addiction severity (viz., number of addiction diagnostic symptoms).

The Formation of Groups Used for Each Set of Analysis

This study consists of three waves of data collection that each have a different sample size. In order to maximize power, analyses were always conducted on the largest appropriate sample. An outline of which analyses were conducted on which sample will now be provided. Firstly, repeated measures ANOVA analyses of the experimental intervention (i.e., manipulation check and main analyses on agency, stigma, and shame) were conducted on the 81 participants who completed both Wave 2 and Wave 3 questionnaires. This sample size is substantially smaller than that required ($n = 130$), according to our power analysis, for an 80% chance of detecting a moderate effect ($|d| = .50$). While we planned for a final sample size of around 130 participants, this proved to be more difficult than expected, with only about half of our sample of mild to moderate alcoholics (81 out of 159) successfully completing the entirety of the study. Given this lack of power, trends toward significance/marginally significant effects ($p < .10$) are reported. Secondly, correlational analyses examining the relationship between addiction beliefs and various demographic factors were restricted to the 28 participants who were in the Wave 2 control condition. Analyses were restricted to the control group, as this is the only neutral group that remained uninfluenced by the manipulation; likewise Wave 2 (rather Wave 3) participants were used to avoid confounds resulting from practice effects. Thirdly, correlational analyses examining the relationship between addiction beliefs and personality traits, life satisfaction, and just world beliefs were conducted on the entire Wave 2 sample (91 participants), as these

measures were completed prior to the manipulation. Lastly, correlational analyses comparing various demographic variables and addiction severity were conducted using the 1894 participants in Wave 1.

Main Analyses of the Experimental Intervention

A set of mixed-model repeated measures ANOVAs represented the main analyses of this study. In these analyses, experimental condition was the between-groups factor with three levels (disease, psychosocial, and control) and wave was the within-subjects repeated measures factor with two levels (Wave 2 posttest, Wave 3 follow-up). No relevant demographic variables (e.g., gender) were found to be significantly related to addiction beliefs and thus none were included as factors (see the later section, “Correlations Between Demographic Variables and Addiction Beliefs”).

Of particular interest are the main effects for condition, which indicate significant differences across groups, at both Wave 2 and 3. Interaction effects were explored by examining the effects of condition at each wave. Main effects for wave were difficult to interpret and likely represent a sort of practice effect that occurred as a result of completing the survey twice. In some cases, when experimental effects change perceptions of agency or stigma from those held by the control group, it is noted that the treatment changed perceptions from those typically held. In these cases, “typical” represents the common, unaltered view (as demonstrated by the control group), held by most mild to moderate alcoholics.

The mixed-model repeated measures ANOVAs were conducted on the 81 participants who completed both Wave 2 and Wave 3. The goal of these analyses was to examine the effects of the experimental manipulation on (a) belief in the disease and psychosocial models of

addiction, (b) personal perceptions of agency (viz., five indices of agency), and (c) personal perceptions of stigma and shame (viz., three indices of stigma and shame).

Belief in the Disease and Psychosocial Models of Addiction

The experimental manipulation randomly assigned participants to read and internalize statements promoting a disease or a psychosocial model of addiction (or unrelated control group). The effects of this manipulation were examined by assessing participants' scores on the ABS disease and psychosocial model subscales.

In terms of belief in the psychosocial model, a mixed-model repeated measures ANOVA found a significant main effect of condition on such beliefs, $F(2, 78) = 6.82, p < .01$. No significant main effect of wave or interaction was found. Follow-up analyses using Tukey's method ($p < .05$) found that the psychosocial condition effected a significantly stronger belief in this model than the disease condition. The control condition, showed a trend toward being significantly different from each of the treatment conditions, with these differences representing medium sized effects (see Table 5). These analyses demonstrate that the disease condition statements decreased belief in the psychosocial model, that the psychosocial condition statements increased such belief, and that the control condition had little effect. These influences persisted 2 weeks later. This suggests that addiction beliefs are amenable to change, even with brief exposure to a few short statements, and that these are more than momentary changes.

Table 5

Psychosocial Model Beliefs (ABS) Main Effect Follow-up Analyses for Condition

Condition 1	<i>M (SD)</i>	Condition 2	<i>M (SD)</i>	<i> d </i>
Disease	0.10 (0.55)	vs. Psychosocial	0.58 (0.48)	0.93*
Disease	0.10 (0.55)	vs. Control	0.34 (0.61)	0.42
Psychosocial	0.58 (0.48)	vs. Control	0.34 (0.61)	0.44

Note. The ABS psychosocial model subscale reflects a -2 (*strongly disagree*) to +2 (*strongly agree*) rating scale.

* $p < .05$.

In terms of belief in the disease model, a mixed-model repeated measures ANOVA found a significant main effect for condition, $F(2, 78) = 18.16, p < .01$, but not for wave. This was qualified by a significant interaction, $F(2, 78) = 5.43, p < .01$. Simple main effects analyses found a significant effect for condition at both Wave 2, $F(2, 78) = 23.34, p < .01$, and Wave 3, $F(2, 78) = 9.95, p < .01$. Follow-up analyses using Tukey's method ($p < .05$) found that the disease condition had a significantly stronger belief in the disease model than the psychosocial condition at both Wave 2 and 3, and that this represented a large effect size; the control condition was significantly different from both the treatment conditions at each of Waves 2 and 3, showing medium effect sizes (the difference between the control and psychosocial conditions only showed a trend toward significance; see Table 6). These analyses demonstrate that the disease condition statements strengthened belief in the disease model, that the psychosocial condition statements weakened such belief, and that the control condition had little effect. These influences persisted 2 weeks later. This mirrors the previous analyses on belief in the psychosocial model, and reinforces the hypothesis that addiction beliefs are amenable to change

and that these changes represent more than momentary effects. It also suggests that such changes can occur with minimal exposure to a few short statements.

Table 6

Disease Model Beliefs (ABS) Simple Main Effects Follow-up Analyses for Condition at Both Waves

Condition 1	<i>M (SD)</i>	Condition 2	<i>M (SD)</i>	<i> d </i>
Wave 2				
Disease	0.75 (0.69)	vs. Psychosocial	-0.42 (0.68)	1.71*
Disease	0.75 (0.69)	vs. Control	0.13 (0.54)	0.98*
Psychosocial	-0.42 (0.68)	vs. Control	0.13 (0.54)	0.90*
Wave 3				
Disease	0.54 (0.62)	vs. Psychosocial	-0.23 (0.73)	1.14*
Disease	0.54 (0.62)	vs. Control	0.08 (0.61)	0.75*
Psychosocial	-0.23 (0.73)	vs. Control	0.08 (0.61)	0.46

Note. The ABS disease model subscale reflects a -2 (*strongly disagree*) to +2 (*strongly agree*) rating scale.

* $p < .05$.

Personal Perceptions of Agency

The chief goal of this study was to assess whether belief in a particular model of addiction influences mild to moderate alcoholics' sense of agency over their addiction. It was hypothesized that promoting a disease model of addiction would attenuate feelings of agency, whereas promoting a psychosocial model would increase feelings of agency. The effects of manipulating addiction beliefs on five different indices of agency were assessed using five mixed-model repeated measures ANOVAs.

In terms of the FAD-Plus, a mixed-model repeated measures ANOVA found a significant main effect for wave, $F(1, 78) = 22.21, p < .01$. No significant main effect for condition or an interaction effect was found. Follow-up analyses found that participants at Wave 2 ($M = 3.31, SD = 0.34$) evinced a stronger belief in free will than they did later at Wave 3 ($M = 3.16, SD = 0.37$), with this difference representing a medium effect size ($|d| = .42$). The lack of main effect for condition suggests that differing addiction beliefs do not significantly influence mild to moderate alcoholics' general tendency to believe in free will. The significant effect for wave is more difficult to interpret and likely represents a sort of practice effect.

In terms of the MHLC, a mixed-model repeated measures ANOVA did not find a significant main effect for wave or for condition. However, there was a significant interaction, $F(2, 78) = 4.25, p = .02$. Simple main effects analyses found a significant simple main effect of condition at Wave 2 but not at Wave 3. Follow-up analyses at Wave 2, using Tukey's method ($p < .05$), found that participants in the psychosocial condition had a significantly stronger internal locus of control than those in both the disease and the control conditions (see Table 7). This suggests that internalizing the psychosocial model of addiction promotes an internal sense of control over alcohol-related behavior. Furthermore, it suggests that mild to moderate alcoholics' perceived locus of control is typically that which is associated with a disease model belief and that advancing the psychosocial model can actually promote a sense of control over addiction-related behavior.

Table 7

Locus of Control (MHLC) Simple Main Effects Follow-up Analyses for Condition at Wave 2

Condition 1	<i>M (SD)</i>	Condition 2	<i>M (SD)</i>	<i> d </i>
Disease	4.35 (0.63)	vs. Psychosocial	4.72 (0.55)	0.63*
Disease	4.35 (0.63)	vs. Control	4.20 (0.49)	0.27
Psychosocial	4.72 (0.55)	vs. Control	4.20 (0.49)	1.00*

Note. The MHLC subscale reflects a +1 (*strongly disagree*) to +6 (*strongly agree*) rating scale.

* $p < .05$.

In terms of the BACQ, a mixed-model repeated measures ANOVA did not reveal any significant effects. The lack of main effect for condition suggests that differing addiction beliefs do not significantly influence mild to moderate alcoholics' general coping style.

In terms of the CDESES, a mixed-model repeated measures ANOVA did not find a significant main effect for wave but did find one for condition, $F(2, 78) = 4.17, p = .02$. This was not qualified by a significant interaction. Follow-up analyses using Tukey's method ($p < .05$) found that the disease condition effected significantly weaker perceptions of drinking self-efficacy than the control condition; no significant differences between the disease and the control conditions were found (see Table 8). A trend toward a significant difference between the disease and the psychosocial conditions, representing a medium effect size, was also observed. These results imply that internalizing the disease model weakens typical perceptions of drinking self-efficacy. The results also imply that internalizing the psychosocial model does not improve self-efficacy above and beyond typical levels.

Table 8

Drinking Self-Efficacy (CDSES) Main Effect Follow-up Analyses for Condition

Condition 1	<i>M (SD)</i>	Condition 2	<i>M (SD)</i>	<i> d </i>
Disease	50.90 (17.40)	vs. Psychosocial	58.65 (18.65)	0.43
Disease	50.90 (17.40)	vs. Control	63.64 (15.74)	0.76*
Psychosocial	58.65 (18.65)	vs. Control	63.64 (15.74)	0.29

Note. The CDSES subscale reflects a 0% (*not at all confident*) to 100% (*very confident*) rating scale.

* $p < .05$.

In terms of the AAES, a mixed-model repeated measures ANOVA did not reveal a significant main effect for wave or for condition; however there was a significant interaction, $F(2, 78) = 3.72, p = .03$. Simple main effects analyses found a significant effect of condition at Wave 2, $F(2, 78) = 3.36, p = .04$, but not at Wave 3. Follow-up analyses using Tukey's method ($p < .05$) found that, at Wave 2, the psychosocial condition had significantly weaker addiction entitization beliefs than the control group (see Table 9). A trend toward a significant difference, with a medium-sized effect, was also found between the psychosocial and disease conditions. These results suggest that internalizing a psychosocial model of addiction can weaken mild to moderate alcoholics' typical addiction entitization beliefs; that is, the psychosocial model beliefs seem to weaken commonly held perceptions that addiction is a stable entity that cannot be influenced with initiative. These results also imply that internalizing the disease model does not promote entitization beliefs above and beyond typical levels. However, these effects did not persist over the 2-week follow-up interval.

Table 9

Addiction Entitization (AAES) Simple Main Effects Follow-up Analyses for Condition at Wave 2

Condition 1	<i>M (SD)</i>	Condition 2	<i>M (SD)</i>	<i> d </i>
Disease	2.52 (0.86)	vs. Psychosocial	2.11 (0.73)	0.51
Disease	2.52 (0.86)	vs. Control	2.63 (0.71)	0.14
Psychosocial	2.11 (0.73)	vs. Control	2.63 (0.71)	0.72*

Note. The AAES subscale reflects a +1 (*strongly disagree*) to 6 (*strongly agree*) rating scale.

* $p < .05$.

Personal Feelings of Stigma and Shame

The second main goal of this study was to assess whether belief in a particular model of addiction influences feelings of stigma and shame. Given the lack of previous experimental research on this subject no hypotheses were made. The effects of manipulating addiction beliefs on three different indices of stigma and shame were assessed using three mixed-model repeated measures ANOVAs.

In terms of the PDD, a mixed-model repeated measures ANOVA did not reveal any significant effects. The lack of a main effect for condition suggests that differing addiction beliefs do not significantly influence, above and beyond typical levels, mild to moderate alcoholics' sense of public perceptions of devaluation of and discrimination towards people with a past alcohol addiction. It should be emphasized that the PDD is an assessment of public perception beliefs concerning an individual with a past history of alcohol addiction. These results suggest that internalizing a psychosocial or a disease model of addiction does not increase feelings of stigma regarding a past history of alcohol addiction.

In terms of the SSMIS, a mixed-model repeated measures ANOVA did not find a significant main effect for either wave or for condition; although there was a significant

interaction, $F(2, 78) = 3.50, p = .04$. However, simple main effects analyses did not find a significant effect of condition at either Wave 2 or Wave 3. Given these null findings, exploratory analyses were conducted on the four SSMIS subscales in order to examine potential group differences among the various types of stigma. Of each of the four subscales, only the aware subscale was found to have a significant effect, with a significant interaction, $F(2, 78) = 4.84, p = .01$ (but no significant main effect for condition or wave). Simple main effects analyses on this subscale found an effect of condition at Wave 2, $F(2, 78) = 4.09, p = .02$, but not at Wave 3. Follow-up analyses using Tukey's method ($p < .05$) found that both the disease and the psychosocial conditions reported higher levels of stigma than the control group, but that these two treatment conditions did not differ from each other (see Table 10). These results provide some evidence to suggest that both the disease and the psychosocial models increase feelings of stigma relative to a control group. Likewise, they suggest that the two treatment models are equally stigmatizing and that neither of the models seems to be protective against stigma.

Table 10

Stigma (SSMIS) Aware Subscale Simple Main Effects Follow-up Analyses for Condition at Wave 2

Condition 1	<i>M (SD)</i>	Condition 2	<i>M (SD)</i>	<i> d </i>
Disease	6.23 (1.53)	vs. Psychosocial	6.29 (1.33)	0.04
Disease	6.23 (1.53)	vs. Control	5.21 (1.68)	0.64*
Psychosocial	6.29 (1.33)	vs. Control	5.21 (1.68)	0.72*

Note. The SSMIS Aware subscale reflects a +1 (*I strongly disagree*) to +9 (*I strongly agree*) rating scale.

* $p < .05$.

These results were found only for the SSMIS aware subscale. This subscale represents the least internalized index of stigma, which measures the extent to which people are aware of stigmatizing public beliefs. No group differences were found among the SSMIS agree, apply, and harm subscales, which measure increasingly deeper and more damaging internalizations of stigma. This may suggest that some forms of stigma are more easily influenced by addiction beliefs than other, more deeply ingrained, forms.

It is also important to note the main difference between the two measures of stigma. The SSMIS assesses stigma regarding an individual with a current alcohol addiction, while the PDD assesses stigma regarding a past history of addiction. The results suggest that addiction beliefs may only negatively influence feelings of stigma for mild to moderate alcoholics' with a current, but not a past, alcohol addiction. Yet, these effects did not persist 2 weeks later, suggesting that the feelings of stigma may represent momentary, rather than enduring, feelings of stigma. However, this null result may also be due to a lack of power.

In terms of the SSGS shame subscale, a mixed-model repeated measures ANOVA revealed a significant main effect for condition, $F(2, 78) = 3.08, p = .05$, but no other effects. Follow-up analyses using Tukey's method ($p < .05$) found that the psychosocial condition reported significantly higher levels of state shame than those in the control group and that the disease condition showed a similar trend toward a significant difference, with each of these differences representing medium-sized effects (see Table 11). No significant differences were found between the disease and the psychosocial conditions. These results suggest that both the psychosocial and the disease models are equally shame-inducing and that neither of these models seems to be protective against feelings of shame. Furthermore, the significant main effect for

condition suggests that the “priming” entailed by either of the treatment conditions can cause enduring feelings of shame.

Table 11

Shame (SSGS) Main Effect Follow-up Analyses for Condition

Condition 1	<i>M</i> (<i>SD</i>)	Condition 2	<i>M</i> (<i>SD</i>)	<i>d</i>
Disease	1.80 (0.93)	vs. Psychosocial	1.91 (0.99)	0.11
Disease	1.80 (0.93)	vs. Control	1.38 (0.58)	0.53
Psychosocial	1.91 (0.99)	vs. Control	1.38 (0.58)	0.64*

Note. The SSGS Shame subscale reflects a +1 (*not feeling this way at all*) to +5 (*feeling this way very strongly*) rating scale.

* $p < .05$.

Exploratory Analyses using the ABSQ Items

The ABSQ consists of 13 very specific addiction-related questions intended to explore group differences on various addiction beliefs as well as explore the effects of terminological influences (viz., medical vs. non-medical terms). No inferential tests were used in analyzing these data because (a) they are exploratory in nature and, (b) more importantly, the very high overall family-wise error that would arise from such a large number of comparisons would question the confidence placed in such findings. As a result, only descriptive statistics (group means, standard deviations, and effect sizes) are reported. In order to calculate Cohen’s *d* effect size, comparisons were restricted to the disease and the psychosocial conditions (see Table 12).

Table 12

ABSQ Exploratory Comparisons Between the Disease and Psychosocial Conditions

ABSQ Items	Disease <i>M (SD)</i>	Psychosocial <i>M (SD)</i>	<i> d </i>
1. brain disease	-0.18 (1.90)	-0.73 (1.74)	0.30
2. social or psychological problem	0.50 (1.58)	1.07 (1.48)	0.37
3. weak will or a lack of self-control	-0.09 (1.83)	0.20 (1.83)	0.06
4. cannot control drinking	1.59 (1.65)	0.17 (1.76)	0.83
5. alcohol problem	-0.76 (1.86)	-1.50 (1.46)	0.44
a. problem to be very serious	-0.15 (1.89)	-1.37 (1.87)	0.65
b. overcome my alcohol problem	1.26 (1.64)	1.70 (1.12)	0.31
c. treatment for my disease of addiction	-0.50 (2.03)	-1.43 (1.63)	0.50
6. disease of alcohol addiction	-1.29 (1.82)	-2.13 (1.28)	0.53
a. this disease to be very serious	0.59 (2.02)	-0.60 (2.37)	0.54
b. overcome my disease of alcohol addiction	0.79 (1.92)	1.57 (1.19)	0.48
c. treatment for my disease of addiction	-0.59 (1.91)	-1.07 (2.05)	0.24
ABSQ Item	<i>n (%)</i>	<i>n (%)</i>	
7. reason(s) drink more than two standard drinks			
a. enjoyment purposes	30 (88%)	28 (93%)	
b. cope with negative emotion/events	24 (71%)	12 (40%)	
c. be more sociable/outgoing/confident	21 (62%)	19 (63%)	
d. fit in with others who are drinking	13 (38%)	10 (33%)	
e. a craving for alcohol	16 (47%)	10 (33%)	
f. never drink more than two drinks	1 (3%)	1 (3%)	

Note. The ABSQ items reflect a -3 (*strongly disagree*) to a +3 (*strongly agree*) rating scale.

These exploratory analyses suggest several tentative implications. First, the results of Items 1-3 suggest that, on average, participants internalized only moderately strong disease or psychosocial model views, such that responses tended to be within the neutral to slightly agree/disagree range (viz., scores of -1 to +1). Second, it appears that the disease group appraised their addiction as more serious (i.e., identified with it, viewed it as more serious, and admitted a need for treatment) regardless of terminological differences that described addiction as a “disease” versus as a “problem.” This suggests these semantic differences are not confounding factors.

Correlational Analyses

Correlations Between Demographic Variables and Addiction Beliefs

Preliminary correlational analyses were conducted to assess the relationship between various demographic factors and addiction beliefs (see Table 13). None of these variables, including age, sex, education, income, religion, or political orientation were significantly related to either belief in the disease or in the psychosocial model of addiction. Similarly, there were no significant relationships between addiction beliefs and the presence of a mental or physical illness, or with ever having received non-alcohol related therapy. Likewise, addiction beliefs were not significantly related to alcohol use or addiction severity. It should be reiterated that these correlations were conducted on the Wave 2 control group (28 participants), which is a small sample size that might, as a result, be obscuring significant relationships.

Table 13

Correlations Between Demographics and Disease and Psychosocial Addiction Beliefs (N = 28)

Variable	Disease <i>r</i>	Psychosocial <i>r</i>
Age	.26	-.17
Sex ^a	-.09	-.30
Education	-.24	.03
Income	-.12	.12
Religion ^b	-.24	.25
Political Orientation ^c	-.18	.11
Therapy for non-alcohol mental illness	.10	-.24
Mental illness diagnosis ^d	-.04	.09
Physical illness diagnosis ^d	.06	.05
Number of drinks in the last 7 days	-.06	.22
Number of DSM-5 diagnostic symptoms	-.01	.16

^acoding: 1 = male, 2 = female; ^bcoding: 1 = atheist, 2 = not religious, 3 = religious; ^ccoding: -3 = very liberal to +3 = very conservative; ^dcoding: 1 = no, 2 = yes.

Correlations Between Attitudinal Beliefs, Traits, and Addiction Beliefs

Further correlational analyses examined the relationship between disease and psychosocial addiction beliefs as well as their relationship with personality traits, life satisfaction, and just world beliefs (see Table 14). Belief in the disease model of addiction was found to be significantly negative related to belief in the psychosocial model, $r(27) = -.42, p = .03$. This suggests that these models of addiction represent related and somewhat opposite models of addiction, albeit they are not incompatible. In terms of personality traits, belief in the

disease model was significantly negatively correlated with both extraversion and (marginally to) neuroticism. This suggests a personality pattern that may be associated with a particular conceptualization of addiction. No significant trends were found between holding a disease model of addiction and life satisfaction or just world beliefs. Belief in the psychosocial model, on the other hand, was not significantly associated with any personality traits, life satisfaction, or just world beliefs. These findings suggest that personal conceptualizations of addiction are not associated with feelings of fairness or general life satisfaction.

Table 14

Correlations Between Addiction Beliefs, Attitudinal Beliefs, and Personality Traits

Variable	Disease <i>r</i>	Psychosocial <i>r</i>
ABS Psychosocial Subscale	-.42*	—
Satisfaction with Life	.15	.29
Belief in a Just World	-.17	.02
Personality Traits		
Extraversion	-.49**	.11
Conscientiousness	-.03	.06
Agreeableness	.09	-.21
Neuroticism	-.34†	.20
Openness to Experience	.01	-.06

** $p < .01$, * $p < .05$, † $p < .10$.

Intercorrelations Between Indices of Agency, Stigma, and Shame

In order to understand the relationship among the various indices of agency and stigma and shame an intercorrelations matrix was calculated (see Table 15). In terms of the relationship

among the various measures of agency, all of the measures were either significantly related in the expected direction or not related (i.e., none of them were related in the opposite direction). This suggests that all indices of agency are tapping positively related facets of agency or unrelated facets of agency. In terms of stigma and shame, none of the measures were significantly correlated. This is difficult to interpret and may suggest that the three indices of stigma and shame are tapping different components of these constructs. Additionally, non-significant relationships may reflect a lack of power given the small sample size for these exploratory analyses.

The relationship between measures of agency and those of stigma and shame were also assessed. These correlations found that (a) internal locus of control, approach coping style, and weaker addiction entitization were all significantly related to lower levels of shame and (b) internal locus of control was the only measure of agency significantly related to stigma (viz., PDD), such that a stronger internal locus of control was related to worse feelings of stigma. These results suggest that stronger feelings of some forms of agency might be associated with increased feelings of stigma but may also be associated with reduced feelings of shame.

Table 15

Intercorrelations Between Indices of Agency, Stigma, and Shame

Variable	1	2	3	4	5	6	7	8
1. FAD-Plus	—							
2. MHLC	.14	—						
3. BACQ	.30	.25	—					
4. CDESES	.31	-.00	.48**	—				
5. AAES ^a	.00	-.59**	-.39*	-.26	—			
6. PDD ^b	-.04	-.42*	.18	.24	.28	—		
7. SSMIS	.17	-.30	-.22	.01	.05	-.10	—	
8. Shame	-.22	-.37*	-.45*	-.29	.49**	-.01	.07	—

^aHigher scores indicate greater entitization of addiction; ^bhigher scores indicate weaker perceptions of stigma.

** $p < .01$, * $p < .05$, † $p < .10$.

Correlations Between Addiction Beliefs and Agency, Stigma, and Shame

Correlational analyses were used to examine the relationship between addiction beliefs and measures of agency, stigma, and shame (see Table 16). The CDESES was significantly negatively related to belief in the psychosocial model and (marginally) positively related to belief in the disease model. This suggests that holding a psychosocial model of addiction is associated with reductions in drinking self-efficacy, while the disease model may be associated with improvements in drinking self-efficacy. The MHLC, on the other hand, was significantly negatively associated with the disease model, suggesting stronger disease model beliefs are associated with a weaker internal locus of control. Thus, it appears that different facets of agency may be differentially related to particular addiction beliefs. Lastly, the different SSMIS

subscales were differentially associated with the two types of addictions beliefs, suggesting that each model may be positively associated with stigma, but in different ways. This aligns with our ANOVA findings, which demonstrated that both treatment conditions led to stronger perceptions of stigma.

Table 16

Correlations Between Addiction Beliefs, Attitudinal Beliefs, and Personality Traits

Variable	Disease <i>r</i>	Psychosocial <i>r</i>
Agency		
Belief in free will	.15	-.14
Free will subscale	-.16	.37*
Scientific determinism subscale	-.04	.26
Fatalistic determinism subscale	-.27	.41*
Randomness subscale	-.13	-.06
Internal locus of control	-.39*	.17
Internal-subscale	-.32†	.23
Chance-subscale	-.08	.21
Powerful Others-subscale	.52**	-.26
Doctors-subscale	.53**	-.41*
Others-subscale	.08	.02
Approach coping style	-.21	-.17
Addiction entitization ^a	.08	-.09
Drinking self-efficacy	.33†	-.65**

Shame and Stigma

Past addiction stigma ^b	.10	-.13
Current addiction stigma	.42*	.09
Aware-subscale	.40*	-.28
Agree-subscale	.44*	.02
Apply-subscale	.15	.02
Harm-subscale	.15	.37*
Shame	.07	.17

^aHigher scores indicate greater entitization of addiction; ^bhigher scores indicate weaker perceptions of stigma.

** $p < .01$, * $p < .05$, † $p < .10$.

Further analyses examined the relationship between agency and stigma and shame variable subscales (see Table 16). It was found that belief in the psychosocial model was significantly positively related to both belief in free will and belief in fatalistic determinism, which suggests that participants were able to hold seemingly contradictory views simultaneously. Furthermore, the MHLC doctors subscale was significantly positively related to belief in the disease model and negatively related with the psychosocial model. These results suggest that a stronger belief in the disease model is associated with a greater reliance on medical professionals to ameliorate one's addiction, whereas a stronger belief in the psychosocial model is associated with less reliance on medical professionals.

In terms of stigma, the disease model was significantly positively related to the SSMIS aware and agree subscales. On the other hand, the psychosocial model was significantly positively related to the SSMIS harm subscale. This suggests that belief in the psychosocial

model is associated with a deeper, more harmful, internalization of stigmatizing beliefs; the disease model, on the other hand, may be associated with relatively less deeply entrenched forms of stigma (see Table 16).

Correlations Between Alcohol Use, Severity, and Agency, Stigma, and Shame

A set of correlations was conducted to explore the relationship between indices of agency, stigma, and shame and current alcohol use (viz., past 7-day alcohol use) and addiction severity (viz., number of diagnostic symptoms; see Table 17). Addiction severity had a significant positive relationship with internal locus of control, such that a stronger internal locus of control was related to a greater number of symptoms. This might, perhaps, reflect a dark side to personal perceptions of agency, in which those more severely afflicted overestimate their ability to control their drinking to their detriment.

In terms of feelings of shame, a greater number of alcohol disorder symptoms was marginally negatively related to shame. This suggests that individuals with a more severe addiction experience less shame, perhaps reflecting a desensitization effect in which discussions of addiction are more common and thus less shame-provoking for these more severely afflicted individuals. Finally, alcohol use was found to be significantly negatively related to controlled drinking self-efficacy, such that higher levels of self-efficacy were associated with consuming fewer drinks. This suggests that mild to moderate alcoholics' perception of being able to control their consumption is related to actual control over consumption.

Table 17

Correlations Between Alcohol Use, Addiction Severity and Agency, Stigma, and Shame

Variable	Alcohol Use <i>r</i>	Addiction Severity <i>r</i>
Agency		
Belief in free will	-.00	.29
Internal locus of control	.01	.47**
Approach coping style	-.17	.02
Addiction entitization ^a	.07	-.25
Drinking self-efficacy	-.45*	-.09
Shame and Stigma		
Past addiction stigma ^b	.08	-.24
Current addiction stigma	-.02	.15
Shame	-.11	-.35†

^aHigher scores indicate greater entitization of addiction; ^bhigher scores indicate weaker perceptions of stigma.

** $p < .01$, * $p < .05$, † $p < .10$.

CHAPTER 4: DISCUSSION

The current study provides the first experimental examination on the effects of addiction beliefs among mild to moderate alcoholics. An experimental manipulation was used to assess the effects of having a disease or a psychosocial conceptualization of addiction. The findings of this study demonstrate that minimal exposure to a set of statements promoting either a disease or a psychosocial model of addiction can significantly influence how mild to moderate alcoholics think about their addiction. These changes in addiction beliefs were shown to have lasting effects over a 2-week interval. Furthermore, these changes in addiction conceptualization led to important changes in alcoholics' perceptions of agency over addiction-related behaviors as well as related feelings of stigma and shame.

Before discussing the results of this study it is important to keep in mind the following factors. First, this study was designed with the intention of exploring an area of research that has, to date, received little experimental study. With this exploratory motivation in mind, the strategy of this study was akin to casting a wide net on a large number of variables in the hope of capturing the important relationships, which could then be more finely examined in subsequent research. One effect of such a wide-net strategy is that the overall family-wise error rate of the sum of our inferential tests exceeds normal limits. Appropriate post-hoc tests (viz., Tukey's method) were used in order to minimize Type 1 error inflation. However, the sheer number of analyses conducted to test our various hypotheses suggests that some findings may represent Type 1 errors. Thus, the conclusions from this study are best considered to be tentative.

Second, the primary experimental analyses of this study are based on a sample size (81 participants) that is much smaller than the sample size required (130 participants), according to a priori power analyses, for an 80% chance of detecting a moderate effect. This limitation is

especially concerning in regards to the correlational analyses, which were restricted to a further subset of 28 participants in the control group. It is due to this lack of power, combined with the exploratory nature of this study, that marginally significant effects are also discussed, albeit tentatively so.

Third, the emphasis of the discussion will concern the experimental results as they provide the most robust assessment of the causal links between addiction beliefs and perceptions of agency and stigma and shame. The results from correlational analyses are provided as supplemental data, discussed in order to further explore experimental findings; when discrepancies between the two arise, the correlational results should be viewed with greater skepticism.

Fourth, the present study was conducted on individuals with a mild to moderate alcohol addiction. The implications of our findings are considered with this population in mind. We presume that at least some of our findings would generalize to those with more severe forms of alcohol addiction; however, substantiating such claims would require further research on this specific population. The results concerning addiction beliefs in general will first be discussed, followed by changes in addiction-related perceptions of agency and stigma and shame.

Manipulating Addiction Beliefs: The Impressionable Nature of Addiction Beliefs

One of the significant contributions of this study is that it provides initial experimental evidence that addiction beliefs, among mild to moderate alcoholics, are amenable to change. Furthermore, it has demonstrated that these changes can occur with only minimal exposure. The fact that changes in addiction beliefs were maintained 2 weeks after our manipulation obviates arguments that participant responses were mere momentary changes and suggests that they represent persisting changes in addiction beliefs.

These experimentally induced changes in addiction beliefs are especially noteworthy given that such changes were observed from a manipulation that was conducted online. This type of manipulation is typically conducted in a laboratory setting (e.g., Baumeister et al., 2009), which provides greater experimenter control and presumably increases the effectiveness of the manipulation. For instance, in-lab manipulations of this sort typically ensure that participants dwell on each of the 15 manipulation statements for a whole minute (e.g., Baumeister et al., 2009); such constraints were not possible in our online manipulation. Thus, the fact that our study was conducted online only serves to bolster our findings that addiction beliefs are quite susceptible to influence. Furthermore, it supports the notion that such manipulations can be successfully conducted online.

The findings that mild to moderate alcoholics tend to hold quite impressionable addiction beliefs have implications for professionals and professional organizations. The results from this study suggest that even minor exposure to addiction beliefs can have significant and lasting effects on alcoholics' views of their addiction. For instance, it is likely that even brief online messages, such as the NIDA's (2012) website, leave lasting impressions. Thus, these results underscore the importance of health professionals providing accurate and helpful information about alcohol addiction.

Typical Addiction Beliefs Among Mild to Moderate Alcoholics

This study employed a control group in order to understand the typical, unmanipulated, addiction beliefs held by mild to moderate alcoholics. It was hypothesized that if one of the two treatment groups did not differ from the control group, that this condition would represent the typically held view of addiction. However, it was found that the manipulation was able to significantly change participants' typical views to be more in line with either the disease or the

psychosocial models of addiction. This suggests that people with a mild to moderate alcohol addiction, who have never received any sort of addiction-specific treatment, do not have incorrigible conceptualizations of their addiction. Rather, their typical view of addiction can be influenced toward either a disease or a psychosocial conceptualization. This further underscores the impressionable nature of mild to moderate alcoholics' addiction beliefs.

The Influence of Addiction Beliefs on Perceptions of Agency

The chief purpose of this study was to assess the effects of different addiction beliefs on five facets of agency. It was hypothesized that internalizing a belief in the disease model of addiction would weaken perceptions of all forms of agency, whereas internalizing the psychosocial model would strengthen such perceptions. Support was garnered for addiction beliefs affecting three of the five indices of agency, namely locus of control, drinking self-efficacy, and addiction entitization. On all of these measures, internalizing the disease addiction model resulted in significantly (or marginally significantly) weaker perceptions of agency relative to those who internalized the psychosocial addiction model. These findings accord with arguments made by psychosocial model advocates (e.g., Fingarette, 1988; Schaler, 2000) who argue that disease models of addiction weaken alcoholics' perceptions of control over their addiction.

It is hypothesized that the two indices of agency that were not influenced by differences in addiction beliefs, namely belief in free will and coping style, were perhaps too general in scope. While the other three measures all tapped perceptions of agency in addiction-specific contexts, these two indices only tapped general perceptions of agency in non-addiction-specific contexts. Thus, it seems likely that these measures lacked sensitivity in detecting differences between experimental conditions. It might be the case that these measures would yield

significant group differences if they were adapted to measure free will beliefs and coping style within addiction-specific contexts. Future research should examine the effects of addiction beliefs on these indices of agency in addiction-specific contexts.

Typical Perceptions of Addiction-Related Agency Among Mild to Moderate Alcoholics

In order to understand the typical perceptions of addiction-specific agency among mild to moderate alcoholics, differences between the two treatment groups and the control group were examined. For instance, the locus of control and addiction entitization measures found that only the psychosocial condition, but not the disease condition, differed from the control group. This suggests that mild to moderate alcoholics are typically characterized by a weaker internal locus of control and stronger addiction entitization beliefs, reflective of those who tend to hold a disease view of addiction. When these individuals are manipulated to internalize a psychosocial model of addiction, their sense of internal locus of control can be strengthened to levels characteristic of a psychosocial view of addiction. Similarly, when internalizing the psychosocial model, addiction entitization beliefs can be weakened to levels characteristic of individuals who hold a belief in the psychosocial model. The opposite pattern was found with drinking self-efficacy, in which only the disease treatment condition differed from the control. This suggests that mild to moderate alcoholics typically have a stronger sense of drinking self-efficacy that is characteristic of those who hold a psychosocial view of addiction, and that this can be weakened by adopting disease model beliefs.

Another point of consideration is the stability of these changes in addiction-related perceptions of agency. Only one of the measures of agency, drinking self-efficacy, revealed group differences that persisted 2 weeks later. It is difficult to ascertain whether the lack of persistence on all three measures is due to a fleeting effect or whether this reflects an under-

powered study. Given that our results demonstrate that changes in addiction beliefs persist over time and that addiction beliefs and perceptions of agency are causally related, it would be expected that changes in agency would also persist over time. Future research should assess the stability of changes in perceptions of addiction-specific agency using a larger sample and perhaps a stronger manipulation (e.g., in a lab setting or with repeated versions of the intervention).

The Influence of Addiction Beliefs on Perceptions of Stigma and Shame

The second main goal of this study was to assess the effects of addiction beliefs on feelings of stigma and shame. Given the lack of previous experimental research on this subject, as well as convincing theories from both perspectives, no hypotheses were made. Some theorists argue that a medical model or a brain disease conceptualization reduces the stigma and shame associated with an alcohol addiction, because it frees the addict from being blamed for having a weak will (e.g., Milam & Ketcham, 1981). Theorists from the other perspective argue that the brain disease model increases stigma and shame by labeling the addict as suffering from an unpredictable, malfunctioning brain abnormality (e.g., Read & Law, 1999). In fact, the results of our study suggest that both camps of theorists have valid arguments.

The results from this study provide experimental evidence that mild to moderate alcoholics may experience equal amounts of stigma and shame from both the disease and the psychosocial conceptualizations of addiction. In terms of stigma, the SSMIS aware subscale provides some evidence that the disease and the psychosocial conditions equally increase stigma relative to a control group. That is, internalizing either the disease or the psychosocial models of addiction appears to worsen perceptions of stigma. While both these treatment groups did experience more stigma on this one subscale, no group differences were found on the other three

SSMIS subscales.

One possible reason for this lack of difference on three of the SSMIS subscales concerns the extent to which feelings of stigma are internalized. The SSMIS aware subscale examines a relatively weaker internalization of stigma than the other three subscales. The aware subscale assesses individuals' perceptions regarding public beliefs concerning stigma towards addiction (i.e., "I think the public believes ..."). The other three subscales, on the other hand, represent progressively stronger internalizations of stigma that assess alcoholics' (a) agreement with stigmatizing beliefs (i.e., "I think most persons with an alcohol addiction ..."; SSMIS agree subscale); (b) the application of such beliefs onto oneself (i.e., "Because I use alcohol or have an alcohol addiction I am ..."; SSMIS apply subscale); and (c) the resulting harm of such beliefs (i.e., "I currently respect myself less because I am ..."; SSMIS harm subscale). In all, the four SSMIS subscales tap a progressive level of internalization of stigmatizing beliefs, from weaker internalized beliefs regarding public perception of stigma to more deeply ingrained beliefs affecting one's self-concept.

It seems probable that our minimally invasive manipulation was sufficiently impactful to influence participants' beliefs regarding public perception of stigma, but not to produce deeper changes in self-perceptions. Further research on this would need to examine the ways in which more invasive addiction messages might influence deeper forms of internalized stigma. Additionally, our study did not find these differences in perceptions of stigma to persist 2 weeks later. Further research would also need to examine whether stronger exposure to various addiction beliefs would create lasting changes in perceptions of stigma; likewise, a larger sample size is needed to test the stability of these effects.

The experimental analyses of this study found that both models of addiction equally

affect feelings of stigma. Yet, correlational analyses also found some evidence that different addiction beliefs may be differentially related to the depth of stigma internalization. For instance, it was found that believing in the disease model was associated with a stronger awareness (viz., SSMIS agree subscale) and agreement (viz., SSMIS agree subscale) with stigmatizing views, while the psychosocial model was associated with a stronger influence of stigmatizing beliefs on one's self-concept (viz., SSMIS harm subscale). Thus, the former, namely the disease model, was associated with relatively less internalized forms of stigma, while the latter, namely the psychosocial model, was associated with deeper, more pernicious internalizations of stigma. Future research examining the effects of addiction beliefs on stigma should also consider the depth of stigma internalization that results from each addiction model. This would provide insight into which model is associated with less damaging forms of stigma.

The PDD represented the other measure of stigma in our study. This measure did not reveal differences between those who internalized disease or psychosocial addiction beliefs. We hypothesize that this null result might be related to whether an individual has a current or past addiction to alcohol. While the SSMIS provides an assessment of perceptions of stigma concerning individuals with a current addiction, the PDD provides a measure of stigma concerning individuals with a former addiction. Thus, it may be the case that alcoholics are less affected by stigmatic beliefs concerning a past addiction. However, our sample consists of individuals who met diagnostic criteria for a current alcohol use disorder and thus we can only speculate on this front. In order to assess the effects of stigma on individuals with a past addiction, further research would need to examine individuals with a past, rather than a current, alcohol addiction.

One important consideration when discussing the effects of stigma is that there are

several different forms of stigma. For instance, one recent review (Kvaale, Haslam, & Gottdiener, 2013) categorized four forms of stigma that can be divided into stigma resulting from (a) blame, (b) perceptions of dangerousness and unpredictability, (c) social distance (viz., unwillingness to enter into relationship with alcoholics), and (d) prognostic pessimism (viz., feelings of hopelessness regarding recovery). The measures used in our study did not differentiate among these different forms of stigma. It is important to interpret our findings in the context of other work suggesting that the disease model may protect against blame-related forms of stigma, while the psychosocial model may protect against stigmatizing perceptions of dangerousness and unpredictability as well as prognostic pessimism (for a review see Kvaale et al., 2013). Differentiating between specific types of stigma is crucial to fully understanding the effects that the disease and the psychosocial models have in this regard. A focus of future research should be on examining the influences of addiction beliefs on different forms of stigma.

The results of our study also provide experimental evidence that addiction beliefs affect feelings of shame. In a similar vein to stigma, it was found that both the disease and the psychosocial models equally increased feelings of shame, relative to a control group. There was also some evidence of these effects persisting 2 weeks later. This finding calls into question the arguments of both disease and psychosocial model proponents who argue that their respective models are the only means to alleviating shame (e.g., Milam & Ketcham, 1981; Schaler, 2000). The results from our study suggest that the two models equally affect feelings of shame. It may, however, be that the underlying source of shame is different for each model. For instance, perceptions of blame thought to be related to the psychosocial model may spark feelings of shame, while perceptions of abnormality and hopelessness thought to accompany the disease model may also spark feelings of shame. Given the detrimental effects of shame on addiction

(Randles & Tracy, 2013), further research should examine the relationship between addiction beliefs and feelings of shame by focusing on understanding the underlying sources for such negative feelings.

Agency and Shame and Stigma

Another factor that is argued to be related to stigma and shame is perceptions of control over one's addiction-related behavior (Milam & Ketcham, 1981). Our study used correlational analyses to examine the manner in which perceptions of control (viz., agency) are related to feelings of stigma and shame. In terms of stigma, some evidence was found that a stronger sense of control over one's actions (i.e., stronger internal locus of control) was positively related to stigma (viz., SSMIS). This accords with disease model proponents' argument that suggesting the alcoholic is in control of their behavior promotes blame and stigma (Milam & Ketcham, 1981). This also accords with empirical research (Kvaale et al., 2013); however, the other ways in which the disease model may be worsening other forms of stigma should also be kept in mind.

While a sense of control over one's addiction behavior was related to stronger feelings of stigma, it was also found to be related to weaker feelings of shame. Perhaps somewhat surprisingly, our study found that a sense of control over one's behavior was associated with lower levels of shame. Likewise other indices of agency, namely approach coping style and weaker addiction entitization beliefs, were also associated with decreased feelings of shame. In this regard, it appears that a sense of agency over one's addiction may be protective against feelings of shame. It may be that a sense of control over addiction or the perception that one is able and actively working towards recovery buffers feelings of shame. This would align with the views of psychosocial model advocates who argue that perceptions of control over one's addiction promote feelings of empowerment (Schaler, 2000). Further research is needed to

examine the ways in which these perceptions of agency may lead to reduced feelings of shame.

The Perceived Importance of Health Professionals: Examining Locus of Control

One of the key differences that emerged between the disease and the psychosocial conditions is the extent to which alcoholics rely on health professionals to overcome their addiction. This was suggested by our finding that the psychosocial group tended to have a stronger internal locus of control, whereas the disease group had a stronger external locus of control. The latter is generally considered to be maladaptive and associated with feelings of learned helplessness (Hiroto, 1974). However, a closer examination of our correlational findings elucidates that it is specifically the MHLC doctors subscale that shares a close relationship to addiction beliefs. On this subscale, those holding a disease model addiction are associated with heavier reliance on medical professionals than those espousing a psychosocial model. Additionally, relevant exploratory ABSQ items also suggest that those internalizing a disease model were more likely to emphasize the need for receiving professional medical treatment than those internalizing the psychosocial model.

The reliance on others to change one's circumstances represents an external locus of control. This is because the location of control is considered to reside outside of oneself, in this case in the external health professional (Luszczynska & Schwarzer, 2005). However, a closer examination of the MHLC doctors subscale suggests that this may not be the case for people with a disease view of addiction. Rather, it may be that, within the addiction context, these subscale items may actually be more indicative of an internal, rather than external, locus of control. For instance, consider one representative item from the MHLC doctors subscale: "Following doctor's orders to the letter is the best way to keep my alcohol use/addiction from getting any worse." This item does, in part, suggest a reliance on doctors (external), however, it

appears that it places the thrust of emphasis for change on the individual maintaining compliance with treatment dictates (internal).

Within the disease conceptualization, dealing with addiction is thought to primarily involve resolving a biological abnormality, which would require professional assistance. Thus, individuals who are acting within this view of addiction are taking the initiative to seek and follow professional advice. We suggest that, for these individuals, this is more appropriately described as an internal locus of control. In this case, relying on professional advice may represent the means to dealing with one's addiction, but the actual locus of change resides within the individual, who is the active agent in seeking and applying professional advice. This is very different than a passive individual, who views the fate of their addiction as entirely dependent on health professionals. Such a case is more reflective of someone with an external locus of control.

This difference in perceptions regarding the role of medical professionals in dealing with addiction seems to represent one of the core differences between the disease and the psychosocial models of addiction. It appears that a reliance on medical professionals may not necessarily represent a diminished perception of agency, in the form of locus of control, but could simply reflect a different strategy in dealing with addiction. It is entirely possible that both groups view their own behavior as central to change, with the psychosocial model group viewing resolving relevant psychological and social factors as the means to effecting change and the disease model group viewing abstinence and medication as the means to effecting change. Indeed, the tendency to strictly emphasize abstinence as the single solution to recovery is a disease model distinctive (Cook, 1988) which may serve to protect against naïve assumptions regarding one's ability to control consumption. Our finding that internal locus of control is positively associated with addiction severity suggests this may be a more adaptive perception of

addiction. Thus, promoting a reliance on appropriate professional support, while maintaining the internal locus of change, may reflect an adaptive version of the disease model. Future research is needed to assess these subtle differences among alcoholics who subscribe to the disease model of addiction.

Perceptions of Drinking Self-Efficacy: Contributions of Disease and Psychosocial Models

A second important difference between the disease and the psychosocial conditions concerns their influence on perceptions of drinking self-efficacy. Experimental analyses demonstrate that the disease model decreases perceptions of drinking self-efficacy. Likewise, relevant exploratory ABSQ items also suggest that individuals who subscribe to the disease model are less confident in their ability to overcome their addiction. Psychosocial model proponents argue that such weaker perceptions of control are maladaptive (e.g., Schaler, 2000) and our study does have some evidence to suggest that this may be the case. For instance, drinking self-efficacy was found to be significantly negatively related to actual alcohol use. This suggests that perceptions of drinking self-efficacy are predictive of actual control over alcohol consumption. In this regard, the psychosocial model, which promotes perceptions of drinking self-efficacy would be considered adaptive.

However, our study also found some evidence to suggest that some aspects of the disease model may also be helpful. For instance, correlational analyses found that the disease, not the psychosocial, model of addiction was positively associated with stronger perceptions of drinking self-efficacy. One hypothesis to explain these findings is that perceptions of drinking self-efficacy may be stronger among those who have vowed to remain abstinent. As such, it may be that those adopting a disease model are more likely to recognize their inability to drink in moderation and are more confident than those holding a psychosocial model as a result of their

commitment to abstinence. In this case, the disease model of addiction would be considered adaptive. Future research is needed to examine the aspects from disease and psychosocial addiction models that may contribute to strengthening perceptions of drinking self-efficacy.

Addiction Entitization: Examining Perceptions of Chronicity and Immutability

The third index of agency that was significantly related to addiction beliefs was addiction entitization. Experimental analyses found that belief in the disease model increased addiction entitization. That is, internalizing the disease model strengthened perceptions of the chronic and immutable nature of addiction. Correlational analyses also supported the view that addiction entitization beliefs were associated with a sense of powerlessness over addiction. For instance, addiction entitization was negatively related to internal locus of control. Further exploration of the MHLC subscales found that addiction entitization was also positively related to the MHLC chance subscale, $r(27) = .40, p = .04$ (but not to the MHLC doctors or others subscales, $-.04 < rs < .26, .18 < ps < .87$). Altogether, these findings suggest that the disease model promotes addiction entitization beliefs and, as a result, increases the perception that addiction behavior is chronic and unchangeable. Within this scheme, it seems that personal effort to pursue either appropriate lifestyle changes or professional medical intervention would be deemed ineffective.

These findings align with disease model critics' concerns regarding the disease conceptualization of addiction. One of their prominent concerns is that such a view propagates the perception that addiction-related behaviors cannot be influenced (e.g., Schaler, 2000). The concern is that viewing one's addiction as unchangeable would lead to feelings of learned helplessness; indeed, it is such cases, in which effort is unrelated to outcome, that tend to breed feelings of learned helplessness (Hiroto, 1974). Furthermore, such a stable view of addiction might discourage self-change narratives, which are so important to alcoholics' wellbeing

(Dunlop & Tracy, 2013). The validity of these concerns is also borne out by findings that biological conceptualizations promote pessimistic prognostic beliefs regarding overcoming various disorders (Kvaale et al., 2013). Furthermore, disease entitization beliefs may be particularly destructive if they contradict the more hopeful message that most mild to moderate alcoholics are able to influence and overcome their addiction (Dawson et al., 2005; Hasin et al., 2007). In these ways, addiction entitization would be considered to promote a maladaptive view of addiction.

On the other hand, some aspects associated with addiction entitization, as was the case with self-efficacy and locus of control beliefs, may confer protective views of addiction. For instance, addiction entitization might confer benefits to the extent that chronic and immutable views of addiction lead alcoholics to commit to abstinence and/or to seek ongoing treatment or support. Such was the case in a study on heroin addicts, in which addiction entitization was associated with treatment attendance (Zeldman et al., 2004). In this way, certain aspects of addiction entitization beliefs might be considered adaptive. Further research is needed to examine the adaptive and maladaptive aspects regarding addiction entitization beliefs and how they might be affected by different addiction beliefs.

The Disease and Psychosocial Models of Addiction: Benefits and Costs

The results of this study suggest that the two models of addiction examined herein may each confer aspects that might be considered both bane and boon for those struggling with an alcohol addiction. Some aspects of the disease model of addiction may be considered adaptive insofar as they serve to (a) diminish unrealistic positive beliefs concerning alcoholics' ability to control their drinking and overcome their addiction, (b) promote the seeking and observance of health professional guidance as well as ongoing support, and (c) reduce perceptions of blame that

might influence feelings of stigma and shame.

On the other hand, some aspects of the psychological model of addiction may be considered adaptive insofar as they serve to (a) promote the changeable nature of addiction behavior and reduce prognostic pessimism, (b) encourage the effortful involvement of making necessary lifestyle changes, and (c) reduce perceptions of unpredictability, dangerousness, and unchangeability that might influence feelings of stigma and shame. The optimal model of addiction would seek to integrate the adaptive elements of each model of addiction. Thus, it is suggested that the addiction model debate needs to move beyond the question, “Which of these addiction models is better?” to, “How can these two models be most optimally balanced and integrated?”

The future of addiction belief research should focus on researching this ideal combination of addiction models. Integrating these two models would seem to be a feasible goal, given that the disease and the psychosocial model beliefs do not appear to represent opposite and mutually exclusive models of addiction. This notion is supported by our study, which found that psychosocial and disease beliefs were sufficiently unrelated to be considered two different constructs. Likewise, past surveys have demonstrated that both health professionals and lay people tend to hold a combination of disease and psychosocial model beliefs (Forman et al., 2001; Lawrence et al., 2013; Pescosolido et al., 2010). Thus, it seems that people have a general tendency toward integrating these two types of addiction beliefs. It is now time for researchers to follow suit.

It would also be more helpful for health professionals and related organizations to work from a more integrated model of addiction. In order to accomplish this, they will need to adjust current biological emphases to incorporate more aspects from the psychosocial model.

Currently, disease model views are outpacing psychosocial model views among health professionals (Lawrence et al., 2013). Likewise, large professional organizations are heavily promulgating disease model beliefs at the expense of psychosocial model components (e.g., NIDA, 2012). This overemphasis of the disease model may be both unintentionally promoting certain maladaptive views of addiction as well as losing out on the benefits that would accompany elements of psychosocial conceptualizations. Realigning current conceptualizations of addiction to place a greater emphasis on the psychosocial view of addiction aligns well with the recommendations of a recent meta-analysis on the matter: Biological explanations “should not be promoted at the expense of psychosocial explanations, which appear to have more optimistic implications” (Kvaale et al., 2013, p. 290).

The conclusion of this study is that neither the disease or the psychosocial model of addiction represents an ideal conceptualization of addiction. They both entail elements that can hinder recovery and worsen stigma. However, each of the models also possesses unique components that reflect adaptive views thought to promote recovery and discourage stigma. The most promising conceptualization of addiction will harness the more adaptive components from each of the psychosocial and the disease models through their integration into a type of biopsychosocial model of addiction. Developing such a holistic conceptualization of addiction is germane to this research enterprise and should be the focus of subsequent study on the matter. Furthermore, our ability to integrate these models of addiction will have implications that reach beyond substance addiction and pave the way for how society comes to think of other forms of behavioral addictions.

REFERENCES

- Alcoholics Anonymous. (2012). *Estimate of A.A. groups and members as of January 1, 2012*. Retrieved from http://www.aa.org/en_pdfs/smf-53_en.pdf
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-4-TR*. Arlington, VA: American Psychiatric Association.
- American Psychiatric Association. (2013a). *Diagnostic and statistical manual of mental disorders: DSM-5*. Arlington, VA: American Psychiatric Association.
- American Psychiatric Association. (2013b). *Substance-related and addictive disorders*. Retrieved from <http://www.dsm5.org/Documents/Substance%20Use%20Disorder%20Fact%20Sheet.pdf>
- American Society of Addiction Medicine (ASAM). (2011). *Definition of addiction*. Retrieved from <http://www.asam.org/for-the-public/definition-of-addiction>
- Babor, T. F., Kranzler, H. R., & Lauerman, R. J. (1989). Early detection of harmful alcohol consumption: Comparison of clinical, laboratory, and self-report screening procedures. *Addiction Behavior, 14*, 139-157. doi:10.1016/0306-4603(89)90043-9
- Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182. doi:10.1037/0022-3514.51.6.1173
- Baumeister, R. F., Masicampo, E. J. & DeWall, C. N. (2009). Prosocial benefits of feeling free: Disbelief in free will increases aggression and reduces helpfulness. *Personality and Social Psychology Bulletin, 35*, 260-268. doi:10.1177/0146167208327217
- Berkson, J. (1946). Limitations of the application of fourfold table analysis to hospital data. *Biometrics Bulletin, 2*, 47-53. doi:10.2307/3002000

- Blackwell, L. S., Trzesniewski, K. H., & Dweck, C. S. (2007). Implicit theories of intelligence predict achievement across an adolescent transition: A longitudinal study and an intervention. *Child Development, 78*, 246-263. doi:10.1111/j.1467-8624.2007.00995.x
- Bogg, T., & Roberts, B. W. (2004). Conscientiousness and health-related behaviors: A meta-analysis of the leading behavioral contributors to mortality. *Psychological Bulletin, 130*, 887-919. doi:10.1037/0033-2909.130.6.887
- Bucholz, K. K., Cadoret, R., Cloninger, C., Dinwiddie, S. H., Hesselbrock, V. M., Nurnberger, J. I., & ... Schuckit, M. A. (1994). A new, semi-structured psychiatric interview for use in genetic linkage studies: A report on the reliability of the SSAGA. *Journal of Studies on Alcohol, 55*, 149-158.
- Buhrmester, M., Kwang, T., & Gosling, S. D. (2011). Amazon's Mechanical Turk: A new source of inexpensive, yet high-quality, data? *Perspectives on Psychological Science, 6*, 3-5. doi:10.1177/1745691610393980
- Butcher, J. N., Mineka, S., Hooley, J. M., Taylor, S., & Antony, M. M. (2010). *Abnormal psychology*. Toronto: Pearson.
- Carey, J. M., & Paulhus, D. L. (2013). Worldview implications of believing in free will and/or determinism: Politics, morality, and punitiveness. *Journal of Personality, 81*, 130-141. doi:10.1111/j.1467-6494.2012.00799.x
- Carr, P. (2012, March 19). How I stopped drowning in drink. *The Wall Street Journal*. Retrieved from <http://online.wsj.com>
- Chiauzzi, E. J., & Liljegren, S. (1993). Taboo topics in addiction treatment: An empirical review of clinical folklore. *Journal of Substance Abuse Treatment, 10*, 303-316. doi:10.1016/0740-5472(93)90079-H

- Conrad, P. (1992). Medicalization and social control. *Annual Review of Sociology*, 18, 209-232.
doi:10.1146/annurev.so.18.080192.001233
- Cook, C. C. (1988). The Minnesota Model in the management of drug and alcohol dependency: miracle, method or myth? Part I. The philosophy and the programme. *British Journal of Addiction*, 83, 625-634. doi:10.1111/j.1360-0443.1988.tb02591.x
- Corrigan, P. W., Kuwabara, S. A., & O'Shaughnessy, J. (2009). The public stigma of mental illness and drug addiction: Findings from a stratified random sample. *Journal of Social Work*, 9, 139-147. doi:10.1177/1468017308101818
- Corrigan, P., & Watson, A. (2004). At issue: Stop the stigma: Call mental illness a brain disease. *Schizophrenia Bulletin*, 30, 477-479. doi:10.1093/oxfordjournals.schbul.a007095
- Corrigan, P. W., Watson, A. C., & Barr, L. (2006). The self-stigma of mental illness: Implications for self-esteem and self-efficacy. *Journal of Social Clinical Psychology*, 25, 875-884. doi:10.1521/jscp.2006.25.8.875
- Cunningham, J. A., Lin, E., Ross, H. E., & Walsh, G. W. (2000). Factors associated with untreated remissions from alcohol abuse or dependence. *Addiction Behavior*, 25, 317-321. doi:10.1016/S0306-4603(98)00130-0
- Davies, J. (1992). *The myth of addiction: An application of the psychological theory of attribution to illicit drug use*. Amsterdam: Harwood.
- Dawson, D. A., Goldstein R. B., & Grant B. F. (2007). Rates and correlates of relapse among individuals in remission from DSM-IV alcohol dependence: A 3-year follow-up. *Alcoholism Clinical and Experimental Research*, 31, 2036-2045. doi:10.1111/j.1530-0277.2007.00536.x

- Dawson, D. A., Grant, B. F., Stinson, F. S., Chou, P. S., Huang, B., Ruan, W. J. et al. (2005). Recovery from DSM-IV alcohol dependence: United States, 2001–2002. *Addiction*, 100, 281-292. doi:10.1111/j.1360-0443.2004.00964.x
- Dean, J. C., & Poremba, G. A. (1983). The alcoholic stigma and the disease concept. *Substance Use & Misuse*, 18, 739-751. doi:10.3109/10826088309027366
- de Bruijn, C., van den Brink, W., de Graaf, R., & Vollebergh, W. A. (2006). The 3-year course of alcohol use disorders in the general population: DSM-IV, ICD-10 and the craving withdrawal model. *Addiction*, 101, 385-392. doi:10.1111/j.1360-0443.2006.01327.x.
- Diener, E., Emmons, R., Larsen, R., & Griffin, S. (1985). The satisfaction with life scale. *Journal of Personality Assessment*, 49, 71-75. doi:10.1207/s15327752jpa4901_13
- Douglas, D. B. (1986). Alcoholism as an addiction: The disease concept reconsidered. *Journal of Substance Abuse Treatment*, 3, 115-120. doi:10.1016/0740-5472(86)90060-7
- Dunlop, W. L., & Tracy, J. L. (2013). The autobiography of addiction: Autobiographical reasoning and psychological adjustment in abstinent alcoholics. *Memory*, 21, 64-78. doi:10.1080/09658211.2012.713970
- Dweck, C. S., Chi-yue, C., & Ying-yi, H. (1995). Implicit theories and their role in judgments and reactions: A word from two perspectives. *Psychological Inquiry*, 6, 267-285. doi:10.1207/s15327965pli0604_1
- Edwards, G., & Gross, M. M. (1976). Alcohol dependence: Provisional description of a clinical syndrome. *British Medical Journal*, 60, 1058-1061. doi:10.1136/bmj.1.6017.1058
- Executive Office. (2013). *National drug control strategy*. Retrieved from http://www.whitehouse.gov/sites/default/files/ndcs_2013.pdf

- Fingarette, H. (1988). *Heavy drinking: The myth of alcoholism as a disease*. Berkeley: University of California Press.
- Fingarette, H. (1990). Why we should reject the disease concept of alcoholism. In R. C. Engs (Ed.), *Controversies in the addiction field* (pp. 48-54). Dubuque, IA: Kendall-Hunt.
- Retrieved from <http://www.indiana.edu/~engs/cbook/chap6.html>
- Finset, A. A., Steine, S. S., Haugli, L. L., Steen, E. E., & Laerum, E. E. (2002). The brief approach/avoidance coping questionnaire: Development and validation. *Psychology, Health & Medicine*, 7, 75-85. doi:10.1080/13548500120101577
- Forman, R. F., Bovasso, G., & Woody, G. (2001). Staff beliefs about addiction treatment. *Journal of Substance Abuse Treatment*, 21, 1-9. doi:10.1016/S0740-5472(01)00173-8
- Foucault, M. (1965). *Madness and civilization: A history of insanity in the age of reason*. New York: Random House.
- Fowler, J. S., Volkow, N. D., Kassed, C. A., & Chang, L. (2007). Imaging the addicted human brain. *Science & Practice Perspectives*, 3(2), 4-16. doi:10.1151/spp07324
- Frances, A. (2010). DSM5 "Addiction" swallows substance abuse. *Psychiatric Times*, 27(3). Retrieved from <http://www.psychiatrictimes.com/>
- Frimer, J. A., Biesanz, J. C., Walker, L. J., & MacKinlay, C. W. (2013). Liberals and conservatives rely on common moral foundations when making moral judgments about influential people. *Journal of Personality and Social Psychology*, 104, 1040-1059. doi:10.1037/a0032277
- Glass, J. E., Kristjansson, S. D., & Bucholz, K. K. (2013). Perceived alcohol stigma: Factor structure and construct validation. *Alcoholism: Clinical and Experimental Research*, 37(Suppl. S1), E237-E246. doi:10.1111/j.1530-0277.2012.01887.x

- Gordis, E. (1995). The National Institute on Alcohol Abuse and Alcoholism: Past accomplishments and future goals. *Alcohol Health & Research World*, 19(1), 5-11.
- Gosling, S. D., Rentfrow, P. J., & Swann, Jr., W. B. (2003). A very brief measure of the Big-Five personality domains. *Journal of Research in Personality*, 37, 504-528.
doi:10.1016/S0092-6566(03)00046-1
- Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, P., Dufour, M. C., Compton, W., & ... Kaplan, K. (2004). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, 61, 807-816.
doi:10.1001/archpsyc.61.8.807
- Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S., Ruan, W., & Pickering, R. P. (2004). Co-occurrence of 12-month alcohol and drug use disorders and personality disorders in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, 61, 361-368.
doi:10.1001/archpsyc.61.4.361
- Hanson, G. R., Leshner, A. I., & Tai, B. (2002). Putting drug abuse research to use in real-life settings. *Journal of Substance Abuse Treatment*, 23, 69-70. doi:10.1016/S0740-5472(02)00269-6
- Hasin, D. S., Stinson, F. S., Ogburn, E., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, 64, 830-842. doi:10.1001/archpsyc.64.7.830

- Hayes, S. C. (2002, April). *The necessary partnership in evidence-based practice*. Paper presented at the meeting of the Center for Substance Abuse Treatment Practice Improvement Collaborative – “Improving Substance Abuse Treatment: Community-Based Approaches to Practice Innovation,” Tampa, FL.
- Hesselbrock, M., Easton, C., Bucholz, K. K., Schuckit, M., & Hesselbrock, V. (1999). A validity study of the SSAGA—A comparison with the SCAN. *Addiction*, 94, 1361-1370. doi:10.1046/j.1360-0443.1999.94913618.x
- Heyman, G. M. (2009). *Addiction: A disorder of choice*. Cambridge, MA: Harvard University Press.
- Hill, S. (1985). The disease concept of alcoholism: A review. *Drug and Alcohol Dependence*, 16, 193-214. doi:10.1016/0376-8716(85)90045-6
- Hiroto, D. S. (1974). Locus of control and learned helplessness. *Journal of Experimental Psychology*, 102, 187-193. doi:10.1037/h0035910
- Hoffman, J., & Froemke, S. (Eds.). (2007). *Addiction: Why can't they just stop?* New York: Rodale Books.
- Horton, J. J., & Chilton, L. B. (2010, June). The labor economics of paid crowdsourcing. In *Proceedings from EC '10: The 11th ACM Conference on Electronic Commerce* (pp. 209-218). New York: ACM. doi:10.1145/1807342.1807376
- Horton, J. J., Rand, D. G., & Zeckhauser, R. J. (2011). The online laboratory: Conducting experiments in a real labor market. *Experimental Economics*, 14, 399-425. doi:10.1007/s10683-011-9273-9
- Institute of Medicine. (1990). *Broadening the base of treatment for alcohol problems*. Washington, DC: National Academy Press.

- Jellinek, E. M. (1946). Phases in the drinking history of alcoholics. *Quarterly Journal of Studies on Alcohol*, 7, 1-88.
- Jellinek, E. M. (1960). *The disease concept of alcoholism*. New Haven, CT: Hillhouse Press.
- Jones, R., & Helrich, A. (1972). Treatment of alcoholism by physicians in private practice: A national survey. *Quarterly Journal of Studies on Alcohol*, 33, 117-131.
- Jones, K., Gill, C., & Ray, S. (2012). Review of the proposed DSM-5 substance use disorder. *Journal of Addictions & Offender Counseling*, 33, 115-123. doi:10.1002/j.2161-1874.2012.00009.x
- Jorm, A. F., & Oh, E. (2009). Desire for social distance from people with mental disorders. *Australian and New Zealand Journal of Psychiatry*, 43, 183-200. doi:10.1080/00048670802653349
- Kingree, J. B., Sullivan, B. F., & Thompson, M. P. (1999). Attributions for the development of substance addiction among participants in a 12-step oriented treatment program. *Journal of Psychoactive Drugs*, 31, 129-135. doi:10.1080/02791072.1999.10471735
- Koivumaa-Honkanen, H., Kaprio, J., Korhonen, T., Honkanen, R. J., Heikkilä, K., & Koskenvuo, M. (2012). Self-reported life satisfaction and alcohol use: A 15-year follow-up of healthy adult twins. *Alcohol and Alcoholism*, 47, 160-168. doi:10.1093/alcalc/agr151
- Knapp, C. (1999). *Drinking: A love story*. New York: Random House.
- Knight, J. R., Wechsler, H., Kuo, M., Seibring, M., Weitzman, E. R., & Schuckit, M. A. (2002). Alcohol abuse and dependence among US college students. *Journal of Studies on Alcohol and Drugs*, 63, 263-270.

- Kvaale, E. P., Haslam, N., & Gottdiener, W. H. (2013). The “side effects” of medicalization: A meta-analytic review of how biogenetic explanations affect stigma. *Clinical Psychology Review, 33*, 782-794. doi:10.1016/j.cpr.2013.06.002
- Lam, D. C., Salkovskis, P.M., & Warwick, H. M. (2005). An experimental investigation of the impact of biological versus psychological explanations of the cause of “mental illness.” *Journal of Mental Health, 14*, 453-464. doi:10.1080/09638230500270842
- Laundergan, J. C. (1982). *Easy does it: Alcoholism treatment outcomes, Hazelden and the Minnesota model*. Centre City, MN: Hazelden Publishing.
- Lawrence, R. E., Rasinski, K. A., Yoon, J. D., & Curlin, F. A. (2013). Physicians’ beliefs about the nature of addiction: A survey of primary care physicians and psychiatrists. *American Journal on Addictions, 22*, 255-260. doi:10.1111/j.1521-0391.2012.00332.x
- Leshner, A. (1997). Addiction is a brain disease, and it matters. *Science, 278*(5335), 45-47. doi:10.1126/science.278.5335.45
- Lester, D. (1989). The heritability of alcoholism. *Drugs & Society, 3*, 29-68. doi:10.1300/J023v03n03_02
- Levine, H. G. (1978). The discovery of addiction: Changing conceptions of habitual drunkenness in America. *Journal of Studies on Alcohol, 39*, 143-174. doi:10.1016/0740-5472(85)90022-4
- Link, B. G. (1987). Understanding labeling effects in the area of mental disorders: An assessment of the effects of expectations of rejection. *American Sociological Review, 52*, 96-112. doi:10.2307/2095395

- Lipkus, I. (1991). The construction and preliminary validation of a global belief in a just world scale and the exploratory analysis of the multidimensional belief in a just world scale. *Personality and Individual Differences, 12*, 1171-1178. doi:10.1016/0191-8869(91)90081-L
- Lipkus, I. M., Dalbert, C., & Siegler, I. C. (1996). The importance of distinguishing the belief in a just world for self versus for others: Implications for psychological well-being. *Personality and Social Psychology Bulletin, 22*, 666-677. doi:10.1177/0146167296227002
- Luoma, J. B., O'Hair, A. K., Kohlenberg, B. S., Hayes, S. C., & Fletcher, L. (2010). The development and psychometric properties of a new measure of perceived stigma toward substance users. *Substance Use & Misuse, 45*, 47-57. doi:10.3109/10826080902864712
- Luszczynska, A., & Schwarzer, R. (2005). Multidimensional health locus of control: Comments on the construct and its measurement. *Journal of Health Psychology, 10*, 633-642. doi:10.1177/1359105305055307
- Mack, A. H., Franklin, J. E. Jr., & Frances, R. J. (2003). Substance use disorders. In R. E. Hales & S. C. Yudofsky (Eds.), *The American Psychiatric Publishing textbook of clinical psychiatry* (4th ed., pp. 309-377). Washington, DC: American Psychiatric Publishing.
- Maisto, S. A., O'Farrell, T. J., Connors, G. J., McKay, J. R., & Pelcovits, M. (1988). Alcoholics' attributions of factors affecting their relapse to drinking and reasons for terminating relapse events following marital therapy. *Addictive Behaviors, 13*, 79-82. doi:10.1016/0306-4603(88)90028-7

Maric, N., Myin-Germeys, I., Delespaul, P., de Graaf, R., Vollebergh, W., & Van Os, J. (2004).

Is our concept of schizophrenia influenced by Berkson's bias? *Social Psychiatry and Psychiatric Epidemiology*, 39, 600-605. doi:10.1007/s00127-004-0803-z

Marschall, D., Sanftner, J., & Tangney, J. (1994). *The State Shame and Guilt Scale*.

Unpublished manuscript, George Mason University.

Martin, C. S., Steinley, D. L., Verges A., & Sher, K. J. (2011). Letter to the editor: The proposed

2/11 symptom algorithm for DSM-5 substance-use disorders is too lenient.

Psychological Medicine, 41, 2008-2010. doi:10.1017/S0033291711000717

Martin, M. W. (1999). Alcoholism as sickness and wrongdoing. *Journal for the Theory of*

Social Behaviour, 29, 109-131. doi:10.1111/1468-5914.00094

Maxwell, M. A. (1984). *The AA experience*. New York: McGraw-Hill.

McKay, J. R., Maisto, S. A., & O'Farrell, T. J. (1996). Alcoholics' perceptions of factors in the

onset and termination of relapses and the maintenance of abstinence: Results from a 30-

month follow-up. *Psychology of Addictive Behaviors*, 10, 167-180. doi:10.1037//0893-

164X.10.3.167

McLellan, A., Lewis, D., O'Brien, C., & Kleber, H. (2000). Drug dependence, a chronic medical

illness: Implications for treatment, insurance, and outcomes evaluation. *Journal of the*

American Medical Association, 284, 1689-1695. doi:10.1001/jama.284.13.1689

Mehta, S., & Farina, A. (1997). Is being "sick" really better? Effect of the disease view of

mental disorder on stigma. *Journal of Social and Clinical Psychology*, 16, 405-419.

doi:10.1521/jscp.1997.16.4.405

- Mewton, L., Slade, T., McBride, O., Grove, R., & Teesson, M. (2011). An evaluation of the proposed DSM-5 alcohol use disorder criteria using Australian national data. *Addiction*, *106*, 941-950. doi:10.1111/j.1360-0443.2010.03340.x
- Milam, J. R. & Ketcham, K. (1981). *Under the influence*. Seattle, WA: Madrona.
- Moore, M. H., & Gerstein, D. R. (1981). *Alcohol and public policy: Beyond the shadow of prohibition*. Washington, DC: National Academy Press.
- Moos, R. H., Finney, J. W., & Cronkite, R. C. (1990). *Alcoholism treatment: Context, process, and outcome*. New York: Oxford University Press.
- Morgenstern, J., & McCrady, B. S. (1992). Curative factors in alcohol and drug treatment: Behavioral and disease model perspectives. *British Journal of Addiction*, *87*, 901-912.
- Moss, H. B., Chen, C. M., & Yi, H. (2007). Subtypes of alcohol dependence in a nationally representative sample. *Drug and Alcohol Dependence*, *91*, 149-158.
doi:10.1016/j.drugalcdep.2007.05.016
- National Institute on Alcohol Abuse and Alcoholism. (2010). Alcohol use and alcohol use disorders in the United States, a 3-year follow up: Main findings from the 2004-2005 Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). *U.S. alcohol epidemiologic data reference manual*, 8(2) (NIH Publications No. 10-7677). Retrieved from <http://www.niaaa.nih.gov/publications>
- National Institute on Alcohol Abuse and Alcoholism. (n.d.-a). *Alcohol use disorders*. Retrieved from <http://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/alcohol-use-disorders>
- National Institute on Alcohol Abuse and Alcoholism. (n.d.-b). *Support & treatment*. Retrieved from <http://www.niaaa.nih.gov/alcohol-health/support-treatment>

- National Institute on Drug Abuse. (2010). *Drugs, brains, and behavior: The science of addiction*. Retrieved from <http://www.drugabuse.gov/publications/science-addiction/drug-abuse-addiction>
- National Institute on Drug Abuse. (2012). *The science of drug abuse and addiction*. Retrieved from <http://www.drugabuse.gov/publications/media-guide/science-drug-abuse-addiction>
- National Institute on Drug Abuse. (2013). *Fear of stigma: Does it hold you back?* Retrieved from <http://teens.drugabuse.gov/blog/post/fear-stigma-does-it-hold-you-back>
- National Institute on Drug Abuse. (n.d.). *Addiction: It's a brain disease beyond a reasonable doubt*. Retrieved from www.drugabuse.gov/sites/default/files/addictionscience.ppt
- National Institutes of Mental Health. (2013, April 23). *Director's update: Mental disorders as brain disorders: Thomas Insel at TEDxCaltech*. Retrieved from <http://www.nimh.nih.gov/about/updates/2013/mental-disorders-as-brain-disorders-thomas-insel-at-tedxcaltech.shtml>
- O'Brien, C., & McLellan, A. (1996). Myths about the treatment of addiction. *Lancet*, 347, 237-240. doi:10.1016/S0140-6736(96)90409-2
- Ogborne, A., Wild, T., Braun, K., & Newton-Taylor, B. (1998). Measuring treatment process beliefs among staff of specialized addiction treatment services. *Journal of Substance Abuse Treatment*, 15, 301-312. doi:10.1016/S0740-5472(97)00196-7
- Paolacci, G., Chandler, J., & Ipeirotis, P. G. (2010). Running experiments on Amazon Mechanical Turk. *Judgment and Decision Making*, 5, 411-419.
- Paulhus, D. L., & Carey, J. M. (2011). The FAD-Plus: Measuring lay beliefs regarding free will and related constructs. *Journal of Personality Assessment*, 93, 96-104. doi:10.1080/00223891.2010.528483

- Peele, S. (1989). *The diseasing of America*. Lexington, MA: D. C. Heath.
- Pescosolido, B., Martin, J., Long, J., Medina, T., Phelan, J., & Link, B. (2010). "A disease like any other?" A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *American Journal of Psychiatry*, 167, 1321-1330.
doi:10.1176/appi.ajp.2010.09121743
- Phelan, J. C. (2005). Geneticization of deviant behavior and consequences for stigma: The case of mental illness. *Journal of Health and Social Behavior*, 46, 307-322.
doi:10.1177/002214650504600401
- Pickens, R. W., & Svikis, D. S. (1988). *Biological vulnerability to drug abuse*. Rockville, MD: U.S. Department of Health and Human Services.
- Randles, D., & Tracy, J. L. (2013). Nonverbal displays of shame predict relapse and declining health in recovering alcoholics. *Clinical Psychological Science*, 1, 149-155.
doi:10.1177/2167702612470645
- Read, J., & Law, A. (1999). The relationship of causal beliefs and contact with users of mental health services to attitudes to the "mentally ill". *International Journal of Social Psychiatry*, 45, 216-229. doi:10.1177/002076409904500309
- Rothman, D. J. (1971). *The discovery of the asylum: Social order and disorder in the new republic*. Boston, MA: Little Brown.
- Ruan, W., Goldstein, R. B., Chou, S., Smith, S. M., Saha, T. D., Pickering, R. P., & ... Grant, B. F. (2008). The Alcohol Use Disorder and Associated Disabilities Interview Schedule-IV (AUDADIS-IV): Reliability of new psychiatric diagnostic modules and risk factors in a general population sample. *Drug and Alcohol Dependence*, 92, 27-36.
doi:10.1016/j.drugalcdep.2007.06.001

- Schaler, J. A. (1995). The addiction belief scale. *International Journal of the Addictions*, 30, 117-134. doi:10.3109/10826089509060737
- Schaler, J. A. (2000). *Addiction is a choice*. Chicago, IL: Open Court Publishing.
- Schomerus, G., Corrigan, P. W., Klauer, T., Kuwert, P., Freyberger, H. J., & Lucht, M. (2011). Self-stigma in alcohol dependence: Consequences for drinking-refusal self-efficacy. *Drug and Alcohol Dependence*, 114, 12-17. doi:10.1016/j.drugalcdep.2010.08.013
- Schomerus, G., Lucht, M., Holzinger, A., Matschinger, H., Carta, M., & Angermeyer, M. (2011). The stigma of alcohol dependence compared with other mental disorders: A review of population studies. *Alcohol and Alcoholism*, 46, 105-112. doi:10.1093/alcalc/agq089
- Schwartz, J. M. (1998). Neuroanatomical aspects of cognitive-behavioural therapy response in obsessive-compulsive disorder: An evolving perspective on brain and behaviour. *British Journal of Psychiatry*, 173(Suppl. 35), 38-44.
- Shapiro, D. N., Chandler, J., & Mueller, P. A. (2013). Using Mechanical Turk to study clinical populations. *Clinical Psychological Science*, 1, 213-220. doi:10.1177/2167702612469015
- Shaw, S. J., Cartwright, A. K. J., Spratley, T. A., & Harwin, J. (1978). *Responding to drinking problems*. London: Croom Helm.
- Sitharthan, T., Job, R. F. S., Kavanagh, D. J., Sitharthan, G., & Hough, M. (2003). Development of a controlled drinking self-efficacy scale and appraising its relation to alcohol dependence. *Journal of Clinical Psychology*, 59, 351-362. doi:10.1002/jclp.10127
- Smith, S. M., Dawson, D. A., Goldstein, R. B., & Grant, B. F. (2010). Examining perceived alcoholism stigma effect on racial-ethnic disparities in treatment and quality of life among alcoholics. *Journal of Studies on Alcohol and Drugs*, 71, 231-236.

- Sobell, L. C., Cunningham, J. A., & Sobell, M. B. (1996). Recovery from alcohol problems with and without treatment: Prevalence in two population surveys. *American Journal of Public Health, 86*, 966-972. doi:10.2105/AJPH.86.7.966
- Stillman, T. F., & Baumeister, R. F. (2010). Guilty, free, and wise: Determinism and psychopathy diminish learning from negative emotions. *Journal of Experimental Social Psychology, 46*, 951-960. doi:10.1016/j.jesp.2010.05.012
- Stillman, T. F., Baumeister, R. F., Vohs, K. D., Lambert, N. M., Fincham, F. D., & Brewer, L. E. (2010). Personal philosophy and personnel achievement: Belief in free will predicts better job performance. *Social Psychological and Personality Science, 1*, 43-50. doi:10.1177/1948550609351600
- Stinson, F. S., Grant, B. F., Dawson, D. A., Ruan, W. J., Huang, B., & Saha, T. (2005). Comorbidity between DSM-IV alcohol and specific drug use disorders in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Drug and Alcohol Dependence, 80*, 105-116. doi:10.1016/j.drugalcdep.2005.03.009
- Substance Abuse and Mental Health Services Administration. (2008). *Summary report: Caravan Survey for SAMHSA on addictions and recovery*. Retrieved from http://www.samhsa.gov/attitudes/CARAVAN_LongReport.pdf
- Szalavitz, M. (2012, May 14). DSM-5 could categorize 40% of college students as alcoholics. *Time*. Retrieved from <http://healthland.time.com>
- Szasz, T. S. (2007). *The medicalization of everyday life: Selected essays*. New York: Syracuse University Press.
- Tangney, J. P., & Dearing, R. L. (2002). *Shame and guilt*. New York: Guilford Press.

- Tracy, J. L., Robins, R. W., & Tangney, J. P. (Eds.). (2007). *The self-conscious emotions: Theory and research*. New York: Guilford Press.
- Tsai, J., Ford, E., Li, C., Pearson, W., & Zhao, G. (2010). Binge drinking and suboptimal self-rated health among adult drinkers. *Alcoholism: Clinical and Experimental Research*, 34, 1465-1471. doi:10.1111/j.1530-0277.2010.01231.x
- Vaillant, G. E. (1995). *The natural history of alcoholism revisited*. Cambridge, MA: Harvard University Press.
- Velten, E. (1968). A laboratory task for induction of mood states. *Behaviour Research and Therapy*, 6, 473-482. doi:10.1016/0005-7967(68)90028-4
- Vohs, K. D., & Baumeister, R. F. (2009). Addiction and free will [Editorial]. *Addiction Research & Theory*, 17, 231-235. doi:10.1080/16066350802567103
- Vohs, K. D., & Schooler, J. W. (2008). The value of believing in free will: Encouraging a belief in determinism increases cheating. *Psychological Science*, 19, 49-54. doi:10.1111/j.1467-9280.2008.02045.x
- Wakefield, J. C. (2013). DSM-5: An overview of changes and controversies. *Clinical Social Work Journal*, 41, 139-154. doi:10.1007/s10615-013-0445-2
- Wallace, J. (1996). Theory of 12-Step-oriented treatment. In F. Rotgers, D. S. Keller, & J. Morgenstern (Eds.), *Treating substance abuse: Theory and technique* (pp. 13-36). New York: Guilford Press.
- Waller, N. G., Kojetin, B. A., Bouchard, T. J., Lykken, D. T., & Tellegen, A. (1990). Genetic and environmental influences on religious interests, attitudes, and values: A study of twins reared apart and together. *Psychological Science*, 1, 138-142. doi:10.1111/j.1467-9280.1990.tb00083.x

- Wallston, B. S., & Wallston, K. A. (1978). Locus of control and health: A review of the literature. *Health Education & Behavior*, 6, 107-117.
doi:10.1177/109019817800600102
- Wallston, K., Stein, M., & Smith, C. (1994). Form C of the MHLC scales: A condition-specific measure of locus of control. *Journal of Personality Assessment*, 63, 534-553.
doi:10.1207/s15327752jpa6303_10
- Willenbring, M. L. (2010). The past and future of research on treatment of alcohol dependence. *Alcohol Health & Research World*, 33, 55-63.
- Williams, G. C., Gagne, M., Ryan, R. M., & Deci, E. (2002). Facilitating autonomous motivation for smoking cessation. *Health Psychology*, 21, 40-50. doi:10.1037//0278-6133.21.1.40
- Williams, G. C., Grow, V. M., Freedman, Z., Ryan, R. M., & Deci, E. L. (1996). Motivational predictors of weight-loss and weight loss maintenance. *Journal of Personality and Social Psychology*, 70, 115-126. doi:10.1037//0022-3514.70.1.115
- Winters, K. C., Stinchfield, R. D., Henly, G. A., & Schwartz, R. H. (1990). Validity of adolescent self-report of alcohol and other drug involvement. *Substance Use and Misuse*, 25, 1379-1395. doi:10.3109/10826089009068469
- World Values Survey. (2011). *2010-2012 World Values Survey revised October 2011*. Retrieved from http://www.worldvaluessurvey.org/index_surveys
- Zeldman, A., Ryan, R. M., & Fiscella, K. (2004). Motivation, autonomy support, and entity beliefs: Their role in methadone maintenance treatment. *Journal of Social and Clinical Psychology*, 23, 675-696. doi:10.1521/jscp.23.5.675.50744

APPENDIX: MANIPULATION

Psychosocial Model Condition

Instructions: You will now be presented with 15 scientific statements about one of the dominant models of alcohol use and addiction. Please spend about one minute thoroughly reading through and thinking deeply about each statement. After you have read through all of the statements you will need to answer a few questions about them.

Statements

Scientific studies have shown that people addicted to alcohol can learn to control and moderate their drinking and that complete abstinence is not the only way to overcome an addiction to alcohol.

Scientific studies show that problem drinkers who experience physiological withdrawal do not completely lose control of their drinking, but are able to successfully refrain from drinking with enough willpower and determination.

Addiction to alcohol occurs as the result of a long and complex series of life decisions, judgments, and choices that people make over a long period of time.

Science has demonstrated that chronic problem drinkers often voluntarily cut back their drinking or abstain completely, and that their decision to drink depends on the rewards or costs that they believe will follow alcohol consumption.

Consuming large amounts of alcohol is a lifestyle choice, not a medical disease. The choice of problem drinkers to drink is influenced by their perceptions of the short-term and long-term costs and benefits of drinking.

Research shows that heavy drinkers can and do learn to change their alcohol-abusing ways of life, without receiving professional help, far more often than doctors who claim alcoholism is a disease would have us believe.

Alcohol serves a meaningful social, psychological, or emotional purpose in the heavy drinker's life. Although it is maladaptive, heavy consumption of alcohol is a life choice, not a loss of control resulting from a medical disease.

Heavy drinkers are not victims of a disease, passive patients who need to be treated by medical experts, but individuals who are capable of exercising control and assuming responsibility over their alcohol problem.

Research has shown that problem drinkers who are motivated to change and who are persistent in the face of setbacks can overcome their alcohol problems if they are given the appropriate tools and strategies for reshaping their lives.

Overcoming an addiction to alcohol is a difficult undertaking that requires determination to reorganize one's lifestyle and to learn to live differently.

Problem drinkers are not helpless victims of a disease; rather their drinking plays a meaningful role in how they cope with life.

The heavy drinker's choice to drink alcohol depends on a variety of psychological, social, cultural, and religious variables. Therefore, the drinker's choice to drink alcohol on any given occasion is influenced by the similar sorts of variables that generally influence us all.

There is no one biological cause of alcoholism; alcohol abuse is the result of a range of physiological, psychological, and social characteristics that together predispose a person to drink to excess.

In order to gain control of their drinking habits, heavy drinkers must take personal responsibility for their alcohol addiction. This resolve must be followed by actions to reshape their way of life, replacing alcohol with other ways of meeting life's challenges.

Research has shown that in order to overcome an addiction to alcohol, problem drinkers must accept responsibility and take an active role in overcoming their problem.

Questions

Instructions: Please answer the following questions about the statements that you just read. According to the scientific statements that I just read ...

Note: To be included in data analyses participants need to answer at least three of the following four questions correctly, including Question 4.

1. What is the primary cause of alcohol addiction?

Answer: lack of self-control/willpower/determination/persistence, decisions/social surroundings/lifestyle, social/emotional/psychological reasons

2. Why do some people with an alcohol addiction continue to drink too much even if it hurts their life?

Answer: lack of self-control/willpower/determination/persistence; decisions/social surroundings/lifestyle; social/emotional/psychological reasons

3. What does someone with an alcohol addiction need to get rid of their addiction?

Answer: learn to control/moderate drinking; self-control/willpower/determination/persistence; change decisions/social surroundings/lifestyle; learn other ways to deal with social/emotional/psychological issues; take responsibility/active role in problem; self-control or lifestyle change related therapy

4. What is the amount of control someone with an alcohol addiction has over their alcohol consumption?

Response scale (5-point scale): 1 (no control), 2 (not much control), 3 (neutral), 4 (a fair bit of control), 5 (full control)

Answer: 4 or 5.

Disease Model Condition

Instructions: You will now be presented with 15 scientific statements about one of the dominant models of alcohol use and addiction. Please spend about one minute thoroughly reading through and thinking deeply about each statement. After you have read through all of the statements you will need to answer a few questions about them.

Statements

There is overwhelming scientific evidence that alcoholism is a genetic physiological disease.

Alcoholism is a chronic genetic disease which progresses from an early physiological susceptibility into an addiction characterized by physiological dependence and a loss of control over drinking.

Scientific studies have shown that people addicted to alcohol cannot learn to control and moderate their drinking and that complete abstinence is the only way to overcome an addiction to alcohol.

The alcoholic suffers from a disease just like any other disease, such as high blood pressure, epilepsy, and diabetes.

Alcoholism is a physiological disease that is not the result of psychological, social, or cultural problems. The real problem is the physiological dependence on alcohol, which then creates these other problems.

Psychological, cultural, and social factors have no effect on whether or not a person becomes an alcoholic. Physiology, not psychology, determines whether one drinker will become addicted to alcohol and another will not.

The idea that alcoholics lack the strength of character to change their ways is a myth with no firm basis in fact. Science shows that alcoholism is a disease that rules over its victims.

When an alcoholic looks back on life, the individual should say, "That's what alcoholism did to me," not "That's what I did." Saying the words "I am an alcoholic" should have the same moral overtones as the words, "I have an allergy to pollen."

Alcoholics can no more control their drinking behavior than epileptics can control their seizures. Since physiological dependence controls alcoholics' behavior, they are powerless over addiction and cannot be held responsible for it.

The alcoholic is a sick, not a bad, person. Such a person is both physically and psychologically sick and it is the disease alone that is responsible for the person's behavior.

Alcoholics are not morally or psychologically defective people, but innocent victims of a chronic and progressive disease.

Alcoholics who commit crimes are not primarily criminals or mentally disturbed individuals but victims of an addiction they are helpless to control.

The alcoholic's anger, fear, depression, immaturity, and defensiveness are all the result of a sick brain.

Alcoholism is a serious and potentially fatal illness. It is not the alcoholic but rather the person's illness that should be blamed for inappropriate behavior.

Alcoholism is not a self-inflicted illness but a very serious physiological disease that fully deserves the best in medical protection and treatment.

Questions (*Note: Questions are identical to those posed in the psychosocial model condition; correct answers are different, of course.*)

Instructions: Please answer the following questions about the statements that you just read. According to the scientific statements that I just read ...

Note: To be included in data analyses participants need to answer at least three of the following four questions correctly, including Question 4.

1. What is the primary cause of alcohol addiction?

Answers: Genetic/physiological/biological problem or disease or sickness; brain disease; sick brain; illness; dependence; loss of control; alcohol addiction; alcoholism

2. Why do some people with an alcohol addiction continue to drink too much even if it hurts their life?

Answers: Genetic/physiological/biological problem or disease or sickness; brain disease; sick brain; illness; dependence; loss of control; alcohol addiction; alcoholism; helplessness

3. What does someone with an alcohol addiction need to get rid of their addiction?

Answers: abstinence; treatment; medicine; hospital; medical treatment or care; abstinence-related therapy or support groups

4. What is the amount of control someone with an alcohol addiction has over their alcohol consumption?

Response scale (5-point scale): 1 (no control), 2 (not much control), 3 (neutral), 4 (a fair bit of control), 5 (full control)

Answers: 1 or 2.

Neutral Condition

Instructions: You will now be presented with 15 scientific statements one at a time. Please spend about one minute thoroughly reading through and thinking deeply about each statement. After you have read through all of the statements you will need to answer a few questions about them.

Statements

Wyoming has a population of around half a million residents — a population dozens of times smaller than America’s most populous state, California, making it the state with the lowest population.

Though Minnesota is known as the Land of 10,000 lakes, Wisconsin actually has substantially more lakes, giving it the highest number of lakes outside of Alaska. There are many lakes so tiny that they don’t even have names.

Buenos Aires, Los Angeles, Cape Town, and Sydney are each thousands of miles apart and are known for having unusually pleasant year-round climates, and they are all almost *identical* distances from the Equator.

California, the Golden State boasts hundreds of miles of picturesque coastline, but only one point registers a record-setting depth below sea level: Death Valley.

Kola Superdeep Borehole which is well over ten thousand meters deep is deemed as the deepest hole drilled for the purpose of scientific research. A massive hydrogen deposit was surprisingly discovered during the drilling.

Outside of Alaska’s enormous coastline, the longest coastline in the U.S. is located in the Sunshine State, Florida, with over a thousand miles of beach access. Beachgoers can swim in the Atlantic Ocean to the east and the Gulf of Mexico to the west.

Though Mt. Everest is the highest altitude in terms of sea level on the planet, Mount Chimborazo is the closest to the moon. The Marianas Trench is the lowest place on earth.

The second longest geographical name that is accepted in the world is “Taumatawhakatangihangak oauauotamateaturipukaka pikimaungahoronukupokaiwhe nua kitanatahu” which is a hill in New Zealand.

The flag of the Philippines is the only national flag that is flown differently during times of peace or war. A portion of the flag is blue, while the other is red. The blue portion is flown on top in times of peace and the red portion is flown in war time.

The only city in the United States to celebrate Halloween on the October 30th instead of October 31st is Carson City, Nevada. October 31st is Nevada Day and is celebrated with a large street party.

The roads on the island of Guam are made with coral. Guam has no sand. The sand on the beaches is actually ground coral. When concrete is mixed, the coral sand is used instead of importing regular sand from thousands of miles away.

The Vatican city is the smallest country in the world at less than one square mile. That is smaller than the average city! The largest country is Russia.

The Rainbow Bridge is the world's largest natural bridge. It is located at the base of Navajo Mountain, Utah and is as long as a football field.

The shortest place name is "Å" which is located in both Sweden and Norway. In Scandinavian languages, "Å" means "river."

Alaska is the most Northern, Eastern and Western state in all of America. It is the only state that enters the "Eastern Hemisphere" making it both the most Eastern lying and Western lying state.

Questions

Instructions: According to these scientific statements ...

Note: Only participants answering at least three of the questions correctly will be included in the data set.

1. What is the smallest country in the world?

Answer: Vatican city.

2. The flag of which country is flown differently in times of war and peace?

Answer: Philippines.

3. What does the place name "Å" stand for?

Answer: River.

4. Which of the American states is the only one to enter the "Eastern Hemisphere"?

Answer: Alaska.