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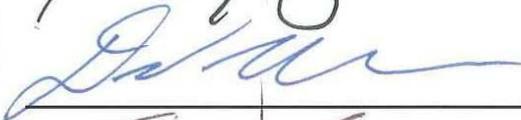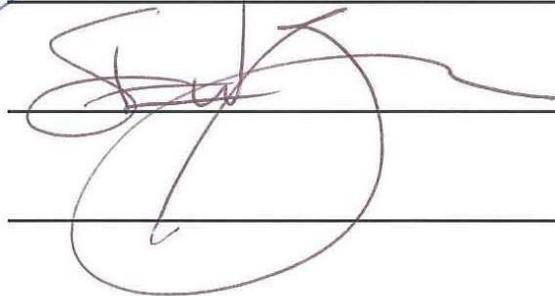
Does Clinical Distress Impact Attempts to Moderate in Problem Drinkers?

Analysis of Outcomes from a Randomized Clinical Trial

BY

WILLIAM P. CAMPBELL

B.A., English, Reed College, 1988

THESIS

Submitted in Partial Fulfillment of the
Requirements for the Degree of

**Master of Arts
Psychology**

The University of New Mexico
Albuquerque, New Mexico

July, 2010

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ABSTRACT

This study examined the relationship between participant's level of psychological distress and their attempts to control their problematic alcohol consumption in a randomized clinical trial comparing two web-based Behavioral Self-Control Training (BSCT) interventions. Potential participants in the trial were screened for both severe mental illness and symptoms of alcohol dependence. Based on prevailing theories, as well as research on individuals diagnosed with co-morbid anxious or mood disorders with alcohol use disorders, it was hypothesized that participants reporting higher levels of clinical distress would have worse outcomes. However, the results of this trial demonstrated that participants reporting higher levels of clinical distress at intake, as assessed by the Brief Symptom Inventory-18 (BSI), were more successful and reducing both number of drinks they consumed per drinking occasion as well as the number of days they drank per drinking period at 3 and 12-month follow-up, relative to those who reported less or no clinical distress at intake.

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Introduction

It's commonly assumed in the world of substance abuse treatment that patients suffering co-morbid mental illness are beset with more difficulties, faced with more uncertain outcomes from treatment, and are more likely to relapse into use and or distress (Kranzler et al, 2003; Grant et al, 2006; Tiet & Mausbach, 2007). Moreover, much less is known about etiology, moderators, mechanisms and interactions of co-morbid disorders than about individually occurring clinical disorders (Kranzler et al, 2003; Rosenthal & Westreich, 1999). One of the many questions still unanswered amidst all this uncertainty is what influence the level of substance use has on treatment outcome. In particular, there is little evidence to help clinicians determine whether the treatment of depressed or anxious individuals who *abuse* alcohol will follow the same, albeit less severe, course as those depressed or anxious individuals who are *dependant* on alcohol. Looked at another way, it is not known whether alcohol-abusing individuals who are severely depressed or anxious are less likely to cut back on their drinking than abusers who are only marginally anxious or depressed.

The question of alcohol abuse with co-morbid psychological distress is important, in part, because it is frequently overshadowed by co-morbidity associated with the stereotypic case of alcohol dependence. It is typical for researchers and clinicians to imagine serious co-morbid cases when thinking of depressed or anxious alcoholics, but this focus is, actually, on the exceptional case, rather than the normative one. Epidemiological research reveals that there are four times as many individuals with alcohol problems who do not exhibit symptoms of dependence as there is who meet the criteria for dependence (National Institute in Alcohol Abuse and Alcoholism,

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2000). Moreover, a more recent report indicates that the number of abusive drinkers is increasing while the prevalence of alcohol dependence is declining (NIAAA, 2004). The Institute of Medicine's (I990) twenty year-old characterization these non-dependent "problem drinkers" as a large but under-served group is more true today than ever, as is it's identification of the pressing need to conduct more research upon, and develop more interventions for, this group.

The Concept of an Alcohol Use Disorder

The categorical distinction between the diagnoses of abuse and dependence currently employed by the DSM-IV TR was originally derived from the "bi-axial" concept of alcohol and drug disorders proposed for the DSM-III by Edwards (1986). DSM-III diagnostic criteria revolved around efforts to reliably identify the "addict" presenting for treatment, and characterized substance abuse along the two orthogonal dimensions of psychological and physiological dependence on one axis and socio-personal consequences on the other. The concept of "abuse" in current use is still focused largely on the latter, and entails repeated use of alcohol that results in and recurs despite negative social, occupational or legal consequences. The diagnosis of dependence may or may not entail the same patterns of pathological use as the abuse diagnosis, but includes other physiological and behavioral symptoms that characterize the dominance the alcohol has assumed over the user's life, such as tolerance, which is defined as the need for and consumption of more alcohol to achieve the desired effect, as well as withdrawal, which is defined as a severe adverse reaction to insufficient doses of alcohol.

One problem with the abuse/dependence distinction, however, is that while the diagnosis of alcohol dependence has excellent proven clinical reliability and validity, the diagnosis of abuse does not (see Hasin, 2003 for review of studies examining this issue). Moreover, there are no clearly distinct markers that distinguish abusive patterns from dependent ones. For example, a hangover may be construed as a form of withdrawal, but it is unclear how many hangovers an individual must have before his or her overall use is best characterized with withdrawal symptoms. Largely for this reason, the two traditional axes proposed by Edwards will be collapsed onto one continuum in the new, more dimensionally inclined DSM-V, with the symptoms of tolerance and withdrawal appearing as but two of eleven possible criteria for diagnosis of "alcohol use disorder" (DSM-V task force, 2010). Further, some researchers, speaking through NIAAA publications, have conceptualized the continuum of use disorder as one that begins with relatively isolated "incidents", and extends into an ever more heavy pattern of consumption *that leaves the drinker increasingly unresponsive to external circumstances* (Hasin, 2003).

The idea that the relative severity of an alcohol use disorder may best be gauged by an increasing unresponsiveness to non-alcohol related external stimuli is central to this thesis. That is, with regards to alcohol use disorders, it may not so much matter how pathological use or psycho-social impairment are topographically defined; the key may actually be, from a functional point of view, that severity be conceptualized as increasing unresponsiveness, or perhaps even as a maladaptive pattern of responsiveness, to external circumstances and adverse consequences of behavior due to the control that alcohol is having over the drinker. That is, responsiveness to other

environmental stimuli significantly decreases relative to the stimulus of alcohol, to the detriment of the individual; thus the DSM-IV categorical distinctions abuse and dependence, when viewed on a continuum, would represent lesser to greater unresponsiveness to non-alcohol related stimuli. Conceptualizing alcohol use disorders in this way is central to the concept of "self-regulation" (about which more will be discussed below), as well as to the behavioral protocols of self-control, upon which the interventions tested in the present study are based.

The Current Study

As mentioned above, an unanswered question about drinkers at the less severe end of the alcohol use disorder spectrum is the effect that co-morbid clinical distress has on the etiology and maintenance of their drinking. Most clinicians, in light of the significant impairment and intractable course typical to those suffering from dual diagnoses, frame their research and treatment in terms of trying to understand what is assumed to be a mutually antagonistic relationship between alcohol misuse, depression and anxiety. It is also typically assumed that co-morbidity affects alcohol use disorders similarly across the severity spectrum.

Just such a "downward inertia" hypothesis was formulated for this study about a group of problem, non-dependant drinkers recently recruited into a randomized clinical trial of an intervention for problem drinkers. The trial compared two web-based interventions based on Behavioral Self-Control Training (BSCT) protocols, with a treatment goal of either moderate drinking or abstinence. In addition to being screened for level of severity of drinking, participants in the trial were screened for severe mental illnesses and, once in the study, administered the Brief Symptom Inventory 18

(BSI-18, Derogatis, 2000), a brief screening instrument designed to assess levels of psychological distress in community samples. Just as level of drinking severity, as measured by a Michigan Alcohol Screening Test (MAST, Selzer, 1971) score of <20, has been found to be highly predictive of successful moderate drinking outcomes (Miller et al, 1992), it was hoped that administration of the BSI-18 would provide further incremental validity with regards to who is more likely to succeed with the moderation protocol. The supposition, following standard co-morbidity theory, was that those participants presenting with higher levels of clinical distress would have poorer outcomes than those participants who reported no psychological distress; the BSI-18 was administered in the hopes that it might prove its worth as an additional intake screen for individuals seeking treatment by providing a cut-off score above which clinicians could rule out the recommendation that the individual attempt BSCT or some similar moderation protocol.

However, this assumption was based on limited empirical evidence. To our knowledge, there has never been a study to test specifically whether non-dependent problem drinkers' attempts to control their drinking are moderated by level of clinical distress. Further, the co-occurrence of alcohol use with anxiety and depression at sub-clinical levels is so prevalent as to obtain a certain degree of cultural orthodoxy. Declaring that one "needs a drink" to soothe one's nerves or lift one's spirits would likely elicit more sympathy than concern from anyone listening. How this relatively normative behavior devolves into a co-morbid clinical disorder remains an open question.

In order to address this issue, we will first of all review the epidemiological evidence on the prevalence of co-morbid alcohol use disorders with anxiety and depression, and then proceed to examine some of the research that attempts to explain the interaction between alcohol use disorders, anxiety and depression, and negative moods in general. Then we will elucidate the concept of self-regulation and its relationship to the processes of both addiction and affective regulation, specifically with regards to the role of a drinker's increasing difficulties in managing his or her relationship to external stimuli. This exposition of self-regulation is important here, since it is foundational to the rationale of BSCT, a primary intervention for non-dependant problem drinkers, and the basis of the protocols tested in this study.

Co-morbid Alcohol Abuse, Depression and Anxiety: Epidemiology

The National Epidemiologic Survey on Alcohol and Related Conditions, the first wave of which was conducted by the NIAAA during 2001-02, comprised a sample of 43,093 ethnically and geographically diverse respondents. It is the first national epidemiological survey to control, according to stringent DSM-IV criteria, for onset and interaction certain mood (major depression, dysthymia, mania or hypomania) and anxiety (Panic disorder with or without agoraphobia, social phobia, specific phobia or generalized anxiety) disorders with substance use disorders (Grant, et al, 2006). That's the good news. The bad news for our current study is that by focusing on diagnostic thresholds for disorders, not much light is shed upon sub-threshold issues of co-morbidity.

With regards to clinical levels of the above specified disorders, the national 12-month prevalence of mood, anxiety and alcohol use disorders were 9.21, 11.08 and 9.35

percent, respectively. A person suffering from one of these mood or anxiety disorders is on average two-and-a-half times more likely to have an alcohol use disorder than a person without clinical levels of anxiety or depression. Looked at the other way, a person suffering from an alcohol use disorder is 17 times more likely than a non-disordered individual to have a mood disorder, and 13 times more likely to have an anxiety disorder. Interestingly, the above rates are double, across the board, for those respondents who sought treatment in the prior 12 months (i.e. 40.7 percent of individuals with AUD seeking treatment had at least one current mood disorder, while 33 percent had at least one current anxiety disorder). *The exception to the overall pattern* is the level of association between alcohol abuse and these specific disorders; this relationship was non-significant in almost all cases. Thus, the results reported here are driven almost entirely by dependent drinkers (Grant et al, 2006).

The survey also determined that in the overwhelming majority of cases, anxiety and mood disorders precede or occur independently of substance use disorders; less that 1.0 percent of all mood or anxiety disorders were substance-induced. This finding was in contrast to previous epidemiological studies (Kessler, 2004; Kranzler and Rosenthal, 2003; Schuckit et al, 1997) which reported a much more mixed picture with regards to the course and causal relationship between the two disorders.

These epidemiological findings have two important implications for the current study. The first is that there is little population-based evidence to support the notion that co-morbid depression and anxiety are a likely result of problematic drinking. The second is that there is a bias in the conceptualization of both the etiology and treatment of co-morbid illness that derives from what is learned about the more severe cases.

Co-morbid Alcohol Abuse, Depression and Anxiety: Theory and Research

There are a variety of theories proffered to explain the deleterious relationship between psychological disorders and substance abuse (see Muesser, et al for a review), but none are more common than the self-medication hypothesis (Khantzian, 1985), which posits that psychologically distressed individuals abuse alcohol and/or drugs in an attempt to alleviate the intolerable cognitive and affective symptoms that arise from their primary disorder. This hypothesis makes the assumption that co-morbid addiction evolves both from the diminishing returns of the chosen substance's effectiveness in alleviating symptoms, as well as from the already low functioning individuals' ability to manage their symptoms on their own. In a broad sense, the self-medication hypothesis reflects a general conception of the problematic interaction between substances and psychological distress, in so far as dysphoric individuals are thought to maladaptively use alcohol or drugs to regulate intolerable and overwhelming physiological and psychological states (Henwood & Padgett, 2007).

The self-medication hypothesis has been tested directly on specific diagnoses, and has been found to have solid, if paradoxical support. For example, Carrigan and Randall, in an extensive review of studies exploring the relationship between alcohol use and social phobias, found that individuals suffering from the disorder did indeed use alcohol to cope, but found less than conclusive support for the notion that drinking actually reduced anxiety (Carrigan and Randall, 2003). Similarly, Thomas et al found that administering paroxetine effectively reduced anxious symptoms among individuals suffering from co-morbid alcohol use disorders and social anxiety, but this relief did not then also reduce their drinking (Thomas et al, 2008). Finally, Bolton et al, in an analysis

of the NESARC data, likewise found that one quarter of individuals in the general population suffering from a mood disorder used substances in an attempt to reduce their symptoms, even though doing so exacerbated their illness (Bolton et al, 2009). The question arises as to why individuals suffering psychological distress persist in medicating themselves when the treatment essentially doesn't work or in fact makes them worse.

An answer to this question is potentially provided in less explicit investigations of the self-medication dynamic; that is, in research on the relationship between substances and non-clinical, negative mood states in general. First of all, there is the issue of a drinker's expectations about the effect he or she will receive from alcohol. Evans and Dunn (1995) found that drinkers' positive expectancies about the effects of alcohol on their mood were associated with an escalation in both their alcohol consumption and their alcohol related problems; that is, positive expectation lead to more drinking, but also to the individual's self-obfuscation of the consequences of drinking. Thorberg and Lyvers (2006) showed that these expectations co-existed in addicts along with lower negative mood regulation expectancies, as well as higher reported levels of stress, anxiety and depression compared to controls. So when it comes to dysphoria, addicts report greater negative affect, less faith in their ability to regulate it, and higher expectations about what substances can do to improve their mood. Moreover, in a frequently cited study, Cooper et al (1995) found that there is a direct relationship between the reasons people drink and the problems that arise from their drinking. She showed that people who drink to cope with negative emotions are at far greater risk to develop drinking problems then are people who drink to enhance

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pleasurable experiences; and that, further, the use of alcohol to cope with negative

emotions was inversely related to the ability to cope in other, more adaptive ways.

Grant et al extended this finding by showing that people who drink to cope with

depressed feelings do drink more on days when they are more depressed, and people

who drink to cope with anxiety do in fact drink more on evenings following days with

greater anxious mood, thus specifying sub-types of negative affect self-medication

(Grant, Stewart and Mohr, 2009).

Grant et al's findings, as well as Bolton's, are important in that they show a

significant relationship between depressed mood and drinking to cope with depression.

Prior researchers have not been so successful in showing this relationship. For example,

Swendsen et al, using prospective, experience sampling method, found a distinct

relationship between stress and the self-medication hypothesis, but no support for a

relationship between sadness or broader conceptions of negative affect and drinking

(Swendsen et al, 2000). This may be a result of the fact that the relationship between

depression and alcohol is complicated by the fact that depression has been reported to

be the result of as much as the precursor to alcohol consumption (Merikangas et al,

1996).

There has been a great deal of research conducted around individuals who

report that they drink specifically to reduce daily tension, but again the findings are

somewhat counter-intuitive. The theory of drive reduction (Logan, 1973), has served as

a sort of behavioral equivalent of the self-medication hypothesis, in as much as it posits,

with regards to alcohol consumption, that drinking alcohol is negatively reinforced by

the reduction in tension that follows, whether that tension be associated with stress

(Finn & Phil, 1987), negative self appraisals (Hull, 1981), a subjective feeling of helplessness (McClelland et al, 1972) or simple negative daily events (Carney et al, 2000).

As with the research mentioned above, recent studies, using prospective and daily diary methods rather than cross-sectional or between subjects designs, have found that drinkers who report drinking in order to reduce their stress don't drink significantly more than drinkers who don't equate any particular emotion with their drinking (Schroder & Perrine, 2007). What Shroder and Perrine did find in this population is significantly higher numbers of individuals who report current and lifetime abuse and dependence symptoms. Again it would appear that the self-medication hypothesis functions more by intention than consequence.

With regards to the relationship between depression and anxiety and the onset of problematic drinking, it is evident from the above summary that much has been hypothesized, tested, and written. But questions about the self-medication hypothesis remain. Further, much less has been explored about the relationship between increased depression and anxiety and the *cessation* of drinking. This bias against investigating elevated psychological distress and obtaining control over problem drinking may be due to the fact that most research on co-morbidity assumes a mutually antagonistic and exacerbating relationship between the two disorders. I will now turn a brief overview of self-regulation theory as it pertains to addiction

Self-Regulation Theory

A common factor across the above investigations of the self-medication hypothesis, whether taken narrowly, in relation to a clinical diagnosis, or more broadly,

in relation to the mitigation of general dysphoria, is the individual's use of substances in response to intolerable psychological states. The regular and maladaptive recourse to alcohol as a response to aversive stimuli is construed as a dysfunction of *self-regulation* by many addictions researchers. Since this concept is relevant to the rationale of Behavioral Self-Control Training, it is appropriate that we turn now to a discussion of its theoretical basis and empirical support, as well as its relationship to the etiology and treatment of affective regulation in general, and addictive behaviors in particular.

Miller and Brown (1991) make the case that "self-regulation offers a promising starting point for understanding the interaction of volition with the other determinants of behavior", especially addictive behaviors. At the risk of tautology, addiction may be thought of precisely as a diminished capacity for healthy self-regulation. Here we will assume that addictive behaviors are fundamentally just like any other behaviors, occurring amidst and interacting with a complex array of determinants and consequences (Drew, 1989), but different in precisely this regard; that they entail a degree of failure in self-regulatory processes, specifically with regards to the inability (as defined by subjective intent) to obtain distal reinforcers in the presence of a given proximal reinforcer (Raichlin, 2000).

In the broadest sense, an understanding of self-regulation starts with the assumption that people are either passive observers or active participants in their experience. In so far as they are active participants, they will seek to suppress some phenomena and increase others, a conception of behavior that assumes an interaction between goal-directed behavior and the contingencies of the environment (Miller and Brown, 1991). Self-regulation may be defined as an individual's ongoing management of

this interaction through both automatic and deliberate processes (Kanfer and Gaelick, 1986). Conceived thus, self-regulation encompasses a set of behaviors that includes the ability to form intentions, plan actions and flexibly modify behaviors in response to changing circumstances (Kanfer, 1970). Self-regulation, while often thought of as a dispositional quality in an individual, is not merely a set of learned stimulus-response relations, nor is it a pre-determined result of genetic precursors; it is more accurately thought of as a "level of behavioral/functional organization achieved through complex interactions of biological and social processes" (Diaz & Fruhauf, 1991).

There have been several accounts of the development of self-regulation (e.g. Bowlby, 1960; Piaget, 1976) but one put forward by Diaz and Fruhauf is particularly useful in helping to understand how certain developmental pathways put individuals at risk for the sort of dysregulatory patterns that often give rise to addiction. Like most theories of self-regulation theirs begins with the conception of the homeostatic organism; not as a given outcome, but as a tendency or potential that must be developed. Theoretically, contingent responding by the *caregiver* initially provides a regulatory pattern for the infant's arousal, security and behavioral motor systems. As the child grows, these patterns are gradually internalized, with a significant final stage occurring when he or she begins to represent the patterns internally with private speech. This final stage of development is significant in so far as the child is then considered to have acquired the basics of a repertoire that will allow him or her to respond adaptively and flexibly when "internal regulatory mechanisms are overtaxed by unusually demanding environmental events or situations" (Diaz & Fruhauf, 1991).

The self-regulating response, based on the internalized repertoire, thus enables an individual to manipulate the situation in such a way to restore lost equilibrium.

The utility of this model lies in how it allows the theorist and therapist alike to frame any given context in which "addictive" behaviors occur. Assuming only that organisms tend to seek balanced states—hardly a gross assumption—we are able to conceptualize the function of an individual's maladaptive substance use as a three-fold process: a situation arises which over-taxes a given capacity of "automatic", internal regulatory processes, the individual possesses a deficient repertoire of self-regulatory behaviors, and exhibits an excessive recourse to external structures (substances, relations, institutions) in order to achieve homeostatic functioning. Here "excessive" simply means a pattern that ultimately devolves into a diminished self-regulatory repertoire, further increasing the probability that the addict will repeatedly pursue this same chain of behaviors (Diaz & Fruhauf, 1991). The unique status given to drugs and alcohol in this process derives in part from their potent and immediate reinforcing effects, which serve to increase the probability that these inimical behavioral chains will persist.

Many theories of substance use place emphasis on aspects of addiction (e.g. the drug's biochemical qualities, genetic vulnerabilities, etc.; see Miller & Hester, 2003 for a review) that emphasize organismic or dispositional deficits, but these are different from the organism-environment interaction captured by the concept of self-dysregulation. These other theories are typically construed by researchers as involving deficits that inhering primarily within the individual, regardless of context. Some examples are insensitivity to the interoceptive cues that indicate degree of intoxication (Paredes,

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Jones & Gragory, 1974; Schuckit, 1980), insensitivity to arousal in general (Luria, 1973),

or even to actual diminished responsiveness to ethanol (Schuckit, 1985). Deficits have

also been identified among the children of alcoholics in cognitive abilities critical to self-

regulation behaviors, such as the ability to plan and monitor goal directed behavior

(Tarter et al, 1989), as well as in the capacity to abstract and formulate rules for

governing behavior (Miller and Saucedo, 1983; Tarter, 1975). Though consideration of

these so-called dispositional factors may be a helpful part of an intervention, the

framework provided by self-regulation theory is more comprehensive in that it

combines these dispositional factors with the context, manner and consequence of use

(Diaz & Fruhauf, 1991).

Addictive behaviors, like all operants, will also be influenced by a host of

discriminative stimuli and contextual factors including environmental cues (Cohen et al,

1973; Miller et al, 1974; Bach & Schaefer, 1979), social influences (Swaim et al, 1989;

Caudill & Marlatt, 1975), availability of other reinforcers (Vuchinich & Tucker, 1988),

affective arousal states (Marlatt et al, 1975; Higgins & Marlatt, 1975) and expected

effects (Critchlow, 1986; Brown et al, 1985; Marlatt et al, 1973 and Schuckit, 1987). But

as with the so-called dispositional qualities of the individual, these contextual factors

will, according to self-regulation theory, obtain utility in intervention primarily by

understanding how they function as part of an addicted individual's pattern of use.

Self-Regulation as a Basis for Intervention

Kanfer's model of self-regulation was one of the first to be formulated

specifically for treating addictive behaviors (Kanfer, 1970, 1986). His model crucially

distinguishes between two modes of regulatory functioning: automatic processing and

controlled processing (Kanfer and Gaelick, 1986; Miller & Brown, 1991). Automatic processes are those posited to require little or no conscious control (though they might have at one time); they include such routine activities as eating, driving, getting dressed and, for experienced drinkers, the behaviors associated with drinking alcohol (such as rate of consumption, or cue response). Controlled processes are behaviors we emit in order to accomplish new or highly demanding tasks, including, importantly, disengaging or modifying automatic processes. Thus the development of self-regulation in Kanfer's model entails a focus on the judicious application of controlled processes (Kanfer, 1987; Miller and Brown, 1991).

This deployment of controlled processes to alter automatic processes is mediated by the development of adaptive private verbal behaviors in the addict, which include the recognition of an impasse between his or her automatic processes and desired outcomes. The techniques employed entail orientation towards pertinent automatic processes and self-monitoring of same, a process meant to enhance the addict's deficient repertoire of self-awareness. Once the ability of self-awareness is developed, further learned processes of self-evaluation help to develop awareness of discrepancies between one's current behavioral patterns and one's goals. Miller & Brown (1991) put it succinctly, "The registration of deviation from a standard is, in fact, a crucial element of any homeostatic system". The third stage of Kanfer's model, termed "self-reinforcement", involves using the newly perceived discrepancies to chart a course for controlled processes and repertoires of behavior that will achieve more *valued outcomes* by cultivating awareness of reinforcers that are able to compete with those offered by the addict's drug of choice (Kanfer & Grimm, 1980). Following this

recognition of the need to change, the subsequent stages of Kanfer's model mirror the development of typical self-regulatory behaviors: searching for alternative behaviors, finding a plan of action to cultivate those behaviors, engaging in those behaviors and monitoring the results of the new repertoire. All of these activities will be discussed in more detail in the following section on BSCT.

Obviously, the third stage is the most problematic of the model. Indeed, if merely arriving at the awareness of a discrepancy between behavior and values were sufficient to instigate the subsequent development of alternative repertoires and lasting change in addictive behaviors, addiction to drugs and alcohol would not be the problem it is. Here we arrive at the uncertain observation that the deliberate development of an addict's self-regulatory repertoire is itself not necessarily enough to promote behavioral change. While theoreticians and researchers alike have spilled much ink on the subject (e.g. Hunt and Mazarro, 1970; Premack, 1970; Bandura, 1977, 1988; Miller, 1985a, 1985b) it remains an insoluble dilemma for many a clinician and addict alike. This subject will be addressed in more detail in the discussion section below, when considering the factors that underlie the success of those participants in the study who were able to bring their drinking under control.

Behavioral Self-Control Training

BSCT is a treatment approach designed to help problem drinkers gain control of their drinking, either to attain non-problematic drinking, or as a stepping-stone to abstinence. It has been found to be particularly effective for non-dependent problem drinking. Like other cognitive-behavioral treatments for alcohol use disorders, BSCT is based on social learning theory; its protocols derive from the assumption that alcohol

use, like all behaviors, is mediated by environmental stimuli, public and private verbal

behavior, and various forms of reinforcement. Its focus is on: identifying and altering

the environmental and behavioral contingencies that support problematic drinking;

developing drinking-related skills to mitigate the harmful effect of these contingencies;

and assisting in the identification and pursuit of activities that moderate or replace

drinking behaviors. Thus, BSCT protocols typically include techniques such as drinking

cue identification, self-monitoring of urges and responses, goal setting, consumption

management, development of coping skills and cultivation of alternate reinforcers. It

can be delivered by a therapist, a self-help manual, or by computer (Hester, 2003).

Delivered in these various formats, BSCT has been tested in 47 controlled clinical

trials, and its efficacy has been consistently supported by this empirical research (see

Hester, 2003 and Miller, Wilbourne, & Hettema, 2003 for reviews). Miller and

colleagues conducted a long-term (2-8 years) follow-up of individuals who had been

involved in BSCT studies and found that many individuals who were initially successful

at moderating their drinking eventually stopped drinking entirely; for them, successful

moderation was indeed a stepping-stone to abstinence. They also identified individuals

who were able to moderate their drinking without any continued signs of alcohol-

related problems. Importantly, they found pre-treatment client predictors of success

with BSCT: a less severe level of dependence and a shorter history of alcohol-related

problems. Specifically, individuals' scores on the MAST at baseline were predictive of

long-term outcomes (Miller et al, 1992). Finally, a meta-analysis (Walters, 2000)

reviewed 17 BSCT RCTs and found a small to medium aggregate effect size of (d = .33)

across all studies.

These studies collectively show that some problem drinkers respond favorably to this approach, sustaining moderate and non-problematic drinking over extended periods, whereas others, following BSCT with a moderation goal, eventually opt for total abstinence (some with and some without additional treatment). Heather and colleagues have noted, "BSCT has become the standard moderation-oriented treatment in countries in which moderation as the goal is an accepted part of treatment services" (Heather et al., 2000).

Objective

The purpose of this study was to evaluate whether the outcomes of participants in the clinical trial comparing the effectiveness of two BSCT protocols, ModerationManagement.com and Moderate Drinking.com, would be moderated by the level of clinical distress they reported upon entrance into the trial.

Hypotheses

Participants in both the experimental and control groups who express higher levels of clinical distress will not reduce their consumption of alcohol as much as those who express little or no clinical distress relative to baseline levels at follow-up.

Methods

Screening and Intake Protocol

The data for this study were collected from a randomized clinical trial that evaluated the effectiveness of two computer-based interventions for heavy but non-dependant drinkers who desired to moderate their drinking (Hester, et al, 2009). For the study, participants were recruited from the Albuquerque NM metro area primarily with a display ad in the weekly TV guide section of the local newspaper. The ad ran a headline asking, "Are you concerned about your drinking?" and invited those interested to join a federally funded study.

Because the trial was run as an effectiveness study, there were minimal inclusion and exclusion criteria, most of which focused on excluding alcohol dependent drinkers. The inclusion criteria were: 1.) Potential subjects must score greater than 7 on the Alcohol Use Disorder Identification Test (AUDIT); 2.) They must drink at least 10 standard (14 grams) drinks per week; 3.) They must not be currently abstaining; 4.) They must have an expressed interest in moderating their consumption of alcohol; 5.) They must have a computer with Internet access at home, and; 6.) They must be at least 21 years of age.

The rationale for the inclusion criteria is as follows: an AUDIT score of 8 or more suggests the presence or risk for of alcohol-related problems (Saunders et al, 1993). The 10 drinks per week minimum was an arbitrary cut-off for inclusion, but one based on the experience of the study's principle investigator, who has found in prior moderation training trials that individuals drinking less than this amount per week were less likely to respond to a moderate drinking protocol. Thus, those individuals

who contacted us but were abstaining, and whose chief concern was that they might relapse, were encouraged to continue abstaining and given referrals to abstinence-oriented resources in the community. Minors were not recruited because it was felt that a moderate drinking program would have to be specifically tailored to their developmental stages. Finally, participants were required to have a computer with internet access at home so that they could access the online MM community and the MD web application from there.

The exclusion criteria were chosen primarily to exclude participants who were most unlikely to benefit from the protocols, given prior research in moderation. The criteria were: 1.) Potential subjects may score no higher than 19 on the Michigan Alcoholism Screening Test (MAST) (Selzer, 1971); 2.) They must not have a history of prior treatment for substance abuse; 3.) They likewise must not have had a past or current diagnosis of drug abuse or dependence; 4.) Further, they must not have ever been hospitalized for alcohol or drug dependence or detoxification or; 5.) They must be lacking evidence of past or present physical dependence (major withdrawal symptoms: DTs, seizures, hallucinations); 6.) They must not have any current diagnosis or indication of psychosis or bipolar disorder; 7.) Nor may they exhibit evidence of significant cognitive impairment from brain dysfunction (based on self-report and observation during screening interviews); 8.) They must not report or exhibit evidence of health oriented contraindications to any further drinking (e.g., taking MAOIs, Hepatitis C, pregnancy, congestive heart failure); 9.) They must not have an English reading level below the 8th grade (assessed using the Slossen, 1990); 10.) They must be available for follow-ups at 3, 6 and 12 months following entrance to the study; 11.)

They must be willing and able to provide one significant other (SO) for corroboration of their self-report of drinking, and; 12.) Individuals inquiring about the study must not currently be members of MM.

Potential participants were initially screened over the phone using the Quick Screen from Project MATCH, and a questionnaire addressing inclusion criteria 2-6 and exclusion criteria 2-5, 8,10, 11, and 12. Those who passed the phone screening were invited to schedule an appointment for a face-to-face meeting for the remaining screening items, to discuss the clinical trial in more detail, and review and sign the Informed Consent. The in-person screening included the MAST, the AUDIT, the Brief Symptom Inventory-18 (Derogatis, 2000), a brief medical history questionnaire, and the gathering of demographic data. Finally, we asked participants for the name of a "significant other" (SO) that we could interview to corroborate the participant's self-reported drinking data. Participants were informed that we would also asked the SO to contact us if he or she became aware that the participant's drinking was increasing or that the participant was experiencing new or worsening alcohol-related problems.

Once the screening was completed, individuals were randomized into the study by stratified random assignment. Participants were classified into blocks based on the three control factors of gender (male or female), ethnicity (Anglo, Hispanic, or Other), and problem severity as measured by their AUDIT scores, with a score of 15 or less allocating them to the "low" group, and a score of 16 or above into the "high" group. Overall, 191 people were screened for eligibility, 107 were excluded, 40 were allocated to the experimental group and 44 to the control. For further details about the flow of participants through the study, please refer to the chart (Figure 1).
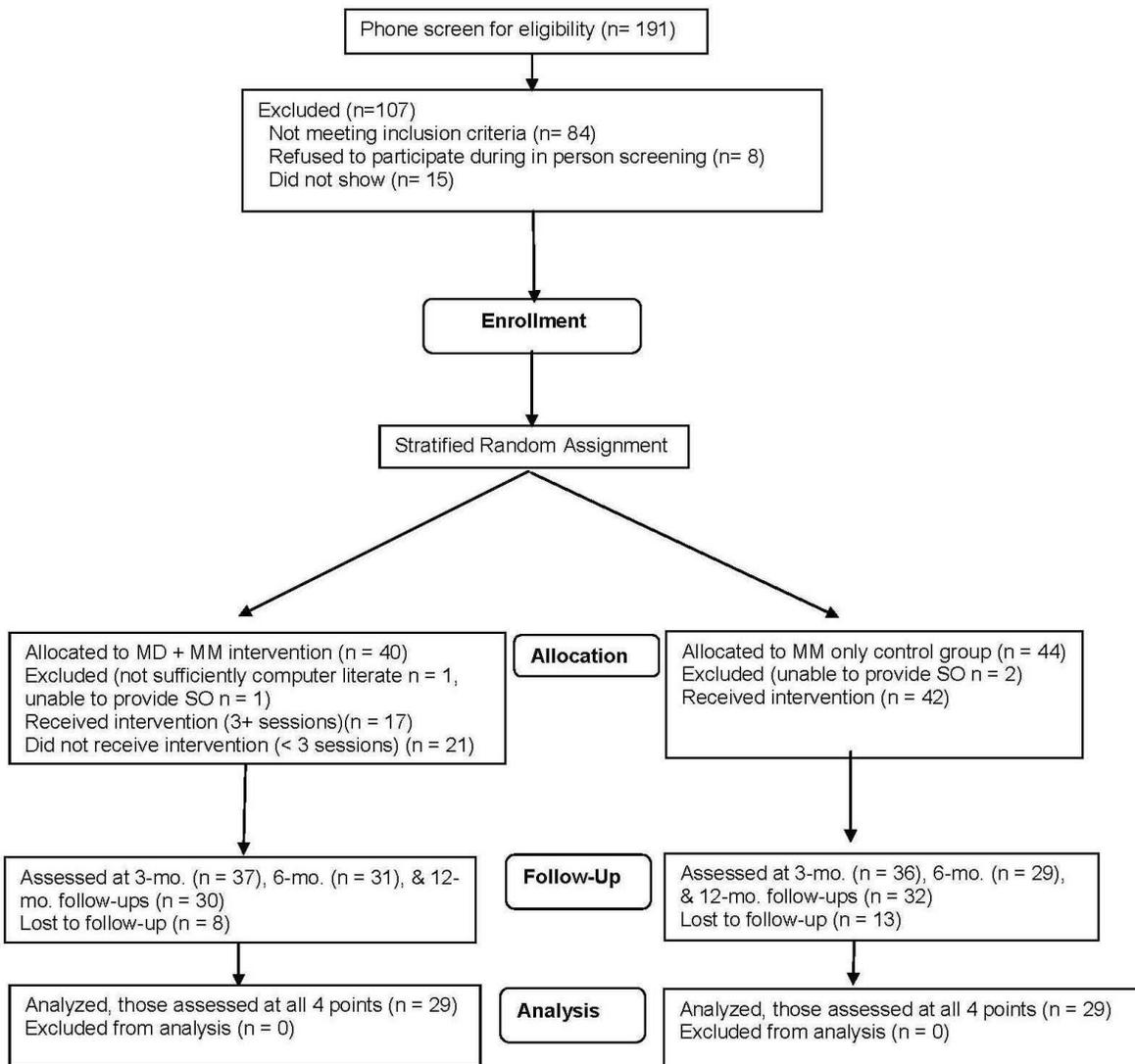
Figure 1. Flowchart of Participation through Trial.

Intake and Assessment

Once participants were enrolled, they were given a formal baseline assessment of their drinking and drinking-related issues using the Drinker's Evaluation program, a separate web application developed to collect baseline and follow-up data. The program deployed the following instruments for baseline and follow-up assessment: the AUDIT (baseline & 12 mo f-up); the Brief Drinker's Profile (BDP) which measures quantity/frequency of drinking and drug use, family history, and other risk factors (baseline, 3,6, & 12 mo f-ups) (Miller & Marlatt, 1987a); the Drinker's Inventory of Consequences (DrInC) which measures both lifetime and recent (previous 3 months) consequences from drinking (baseline, 3,6, & 12 mo f-ups) (Miller et al., 1995); the Severity of Alcohol Dependence (community sample) (SADQ-C) which measures symptoms of alcohol dependence (baseline, 6, & 12 mo f-ups) (Stockwell et al., 1994); the Stages of Change and Readiness and Treatment Eagerness Scale (SOCRATES) which measures motivation for change (baseline, 3,6, & 12 mo f-ups) (Miller & Tonigan,1996); demographic data; and additional questions from the Form 90 Miller, 1996) that ask about additional treatments (outside of the study) they have received during follow-up. SO data was collected later by phone using the Collateral Interview Form (CIF) (Miller & Marlatt, 1987b). The CIF is a parallel instrument to the BDP for use with collaterals at baseline and follow-up.

Introduction of the Intervention

Upon completion of the baseline assessment, participants are offered a break, informed of their treatment condition and presented with the appropriate intervention for their group. Both interventions are based on BSCT. Control subjects are introduced

to Moderation Management (MM). On the site (www.moderation.org) MM describes itself as a "behavioral change program and national support group network for people concerned about drinking and who desire to make positive lifestyle changes. MM empowers individuals to accept personal responsibility for choosing and maintaining their own path, whether moderation or abstinence." MM provides basic concepts of and guidelines for moderation, offers an online mutual-help support community via an active listserv and a moderated forum, as well as information about face-to-face meetings, and written materials describing MM's program of behavioral change, as well as links to various other resources, including an interactive program that allows users to report their abstinence and moderate drinking days in a calendar online (for others to see). MM is the most widely known on-line self-help group supportive of moderation in drinking. While MM is also supportive of abstinence, it was founded on the premise, supported by empirical research, that most non-dependent drinkers can cut back on their drinking when they decide to change (Klingemann & Sobell, 2007).

Participants were given a brief tour of the site and enrolled in its listserv. In order to ensure that they received a minimum dose of exposure to the treatment, participants were asked to read and/or post to the listserv at least twice a week for at least the first 12 weeks of the study. They were not asked to self-monitor this activity on a daily basis, since this might compromise the external validity of the trial's results. Instead the Drinker's Evaluation program sent them a monthly email that asked how much they had accessed the MM website and how much they felt they had benefitted from it.

Experimental subjects were given this same tour of MM, and then introduced to the ModerateDrinking.com (MD) web application. MD is the product of 15 years of research and testing by the principle investigator. When initially developed as a program for the Windows operating system, Behavioral Self-Control Program for Windows (BSCPWIN) was evaluated for its effectiveness and found to have comparable treatment effect sizes to BSCT provided in face-to-face individual therapy and in-group settings (Hester & Delaney, 1997).

Moderate Drinking.com employs a protocol similar to the one advocated by MM (Rotgers, Kern, & Hoeltzel, 2003). It is divided into modules which focus on the following topics, which are typical of BSCT protocols: Building motivation and self-confidence; setting drinking goals/limits; "doing a 30" (an initial month long period of abstinence); self-monitoring drinking (with personalized feedback relative to the goals the drinker has set); rate control; setting personal drinking rules; self-monitoring urges to drink (with personalized feedback); identifying and managing triggers; developing alternatives; general problem solving; dealing with lapses and/or relapses; considering abstinence; and self-monitoring one's own mood using the Positive and Negative Affect Scale (Watson, Clark & Tellegen, 1988) (with feedback relative to baseline levels).

Once users register on the site, the MD program recommends first choosing a goal (abstinence or moderation), building motivation for change, "doing a 30," setting moderate drinking goals/limits (if that is the desired goal), and then self-monitoring one's drinking. Users are prompted to enter their self-monitoring data when they log back onto the site, and then get feedback about their progress towards their goals. The MD protocol is designed in such a way that users can either go through the program in

any sequence they prefer, or simply pick and choose which modules are most pertinent to them.

Once the participants were shown their respective interventions, they were asked if they had any questions or concerns. Participant response to this open-ended part of the protocol varied widely and was only loosely controlled. Some participants had no questions, some had only technical questions about how to operate the program, and some were in fact eager to discuss their drinking problem with the research assistant (RA). This initial discussion at the outset of the study was problematic. Any discussion with the participants about their concerns with their drinking at this point in the study might well have constituted a treatment effect that would "cloud" the study's results; on the other hand, ignoring the participant's sincere concerns at this point might have diminished his or her engagement with the materials or the study itself. The middle path decided upon for the RA was to address, as briefly as possible, any concerns the participant had about their drinking, not to solicit any further discussion, and to always direct participants to the resources being made available to them.

Those interested in the outcome of the clinical trial, as well as other, supplemental analysis, will find the results published elsewhere (Hester et al, 2009; Hester et al, under review).

Outcome Measures: Clinical Distress

As mentioned above, participants were administered the BSI-18 when they were screened into the study, just prior to their full assessment. The BSI-18 is described by its authors as "a brief, highly sensitive self-report symptom inventory designed to serve as a screen for psychological distress and psychiatric disorders in medical and

community populations" (Derogatis, 2000). It is an abbreviated version of the 53-item BSI (Derogatis, 1993), which is, in turn, a shortened version of the 90-item Symptom Checklist-90-Revised (SCL-90-R: Derogatis, 1994). The BSI-18 was developed primarily to tap anxiety and depression, and includes a somatization scale as well since anxious and depressed clients frequently also present with a profusion of somatic complaints (Derogatis and Wise, 1989; Kirmayer et al, 1993) as do Hispanics (Asner-Self et al, 2006). Derogatis reports that the measure exhibits satisfactory reliability, and very high convergent validity with its progenitors, both the BSI and the SCL-90-R, which, in turn, have each exhibited good convergent and discriminative validity with a variety of clinical and community samples (e.g. Derogatis, 2000; Royse and Drude, 1984; Zimmerman et al, 1996 and Grassi et al 1998).

As the name suggests, the BSI-18 is composed of 18 items, 6 each across the three dimensions of anxiety, depression and somatization. The instructions ask test-takers to report on their level of distress over the preceding seven days, on a 5-point Likert scale (see appendix 1). The measure is meant to yield three meaningful sub-scale scores as well as a global index of psychological distress (GSI). However, there has been controversy on this point. What Asner-Self refers to as the BSI's and SCL-90-R's "long history of mixed psychometric soundness" has, apparently, been inherited by the BSI-18 (Asner-Self et al, 2006; Boulet and Boss, 1991). Commentators thus suggest that while the three subscales may offer some insight into the character of the client's suffering, the GSI may nonetheless be relied upon as a good indicator of psychological distress.

Outcome Measures: Drinking Frequency, Severity and Quantity

Of the six within-subject quantity/frequency measures of drinking used in the RCT, three were chosen as the dependent variables for the present analysis. The three chosen measured number of drinks per drinking day (DPDD), Mean Peak BAC (blood-alcohol content) per Drinking Day (MPBDD) and percent days abstinent (PDA). Three were chosen to simplify the analysis, and because they were considered sufficient to test the hypothesis.

Each one of the three dependant variables taps a different dimension of use. DPDD is a simple gauge of quantity, easily identifiable to the participant, but obviously relative, since the participant's weight mitigates the intensity of the effect of the alcohol. MPBDD is more sensitive in this regard, since it is derived both from the participant's weight and the intensity of his or her drinking; thus it is a better measure of degree of intoxication. Finally, PDA is a global measure of how often the participant drinks, an especially sensitive measure for participants who would rather moderate instances of drinking than the quantity or intensity of drinking. That is, it is assumed that there are multiple paths to moderation, and a client may achieve it by more than one path.

Results

Sample Characteristics

A total of 80 people participated in the study analyses were conducted on the 58 participants having data at all three assessments (Baseline, 3, and 12 month follow-ups). Figure 1 summarizes the sample sizes at various stages throughout the study. Overall follow-up rate (58 of 80) was 74%. Of the original 80 participants, 45 were females (56% of sample) and 35 were males (44%); of the final 58, 29 were female and 29 were male. Participants included 63 (79%) non-Hispanic Caucasians, 15 (19%) Hispanics, and 2 (2%) other. The mean age of participants was 50 years, and mean number of years of education was 15. We classified participants as binge drinkers if their baseline level of drinking, on average, met NIAAA's definition: a mean number of drinks per drinking day as 5 or more for males and as 4 or more for females. No significant differences were found across groups on demographic variables or any of the continuous measures assessed at baseline (see Table 1).

Table 1. Means on Continuous Measures at Baseline.

Measure	Group	
	MD + MM	MM only
Age	48.7	52.1
Education in Years	15.7	15.1
BSI score		
Drinks (SECs)/Week	33.0	35.4
Percent Days Abstinent (PDA)	16.3	16.2
Mean Drinks per Drinking Day	5.5	6.1
Hours BAC > 80 mg%	21.9	26.1
AUDIT score	17.7	18.3
Bingers		

Results of Primary Analyses

To test the hypothesis, outcomes were analyzed by means of a set of 3 X 2 X 2 X 2 analyses of covariance (ANCOVA) utilizing one continuous variable of clinical distress (BSI Total score) entered as a covariate, three two-level categorical variables (treatment condition, sex and binge status) and one three-level repeated factor (time of assessment: baseline, 3 month, 12 month). This analysis was conducted on three primary alcohol consumption measures: Percent Days Abstinent (PDA), log Mean Drinks per Drinking Day (DPDD), and log Mean Peak BAC (blood-alcohol content) per Drinking Day (MPBDD).

Treatment condition was of obvious interest due to the hypothesis of the clinical trial, that a more thorough BSCT protocol would enhance the results of those attempting to moderate their drinking; binge status was of interest here because it was found in a post-hoc analysis of the RCT's results that non-bingers differentially benefitted from the experimental condition more significantly than did bingers in either group. Finally, it was thought that the sex of participants might bear on outcomes, in so far as women are known to express higher rates of depression than men (Nolen-Hoeksema, 1987).

For each analysis, two contrasts of the within-subject factor were conducted to explore two a priori hypotheses: one contrast examined whether change occurred between baseline assessment and time points following treatment, which was defined as an average of follow-up assessment at 3 and 12 months, another contrast examined whether the two follow-up time points differed significantly, so that, if it was indeed the case, the course of change, whether delayed improvement or deterioration of improvement, might be further examined. The first contrast will here be referred to as the *before-after treatment* (BAT) contrast, and the second will be referred to as the *delayed-or-deteriorated effect* (DDE).

Transformation of Skewed Variables

Two of the measures of drinking were very positively skewed. DPDD had a skewness at baseline of 2.0, and MPBDD was positively skewed > 2.5. Thus, such measures were log transformed before analysis. This resulted in reducing the skewness for the log transformation of the baseline variables to less than 1.0 in absolute value.

Test of the Hypothesis: Clinical Distress Moderates Drinking Outcome:

Drinks per Drinking Day

As reported elsewhere, multivariate tests of the overall reduction in alcohol consumption averaging across both groups in the study was highly significant (Hester et al, under review). However, when predicted by BSI score, participants were more likely to reduce the number of drinks they consumed per drinking day given a *higher*, rather than lower, measure of clinical distress: DPDD, F (1, 48) = 7.20, p < .010. The locus of the change over time was from the baseline to the average of the two follow-ups (BAT), with there being no significant differences among the 3 follow-up periods, F (1, 48) = 1.821, .183.

As stated above, analysis were also run to examine whether BSI score interacted with the treatment condition, drinking style or sex of the participant with regards to mean number of drinks consumed by participants per drinking occasion. None of these interactions were significant from baseline to the average of the two follow-ups: neither for DPDD x Treatment x BSI F (1, 48) = .075, p = .786; nor for DPDD x binge status x BSI F (1, 48) = .572, p = .453; nor for DPDD x sex x BSI F (1, 48) = .572, p = .453.

Percent Days Abstinent

Just as with DPDD, with regards PDA, when predicted by BSI score, participants were more likely to reduce the number of days they consumed alcohol given a *higher*, rather than lower, measure of clinical distress: PDA, F (1, 48) = 10.054, p = .003. The locus of the change over time was again from baseline to the average of the two follow-ups, but in this case there was a significant difference between the two follow-up periods, F (1, 48) = 6.162, p = .017. Further analyses were then conducted to determine

whether the significance of the DDA result constituted a delay in improvement (significant increase in PDA from 3 to 12 months) or deterioration of initial improvement (significant decrease in PDA from 3 to 12 months). To test this finding, a two-tailed Pearson correlation matrix was created which revealed a significant relationship between BSI and the final 12-month follow-up time point (.452, <. 000), suggesting that elevated BSI scores correlated highly with a significant but delayed increase in PDA.

Analyses of whether BSI score interacted with the treatment condition, drinking style or sex of the participant with regards to PDA was conducted and, as with DPDD, it was found again that none of these interactions were significant from baseline to the average of the two follow-ups: neither for PDA x Treatment x BSI $F (1, 48) = 2.063$, $p = .157$; nor for PDA x binge status x BSI $F (1, 48) = .005$, $p = .942$; nor for PDA x sex x BSI $F (1, 48) = 1.239$, $p = .271$.

Mean Peak BAC per Drinking Day

Unlike DPDD and PDA, MPBDD was not affected by baseline BSI score $F (1, 48) = 1.633$, $p = .206$. Not surprisingly, when analyses of whether BSI score interacted with the treatment condition, drinking style or sex of the participant with regards to MPBDD it was found that none of these interactions were significant from baseline to the average of the two follow-ups: neither for MPBDD x Treatment x BSI $F (1, 48) = .002$, $p = .957$; nor for MPBDD x binge status x BSI $F (1, 48) = .008$, $p = .824$; nor for MPBDD x sex x BSI $F (1, 48) = .098$, $p = .085$.

Discussion

This study examined the moderating effect of self-reported psychological distress on alcohol abusers who attempt to reduce their drinking. Because both epidemiological findings and intervention research supports the view that patients suffering from depression and anxiety with co-morbid alcohol use disorder generally have poorer outcomes, we hypothesized that participants in a randomized clinical trial who reported more clinical distress would be less likely to reduce or curtail their drinking than those participants who reported less distress. In fact, our analysis found partial support for just the opposite conclusion. To wit, taken as a continuous predictor, higher scores for general psychological distress were more likely to correlate with successful outcomes as measured by two of the three drinking measures used for this study. Our findings indicate that dysphoric non-dependent problem drinkers are more successful at some forms of moderating their drinking than those who are not dysphoric.

Specifically, we found that more clinically distressed individuals were better than non-distressed problem drinkers at reducing the number of drinks they consumed on a typical drinking occasion; they were also more successful at reducing the number of days they actually consumed alcohol, although this change took longer to develop. Interestingly, distressed problem drinkers were not anymore successful than non-distressed drinkers at reducing the intensity of their drinking, as measured by their typical peak BAC per drinking occasion.

There are at least two possible reasons to explain this failure of clinically distressed individuals to reduce the intensity of their drinking. One may be that the

positively skewed distribution of this measure had too little variability across a range of responses to drive an effect. Another reason could be that this group of problem drinkers, working to acquire a self-regulatory repertoire, may be more successful with the sort of rule-governed behavior that is reflected in counting drinks per day or number of drinking days than they are at controlling their drinking once under the influence of the alcohol. This explanation is especially interesting since it suggests limits on how much self-regulation might have been acquired through the intervention over the course of the trial.

Clearly, these findings are cursory, and must be interpreted with caution. The measure of clinical distress was derived from the BSI-18, which is a screening tool, and only three quantity/frequency measures of drinking were used. The BSI has shown good validity as a measure of clinical distress, so there is good reason to believe it provided a good measure in that regard, but it has no diagnostic specificity, and potential participants with a history of severe mental illness were excluded from the study. Nonetheless, the results of this study stand in stark contrast to the conventional wisdom of the co-morbid literature, and warrant some consideration.

One possible insight to these results comes from some research that has explored grounds for a non-linear relationship between alcohol use and mood and anxiety disorders. Rodgers et al (2000b) reported two prominent findings in a study designed specifically to look at rates of co-morbid clinical distress across the spectrum of alcohol use, from abstinence to heavy drinking. First of all, using multiple measures to assess both depression and anxiety, they found higher levels of consumption were associated with more severe symptoms of both anxiety and depression; they also found

that non-drinkers had higher anxiety and depression scores than infrequent and moderate drinkers. That is, they found a clear, U-shaped relationship between both degree of anxiety and depression and level of alcohol consumption.

Secondly, using the same sample, Rodgers et al (2000a) then looked at a range of social, personal and socio-economic factors to try and account for the U-shaped relationship. Specifically, they attempted to determine which of three possible models might best explain the U-shaped effect: whether alcohol use (and/or its absence) leads to dysphoria; whether dysphoria leads to alcohol use or avoidance, or whether there might be some common factors that give rise to dysphoria at both ends of the use spectrum. They found that both education and employment "status" were significantly associated with moderate drinking. Looking at social factors and life event variables, they found again that non-drinkers differed from moderate drinkers on a variety of factors known to correlate with psychological distress, but not so with regards to personality factors. Overall, the results suggested that moderate drinkers differ from abstainers on a variety of factors that are known to be linked with psychological distress and poorer health in general, while effects with heavy drinkers, though also differing from moderate drinkers, could not also be accounted for by these same factors. This suggests that different factors are most likely to account for elevated negative affect in both abstainers and heavy drinkers. Thus, a most parsimonious explanation for the U-shaped effect, that alcohol has a truly self-medicating effect when taken in moderate doses, is not well supported by these findings. Instead, Rodgers suggested that moderate drinkers might be more proficient, as a group, at self-regulation.

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Turning the issue around, a body of research has lately emerged in the DUI treatment literature indicating that depressed offenders mandated to treatment are both more receptive to treatment (Wells-Parker et al, 2006), more likely to change their drinking than non-problem drinkers if given slightly enhanced, personalized treatment (Wells-Parker & Williams, 2002), and both more motivated to change their drinking upon entering treatment and more likely to have better outcomes (Holt et al, 2009). One unexpected finding in these studies was that depressed offenders scored lower on measures of self-efficacy, which usually predicts worse outcomes. The counter-intuitive outcomes here were thought by Wells-Parker to be understandable in motivational or "readiness to change" terms. That is, Well-Parker posited that depression in this population indicates awareness of discrepancy between goals and behaviors and thus presents a "teachable moment" which these individuals take advantage of in order to change.

Blume, Schmaling and Marlatt laid the groundwork for this train of thought with an influential paper (Blume et al, 2001) that attempted to parse, in terms of the Transtheoretical Model of Change (Prochaska & Diclemente, 1982) the divergence in this population between decreased self-efficacy and enhanced motivation to change. Their findings indicated that higher assessed depression did significantly associate with higher Readiness to Change "action" scores. Sheilds and Hufford decided to test this idea directly. When they compared a sample of individuals with alcohol use disorders to a sample of individuals with alcohol use disorders and co-morbid depression, they found that the latter group scored higher on a measure of "readiness to change", but

they also found a discrepancy between readiness to change and self-efficacy in the depressed group.

One possible way to make sense of the above findings, taken together, is that depressed individuals suffering from an alcohol use disorder are motivated to change, but benefit from a treatment that deals specifically with issues of self-efficacy and structured recourse to self-regulation. The above studies did not control for level of consumption, but given what we know about the U-shaped relationship between psychological distress and levels of drinking, it may be that drinkers who typically moderate their drinking, but are in an active phase of abusive drinking, are both aware of the discrepancy between their behaviors and their values, and are in the best possible position to do something about it. That is, drinkers who typically moderate, or at least who have no history of severe abuse or dependence, are more likely to have the resources to effect change when they do enter a phase of problem drinking, and the clinical distress they express derives from their recognition of the discrepancy between their current dysregulated behaviors and past success at controlling their drinking.

This finding has important clinical implications because it may allow clinicians to distinguish more clearly which co-morbid cases may have a relatively easier course of treatment. That is, an individual presenting for treatment, who does not have a current diagnosis of alcohol use disorder with symptoms of dependence, but who is co-morbid with symptoms of general dysphoria, may be a better candidate for a relatively brief, self-directed or efficacy focused treatment such as is afforded by BSCT. This possibility follows from the fact that BSCT provides excellent structure for individuals

already motivated for treatment, but who may lack the skills or the awareness to alter their behavior.

There are limitations to the current study that constrain the veracity of this finding, however. The first derives from the fact that a relatively simple screening measure was used to obtain the measure of psychological distress for this study. While the measure itself exhibits good validity for dysphoria along the depressed-anxiety continuum, and the effectiveness of a screening measure would be ideal for application to treatment seeking populations, the measure itself is not sensitive enough to offer insight into the exact nature of the interaction of the psychological distress with the alcohol use disorder. To really understand the interaction here between this particular co-morbid case profile and treatment outcomes, more sensitive measures of anxiety and depression would be helpful. Depression, for example, is a broad term encompassing diverse symptomologies; it might be important to know whether, for example, anhedonia was a better predictor of readiness to change than pervasive rumination.

Similarly, though the clinical field is progressing toward an understanding of both depression and anxiety as sharing many subjective similarities, and frequently co-occurring, the conflation of those two constructs in this study may be obscuring other useful clinical insights. The most that can be said of the current findings is that research may usefully be conducted on the various differences between such states as stress and irritability, or poor concentration and lassitude, in relation to moderate drinking treatment outcomes.

Another limitation of the current study is that it did not differentiate between various other possible mechanisms of change that could be present in the clinical trial. For example, there has been no control over whether incoming participants benefitted more from their motivation for treatment, or from the treatment itself. Likewise, the assessment at intake was extensive; it may be that participants coming into the study simply benefitted from the self-awareness provided by the process of completing such an assessment, in so far as that event alone increased their sense of discrepancy enough to motivate change.

If we accept the logic of self-regulation theory, these findings do suggest a future potential line of research. First of all, it would not be illogical to assume that those moderate drinkers at the "bottom of the U" could be fairly conceptualized as good at regulating both negative affect and the powerful positive reinforcement of alcohol in general. That is, non-distressed, moderate drinkers are *de facto* good "self-regulators". It is not unreasonable that, given the natural variability and vicissitudes of life, anyone of these naturally effective self-regulators might become out of balance. They might drift into a pattern of excessive social drinking, and begin to experience negative emotions from the consequences of too much alcohol consumption. Conversely, they might suffer some adverse consequences in life, for example lose their job, or a loved one, and compensate with more drinking than usual. It would be interesting to explore whether one precipitant to dysphoria was more correctible through a protocol like BSCT than the other.

Another natural avenue of research, beyond replication of these results, would be the exploration of whether there are cut-offs for the effect found in this study. That

is, at what point severity of either psychological distress or level of consumption

reverses the counter-intuitive result found here, and the standard theories of co-

morbidity reassert themselves; and further, to specify the dispositional and contextual

factors that influence those outcomes.

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