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DEPRESSION AS A MEDIATOR OF MEMORY
BIASES IN ALCOHOLICS

by

Henry Jay Richards

A Dissertation Submitted to the Faculty of the Graduate
School of Loyola University of Chicago in Partial
Fulfillment of the Requirements for the Degree of
Doctor of Philosophy

November

1987

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VITA

The examinee, Henry Jay Richards, is the son of Robert Leon Richards and Thelma Marie Richards. He was born on July 8, 1951, in Waukegan, Illinois.

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Mr. Richards has worked in a wide variety of positions, including lecturer in African literature at the College of Education, Port Harcourt, Nigeria. He has worked as a manager and hospital department head with Servicemaster Management Services Corporation, Downers Grove, Illinois. Within the field of psychology, he has worked as an adjunctive therapist at Michael Reese Medical Center, Chicago Illinois. He was a research assistant at three institutions: Lutheran Center for Substance Abuse, Park Ridge, Illinois; Lakeside Veterans Administration Hospital, Chicago; and Ravenswood Community

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CHAPTER I

INTRODUCTION

Pathological memory functioning in alcoholics has been viewed as both cause and consequence of prolonged abuse. Specific differences in alcoholics from nonalcoholics in the processing of emotionally charged stimuli have been implicated in the addictive process. In a study conducted by this author and preliminary to the present study, differences between alcoholic and nonalcoholic subjects in the accuracy of their memories for the frequency of occurrence of emotionally charged stimuli were demonstrated. The target stimuli used in the preliminary study were the subjects' own mood states as recorded on self-report forms over a two week period. A memory task was later administered, in which the subjects estimated the frequency of occurrence of their moods during the recording period.

When these memory estimates were compared to the previously recorded rates of mood occurrence it was found that the estimates from subjects in both groups were highly accurate. Some evidence was found for a small decrement in memory accuracy in the alcoholic group. However, when accuracy was examined by the emotional content of the estimated stimuli, larger group differences emerged.

Although the alcoholics appeared to have more random error in their estimates, they also demonstrated less systematic error than the non-alcoholic subjects, whose judgments could be called defensive, in that their estimates maximized positive moods and minimized negative moods. The systematic error demonstrated by the alcoholic subjects was in the same direction as that of the nonalcoholics less extreme. Since the preliminary study focused on possible cognitive deficits in alcoholics and therefore on accuracy, an extensive investigation of the effects of mood relevant content of items on memory bias was beyond its scope.

The literature on the effects of depression on cognitive processes, however, suggested a possible explanation of these results. That is, the two groups may have differed in level of depression, resulting in different degrees of accuracy and bias for various kinds of affective memories. When the literature on depression and cognition was reviewed, several theoretical perspectives on depression and cognitive performance emerged (such as the learned helplessness model of depression, the depressive realism model, and theories of mood selectivity effects) which predict different degrees and types of bias in affective memories for depressed and nondepressed persons. Separate trait and state depression effects on memory were hypothesized to mediate these predicted differences. The literature on the incidence and etiology of depression in

alcoholics, and the escape theory of alcohol addiction suggested that these predictions and hypothesized processes may be relevant to alcoholics. The present study combined these related lines of research and theory in order to make and test predictions about the relationship between depression and memory biases.

The present study used archival data to investigate the role of depressed affect as a variable mediating the differences found in the preliminary study. Trait depression as measured by the Minnesota Multiphasic Personality Inventory (MMPI) and state depression as measured by the Symptom Checklist 90 (SCL 90) were used to measure specific types of depressed mood and mood predispositions. An adapted version of the Experience Sampling Form (ESF), an inventory similar to the Program of Mood States (POMS) was used to measure positive and negative affect more generally over the two week reporting period described earlier.

The present study focused on bias rather than accuracy in memory. Also, in addition to self-reports and memory estimates, behavioral measures were developed in an attempt to understand the amount and kinds of cognitive strategies used by alcoholics and nonalcoholics in their performance of memory tasks, and how these differences might be related to specific types of depression, and to negative affect more generally.

CHAPTER II

REVIEW OF THE LITERATURE

Alcoholism and Memory for Mood Occurrence

In the literature on cognitive functioning of alcoholics, there is a growing consensus that neurological damage to some extent accounts for their usually poorer performance on a wide variety of tasks, including those involving memory functions (Becker & Kaplan, 1986; Butters & Cermak, 1980; Goldman, 1983; Parson & Farr, 1981). There is evidence that alcoholics may be specifically impaired in certain types of memory for emotional events (Cowan, 1983; Ellis, Thomas, McFarland, & Lane, 1985; Johnson, Kim, & Risse, 1985; Markowitsch, Kessler, & Bast-Kessler, 1984; Markowitsch, Kessleer, & Dezler, 1986; Warrington, 1986). Yet access to memory for moods and emotional events may be essential to the treatment of alcoholism and prevention of relapse (Freed, 1978; Goldman, 1983; Sussman, Rychtarik, Mlueser, Glynn, & Prue, 1986) as it is to the development of changes of self in all individuals (Rogers, Kuiper, & Kirker, 1977).

One basic memory function which has been meagerly investigated in alcoholic subjects is memory for frequency of

occurrences. This is the kind of memory required to answer such questions as: were you sad more often this week, or in the previous week. A substantial body of experimental evidence suggests that adult humans are highly sensitive to the frequency of occurrence of events (Hasher & Zacks, 1984; Greene, 1986; Naveh-Benjamin & Jonides, 1986). In the verbal learning paradigm that has most often been used to evaluate the abilities of persons to accurately estimate frequencies of presented target items, correlations between actual and estimated frequencies of occurrence have typically been in the high .80's (Zechmeister & Nyberg, 1982). However, because of the limited procedures and stimulus materials that have been investigated, it is difficult to generalize from existing laboratory studies to naturalistic settings.

An investigation of memory for frequency of occurrences in which alcoholic subjects were compared to non-alcoholic subjects was conducted by the present writer (Richards, 1986). The study focused specifically on memories for one's own mood states and experiences, as in the example question above. There were two objectives in conducting this study. The first objective was to determine if the high correlation between estimated and actual rates of occurrence obtained under laboratory conditions could be obtained in a more ecologically relevant setting. The second objective was to examine differences in judgment accuracy between alcoholic and non-alcoholic subjects.

Several theories were used to develop hypotheses about group performance on a task of memory for frequency of occurrences of moods. Automatic Processing Hypotheses predicted high correlations between estimates and recorded moods for all subjects, regardless of alcoholism status (Hasher & Zacks, 1984). Availability Heuristic Hypotheses, predicted differences in accuracy related to stimulus content, with subjects overestimating the frequency of stimuli that are relevant or salient to them (Tversky & Kahneman, 1973). Availability Heuristic Hypotheses in this context, assume that the content of salient stimuli is different for alcoholics and non-alcoholics. A third set of hypotheses were generated from the prediction of impaired performance in alcoholics paralleling the cognitive impairments found in other types of memory tasks (Goldman, 1983).

Self-reported mood state data was collected for alcoholic and non-alcoholic subjects over a two week period. As they conducted their usual daily routines, subjects were cued by means of long-range pagers to pause and record their moods. The record was made on a standard form that included a ten item self-rating of mood at the time of the cue. At the end of two weeks, subjects were asked to estimate their recorded moods from memory, using another standard form. Difference scores for each subject on each mood item were derived by subtracting each frequency from its corresponding

estimate. Both signed and absolute values of differences were used to investigate group performance.

Both groups tended to overestimate positive items and underestimate negative items. Estimates correlated with actual frequencies at .82 across groups, with some subjects having correlations as high as .99. Evidence for relative memory impairment in alcoholic subjects was present but weak. Stronger evidence was found for differences between groups related to content of stimuli. A trend was demonstrated for nonalcoholic subjects to systematically underestimate negative moods and systematically overestimate positive moods more than alcoholic subjects. For positive mood items this overestimation difference approached significance. Although alcoholic subjects demonstrated overestimation and underestimation in the same direction as other subjects, their systematic distortions were not as extreme and estimates by alcoholics contained more error that was apparently unsystematic. The nonalcoholic group's accuracy appeared more sensitive to evaluative content than the alcoholic group.

These patterns of error were surprising in that they appeared inconsistent with Availability Heuristic Theory, which predicted that group biases in accuracy would depend on the relevance of item content, with subjects overestimating items more relevant to their concerns, due to the increase of salience at encoding and availability at recall of such

items, resulting from biases in set. Instead, the groups were found to be similar in direction of bias, but different in degree. It was not apparent that Availability Heuristic Theory could provide an explanation of these group biases, except by resorting to the rather untenable argument that alcoholic subjects had experienced all items as less salient than nonalcoholic subjects. Other theoretical approaches toward cognitive performances were sought to account for the observed group differences.

Depression as a Possible Mediator of Differences in Memory Biases

A related area of research suggested a possible explanation of the results described above. Effects similar to the overestimation and underestimation trends found in the preliminary study have been reported in the literature on human learning and depression. Depressed subjects have demonstrated the kinds of biased, systematic distortions in learning predicted from social psychological theories of judgment biases, while depressed subjects demonstrated such distortions to a lesser extent. A kind of depressive realism was described by Alloy and Abramson (1979). In their studies of estimation of contingency of reward on behavior (to be described in detail later in this review), depressed subjects were more accurate in their estimates of contingencies of outcomes than were non-depressed controls, who tended to overestimate contingencies in the direction that would be

more desirable or beneficial to themselves. The researchers concluded that nondepressed subjects succumb to various "cognitive illusions" more easily than do depressed subjects, and that depressed persons may be in a sense "sadder but wiser".

The results of both Hasher and Zacks (1979) and Alloy and Abramson (1979) contradict in similar ways predictions of some cognitive theories of depression. These theories emphasize the depressed person's inability to accurately perceive events and reconstruct reality in a consensual way (Beck, 1974). The depressed person is viewed as consistently distorting the future, ongoing events, and the past in ways that both place the self in a negative light, and reflect the anticipation of failure and defeat.

In regard to cognitive performance on hedonically charged tasks, several studies support the hypothesis that increased inaccuracy of perception of frequency of reinforcement may result from depression (Buchwald, 1977; Wener & Rehm, 1975). Other studies have found the picture far more complicated, and in some ways approaching the greater accuracy for depressives found by Alloy and Abramson (1979) in studies of perception of contingency of reinforcement. Rather than following contemporary theories of depression (e.g., Beck, 1976) by displaying a self-blaming attributional style, depressed subjects have often been found to be less biased in their attributions about causes of

success or failure, and less biased in their judgments of contingency of reinforcement than nondepressed subjects (Alloy & Abramson, 1979; Abramson & Alloy, 1981; Kuiper, 1978; Raps, Peterson, Reinhard, Abramson, & Seligman, 1982; Tennen & Hezberger, 1987). Nelson and Craighead (1977) for example, found depressed subjects to be more accurate about frequency of punishment than nondepressed subjects (who underestimated the amount of both punishment and non-reinforcement), while simultaneously significantly underestimating the amount of positive reinforcement.

If such findings about estimates of contingency and reinforcement frequency can be generalized to estimates of mood occurrence, it is conceivable that the differential biases found in the preliminary study were mediated by different levels of depression in the two groups, while differences in unsystematic error (caused by cognitive deficits in alcoholics) were obscured by the same depression-related biases.

Some evidence supporting the hypothesis that the nonalcoholic and alcoholic groups differed in level of depression was found in the preliminary study data. Rough inferences about the level of depression can be made based on differences in the frequencies of moods recorded by the subjects in each group. The two groups were not significantly different in mean occurrence of combined positive mood states, with the nonalcoholic group having a

mean of 53.8% with a standard deviation of 19.4%; whereas the alcoholic group had a mean of 58.42% with a standard deviation of 16.5% . The two groups were significantly different in mood states that were neither positive nor negative, with the nonalcoholic group having mean of 29.3% with a standard deviation of 17.1%, whereas the alcoholic group had a mean of 18.3% with a standard deviation of 13.5%.

The differences in the occurrence of combined negative moods approached significance, with the nonalcoholic group mean being 16.8% , with a standard deviation of 8.1%, whereas the alcoholic group had a mean of 23.3% and a standard deviation of 13.5%. When individual negative mood items were examined, three of ten resulted in significant differences at the .05 level (i.e., Angry, Confused, and Ashamed) and one (Tense) resulted in a significant difference at the .01 level with the alcoholic group having the higher mean for all ten negative mood states over two weeks. The alcoholic group could be described as experiencing more negative affect, similar levels of positive affect, and less "neutral" emotional time relative to the nonalcoholic group. These differences are similar to those expected between two groups with different levels of negative affectivity (Watson & Clark, 1984) a variable known to be highly associated with depression.

With these differences in mood experiences in mind, a post hoc attempt was made to determine if they were related

to the differences in accuracy of memory for moods, as various theories of depressed affect would predict. The emphasis of the preliminary study was on accuracy rather than systematic bias related to mood content. Because of this, no measure of total bias by mood content was used in analyses. A related measure that captures much of the same information, the correlation coefficient normalized by the r to z transformation (Hays, 1973) was used in a subsequent analyses of the data relevant to the current discussion. Often referred to as a discrimination coefficient in the literature on frequency of occurrences (Flexnor & Bower, 1975) the correlational measure of accuracy is a measure of relative accuracy. It answers the question of how strongly related are the subjects estimates and their target items. A high correlation can result from either high absolute accuracy, or systematic inaccuracy.

The correlation of each subject's estimates with the corresponding actual rates of occurrence of their moods across 30 items was calculated. Based on this measure, the two groups were virtually identical in relative accuracy: the nonalcoholic group mean was .83 with a standard deviation of .15, while the alcoholic group mean was .80 with a standard deviation of .18. Group status alone had a nonsignificant correlation with accuracy of only .03. To investigate the possibility that evaluative direction might be interacting with group status and level of frequency of various moods to

influence accuracy, several correlational analyses were performed. Since an interaction with group was suspected, separate analyses by group were conducted initially. The correlation of level of positive mood with accuracy was determined to be nonsignificant in the nonalcoholic sample ($r = .23$), but significant in the alcoholic sample ($r = .555$, $p = .05$).

This differential predictability having confirmed an interaction between group, mood state occurrence and accuracy, a multiple regression analysis was conducted with the subjects from both groups combined. Overall accuracy as measured by the above described discrimination coefficient was predicted from each subject's mean frequency of positive moods, mean frequency of negative moods, and mean frequency of mood responses in neither the positive or negative category, with group membership and interaction terms as additional predictors. The final stepwise equation accounted for 57.8% of the variance in accuracy scores, with the rate of positive mood accounting for the largest percent of explained variance, the rate of negative mood accounting for the second highest percent of variance, and several interactions between mood rates and group status accounting for other significant proportions of variance in accuracy scores.

Taken together, these three correlational analyses supported the hypothesis that the frequency of mood

experienced over the two week interval was related differently to accuracy in the alcoholic and nonalcoholic groups. It appeared that positive mood frequency and negative mood frequency interacted with group status to moderate differences in mood accuracy. The low sample sizes and low probabilities of Type I error suggested that these relationships were fairly robust.

Overall, several aspects of the preliminary study implicated depression as a variable of interest in memory differences between alcoholics and nonalcoholics. With this background, the next three sub-sections will review areas of psychological literature supporting these implications. First the literature on the incidence of depression among alcoholics will be reviewed, to be followed by a review of the literature on the role of depression, affect, and affective memories in the etiology of alcoholism. A sub-section describing the literature on cognitive performance in depression will follow, outlining typical findings and their similarities to and differences from the findings of the preliminary study.

The Incidence of Depression in Alcoholism

The effects of depressed mood on memory would be irrelevant to the study of memory in alcoholics if the incidence of depression in alcoholism was not substantial. However, the higher incidence of trait depression among alcoholics when compared to nonalcoholics has been a

cornerstone of some theoretical and treatment approaches to alcohol addiction and was therefore viewed as a variable that might have mediated the differences between alcoholics and nonalcoholics detected in the preliminary study (Jaffe, & Ciraulo, 1986; Jones, 1968, 1971; Keeler, Taylor & Miller, 1979; Neriano, 1981; Neriano, McCarthy & McCarthy, 1980; Wikler, 1973; Woodruff, Guze, Clayton, & Carr, 1973).

Determining the incidence of depression among alcoholics and finding ways for screening for depression early in the recovery process has been of interest recently, since some investigators have suggested that among alcoholics, depressed alcoholics are most in need of intensive, long-term therapeutic programs (Willenbing, 1986). Some investigators, however, see no difference in treatment outcome and course of illness between depressed and nondepressed alcoholics (Hesselbrock, Hesselbrock, Tennen, Meyer, & Workman, 1983 ; Schuckit, 1983). Substantial evidence suggests that depressed alcoholics have longer histories of problem drinking, more previous treatments for alcohol misuse, more trouble in resisting use of alcohol, more marital problems, and more physical symptoms related to alcohol abuse than other alcoholics (McMahon & Davidson, 1986).

A recent study of depressed alcoholics found them to be more anxious, tense, restless, apprehensive, and having more somatic symptoms than nondepressed alcoholics (McMahon &

Davidson, 1986). There is also evidence that as a group they are more apt to be interpersonally detached with avoidant or asocial personality traits, to have disorganized and distracted cognition, and to have a negativistic self-image (McMahon & Davidson, 1985).

In their review of the literature on the relationship between alcoholism and depression, Jaffe and Ciraulo (1986) noted that the percent of alcoholics considered clinically depressed depends on the diagnostic criteria and conceptual frames of the investigator, as well as on the point in the cycle of alcohol use and withdrawal in which the patients are assessed. Depressive symptoms may be very common and very intense in alcoholics without warranting the diagnosis of a separate affective illness. These depressive symptoms may clear up very quickly after detoxification. For example, one study reported as many as 98% of recently admitted patients reported depressive symptoms which waned after a few days to several weeks to normal levels (Shaw, Donley, Morgan, & Robinson, 1975). This has led some to the view that only a small percent of alcoholics have persistent severe depression (Keeler et al, 1979; Schuckit, 1979).

Studies that attempted to examine the occurrence of more stable kinds of depression than depressive symptoms after detoxification have produced a wide range of estimates (Cadoret, Troughton, & Widmer, 1984; Freed, 1978; O'Sullivan, Daly, Carroll, Clare & Cooney, 1979; Schuckit, 1979, 1983).

For example, Weissman and Myers (1980) found 44% of community alcoholics had major depression, 15% had minor depression, 6% had bipolar depression and 18% were considered to have depressive personalities. Midanik (1983) found 33% of female problem drinkers and 17 % of male problem drinkers to have a coexistent depression. When the same study examined persons who were alcohol dependent, 56.6% of the females and 19% of males met the criteria for both disorders. Patients may also be divided into those who develop depression before alcohol use and those who develop it after chronic abuse, with the first group being considered primary depressives. In studies where primary depression was used as a criterion, estimates of the proportion of depressed patients ranged between 3% and 46%, with the incidence of primary depression consistently more frequent among female alcoholics (Beck, Steer, & McElroy, 1982; Hesselbrock et al., 1983; Schuckit, 1983; Winokur, Rimmer & Reich, 1971).

In summary, various measures of depressive symptoms taken at different points in the recovery process have resulted in widely different estimates of the incidence of clinical depression and depressive symptoms in alcoholic subjects. Depressive symptoms appear to be most extreme upon admission for treatment, with some gradual decrease over the treatment period. Despite this gradual decline in depressed affect, strong evidence exists that a substantial number of alcoholics also have long-standing clinical or sub-clinical depression.

The Role of Depressed Affect and Affective
Memories in the Etiology of Alcoholism

The high correlation between depressed affect and alcoholism suggests a possible causal connection. Jaffe and Ciraulo (1983) listed ten possible causes for the high incidence of depression among alcoholics: 1) the direct toxic effects of alcohol on the brain; 2) indirect toxic effects, via other organs and body systems; 3) effects of alcohol withdrawal; 4) central nervous system (CNS) effects of drugs (other than alcohol) related to the treatment or use of alcohol; 5) CNS effects of injury or anoxia associated with alcohol-related trauma and/or suicidal gestures; 6) the effects of social losses related to alcohol use; 7) psychological responses to physical impairment related to alcohol use; 8) a personality disorder antedating the alcohol use, and perhaps resulting in alcohol abuse; 9) the effects of an independently transmitted affective disorder; and 10) the effects of a genetically transmitted vulnerability to both affective symptoms and alcoholism.

Jaffe and Cirulo emphasized in their review the difficulty inherent in trying to investigate the relative importance of these possible contributing causes, underlining the difficulty in forming groups of alcoholics that are comparable in terms of the origins of their depressive symptoms, problems in the diagnoses of personality disorders, and problems in identifying the temporal order of onset in

persons with both alcoholism and depression. To this list of difficulties can be added the differences in drinking history and length of abstinence encountered in clinical studies with alcoholics. Not all of the causes for the high relationship between alcoholism and depression suggested here are of direct relevance to the present study. Only two will be described in further detail: biological predispositions to both depression and alcoholism, and predisposing personality characteristics.

The separation of primary depressives from other depressed alcoholics is of special importance in biological studies, because of its inferential value relative to understanding the causal relationship between alcoholism and depression. At one time, the apparent high incidence of primary depression in alcoholics and familial aggregation of both major depression and alcoholism was seen from a biological perspective as evidence that depressive and dysphoric mood played a causal role in the development of alcoholism, and that primary depression and alcoholism were manifestations of the same underlying genetic vulnerability (Bohman, Cloninger, von Knorring & Sigvardsson, 1984; Merikangas, Leckman, Prusoff, Pauls, & Weissman, 1985; Schuckit, 1979). More recently, there is some evidence from the same perspective that, by a process of assortative mating, some individuals inherit independent predispositions to alcoholism and or personality disorders (Bohman et al.,

1984; Cadoret, O'Gorman, Troughton, & Heywood, 1985; Lorantger & Tulis, 1985; von Knorring, Cloninger, Bohman, & Sigvardsson, 1983).

Several theories accounting for alcohol addiction suggest that alcohol is used to escape, or forget, painful emotional experiences (such as depressed, tense affect) rather than the drug primarily being used for its euphoria inducing quality. These theories stress the role of predisposing personality characteristics, such as avoidance, or unmet dependency needs (Freed, 1978). Focusing on the need to escape memories rather than a need to alter or escape current experience, Cowan (1983) tested the hypothesis that alcohol may permit the drinker to forget his previous feelings, both good and bad, rather than make him feel euphoric. He hypothesized that the primary action of alcohol on the emotional system may be to reduce the impact of past experience by blocking emotional memories and associated cognitions, keeping them from intruding on current experience. This would allow current experience to change in accordance to the drinkers expectations and the drinking situation, rather than being dominated by previous emotional experience. According to Cowan (1983):

Euphoric and dysphoric current feelings of various types, as well as increased emotional lability and "disinhibition," can all result from a drug-induced impairment (operationally, a decrease in accuracy) of memory for particular kinds of feelings. For the sober problem drinker, many of these memories are related to his problems, and are therefore unpleasant; forgetting these may be particularly reinforcing. (p.41).

Cowan tested hypotheses related to his theory by using in vivo alcohol doses either during a learning session or a recall session. He randomly assigned 32 non-alcoholic students to one of four drug conditions over the two sessions: placebo-placebo, placebo-alcohol, alcohol-placebo, alcohol-alcohol. Each subject was administered the Profile of Mood States (POMS) (McNair, Lorr, & Droppleman, 1971) five times: during session one before ingestion of drink, and at the end of the session; during session two before ingestion of drink, another for current mood at the end of the session, and a final measure reflecting the subject's memory of the POMS given at the end of the previous session. The POMS is a checklist containing 65 mood adjectives on six scales: Tension-Anxiety, Depression-Dejection, Anger-Hostility, Vigor, Fatigue, and Confusion-Bewilderment.

During each session, subjects participated in several intentional verbal and pictorial memory tasks including free recall of a word lists after one exposure, or several exposures, and four-alternative forced-choice recognition of pictures of men's faces. None of the verbal and pictorial memory tests resulted in significant effects due to alcohol before testing, or before learning. Alcohol produced no significant changes in feelings of any of the POMS scales.

However, there were significant differences in affective memory, as measured by the subjects' accuracy in reconstructing previous POMS ratings. Cowan divided memory

error into two kinds: memory bias, or signed error in estimates of the intensity of previous emotions; and inaccuracy, or absolute error. Both bias and inaccuracy effects of alcohol ingestion on affective memories were demonstrated. Persons given alcohol during incidental learning of moods (session one) exaggerated angry affect at session two significantly more than other subjects. Alcohol ingestion during the learning session caused significantly more inaccuracy on four of the six moods scales (Confusion, Vigor, Depression-Dejection, and Tension-Anxiety, in order of most inaccuracy). Alcohol given before testing increased inaccuracy for moods even more strongly, significantly effecting Fatigue, Confusion, and Vigor, in descending order. One significant interaction of learning and testing states was in contrast to what might have been expected if state-dependent retrieval had occurred: the same drug condition groups showed less accurate memory for Vigor than those that changed condition across sessions. When Cowan computed a "Total Memory Inaccuracy Score" by adding the absolute value differences between learning and test session POM's across the six scales, alcohol produced significant effects both during learning and testing.

Cowan considers this study to be "the first study, performed with a well established and extensively validated mood scale, which demonstrates that alcohol directly affects memory for feelings" (Cowan, 1983, p. 45). He cites five

lines of evidence from his experiment which indicate that alcohol has specific and selective effects on memory for emotional events beyond the general performance impairment known to be caused by alcohol ingestion: 1) Alcohol's effects on memory accuracy are stronger than those on memory bias; 2) Alcohol has different pattern of effects on emotional memory than on verbal and pictorial memory; 3) Alcohol's effects on both learning and testing conditions are specific to certain mood scales; 4) Alcohol induced inaccuracy for moods does not parallel the normal forgetting curve over time, therefore alcohol does not merely potentiate the effects of time on memory for moods; and 5) Alcohol does not alter current feelings while impairing memory for earlier emotional events.

Cowan's research is important in that it attempts to directly measure the psychopharmacological impact of alcohol on memory for moods in order to establish a etiology for pathological drinking that takes into account much that is known clinically about the personalities of alcoholics. However, procedural and measurement problems inherent in his research has caused some workers to cast doubt on his conclusions. His data have, in fact, been analyzed in a manner to support the hypothesis that alcohol enhances memory for the affect current immediately before the ingestion of alcohol (Mueller & Klajner, 1984), supporting the view that persons most at risk for alcoholism feel their best immediately before intoxication (Parker, Birnbaum, Weingartner, 1980).

If Cowan is correct, however, and alcohol use is at least partially motivated by the reinforcing effects of memory impairment, it is possible that depressive realism as described by Alloy and Abramson (1979) has a causal role in the development of some cases of alcoholism. The asymmetrical effects of alcohol on different kinds of affective memories and the asymmetry of mood selectivity effects on memory may also have a role in the development of some variants of alcoholism. Depressed persons may be particularly vulnerable to the abuse of alcohol in order to take advantage of its specific effects on affective memories, which otherwise would intrude on ongoing experience, unaltered by self-protective biases. Some cases of alcoholism, then, might result from alcohol use during attempts at self-medication for excessive realism related to depression.

Several theories have been reviewed to account for the apparently high incidence of depression in alcoholics. Some investigators view this high incidence of depressive symptoms as being related to the phase of the illness in which alcoholics are apt to present for treatment. They suggest that alcoholics obtain treatment at times of reaching "rock bottom", that is when severe physical and emotional symptoms result in acute depression and distress that soon lifts after detoxification. A biomedical perspective suggests that depression and alcoholism have a common

physiological, perhaps genetic, basis that may also be related to the development of personality disorders. Personality traits predisposing individuals to both alcoholism and depression have been suggested, such as dependency, interpersonal ambivalence, and avoidance. Finally, a theory related directly to alcohol's impact on memory for affects was reviewed, suggesting that alcoholism may result from reliance on alcohol to prevent intrusion of negative affects, including depressed affect, into ongoing experience.

Depression and Cognitive Performance

Hasher, Rose, Zacks, Sanft and Doren (1985) proposed that there are two independent frameworks that make predictions about the impact of depressed mood on performance in the memory field. One framework is based on limits in capacity for cognitive tasks (Kahneman, 1973) with depression reducing total capacity, or causing additional demands on available capacity (Hasher & Zacks, 1979). The second framework emphasizes the use of mood as a organizing principle for processing new information and guiding retrieval of memories (Beck, 1967; Kuiper, MacDonlad, & Derry, 1983; Teasdale, 1986). The solid empirical findings supporting each framework are discussed under separate sub-headings below.

Reduction of Cognitive Capacity Due to Depression

The cognitive literature on depression contains

widespread reports that may be interpreted as declines of capacity due to depression, including deficits in problem solving, memory, and rate of learning (Dobson & Dobson, 1981; Cohen, Weingartner, Smallberg, Pickar, & Murphy, 1982; Stromgren, 1977; Weingartner, Cohen, Murphy, Martello, & Gerdt, 1981).

Specifically in alcoholics, some investigators note that it is often difficult to separate the effects of clinical depression and neurological impairment (Gass & Russell, 1986; Hesserlbrock, Hesserlbrock, Meyer & Workman, 1983). Both depression and a history of alcohol abuse have been demonstrated to lower both new learning and immediate memory on psychological tests (Query & Megran, 1984). Recent investigations (Clark & Teasdale, 1982; Coyne & Gotlib, 1983; Riskind, Rholes, & Eggers, 1982) suggest that depressed mood alone may interfere with the retrieval of positive (pleasant) memories from Long Term Memory (LTM). A more recent study using more neutral materials (the Digit Span subtest from the Wechsler Intelligence Scale-Revised and the Logical Memory subtest from the Wechsler Memory Scale) found little additional impact of depression above that of organicity, including organicity due to alcohol abuse. The investigators concluded that the clinical lore associating depression with memory impairment is mainly due to the exaggerated memory complaints of depressed patients (Gass & Russell, 1986). Gass and Russell reached a conclusion that appears to

overlook the possibility that the neutral stimuli used by the investigators would be least likely to uncover distortions related to depression, which may be strongest when materials are emotionally charged and personally relevant. These are the factors emphasized in the second major framework for understanding the effects of depression on memory: the mood selectivity framework.

Mood Selectivity Effects on Memory

Bower's (1980) work on mood and memory has proved seminal in investigating the relationship between mood and memory from the second framework described by Hasher et al. (1985), that of mood as an organizing principle for encoding and retrieval of memory contents. Bower used hypnosis and reading of emotionally charged self-reference statements to induce happy or sad mood states prior to a memory task. He demonstrated that persons so induced had better recall for material that was similar in evaluative content to their mood state. He has labeled this effect of better recall of mood-congruent material a "mood-state-dependent memory" effect. Salience of material that is similar in content to the induced mood has been demonstrated by Bower and associated workers, and has been labeled "the mood congruity effect" (Bower, 1981). Bower frames his work as an extension of the Availability Heuristic Theory, and defined both mood-state-dependent memory effects and mood congruity effects as "automatic". His work is thus in part an

extension of two theoretical perspectives used in the preliminary study (Automatic Processing Theory and Availability Heuristic Theory) by the inclusion of the effects of mood on memory. Although not of direct relevance here, state dependent learning has been used in some models to explain the addictive process and the unfolding of affective memories in psychotherapy (Liard, Wagener, Halal & Szegda, 1982).

In an experiment investigating memory for personal episodes, Bower and associates had subjects record emotional events in a diary for one week. At the end of the week period, hypnosis was used to induce either a pleasant or unpleasant mood in subjects according to random assignment. When subjects were asked to recall recorded emotional events, the number of incidents recalled depended on the original rating of the incident by the subject (either pleasant or unpleasant) and the manipulated mood state at time of recall. Percent of recall was highest for the unpleasant mood condition for both kinds of incidents. In both mood state conditions, recall for incidents that had the same mood content as the manipulated recall condition was much higher. This effect was stronger in the pleasant mood condition, with subjects recalling 92% more pleasant than unpleasant moods. In the unpleasant mood condition the bias was less severe; subjects recalled only 52% more unpleasant incidents than pleasant incidents.

Recently the generalizability of these mood dependent effects has been called in question. In a series of three experiments, Hasher, Rose, Zacks, and Doren (1985) attempted to clarify whether depression reduces overall capacity, whether mood congruent selectivity occurs, and if so, at what point in the memory process (encoding or retrieval) selectivity operates. The methodology differed in these experiments from other investigations in the mood and memory with normal subjects in that the BDI and MCL were used to form groups of naturally occurring depressed mood, rather than resorting to some experimental induction of mood states. The results across all three experiments were that no significant differences were found between mildly depressed college students and nondepressed students on recall of verbal material of differing mood contents.

Bower and Mayer (1985a) have also reported a failure to replicate mood congruent recall, using the original methodology (Bower, Monteiro, & Gilligan (1978). This failure contradicted Bower's (1981) theory of mood as an active retrieval cue and led the authors to view mood dependent recall "an evanescent will-o-the-wisp, and not the robust outcome suggested by earlier reports." (Bower & Mayer, 1985a, p.42). Isen (1985) and Ellis (1985), commenting on these failures to replicate, have stressed the importance of both a possible asymmetrical effect for positive mood and negative mood on memory, with negative mood having a less powerful

selectivity effect than positive mood (Isen, Shalcker, Clark, & Karp, 1978), and the absence of a true continuity of mood from clinical depression to mildly depressed college students. Hasher et al., (1985) do not view their findings as evidence against mood congruent effects in clinical populations, but as a caveat about the consistency of such effects at a lower level in normals.

More recently, Bower and Mayer (1985b) have disagreed with these critiques, arguing that differences in methods between the experimental use of naturally occurring moods and manipulated mood is a potent factor obscuring the detection of such effects in a normal college population. Also, according to their model of spreading activation of associates, it is the present mood state that is expected to result in mood congruent effects, and Bower and Mayer suggest that the BDI and MAACL used by Hasher et al., (1985) are, in part, trait measures of personality.

In an article examining the relationship between mood state and severity of psychopathology in depression and mania, Johnson and Magaro (1987) provided some interesting concepts related to this debate. In their review of the literature, they found trends indicating unsystematic cognitive disruption in mania, and increased severity of pathology leading to increased memory deficit in both depression and mania. They found depressive performance on recognition tasks as characterized by a conservative response

bias, indicating that depressives had more stringent response criteria for reporting recognition than did nondepressed persons. In regard to selectivity effects they concluded that mood, not clinical depression, exerts the most profound influence on the content of material recalled, cutting across diagnostic categories. They conceptualized mood and severity of affective disorder as discrete entities, although related. Mood state may function in a somewhat autonomous manner from diagnosis with regard to recall in memory. They viewed mood as analogous to a train yard switchman, determining the direction of the "train of thought":

Therefore, memory content is hypothesized to be determined by two dimensions, mood state and severity of psychopathology. Both produce their effect by altering the manner in which information is processed --mood by providing contextual cues and schema activation, severity through low levels of effort and the disruptive effects of the presence or severity of psychiatric illness. In addition, we can further hypothesize a relation between memory content in consciousness and mood, such that a positive feedback loop is created (Beck, 1967; Bower, 1981). That is, mood increases the likelihood that thoughts present in consciousness will be congruent with hedonic (mood) state, which in turn will affect mood state such that these thoughts will intensify the mood. (Johnson & Magaro, 1987, p. 38).

In the terminology used earlier in this review, Johnson and Margaro are suggesting that current mood influences content and results in selectivity, whereas severity of psychopathology influences capacity. The current study adopts a similar view of the independence of mood and diagnostic severity by separating state depression from trait

depression. In addition to the capacity reducing effects of depression, the present study incorporates the effects of depressive realism on memory, to be described with the learned helplessness literature, later in this review.

Depression and Memory for Frequency of Occurrences

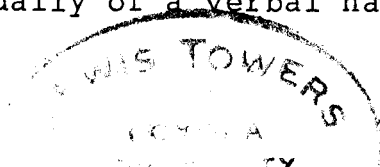
Several studies have been conducted to investigate the impact of depression on memory for frequency of occurrences, often combining concepts and methods from both frameworks described by Hasher et al. (1985). Studies of contingency of reinforcement can also be considered frequency studies, in the sense that the subject's ability to distinguish the frequency of reinforcement in the contexts of various rates and types of responding are the focus of investigation. From this point of view, Alloy and Abramson's (1979) investigation of perception of contingency in depression is a frequency study, although the emphasis is on learning (especially abstraction and generalization) rather than on memory. Perhaps the most influential investigation of the effects of depression on frequency information from a memory framework (with the emphasis on encoding and retrieval) is that of Hasher and Zacks (1979).

Hasher and Zacks (1979) classified subjects as depressed or nondepressed from scores on the Beck Depression Inventory (BDI). Pictures of common objects were presented at controlled frequencies over eight study trials. Study trials were alternated with imaginary trials to determine if

the occurrence of imagined events would influence frequency estimation of presented objects. Results showed no differences between depressed and nondepressed persons.. Also, imagined trials increased the estimate of frequency similarly in both groups. The authors concluded that depression does not influence the ability to accurately estimate event frequency.

Based on this finding related to depression and other findings of no effect on memory for frequency of performance on a large number of subject variables, and learning conditions, Hasher and Zacks (1979) proposed that frequency for memory of occurrences is one of several "automatic processes" that place minimal demands on the capacity of the cognitive processing system, and therefore are not influenced by reductions in capacity, as are more capacity demanding processes (named "effortful" or "controlled" processes in their framework).

In an earlier attempt to clarify issues of capacity and mood selectivity effects for depressed affect specifically for memory for frequency of occurrences (the type of memory investigated in the present study), Curt (1982) categorized studies of frequency into two types: "frequency studies" and "depression studies". The primary focus of frequency studies, in her typology, is the ability to make absolute or relative judgments about the occurrence of specific stimuli, using innocuous, neutral stimuli, usually of a verbal nature.



Depression studies, on the other hand, use emotionally-charged events, feedback about performance, personally relevant stimuli, and require judgments about the occurrence of categories or types of items rather than individual item frequency. In a study designed to investigate the findings of the two kinds of studies by combining all of their elements into one design (using pleasant, neutral, and unpleasant self-statements, category judgments and item frequency judgments) she found that depressed subjects (as determined by BDI scores) did not differ significantly from non-depressed subjects, either in the recall of items, or in the accuracy of their judgments. She interpreted the absence of differences as being due to the non-reinforcing quality of her stimuli (self-reference statements).

Another possibility for the absence of findings of differences between depressed and nondepressed subjects in this study (as well as Hasher & Zacks, 1979) is the use of the BDI to form a "depressed" group from non-clinical college students. Recent research suggests that the BDI is not appropriate for this purpose, due to its vulnerability to social desirability effects and low correlation with independent measures of depression (Tanka-Matsumi & Kameoka, 1986). Hasher et al. (1985) suggests that a non-clinical population is not appropriate for investigating naturally occurring mood state and memory selectivity effects. Also Bower and Mayer's (1985b) proposition that mood biasing

effects would most likely be detected using state measures is relevant here.

The literature on the effects of depressed affect on cognitive performance have been reviewed from the point of view of memory. The typical findings in this area of research have been shown to be related to two guiding theoretical frameworks. Two major interpretations of the clinical relevance of these findings (learned helplessness and depressive realism) will be reviewed in further detail.

Learned Helplessness Theory and Depressive Realism

The learned helplessness theory of depression is based on the similarities between naturally occurring depression in humans and human performance under conditions of being exposed to noncontingent adverse events (Maier & Seligman, 1976; Seligman, 1976, 1975). Under conditions of noncontingent adversity, humans and animals behave as if they have learned that their responding does not matter. They display a reduced incentive for initiating voluntary responses and appear to show cognitive deficits in learning future response-outcome contingencies. Organisms that have learned that outcomes are not contingently related to responses, according to the learned helplessness theory, demonstrate emotional disturbances similar to depression (Abramson, Seligman, & Teasdale, 1978; Miller, Rosellini, & Seligman, 1977; Seligman, 1975a, 1975b; Seligman, Klein, & Miller, 1976).

Learned helplessness theory predicts that depressed subjects will underestimate the degree of contingency between outcomes and their personal responses. Seligman and his colleagues have tested this prediction by means of the chance-skill method, a method involving a series of tasks with outcomes that appear to be determined by either chance or skill. After performing several trials at each kind of task, subjects report their expectations about future trials. The typical finding in studies using this method is that outcomes that appear to be dependent on responses have a greater impact on expectancies for future success than chance determined outcomes.

For example, Miller and Seligman (1976) exposed college students to noise under three conditions: contingent noise, noncontingent noise, and no noise. In a later task with contingent conditions, students exposed to noncontingent noise showed less expectancy change toward success than other students, which the authors interpreted as resulting from a generalized learned expectancy of response-outcome independence. Similarly, depressed subjects have shown less change toward expectancy of success after both success and failure at a task than nondepressed subjects (Klein & Seligman, 1976); and unipolar depressives have shown smaller expectancy changes in a contingent task relative to other hospitalized control subjects and schizophrenics (Abramson et al., 1978).

In an investigation of the learned helplessness model of depression in an alcoholic population, O'Leary, Donovan, Kreger, and Cysewski (1978) advanced reasons for their choice of this population to investigate depression similar to the reason advanced in the present study for viewing depression as a factor in memory biases in alcoholics:

Alcoholics were chosen due to the high relation between depression and this disorder (Weingold, Lachin, Bell, & Coxe, 1968), the similarity between the self-reported affect described by alcoholics and depressives (Gibson & Becker, 1973), and the apparent applicability of the learned helplessness model to this population. (p. 111).

Sixty-two male alcoholics in an inpatient rehabilitation unit were divided into low, medium, and high depression groups based on Beck Depression Inventory (BDI) scores. Procedures identical to those used by Miller and Seligman (1973) were employed to create chance and skill tasks wherein success or failure could be manipulated covertly by the experimenters. The chance condition consisted of a task requiring the subject to predict which of two letters would appear on a slide projector screen. The skill condition involved the manipulating of a string by the subject in an attempt to raise a platform without causing a ball to fall from its resting position on the platform. Success was controlled covertly by means of an electromagnet attached to the platform and a metal strip attached to the ball. In the

chance condition success was manipulated by the experimenter covertly choosing the advance or backward switch on the slide projector.

Five dependent measures for each task condition were developed to measure changes in expectancy : (a) an initial expectancy of success self-rating, performed before the first trial on a zero to ten scale, with zero indicating certainty of failure, and ten indicating certainty of success; (b) The difference in expectancy after the first trial; (c) The total value of "appropriate expectancy shifts" across ten trials (i.e., the sum of increases in expectancies after a success and decreases of expectancies after failures); (d) the total value of "inappropriate" shifts across trials (summed increases in expectancies after a failure and decreases of expectancies after success: an equivalent to the "gambler's fallacy"); (e) the final expectancy after all trials. Successes and failures were alternated, resulting in each subject beginning with a success trial and ending in a failure trial.

Unlike the results of Miller and Seligman (1973) (wherein depressed college students showed consistently lower expectancy shifts of the types described above) the only significant differences were for task condition and end expectancy. The skill task condition resulted in significantly higher expectancies for all levels of depression. Alcoholics with lower levels of depression had

significantly higher end expectancies than those with higher levels of depression. Two significant correlations ($p < .05$) of dependent measures with depression score were found: that with initial expectancy ($r = -.28$), and that with end expectancy ($r = -.25$). The investigators concluded:

Contrary to Miller and Seligman's (1973) results and Klein and Seligman's (1976) contention, present findings do not support the direct applicability of the learned helplessness model to a population of depressed subjects with other forms of psychopathology. While the self-reported affective features are apparently similar between alcoholics and depressives (Gibson & Becker, 1973); the present sample of depressed alcoholics did not evidence the response-outcome independent deficits in the skill task as previously demonstrated by Miller and Seligman (1973) among college students. (p. 112.)

Recognizing the challenge to his theory inherent in these findings, Seligman (1978) requested that O'Leary reanalyze the data using MMPI Hypomania scale as a controlled variable to "purify" the BDI depression measure. In a personal communication, O'Leary informed Seligman that after using post hoc blocking, the high-manic/high depressed subjects demonstrated significantly higher expectancy shifts than did low-manic/high depressed subjects, and that depressed alcoholics showed higher initial expectancies and final expectancies and are "more unrealistically optimistic than matched depressives who are not alcoholic." (Seligman, 1978, p.168). Seligman interpreted these findings as consistent with the learned helplessness modeling of expectancy in depression.

Seligman was correct in perceiving a threat to the relevance of learned helplessness to understanding depressed alcoholics in O'Leary et al.'s (1978) study, but may actually be increasing that threat by his requested reanalysis. A reinterpretation of O'Leary et al.'s findings based on a critique of the experiment's dependent measures, and the relationship between expectancy and contingency clarifies the consistency of the original results and the subsequent reanalysis with the description of depressive realism later offered by Alloy and Abramson (1978). Such a critique and reinterpretation are as follows:

In the described method, noncontingent outcomes alternate in pairs, making the actual probability of success (or failure) equal to .5. Expectancy should be influenced by contingency if subjects are responding "realistically". Therefore, a totally realistic expectancy in the described experiment, would be 5 on the described self-rating scale: reflecting the actual probability of positive reinforcement (.5) and the amount of control (none). A criticism made against many learned helplessness studies is relevant in this case. In both task conditions, outcomes are noncontingent: the subjects are in fact helpless, yet expectancies which appear to reflect recognition of this fact are interpreted as due to a pathogenic process, learned helplessness.

The dependent measures of expectancy change were designed to detect biases in the direction predicted by

Seligman's theory, not to detect changes in expectancy as reflections of accurate perception of contingency and rate of reinforcement. The raw expectancy means, however, are more useful for examining expectancy as a reflection of contingency and frequency of reward. Assuming that initial expectancy (with no knowledge of the task) may reflect subject optimism, or pessimism more accurate perceptions of contingency should have resulted in end expectancy means closer to the self-rating scale mean of 5 than initial expectancy means. Inspection of the reported expectancy means and correlations indicate that subjects with higher levels of depression had end expectancies significantly different than subjects with lower levels of depression, and that were more consistent with contingency: i.e. were closer to the expectancy scale mean of 5. This was true in both task conditions, but more strongly the case for the skill condition. The link between contingency and expectancy may be as strong in the chance condition, but because of the transparent lack of contingency in what could be described to be a task of clairvoyance or telepathy, there was almost no difference between the high depressed subjects' initial and end expectancies, reflecting the maintenance of their initial accurate perception of contingency. In both conditions, (wherein both the paraphernalia and outcomes approximate events in a typical carnival game of chance) the higher end expectancy means for the less depressed subjects may reflect

these subjects' greater inability or unwillingness to perceive the underlying contingency realistically.

Seligman, in his comments on this study, (1978) did not report the expectancy means of the requested reanalysis with hypomania as a controlled variable, and did not make it clear how the comparison with non-alcoholic depressives was accomplished (O'Leary et al.'s study included only alcoholic subjects). This leaves open the possibility that the low-manic/high-depressed subjects were more realistic than the high-manic/high-depressed subjects, if the former's lower expectancy shifts were toward the scale mean of 5, reflecting more accurate perception of the prevailing contingency and frequency of reinforcement.

Note that this study (O'Leary et al, 1978) challenges the learned helplessness model and is consistent with the description of depressive realism that later emerged from such findings. Alloy and Abramson's (1979) article was seminal in suggesting that such findings may be related to the absence in depressed subjects of normal selfprotective biases.

Working within the framework of studying contingency of reinforcement in learning, Alloy and Abramson garnered evidence that raised serious challenges to the learned helplessness model of depression. In a set of four experiments designed to investigate the relationship between actual and perceived reinforcement, they examined depressed

and nondepressed students' abilities to detect the degree of contingency in a task under differing conditions of outcome frequency and desirability. Severity of depression was determined by use of the BDI.

In Experiment 1, the task used was making a green light come on by the pressing of a button. Each subject performed different "problems" in estimating contingency (amount of control) and rate of reinforcement (the lighting of the green bulb) using this task, having been told that control and number of green lights might vary between problems. Subjects were later asked to make judgments of the percent of control (contingency) they had over the light coming on. In the Experiment 1, the reinforcement was manipulated mechanically to be negatively related to the actual degree of contingency, which varied among the three problems performed by subjects. Contrary to the predictions of the learned helplessness model, ratings of contingency by the subjects were found to be highly accurate, with no significant differences between depressed and nondepressed subjects.

In Experiment 2, the subjects' task was the same, but the experimental goal was to assess judgments of noncontingency rather than contingency as in Experiment 1. The learned helplessness model also predicted that depressed persons would be accurate in assessing noncontingency, whereas nondepressed persons would overestimate contingency. Rates of reinforcement differed across problems in this

experiment, but were noncontingent on subject responses. It was found that depressed persons were accurate in their assessment of noncontingency regardless of level of reinforcement. Nondepressed persons, however, overestimated contingency under high rates of reinforcement, but not under conditions of low reinforcement (this trend being more powerful for males than females), thus apparently providing partial support for the learned helplessness model.

Experiment 3 was designed to further examine the illusion of control found among nondepressives in Experiment 2. The task in this experiment was similar except the green light was now associated with the gain or loss of money. In one problem, the light signified a 25¢ loss from an initial \$5 provided by the experimenter (lose problem). In the other problem, the light signified a 25¢ gain (win problem). Frequency of reinforcement was held constant across problems. The Multiple Affect Adjective Check List (MAACL) (Zuckerman & Lubin, 1965) was used in combination with the BDI to form depressed and nondepressed groups. In addition the MAACL was administered again both before and after each problem to assess affect changes related to the rate of reinforcement under noncontingency, yielding a depression change score, a hostility change score, and an anxiety change score. Depressed subjects accurately detected noncontingency of their responses, whereas nondepressed people demonstrated illusions of control. Both groups judged reinforcement to be

higher in the win problem. Both groups showed significant change toward dysphoria in the lose situation, with nondepressed subjects showing greater change in the dysphoric direction in the lose situation, and depressed subjects showing greater change in the euphoric direction in the win situation. The investigators concluded from these findings that under conditions of noncontingency involving hedonistic rewards nondepressed subjects err by overestimating both contingency and outcome frequency.

In Experiment 4, the learned helplessness model hypothesis that depressed subjects would underestimate contingency relative to nondepressed subjects under hedonistic reward conditions was tested. The procedure was similar to experiment 3, but contingency was set at 50% in both problems. It was found that depressed subjects were more accurate than nondepressed subjects in judging contingency of reward. Nondepressed subjects overestimated control in the win problem (especially when the active strategy of pushing a button was most effective) and greatly underestimated control in the lose problem, whereas depressed subjects were accurate about the degree of control regardless as to amount of contingency or the kind of response that was most effective in gaining reinforcement (actively hitting the button, or passively not hitting it and waiting for reinforcement).

Across all four experiments the learned helplessness

hypotheses that depressed persons would underestimate control and that nondepressed persons will overestimate control were not supported. Depressed subjects were consistently accurate in their estimates of control, while nondepressed subjects showed both illusions of control and illusions of no control depending on experimental conditions.

Alloy and Abramson (1979) proposed a revision of the learned helplessness model that would incorporate these findings. The revised hypothesis maintains that there is a motivational deficit in depression that works without perceptual distortion, that is depressives are less apt to initiate successful responses, but are not less able to perceive what the required response would be. The revised hypothesis predicts that depressed subjects will initiate fewer instrumental responses when the required response is complex, due to their motivational impairment. The helplessness experienced by depressives, according to the new model, is not entirely due to the experience of noncontingency filtered through perceptual, attributional, and expectational processes, but may also result from hormonal and physiological sources.

An alternative framework was also proposed to account for the fact that nondepressives were inaccurate in assessments of contingency. Self-esteem maintenance and self enhancement are the cornerstones of this alternative view. The results in all four of Alloy and Abramson's (1979)

seminal experiments can be explained if one hypothesizes that nondepressed persons are motivated to maintain their self-esteem, whereas depressed persons are not. Roots to this viewpoint include Bibring (1953) who argued that depressives are not motivated to retain self-esteem because the mechanism for self-deception has broken down. Depressives have taken off their rose colored glasses, and are "sadder but wiser" according to Alloy and Abramson (1979). The literature on self-esteem is consistent with the view that persons with low self-esteem lack protective perceptual biases. Zuckerman (1979) concluded that self-esteem is maintained by the kind of self-serving attributional biases seen in nondepressed subjects. An attributional style of evenhandedness (willingness to attribute success or failure equally to either the task situation or to the self) has also been observed among subjects with low self-esteem (Fitch, 1970; Ickes & Layden, 1978; Tennen, Herzberger, & Nelson, 1986).

The direction of the causal link between depression and helplessness is still under debate in the depressive realism literature. Building on the revised version of the learned helplessness model of depression which emphasizes the perception and generalized expectancy of noncontingency in the development of depression, Schwartz (1981, 1982) has argued that helplessness can not lead to any form of depression because non-depressed persons do not experience

noncontingency even when it is present. According to his view, depression causes helplessness by producing a deficit in initiating formal hypotheses about ongoing experiences, and therefore preventing the usual bias toward confirmation that accompanies hypothesis testing (Kahneman & Tversky, 1973). According to Schwartz, it is actually an "inferential handicap" that makes depressives appear "wiser". He cites a series of experiments by Reber (1967, 1968, 1976) demonstrating that incidental learning of patterns and abstract principles can be superior to intentional learning of the same ideas, because of the distorting influences of confirmation biases evoked by hypotheses generated by subjects under intentional learning conditions. He views the depressed person as similarly operating permanently under conditions of incidental learning because of a failure to initiate hypotheses about the learning situation. At least one empirical study investigating the role of hypothesis testing in judgments of contingency by depressive and nondepressives supports Schwartz's view, in that depressed subjects demonstrated the same biases as nondepressed persons after they were provided hypotheses to test in relationship to their judgments of contingency (Abramson, Alloy, & Rosnoff, 1982).

Abramson and Alloy (1981) do not subscribe to this view, but see the optimistic biases of nondepressives as a pervasive aspect of human cognition that accounts for their

inability to perceive noncontingency. To them the depressive does not possess a depressogenic bias: "but rather that he or she suffers from an absence of nondepressive cognitive bias" (Abramson & Alloy, 1981, p.444). Recently, however, Abramson and Seligman along with other researchers (Raps et al., 1982) have presented evidence that a depressogenic attributional style (attributing causality of negative events to internal, stable, and global causes) may lead to both helplessness and to depression.

The present study addresses several issues raised in the reviewed literature. The high incidence of depression among alcoholics suggests that some cases of alcoholism may result from depression. One line of investigation suggests that the specific influences of alcohol on affective memories may reinforce the abuse of alcohol among persons with intrusive negative emotional memories, including memories of depressed affect. The literature on depression and cognitive performance outlines several results of depressed affect on and memory. Total capacity may be reduced, resulting in inaccuracy of memory. Biased processing toward a depressive world view may occur, resulting an increase of negative emotional contents in memory. The failure to produce self-protective biases may cause depressed persons (and therefore alcoholics that are depressed) to be more realistic about previous events than others. Considered together, the reviewed research findings and theoretical formulations

suggest that depression may have potent effects on memory, and that differences in memory performance between alcoholics and nonalcoholics that were demonstrated in the preliminary study might be explained by differences in levels of depression between the two groups.

CHAPTER III

THE PRESENT STUDY

General Assumptions

Although differing explanations of the relative objectivity of depressed persons in various learning situations continue to exist, the findings in the literature described earlier are very similar to some of the findings in the preliminary study. The present study will investigate the role of depression in memory differences between alcoholics and nonalcoholics, by testing predictions based on viewing depression as a mediator of cognitive biases and a moderator of apparent cognitive deficits. In order to do this, a very complex interaction of many variables will be limited to a focus on only a few. Therefore, all systematic error will be viewed as related to the effects of level of depression (or conversely to the level of positive affect) although there may be other sources of bias. Similarly, all unsystematic error will be viewed as related to cognitive impairments, as was the case in the preliminary study.

The hypotheses to be tested assume two separate but related depressive processes acting on memory and cognitive strategies related to memory tasks. None of the memory or bias measures is expected to be solely influenced by one

process and not the other. Instead, each hypothesis about the effects of depression requires an assessment of the relevance of trait depression relative to the effects of current depressed mood in the particular memory production in question. In general, the effects of depression on memory are expected to be stronger than state depression effects. Trait depression is expected to interfere with self-protective biases, which minimize negative events and maximize positive events in memory, whereas state depression is expected to increase depressive biases which maximize negative events in memory. Trait depression is seen as primarily disruptive and limiting of normal cognitive processes, whereas state depression is seen as primarily productive of mood specific bias effects.

Alcoholics are expected to have a greater frequency of negative affect over two weeks than the nonalcoholic subjects, due to trait depression. Trait depression and (less significantly) cognitive inflexibility related to cognitive impairments are expected to result in alcoholic subjects having less mood variability over two weeks. Trait related depression effects are expected to result in higher accuracy due to depressive realism, which in this context is assumed to be due to the failure of self-protective biases. A related prediction is made that the extremes of emotions experienced by subjects will have less of a distorting impact on memory for alcoholic subjects than for nonalcoholic

subjects. The nonalcoholics, on the other hand, are expected to have greater distortions in memory toward depicting the self as happier, more confident, and secure, i.e., to demonstrate self-protective bias.

State related depression is predicted to result in overestimation of depressive content, resulting from the greater availability of depressive items, via mood selectivity effects on memory. State depression and, therefore, depressive bias is expected to be greater in the alcoholic group. The latter difference, moderated by trait depressive realism and resulting from a weaker process, is expected to be less dramatic and to be exhibited mainly in the overestimation of negative moods. This prediction is made because trait depression related realism about negative events and state depression selectivity for negative events are expected to combine, resulting in increased overestimation of negative moods. The preliminary study found that the groups did not differ significantly from each other on positive mood item occurrence, but that the alcoholics had significantly more negative mood occurrences. Thus the selectivity effect for positive moods, although perhaps stronger than the selectivity effects of negative affect, is not expected to be different between groups, whereas the (perhaps weaker) selectivity effect of negative moods is expected to result in group differences in memory estimates.

The cognitive strategies used by subjects to perform the memory task are also investigated in the present study. Strategic processing of the memory task is assumed to be reflected in the order in which subjects performed sub-items of memory judgments about the occurrence of various bipolar mood states. Although the actual procedure used will be described more fully in the Methods section, an example using the bipolar item Happy-Sad will be used here to illustrate the concepts the strategic processing measures. Each bipolar mood item was divided into three sub-items: judgments of the rate of occurrence of positive affect (e.g., Happy), neutral affect (e.g., neither Happy nor Sad), and negative affect (e.g., Sad). For each of ten mood items, subjects chose to either perform the sub-items in the order presented on a printed form, or to perform them in another order. Deviations in sub-item performance from the order presented to the subject is assumed to be the result of strategic cognitive processing of the task. Differences in strategic processing are assumed to result in accuracy and bias differences by order of sub-item performance. Memory for frequency of occurrences will be viewed as a function requiring relatively low demands on cognitive capacity, i.e., will be considered an automatic process. Strategic processing is conceptualized here as a complex of cognitive functions requiring relatively higher demands on cognitive capacity, i.e., is considered a effortful process.

Depression and alcoholism are expected to have some negative impact on both amount of strategic processing and unsystematic memory error (due to decreased motivation and cognitive deficits respectively), but the present study will focus only on the effects of depression. Again, two separate depressive processes are assumed, with trait depression inhibiting any kind of processing, and state depression inhibiting a tendency to process information along the positive direction, i.e., to use the positive pole as the first, or anchoring sub-item. Although state depression is assumed to produce increased processing along the negative direction (i.e., to use the negative pole as the first, or anchoring sub-item) this effect is assumed to be weaker than the similar effect of positive mood, due to the asymmetrical nature of mood selectivity effects.

Specific Hypotheses

Trait Depression Hypotheses

Hypothesis 1. The alcoholic group will have a significantly higher level of depression than the nonalcoholic group, as measured by Scale 2 of the MMPI.

Hypothesis 2. Alcoholic subjects will have significantly lower self-protective bias scores than nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

Hypothesis 3. Alcoholic subjects will have

significantly lower absolute accuracy of memory than nonalcoholic subjects, after depression has been controlled.

Hypothesis 4. Alcoholic subjects will have significantly higher rates of negative affective states on ESM records than nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

Hypothesis 5. Alcoholic subjects will have significantly lower variation in moods on ESM records than nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

Hypothesis 6. Alcoholic subjects will demonstrate in their estimates significantly less sensitivity to extremes of ESM mood state occurrence than will nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

State Depression Hypotheses

Hypothesis 7. The alcoholic group will have a significantly higher level of state depression than the nonalcoholic group, as measured by the DEP scale of the SCL 90 at the time of the memory task.

Hypothesis 8. Alcoholic subjects will have significantly higher depressive bias scores than nonalcoholic subjects. This difference will be significantly attributable

to state depression (measured by the SCL 90 DEP scale) as a mediating variable.

Hypothesis 9. Alcoholic subjects will be significantly higher than nonalcoholic subjects in overestimation of negative moods but will not be significantly different in overestimation of positive mood items. This difference will be significantly attributable to state depression (measured by the SCL 90 DEP scale) as a mediating variable.

Strategic Processing Hypotheses

Hypothesis 10. Alcoholic subjects will demonstrate significantly less strategic processing of items in the behavioral observations of their memory tasks than will nonalcoholic subjects. This difference will be significantly attributable to trait and state depression as mediating variables.

Hypothesis 11. Across groups, the first sub-item judgement performed will be significantly different in accuracy than subsequent sub-item judgments.

Hypothesis 12. When overestimation and underestimation are considered by order of sub-item judgment, the first sub-item judgment will demonstrate significantly less underestimation and significantly more overestimation than other sub-item judgments.

Hypothesis 13. Nonalcoholic subjects will demonstrate significantly higher use of positive mood states as the first sub-items performed in behavioral observations of their

memory tasks. This difference will be significantly attributable to trait and state depression as mediating variables.

Hypothesis 14. Alcoholic subjects will demonstrate strategic processing biases as stated in Hypothesis 13 to a significantly lesser degree, but in the same direction as nonalcoholic subjects. This difference will be significantly attributable to trait and state depression as mediating variables.

CHAPTER IV

METHOD

The Preliminary Study

The present study uses the self-report data and accuracy scores from the preliminary study described in the review of the relevant literature. These data are combined with archival data not used in the preliminary study consisting of behavioral observations of the memory task and psychological test data. Although reported elsewhere (Richards, 1986), the methods of the preliminary study will be described here in considerable detail.

The preliminary investigation of memory for frequency of occurrences in alcoholics, was part of a larger, programmatic investigation of the recovery process begun in November 1983, at Parkside Lutheran Center for Substance Abuse in Park Ridge, Illinois. This center is a private hospital specializing in alcoholism treatment. The center was investigating patterns of recovery in alcoholics by use of intensive self-reports measures and structured interviews. The collection of much of the self-report data depended on subjects carrying long-range pagers, used to cue their completion of a standard self-report inventory.

Clinical subjects for this larger investigation were

volunteers recruited from the inpatient population who met two criteria: (a) geographic ease of access to the center for periodic interviews and exchanges of experimental materials, (b) the absence of any clinical judgment on the part of the treatment team that participation would be disruptive of the potential subject's adjustment after discharge, and (c) the absence of psychopathology so severe that it would preclude meaningful participation. Potential subjects excluded from recruitment due to the second criteria were extremely rare. Subjects were introduced to the experiment's purpose and methods in an information meeting, where the voluntary nature of their participation, confidentiality of subject information, and the independence of the study from the facility's treatment activities were emphasized. Participating subjects received a total of \$50 for transportation and other expenses related to their participation. This involved two disbursements, one of \$20 at discharge and a second of \$30 at the investigator's receipt of all experimental materials at the completion of the 90-day participation.

A community sample was recruited from the surrounding residential area to serve as a nonalcoholic comparison group. These subjects received \$25 at the end of their two-week participation. An attempt was made to obtain a reasonably representative sample across the age, gender, and SES ranges typically served by the center. When subjects agreed to

participate, they were given several standard psychological tests, including the Minnesota Multiphasic Personality Inventory (MMPI). All subjects participated under a signed consent and all experimental procedures were reviewed and approved by the hospital's Human Subjects Committee, and were in accordance with the ethical guidelines of the American Psychological Association (APA).

The clinical subjects were randomly assigned to one of three groups. Subjects in Group I carried the pager each day for the entire 90 days. Subjects in Group II followed the same protocol of filling out self-reports when paged, but carried pagers on a two weeks "on," two weeks "off" schedule. Subjects in Group III served as a clinical control group and did not carry a pager at any time. In addition to day-to-day self-reports, Groups I and II were assigned contact schedules for brief biweekly, on-site testing and interviews, alternating with biweekly telephone contacts conducted by trained, supervised research assistants. Group III was assigned only one telephone contact per month and a final on-site interview with testing.

For Groups I and II and the nonalcoholic group, a random sample of the subject's moods and experiences was obtained by means of long-range pagers that were triggered randomly four times per day between the hours of 8:00 am. and 10:00 p.m., seven days per week. Subjects who were scheduled to be "on the beeper" for a given period were to complete a

Daily Activity Report each time they were paged. This report is a self-report measure of mood states, thoughts, and experiences, based on the Experience Sampling Form (ESF), an inventory designed to be used to systematically sample experience by periodically cueing self-reports (Figure 1). The ESF includes items composed of adjectives describing mood state opposites on each pole of a Likert scale. Subjects indicated their mood state and its intensity by placing a mark somewhere along the continuum formed between the two mood extremes.

At the end of two weeks of participation, subjects completed a series of self-report inventories including the Symptom Checklist 90 (SCL 90) (Derogatis, 1977). During the same session, subjects performed a memory task requiring estimates of the percent of occurrence of their previously recorded mood states. These estimates were collected by means of a paper and pencil instrument titled "Memory Task Moment-to-Moment Beep", which divided the above described bipolar adjective items into three categories of mood occurrence: the percent of one mood state, the percent where neither mood item applied, and the percent of the opposing mood state (Figure 2). For example, the Alert-Drowsy bipolar adjective item on the ESF is divided into three ranges: (a) very to somewhat alert; (b) neither alert nor drowsy; and (c) somewhat to very drowsy. Subjects were told to estimate the occurrence of their recorded moods in percentages, with 100%

Figure 1. Modified Experience Sampling Form

What were you thinking about? _____

Where were you? _____

What was the MAIN thing you were doing? _____

	Not at all	Some- what	Quite	Very					
How much choice did you have in selecting this activity?	+-----+-----+-----+-----+-----+								
Did you feel in control of your activity?	+-----+-----+-----+-----+-----+								
How guilty did you feel?	+-----+-----+-----+-----+-----+								
How vulnerable did you feel?	+-----+-----+-----+-----+-----+								
How self-conscious were you?	+-----+-----+-----+-----+-----+								
How much were you concentrating?	+-----+-----+-----+-----+-----+								
How satisfied did you feel with yourself?	+-----+-----+-----+-----+-----+								
	0	1	2	3	5	6	7	8	9

Describe your mood as you were beeped:

	Very	Quite	Some	Neither	Some	Quite	Very	
Alert	0	o	.	-	.	o	0	Drowsy
Happy	0	o	.	-	.	o	0	Sad
Irritable	0	o	.	-	.	o	0	Cheerful
Strong	0	o	.	-	.	o	0	Weak
Angry	0	o	.	-	.	o	0	Friendly
Active	0	o	.	-	.	o	0	Passive
Lonely	0	o	.	-	.	o	0	Sociable
Adequate	0	o	.	-	.	o	0	Inadequate
Free	0	o	.	-	.	o	0	Constrained
Excited	0	o	.	-	.	o	0	Bored
Proud	0	o	.	-	.	o	0	Ashamed
Confused	0	o	.	-	.	o	0	Clear
Tense	0	o	.	-	.	o	0	Relaxed
Fat	0	o	.	-	.	o	0	Thin

Figure 2. Memory Task Moment-to-Moment Beep

Check one: Total Period First 2 Weeks Last 2 Weeks

General Questions:

1. What percentage of the time did you mark (fill out) your book on the EXTREME RIGHT of the mood rating form? _____%
2. What percentage of the time did you mark (fill out) your book on the EXTREME LEFT of the mood rating form? _____%
3. What percentage of the time did you mark the POSITIVE items on the mood rating form? _____%
4. What percentage of the time did you mark the NEGATIVE items on the mood rating form? _____%

Percentage of Responses

Mood Questions

	very 0	quite o	some .	neither -	some .	very o	quite 0	
alert	_____%	_____%	_____%	_____%	_____%	_____%	_____%	drowsy
happy	_____%	_____%	_____%	_____%	_____%	_____%	_____%	sad
irritable	_____%	_____%	_____%	_____%	_____%	_____%	_____%	cheerful
strong	_____%	_____%	_____%	_____%	_____%	_____%	_____%	weak
angry	_____%	_____%	_____%	_____%	_____%	_____%	_____%	friendly
active	_____%	_____%	_____%	_____%	_____%	_____%	_____%	passive
lonely	_____%	_____%	_____%	_____%	_____%	_____%	_____%	sociable
proud	_____%	_____%	_____%	_____%	_____%	_____%	_____%	ashamed
confused	_____%	_____%	_____%	_____%	_____%	_____%	_____%	clear
tense	_____%	_____%	_____%	_____%	_____%	_____%	_____%	relaxed

Percentage (%) of Responses

	<u>Not at all/Somewhat</u>	<u>Quite/Very</u>
How preoccupied were you with eating?	_____%	_____%
How preoccupied were you with drinking/ using?	_____%	_____%
How confident did you feel about your ability to resist the urge to drink/ use?	_____%	_____%
Did you share your feelings with someone close to you?	_____%	_____%

being the total number of times they responded to the bipolar adjective over the two week recording period. Graduate level research assistants administered the task according to written directions designed to impress on the subject that memory was to be used to perform the task, rather than some other strategy, such as guessing what one might have recorded (See Appendix A).

As subjects performed the above described memory task, the order in which they did the three sub-items tallying to 100% for each bipolar mood item was observed by the research assistant, after the preliminary instructions that they were allowed to perform the sub-items in any order they chose. These observations were recorded by the experimenters, as unobtrusively as possible on a standard observation record (Figure 3).

Accuracy measures were constructed by comparing each subjects estimates and recorded mood percentages in each category. A measure of relative accuracy, the discrimination coefficient, was formed by finding the correlation between estimates and actual mood occurrences. Difference scores were used to measure the amount and direction of error on each judgement. These accuracy measures resulted in the patterns of group differences described earlier in the review of the literature. Means and standard deviations by group of these variables are available in Appendix B (Tables B-1, B-2, B-3).

BEHAVIORAL OBSERVATION OF
MEMORY TEST

We want to observe and record the sequence of the answers to the mood and preoccupation/confident/feeling questions.

As the subject fills out the form, observe how they complete these sections and record that information as follows:

1 =FIRST CHOICE 2 = SECOND CHOICE 3 = THIRD CHOICE

	Columns		
	<u>One</u>	<u>Two</u>	<u>Three</u>
alert	_____	_____	_____
happy	_____	_____	_____
irritable	_____	_____	_____
strong	_____	_____	_____
angry	_____	_____	_____
active	_____	_____	_____
lonely	_____	_____	_____
proud	_____	_____	_____
confused	_____	_____	_____
tense	_____	_____	_____

Not at all/ somewhat = 1

Quite/very = 2

	<u>First</u>	<u>Second</u>
Preoccupied eating	_____	_____
Preoccupied drinking/using	_____	_____
Confident	_____	_____
Shared feelings	_____	_____

The Present Study

Subjects

All subjects were participants in the larger investigation of memory accuracy described above. Subjects consisted of 22 alcoholics and 22 nonalcoholics. Alcoholic subjects were selected at random from a larger pool of Group I and Group II subjects in the context study that had completed two weeks of participation and had taken the memory task. All nonalcoholic subjects that completed the two week participation period and the memory task were included in the present study.

The alcoholic group consisted of 11 white males, 10 white females, and 1 black male; whereas the nonalcoholic group consisted of 10 white males, 10 white females, 1 oriental/white female, and 1 black male. The alcoholic group contained 19 persons whose level of education was at or above that of high school graduate, 2 persons who had below a high school education, and 1 person for whom this data was not available; whereas the nonalcoholic group consisted entirely of high school graduates. The alcoholic group's mean age was 32.0 years with a standard deviation of 9.1 years, whereas the nonalcoholic group's mean age was 26.4 with a standard deviation of 7.8 years.

Archival Data

Archival data of several types were obtained for each of the 44 subjects. These data consisted of demographic

characteristics, records of mood over two weeks, memory accuracy scores, behavioral observations of the memory task and depression scores from the MMPI and SCL 90. Several of these sets of data have been described above under the description of the preliminary study. The ESF as a measure of mood, and not simply as a target stimuli for memory will be described below, along with relevant reliability and validity characteristics of the ESF, the MMPI Scale 2, and the SCL 90 DEP scale.

The Experience Sampling Form: A Record of Mood Occurrences

Mood state data for the present experiment were collected by means of the experience sampling method (ESM) (Csikszentmihalyi & Larson, 1984). The method was developed to study the subjective experience of individuals interacting in natural environments, with an attempt to insure ecological validity.

Long range pagers are used to cue subjects to complete self reports of thoughts, moods, activities and other aspects of experience. The pagers are activated, usually by radio, at random intervals during the day, resulting in the cue being unexpected by the subject. Studies using the ESM have included studies of the phenomenology of everyday life (Klinger, 1978; Hurlburt, 1979); changes in self-esteem (Savin-Williams & Demo, 1983); variation in self-awareness (Franzoi & Brewer, 1984); frequency and intensity of moods (Diener & Larsen, 1984; Diener, Larsen, & Emmons, 1984) and

recovery process in alcoholics (Filstead, Reich, Parrella & Rossi, 1985).

In addition to long distance pagers, the ESM utilizes standardized self-report forms, the Experience Sampling Form (ESF). The form is designed to take no more than 90 seconds to complete. Items include questions about the time when the form was completed, and the environmental circumstances, subject's thought content, and ongoing activities. In addition, the form contains a number of Likert scales measuring mood states, levels of arousal, and other self-perceptions. Item content may vary slightly depending on the area of interest of the researchers. For the present study, the original form was slightly modified, with additional questions about substance use, abstinence related activities, and preoccupation with using drugs or alcohol were added (See Figure 1).

Although of the reliability of ESF data is a complex question, they are highly consistent across time within the same individual and within similar activities and situations, while differing significantly among various individuals, situations and activities. The median correlation coefficient on the original eight Likert scale variables has been reported as .60 for adolescents and .74 for adults (Csikszentmihalyi & Larson, 1984). For ESF data of German high school students across one week, Pawlik and Buse (1982) reported correlation coefficients of .57 for locations, .76

for moods, and .80 for motives. Individual consistency over two years for 28 adolescents was unexpectedly high, with test-retest correlations of individual items ranging from .45 to .75 (Freedman, Csikszentmihalyi, & Larson, in press).

The ESF has demonstrated high concurrent validity with physiological measures (such as heart rate and physical posture), activities (such as work versus play), and social contexts (such as being with friends versus being alone). For example, measures of affect and arousal decrease dramatically when subjects are alone, while measures of friendliness and sociability increase when at school for normal adolescents (Larson, 1979). Convergent validity of the ESF with a variety of other psychometric instruments have been demonstrated, including measures of alienation (Gianinno, Graef, & Csikszentmihalyi, 1983); work satisfaction (Csikszentmihalyi & Larson, 1985); intimacy needs (McAdams & Constantian, 1983); intrinsic enjoyment (Hamilton, Haier, & Buchsbaum, 1984); and self-esteem (Well, 1985). The ESF has shown strong predictive validity in distinguishing group membership based on item responses.

Schizophrenics and non-schizophrenics (Csikszentmihalyi & Larson, 1984); bulimic women and normal women, (Larson & Johnson, 1985); light and heavy T.V. viewers (Kubey, 1984); and underachievers and achievers in high school performance (Robinson, 1985); have been demonstrated to have significantly different ESF profiles. In addition, ESF

reports have detected expected significant differences in ideographic studies of perception and experience before and after important life events, such as a suicide attempt (Csikszentmihalyi & Larson, 1984); a marital separation (Wells, 1985); and personality alternations in a case of multiple personality (Hamilton et al., 1984).

Depression Measures

Several general issues are relevant to evaluating the appropriateness and validity of the specific measures of depression used in the present study. Screening for the presence of depression and depressive symptoms is often accomplished by means of self-rating scales. Cut-off scores on these scales are used to determine whether the diagnosis of depression is warranted in any given case. Instruments often used for this purpose include the Beck Depression Inventory (BDI) (Beck, 1967); the Minnesota Multiphasic Personality Inventory, Scale 2 for depression (MMPI-D) (Hathaway & McKinley, 1951); the Hamilton Depression Scale (Ham-D); the Raskin Depression Scale (Raskin et al., 1969) the Mood Assessment Scale (MAS) (Yesavage et al., 1983); the Self-Rating Depression Scale (SDS) (Zung, 1965), and the Symptom Checklist 90 (SCL 90) (Derogatis, 1977).

Correlations among these self-rating scales tend to be moderate (.60 to .93) (Willenbring, 1986). The wide range of estimates for the occurrence of depression in alcoholics (3% to 98%) has been interpreted as in part related to the lack

of agreement among the various scales used for the purpose of diagnosis (Weissman & Meyers, 1980). Willenbring (1986) argues that, ideally, these instruments should be both sensitive to true positive cases and result in few false positives when depression as determined by the current diagnostic system (DSM-III) is used as a criterion. Yet this is currently not the case (Hesselbrock, Hesselbrock, Tennen, Meyer, & Workman, 1983).

Instruments differ from each other and from the DSM-III in the way in which depression is conceptualized. For example, in a study of the factor structure of the BDI and the SDS, Gibson and Becker (1973) found that although the factors present in alcoholics were for the most part similar to those in depressed patients, an additional factor that they labelled endogenous depression did not occur to the same extent in the data for the alcoholics, suggesting that the alcoholics might exhibit the cognitive disturbance associated with depression, without having a true endogenous depression. Since the DSM-III criteria rely heavily on endogenous-type symptoms to determine the diagnosis of depression, any self-report inventory that measures depressive factors other than the endogenous factor may have a low correlation with criterion, yet nonetheless, reflect a kind of depression.

The measures of depression and affect used in the present study consisted of one trait depression measure, the MMPI, and one state depression measures, the SCL 90, and one

measure of general affect over time, the Daily Activity Report form of the ESF. The appropriateness, validity, and reliability of each of these measures will be reviewed.

The MMPI Scale 2 as a Measure of Trait Depression. The MMPI is commonly used as a measure of depression in studies of depression in alcoholics (Dinning & Evan, 1977; Query and Megran, 1984; Willenbing, 1986). The MMPI is an instrument with an extensive literature, most of which reflects its sound convergent and discriminant validity as a measure of personality traits and symptom patterns (Wiggins, 1966, 1969; Wiggins, Goldberg, & Applebaum, 1971). Although at times used to measure short term symptom patterns, the MMPI was designed and is most typically used to measure enduring personality traits (such as trait depression) and longstanding symptom patterns.

Although the MMPI-D was not originally designed to be used alone to discriminate depressives from nondepressives, elevation of the D scale (T-score = 70, over two standard deviations above the mean) is often used alone or in combination with other elevations as an indication of depressive symptoms (Nerviano et al., 1980, 1981). D scale (Scale 2) contains items reflecting a broad range of depressive symptoms, including dysphoric mood and affect, withdrawal, apathy, somatic concerns, ahedonia, lack of motivation, feelings of hopelessness, suicidal ideation and other cognitive expression of depression.

Based on the criterion of MMPI-D T score greater than 70 indicating depression, estimates of the percent of alcoholics categorized as depressed range from 43% (Hesselbrock et al., 1983) to 62% (Zeeler et al., 1979). In one study comparing the Beck Depression Inventory (BDI) and the MMPI-D in their ability to accurately predict alcoholics diagnosed as also having depression as conceptualized in the DSM-III and as determined by the National Institute of Mental Health Diagnostic Interview Schedule (NIMH-DIS), neither instrument agreed well with the DSM-III, but were moderately correlated with each other ($r = .59$) (Hesselbrock et al., 1983). The investigators interpreted the finding of only a moderate correlation between the MMPI-D and the BDI as due to differences in time frames, symptom-clustering criteria, formats, and modes of administration between the two instruments. Willenbring (1986) found that the agreement among the MMPI-D, BDI and Ham-D was not high ($r = .4$), suggesting to him that they measure related but distinct phenomena, and cited evidence that the BDI is more sensitive to state symptoms rather than enduring personality patterns.

State Measures of Depression: the Symptom Checklist

90. The Symptom Checklist 90 (SCL 90; Derogatis, 1977) is a multidimensional self-report inventory comprised of 90 items, each rated on a five-point dimension of distress (0 to 4) from "not at all" to "extremely". The instrument consists of

items comprising nine symptom dimensions and seven items that do not load on any symptom dimension. The symptom dimensions are Somatization (SOM), Obsessive-Compulsive (O-C), Interpersonal Sensitivity (INT), Depression (DEP), Anxiety (ANX), Hostility (HOS), Phobic Anxiety (PHOB), Paranoid Ideation (PAR), and Psychoticism (PSY). Items in each dimension have face validity with these titles. From responses to these dimensions and the seven additional items, three global indices of pathology are calculated. The global indices are the Global Severity Index (GSI), the Positive Symptom Distress Index (PSDI) and the Positive Symptom Total (PST).

The GSI is considered by the test author to be the best single indicator of the current level of pathological symptoms, and is the grand total of the summed distress scores for the 9 symptom dimensions and additional items. The PST reflects the range of problems identified by the subject, and is the count of non-zero responses to the 90 items. The PSDI is a measure of average intensity of distress, and is the mean level of non-zero responses. The discriminant and convergent validity of the SCL 90 has been investigated with both outpatient subjects and inpatient subjects (Dinning & Evans, 1977). The nine symptom dimensions have been shown to have peak correlations ($r > .4$) with analogous MMPI scales while correlating to a lesser degree ($r < .4$) with nonanalogous scales (Derogatis et al., 1976).

The SCL 90 was developed for use with an outpatient medical and psychiatric population, and its use with inpatient populations has been questioned (Steer & Henry, 1979) as has the use of many popular self-report measures of depression with non-clinical samples (Tanka-Matsumi & Kameoka, 1986). Nonetheless, the SCL 90 is sometimes used for measuring depression and other kinds of distress in alcoholics and prototypical symptom profiles for alcoholics have been developed, with alcoholics typically having profile peaks on DEP as well as the Anxiety and Phobia dimensions and lowest symptom scores on Hostility (Derogatis, 1977). This may be compared to profiles for depressed patients from the same source, who tended to have peaks on Obsessive-Compulsive, Depression, and Anxiety with the lowest score tending to be on Hostility.

Several studies have investigated the factor structure of the SCL 90 (Cyr, 1979; Derogatis & Cleary, 1977; Evanson, Holland, Metha, & Yasif, 1980; Hoffman & Overall, 1978; Holcomb, Adams, & Ponder, 1983) with various results, leading several investigators to conclude that the SCL 90 might best be seen as a general measure of distress, rather than possessing the ability to measure types of distress as suggested by its various dimensions. Despite these reservations, for the purposes of the present study, it is important to note that all factor analytic studies of the SCL 90 have found at least one viable Depression factor, and two

studies (Hoffman & Overall, 1978 ; Holcomb et al., 1983) found an Insomnia factor that was highly correlated with the Depression factor.

The DEP dimension is used as a measure of depression in the current study and is therefore of special interest. It consists of 12 items appearing in Table 1.

In an inpatient population, one study (Dinning & Evans, 1977) found the correlation between BDI and SCL 90 DEP to be high ($\underline{r} = .7$) and its correlation with the MMPI-D to be moderate ($\underline{r} = .4$). The same study also found significant correlations of the SCL 90 dimensions and the L and K scales of the MMPI, indicating that defensiveness and dissimulation result in lower scores on the SCL 90 scales while "fake bad" response sets result in higher scores for distress on the SCL 90.

In a study more relevant to alcoholic patients, Rounsaville, Weissman, Rosenberger, Wilber and Kleber (1979) examined the specificity and sensitivity of five depression screening scales in young drug abusers. The SCL 90 was found to have a 94% true positive rate for current depression, as determined by the Research Diagnostic Criteria (RDC) (Spitzer and Endicott, 1978), with most false positive occurring in persons with a history of depression.

In summary, despite a lack of agreement of how specific measures should best be used to measure depression, there is sufficient evidence that the trait measure (the MMPI Scale 2)

Table 1

Items of the SCL 90 Depression Dimension

No.	Symptom Item
5	Loss of sexual interest or pleasure
14	Feeling low in energy or slowed down
15	Thoughts of ending your life
20	Crying easily
22	Feeling of being trapped or caught
26	Blaming yourself for things
29.	Feeling lonely
30	Feeling blue
31	Worrying too much about things
54	Feeling hopeless about the future
71	Feeling everything is an effort
79	Feelings of worthlessness

and state measure (the SCL 90) to be used in the present study have considerable reliability and validity as measures of depression. Also, the ESF both measures mood states over time with demonstrated sound reliability and validity.

Procedure

Several measures of accuracy and bias were constructed for the purposes of the present study from archival absolute difference and signed difference scores. For each subject composite scores for memory accuracy on positive items and memory accuracy on negative items were constructed by calculating the mean absolute error for items of each type. Four scores reflecting bias were calculated by finding the total overestimation and total underestimation for each mood type, positive and negative. From these four scores two measures of hypothesized cognitive biases were constructed. Depression bias scores were constructed by summing the overestimation of negative moods and the underestimation of positive moods. Self-protective bias scores were formed by summing the overestimation of positive moods and the underestimation of negative moods.

The recorded behavioral observations of the memory task were used to develop several measures reflecting strategic processing. The total number of sub-items performed in the identical order as they appeared on the memory task form was used as a measure of degree of strategic processing, with lower scores indicating more strategic processing. The

number of items in which the first performed sub-item was the last presented sub-item on the memory task form was also used as a measure of increased strategic processing. Two measures of the direction of strategic processing were calculated: the number of times that a subject performed negative sub-items when a positive sub-item was presented, and similarly, the number of times that positive sub-items were performed initially after a negative sub-item was presented on the memory form.

MMPI Scale 2 T scores were extracted from the archive for each subject, as were SCL 90 DEP Scale mean intensity scores, demographic data, and ESF records. Analyses were performed to demonstrated hypothesized group differences on relevant variables, and to demonstrate the relationship of those differences to depression.

CHAPTER V

RESULTS

Preliminary Analyses

Demographic Data

Group differences in educational level, gender and race were examined by means of the chi-square statistic, resulting in no significant differences between groups on any of these demographic variables. When age differences were examined, however, alcoholic subjects were found to be significantly older than nonalcoholic subjects. The mean age in the alcoholic group was 32.0 with a standard deviation of 9.1 years; whereas the nonalcoholic group mean age was 26.5 with a standard deviation of 7.8 years, $t(42) = 2.15$, $p < .04$. These variables are summarized in Table 2.

To assess the strength of relationship of age with variables of interest other than group status, the correlation coefficients between age and other variables (i.e. demographic variables, measures of memory accuracy and bias, and measures of depression) were calculated. No significant correlations were obtained. Despite this evidence of only a weak relationship of age with other variables of interest, age was used as a covariate in subsequent analyses of group differences, because of the possibility of significant interactions.

Table 2

Demographic Data by Group.

<u>Variable</u>	<u>Group</u>	
	<u>Alcoholic</u> (N = 22)	<u>Nonalcoholic</u> (N = 22)
Mean Age	32.0 (9.1)	26.4 (7.8)*
Gender		
Males	11	11
Females	11	11
Race		
White	21	20
Other	1	2
Education		
High School or Higher	19	22
Less than High School	2	0
Information not available	1	0

Note. * $p < .05$

Memory Bias and Memory Accuracy Scores

Memory estimates and recorded mood occurrences were compared to produce several measures of memory accuracy and memory bias. Three measures of memory accuracy were of interest: discrimination coefficients, absolute error on positive mood items, and absolute error on negative mood items. Discrimination coefficients for overall relative accuracy were found by computing the correlation of memory estimates to corresponding mood occurrences across each subject's memory task form. Mood items were divided into two types: positive moods and negative moods. The positive mood items were Alert, Happy, Strong, Active, Proud, Cheerful, Friendly, Sociable, Clear, and Relaxed. The negative mood items were Angry, Irritable, Lonely, Confused, Tense, Drowsy, Sad, Weak, Passive, and Ashamed. The two absolute accuracy measures were calculated by summing absolute differences between memory estimates and mood occurrences across items for each mood content type.

Measures of memory bias were calculated from signed differences between each subject's memory estimates and corresponding mood occurrences. Two overestimate variables were formed by calculating the mean of negative signed differences for each mood item type. Two underestimate variables were similarly calculated from differences with positive signs. A measure of depression bias was formed by summing overestimation of negative items and underestimation

of positive items. A measure of self-protective bias was formed by summing underestimation of negative items and overestimation of positive items.

The measures of memory accuracy and bias described here were used as the dependent measures for testing specific hypotheses about the relationship of depression to cognitive differences between alcoholics and nonalcoholics. State depression and trait depression were expected to have different effects on each memory measure. The means and standard deviations of these measures of accuracy and bias are reported by group in Table 3, along with the same information for the measures of depression to be discussed below.

Measures of Trait and State Depression

The MMPI Scale 2 T-score was obtained for each subject, as was the mean intensity score on the DEP Scale of the SCL 90. The means, standard deviations, and significant differences as determined by t -tests for these two variables is reported in Table 3. Alcoholic subjects had significantly higher trait depression scores, $t(42) = 2.30$, $p < .03$. The alcoholic group also had higher state depression, although the difference between group means was not significant. When the cut-off of MMPI Scale 2 T-score greater than or equal to 70 was used to categorize subjects as trait depressed, no nonalcoholic group subjects were categorized as clinically depressed, whereas 8 alcoholic subjects (5 females and 3

Table 3

Measures of Accuracy, Bias, and Depression

<u>Measure</u>	<u>Group</u>			
	<u>Alcoholic</u>		<u>Nonalcoholic</u>	
	(N = 22)		(N = 22)	
	M	SD	M	SD
<u>Depression Measures</u>				
MMPI Scale 2	63.7	(16.3)	54.2	(11.0) *
SCL 90 DEP	.850	(.686)	.625	(.631)
<u>Accuracy Measures</u>				
Absolute Error: Positives	15.7	(5.6)	16.8	(13.5)
Absolute Error: Negatives	11.5	(6.2)	8.3	(3.9) *
Discrimination Coefficient	.80	(.18)	.83	(.15)
<u>Bias Measures</u>				
Self-Protective Bias	14.5	(8.7)	15.9	(9.4)
Underestimation: Negatives	7.0	(4.9)	5.9	(3.7)
Overestimation: Positives	7.5	(5.5)	10.0	(11.1)
Depression Bias	11.0	(8.7)	6.8	(5.7)
Overestimation: Negatives	4.7	(5.2)	2.3	(2.8)
Underestimation: Positives	6.5	(6.0)	4.3	(.9)

Note. MMPI Scale 2 units are T-scores. DEP scale units are mean intensity scores. All other non-correlational variables are measured in percent in error.

Note. * $p < .05$.

males) were so categorized. When the same T-score on the SCL 90 DEP scale was used to categorize persons as state depressed using the norms for psychiatric outpatients, no subject's score reached criterion. When the nonpatient norms were used, 3 nonalcoholic subjects (1 male and 2 females) and 3 alcoholic subjects (1 male and 2 females) were categorized as state depressed. Only one subject had clinical levels of depression on both state and trait measures, a 27 year old white female, who also had the highest frequency of negative affect among all subjects (55.14%) and the second highest discrimination coefficient (.95 compared to the highest of .96).

Correlational analyses were conducted to investigate the assumption that these measures of state and trait depression were significantly related to measures of memory accuracy and bias. The results of these analyses are summarized in Table 4. Both trait and state depression had significant correlations at the .05 level with several measures of accuracy and bias. For trait depression significant correlations occurred on absolute error on negative items ($r = -.367$), absolute error on positive items ($r = .406$), overestimation of negative moods ($r = .446$), depressive bias scores ($r = .347$), and with state depression ($r = .351$). The latter correlation between the two measures of depression is similar to that found in other studies (for example, Dinning and Evans, 1977 reported the correlation to

Table 4

Trait and State Depression Correlations Across Groups

<u>Measure</u>	<u>Trait</u>	<u>State</u>
<u>Depression Measures</u>		
MMPI Scale 2	1.000	.351 *
Frequency of Negative Affect	.217	.582 *
<u>Accuracy Measures</u>		
Absolute Error: Positives	-.116	-.148
Absolute Error: Negatives	.406 *	.290
<u>Bias Measures</u>		
Self-Protective Bias	-.091	-.244
Underestimation: Negatives	.015	-.032
Overestimation: Positives	-.124	-.296 *
Depression Bias	.347 *	.339 *
Overestimation: Negatives	.446 *	.347 *
Underestimation: Positives	.142	.211
<u>Behavioral Indices</u>		
Performed as Presented	.189	.285
Shift from Positive Pole	-.126	-.189
Shift from Negative Pole	-.025	-.217

Note. * $p < .05$.

be .4). For state depression significant correlations were with overestimation of positive items ($r = -.296$), overestimation of negative items ($r = .347$), frequency of negative affect over two weeks ($r = .582$), and depression bias ($r = .339$). The correlation of state depression with absolute error on negative items approached significance ($r = .290$, critical value of alpha at .05, two-tailed = .292).

Specific Hypotheses

Three sets of hypotheses were tested: hypotheses about the relationship between trait depression and affective memory differences between alcoholics and nonalcoholics; hypotheses about the relationship of state depression to these differences; and hypotheses relating both kinds of depression to the behavioral observations of subjects performing the memory task. Each hypothesis predicted a difference between alcoholics and nonalcoholics on a specific memory production or a behavioral index related to the memory task.

The data analytic approach used, unless otherwise noted, was to first demonstrate the hypothesized group differences by means of t tests or ANOVA's. This was followed by an ANCOVA analysis to test the significance of trait depression and state depression as covariates. Each form of depression was predicted to act as a mediator of specific memory differences, with one type of depression having a significant effect while the other type of depression

would not. Despite the prediction of different effects for the two depression variables, trait depression and state depression were expected to be moderately correlated. Therefore, both depression measures were used as covariates in ANCOVA's in order to evaluate the unique influence of each type of depression on the dependent variables. This strategy addressed an alternative explanation that might be given for any significant mediation by one form of depression: that the other form of depression may also, and perhaps better, account for the observed effect. Age was also included as a covariate in the ANCOVA's to control for the significant group difference in age. (In no case was age a significant covariate in these analyses; age is therefore not discussed further in the discussion of individual hypotheses.) Finally, correlational analyses were conducted to investigate the direction and strength of the relationship of each type of depression to the dependent measures.

Trait Depression Hypotheses

Hypothesis 1. **The alcoholic group will have a significantly higher level of depression than the non-alcoholic group, as measured by Scale 2 of the MMPI.**

When a t -test between groups on MMPI Scale 2 scores was conducted, the groups differed significantly in the predicted direction, $t(42) = 2.30$, $p < .025$, one-tailed. When age was entered as a covariate in an analysis of variance, the resulting statistic for the effect of the covariate was not

significant. The statistic for the main effect for groups was significant, $F(1, 42) = 5.97, p < .02$. The hypothesis was supported.

Hypothesis 2. Alcoholic subjects will have significantly lower self-protective bias scores than nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

Statistics resulting from an ANOVA between groups on self-protective bias scores and from an ANOVA on self-protective bias scores with age as a covariate were not significant with alpha set at .05. Subsequent analyses controlling for one or both kinds of depression also resulted in non-significant F -ratios. The hypothesis was not supported.

Hypothesis 3. Alcoholic subjects will have significantly lower absolute accuracy of memory than nonalcoholics after depression has been controlled.

This hypothesis assumes that trait depression related biases may obscure differences in cognitive efficiency between alcoholics and nonalcoholics. Two measures of absolute accuracy were used: one for positive mood items and one for negative mood items. The difference between groups on absolute accuracy for positive items was not significant. Neither age, trait depression, nor state depression had

significant F -ratios as covariates. However, trait depression was found to be significantly correlated at the .05 level with absolute error on positive items ($r = .446$).

The ANOVA between groups on absolute accuracy on negative items produced a significant main effect for groups $F(1,42) = 6.233$, $p < .02$. Analyses of underestimation and overestimation of negative moods described under state depression hypothesis 9 below clarify the source of this significant group difference as being due primarily to overestimation of negative moods. When an ANOVA was performed controlling for the effects of age, trait depression and state depression before calculating the group effect, the main effect for group was no longer significant $F(1, 40) = 1.466$, $p < .3$. Trait depression was the only significant covariate, $F(1, 42) = 5.275$, $p < .03$. Trait depression correlated significantly at the .05 level with absolute error on negative items ($r = -.367$), as did the occurrence of negative affect over two weeks ($r = .555$).

The hypothesis of group differences in accuracy after the statistical control of depression was not supported for either positive or negative mood items. In fact, the opposite effect was observed. Lower accuracy in alcoholics was found to be significantly attributable to trait depression differences between groups. Across groups, trait depression was found to be significantly correlated with decreases in accuracy on positive mood items and increases in accuracy on negative mood items.

Hypothesis 4. Alcoholic subjects will have significantly higher rates of negative affective states on ESF records than nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

The mean frequency of negative moods was calculated for each subject. The alcoholic group mean was 23.29% with a standard deviation of 13.5%, whereas the nonalcoholic group mean was 16.84% with a standard deviation of 8.2%. When an ANOVA was performed calculating the sums of squares for the grouping variable before the covariate effects were removed, the statistic for the main effect for groups closely approached significance, $F(1, 42) = 4.04, p < .052$. The difference between groups was significant and in the predicted direction when tested by means of t -tests, $t(42) = 1.92, p < .04$, one-tailed. Of three covariates entered in the ANOVA analysis (age, state depression, and trait depression) only trait depression significantly explained variance in frequency of negative mood occurrence, $F(1, 39) = 4.92, p < .04$. When an ANOVA was performed removing the effects of age, state depression, and trait depression before calculating the sum of squares for group effects, the group effect statistic was highly significant, $F = 7.519, p < .01$. Contrary to the prediction of this hypothesis, the control of trait depression increased between group variance in the frequency of occurrence of negative mood states.

Hypothesis 5. Alcoholic subjects will have significantly lower variation in moods on ESF records than nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

The variance of mood item response over two weeks was calculated for each subject. The alcoholic group had a mean variance of 243.69 with a standard deviation of 625.8, whereas the nonalcoholic group had a mean variance of 918.59 with a standard deviation of 1626.5; the difference between groups was significant under the one tailed test provided for in the hypothesis, $t(42) = 1.82$, $p < .05$, one-tailed. The F statistic reflected the same level of group effect, $F(1,42) = 3.121$, $p < .09$. The model no longer demonstrated a trend toward significance when age, state depression and trait depression were statistically controlled, $F(1, 39) = .962$, $p < .66$, NS. However, none of the covariates were significant, including that of trait depression. The hypothesis that lower variation in ESF mood records is significantly attributable to trait depression was not supported.

Hypothesis 6. Alcoholic subjects will demonstrate significantly less sensitivity in their estimates to extremes of ESF mood-state occurrence than will nonalcoholic subjects. This difference will be significantly attributable to trait depression (measured by Scale 2 MMPI) as a mediating variable.

The percent of extreme responses to items on the self-report scale was calculated for each subject. All subject self-ratings that utilized the "Very" intensity marker for any mood state were considered extreme. Due to the arrangement of the bipolar scales, the percent of extreme responses corresponded to the percent of responses using either the far right-hand or far left-hand Likert scale marker.

The alcoholic group had a mean percent of extremes of 7.54 with a standard deviation of 9.02, whereas the nonalcoholic group had a mean of 6.84 with a standard deviation of 10.6. The difference between groups was not significant. Discrimination scores described earlier were used as the measure of accuracy. The alcoholic group had a mean relative accuracy of .80 with a standard deviation of .18, whereas the nonalcoholic group had a standard deviation of .83 with a standard deviation of .15. Analyses were conducted using the z transformations of these scores. The difference between groups was not significant. The correlation of extreme responses with accuracy was .2786 across groups ($p = .067$). For the alcoholic subjects alone, this correlation was .2278, whereas for the nonalcoholic group alone the correlation was .3405. Although these group correlations were in the direction hypothesized, neither the correlations for individual groups nor the difference between these correlations approached significance, perhaps due to

the limit in sample size. The hypothesized significant group differences were not supported, therefore the further hypothesized relationship of such differences to depression were not analyzed.

To summarize the findings on trait depression hypotheses: as predicted alcoholics were more depressed than nonalcoholics. They did not, however, have significantly lower self-protective bias scores. Alcoholics and nonalcoholic subjects were not significantly different on absolute accuracy on positive items, with or without covariates in the analyses. For absolute accuracy on negative mood items, alcoholics demonstrated more error, due to overestimation of negative moods. Contrary to prediction, however, this difference between groups was found to be attributable to trait depression. As predicted, alcoholics were found to have significantly higher negative affect on ESF records and significantly less variability in their ESF records. Contrary to prediction, group differences in ESF negative affect were found to be suppressed by trait depression. ESF record variability differences were not found to be significantly attributable to trait depression. Finally, alcoholic subjects had lower correlations between percent of extreme scores and relative accuracy, although not significantly lower than nonalcoholic subjects.

State Depression Hypotheses

The following hypotheses predict bias related to state depression, and assume state dependent memory effects.

Hypothesis 7. The alcoholic group will have a significantly higher level of state depression than the nonalcoholic group, as measured by the DEP scale of the SCL 90 at the time of the memory task.

The alcoholic group had a mean DEP scale score of .850, with a standard deviation of .686, whereas the nonalcoholic group had a mean of .625, with a standard deviation of .631. Although the means were in the predicted directions, the differences between groups on SCL 90 DEP scale scores did not approach significance, and the hypothesis was not supported.

Hypothesis 8. Alcoholic subjects will have significantly higher depressive bias scores than nonalcoholic subjects. This difference will be significantly attributable to state depression (measured by the SCL 90 DEP scale) as a mediating variable.

The one-tailed test specified in the hypothesis was significant at the .05 level, $t(42) = 2.32$, $p < .04$, with alcoholics having the higher depressive bias scores. When an ANOVA was performed with age, trait depression, and state depression as covariates, state depression was not a significant covariate. As stated earlier, the combined subjects correlation of state depression and depressive bias was found to be significant, and in the expected direction ($r = .339$), but smaller than that with trait depression ($r = .347$). The hypothesis of greater depressive bias in alcoholics was supported. However, this difference was not

significantly attributable to state depression. The second half of this hypothesis was not supported.

Hypothesis 9. Alcoholic subjects will be significantly higher than nonalcoholic subjects in overestimation of negative moods but will not be significantly different in overestimation of positive mood items. This difference will be significantly attributable to state depression (measured by the SCL 90 DEP scale) as a mediating variable.

Comparisons by means of t -tests resulted in no significant differences between groups in overestimation of positive moods. ANOVA's controlling for depression and age resulted in no significant differences between groups on overestimation of positive items.

Overestimation of negative moods was not significantly different when tested by means of t -tests. However, when an ANOVA was performed on negative mood overestimation computing the sum of squares for the grouping variable before controlling for age, trait depression, and state depression, the main effect for groups was significance given the one-tailed hypothesis under consideration, $F(1, 40) = 2.991$, $p < .09$. Among the covariates, only trait depression was significantly related to the overestimation of negative moods, $F(1,41) = 7.03$, $p < .02$. Contrary to prediction, state depression was not a significant covariate. An ANOVA on the same variable, but calculating the sum of squares for the grouping factor after the computation of covariate

effects, resulted in a much lower F value for groups, $F(1,42) = .725$, $p = .40$, NS. The hypothesized group difference in overestimation of negative moods was supported. This difference, however, was attributable to trait depression and not to state depression as had been hypothesized.

To summarize the findings on state depression hypotheses: alcoholics did not have significantly higher levels of state depression than nonalcoholic subjects. Alcoholics had significantly higher depressive bias scores, but this difference was not significantly attributable to state depression and was more related to trait than state depression. The groups were not significantly different in overestimates of positive items. Although the groups were significantly different in overestimation of negative items; the difference was significantly explained by trait depression and not by state depression as predicted.

Strategic Processing Hypotheses

This set of hypotheses assumed that order of sub-item performance on the memory task was a behavioral index of the degree and type of cognitive strategy used by the subjects.

Hypothesis 10. Alcoholic subjects will demonstrate significantly less strategic processing of items in the behavioral observations of their memory tasks than will non-alcoholic subjects. This difference will be significantly attributable to trait and state depression as mediating variables.

Two variables were used to assess this hypothesis: the number of items performed in the identical sub-item order presented on the memory task form, and the total number of items where the sub-item presented last on the memory task form was performed first. Descriptive statistics and significant differences between groups for these variables and other behavioral indices of strategic processing appear in Table 5. On the number of items performed as presented, the alcoholic group had a mean of 5.41 and a standard deviation of 3.12, whereas the nonalcoholic group had a mean of 3.45 and a standard deviation of 1.97. The difference between groups was significant, $t(42) = 2.42$, $p < .001$, one-tailed. An ANOVA on identically performed items with age, state and trait depression as covariates indicated that no covariates were significantly related to this variable.

The number of identically performed items was significantly correlated at the .05 level with absolute error on positive items ($r = -.438$), overestimation of positive items ($r = .475$), and self-protective bias ($r = -.347$). Although neither correlation was significant, the correlation of this variable with trait depression was lower than its correlation with state depression ($r = -.189$ compared to $r = -.285$).

On the number of items where the presented pole was substituted for the opposite pole, the alcoholic group had a mean of .818 and a standard deviation of 1.26, whereas the

Table 5

Behavioral Indices of Strategic Processing

<u>Behavioral Index</u>	<u>Group</u>			
	<u>Alcoholic</u>		<u>Nonalcoholic</u>	
	(N = 22)		(N = 22)	
	M	SD	M	SD
Performed as Presented	5.4	(3.2)	3.5	(2.0) *
Performed with Poles Switched	.8	(1.3)	2.2	(1.7) *
Shift from Positive Pole	.3	(.5)	1.2	(1.1) *
Shift from Negative Pole	.5	(1.0)	1.1	(1.2)

Note. Units are number of bipolar items.

Note. * $p < .005$.

nonalcoholic group had a mean of 2.227, and a standard deviation of 1.72, a difference that was also significant in the predicted direction, $t(42) = 3.11$, $p < .002$, one-tailed.

An ANOVA on the number of items with switched first and third presented sub-items indicated a trend of trait depression to significantly explain the variance, $F(1,40) = 3.968$, $p < .06$. The F value of variance due to groups was smaller when the covariates were computed before the group effects, $F(1,42) = 11.750$, $p < .002$ compared with $F(1, 39) = 12.733$, $p < .002$. The hypothesized group differences in strategic processing were supported. While there was weak evidence that depression was related to strategic processing, the group differences in strategic processing could not be attributed entirely to depression.

Hypothesis 11. Across groups, the first sub-item judgment performed will be significantly different in accuracy than subsequent sub-item judgments.

The sums of absolute error of estimates across items for first sub-item performed, second sub-item performed and third sub-item performed were calculated. Means and standard deviations of these variables and other measures of accuracy and bias by order of sub-item performance appear in Table 6. No significant group differences occurred on these variables, although the means were in the predicted direction.

A repeated measures ANOVA was performed across groups on absolute accuracy by order of sub-item performance. The

Table 6

Accuracy and Bias by Order of Sub-item Performance

<u>Measure by Performance Order</u>	<u>Group</u>			
	<u>Alcoholic</u>		<u>Nonalcoholic</u>	
	(N = 22)		(N = 22)	
	M	SD	M	SD
First	85.3	(58.2)	82.2	(52.2)
Second	54.4	(42.9)	47.4	(31.4)
Third	58.3	(42.1)	75.0	(66.7)
 <u>Underestimation</u>				
First	57.4	(42.9)	49.3	(31.4)
Second	70.0	(35.4)	96.1	(64.7)
Third	58.3	(33.4)	51.0	(33.4)

Note. All units of measure are percent in error.

effect for the repeated measure did not approach significance. However, the univariate F test comparing the third choice with the other two choices approached significance, $F(1,43) = 3.442$, $p < .08$. Subsequent t -tests indicated a trend toward the first choice being significantly more inaccurate than the third choice, $t(43) = 1.79$, $p < .09$. The hypothesis as stated was not supported. However, trends were found for differences in accuracy by order of performance.

Hypothesis 12. When overestimation and underestimation are considered by order of sub-item judgment, the first sub-item judgment will demonstrate significantly less underestimation and significantly more overestimation than other sub-item judgments.

Overestimation and underestimation sums by order of sub-item performance were calculated for each subject. The resulting means and standard deviations by group appear in Table 6. No significant group differences occurred on these variables. A repeated measures ANOVA was conducted on overestimation by performance order by group was conducted. No multivariate main effects or interaction effects approached significance. The univariate F -test for overestimation on the second sub-item performed compared with the other two sub-items across groups was significant, $F(2, 40) = 3.6928$, $p < .04$, with overestimation on the second sub-item being lower than that on other sub-items.

A repeated measures ANOVA was conducted on underestimation by performance order by group. The interaction of group by performance order approached significance, Hotellings F approximation: $F(2, 39) = 2.5143, p < .01$, with the alcoholic group underestimating less on the second sub-item performed.

A repeated measures ANOVA was conducted on underestimation by performance order by group. The interaction of group by performance order approached significance, Hotellings $F(2, 39) = 2.5143, p < .01$, with the alcoholic group underestimating less on the second sub-item performed. The hypothesis as stated was not supported. However, evidence was found for differences in overestimation by order of performance, and for a group by performance order interaction on underestimation.

Hypothesis 13. Nonalcoholic subjects will demonstrate significantly higher use of positive mood states as the first sub-item performed in behavioral observations of their memory tasks. This difference will be significantly attributable to trait and state depression as mediating variables.

The number of items on which subjects performed the positive sub-item first although the negative sub-item was presented initially on the memory task form was used as the dependent variable. Alcoholic subjects had a mean of .5 shifts to positive sub-items with a standard deviation of .964, whereas nonalcoholic subjects had a mean of 1.05 with a

standard deviation of 1.21; the difference between groups was not significant. The hypothesis was not supported.

Although the number of shifts to positive poles was not different between the two groups, several interesting significant correlations of this variable with other variables emerged. The number of shifts to positive poles significantly correlated at the .05 level with the frequency of positive affect over two weeks ($r = .412$), the frequency of negative affect over two weeks ($r = -.332$), the number of shifts to a negative pole ($r = .312$), absolute error on the second sub-item performed ($r = -.338$), absolute error on the third sub-item performed ($r = -.376$), and underestimation of the first sub-item performed ($r = -.293$).

Due to the asymmetrical of positive and negative effects of mood on memory, it was not hypothesized that alcoholics subjects would have higher use of negative moods as the first sub-item performed. When the number of items on which a subject did not perform the presented positive pole, but instead performed the negative pole first was investigated, however, unexpected group differences emerged. The alcoholic group mean was .3182, with a standard deviation of .48, whereas the nonalcoholic group mean was 1.182, with a standard deviation of .1.21. The difference between groups was significant, $F(1, 42) = 12.733$, $p < .0002$, one-tailed, after removal of the effects of age and both types of depression in an ANOVA. Only age was significantly related

to shifting to negative sub-items, $F(1, 39) = 4,623$, $p < .04$. This variable had significant correlations at the .05 level with only the sum of absolute error on the first sub-item chosen ($r = -.335$).

Hypothesis 14. Alcoholic subjects will demonstrate strategic processing biases as stated in hypothesis 13 to a significantly lesser degree, but in the same direction as nonalcoholic subjects. This difference will be significantly attributable to trait and state depression as mediating variables.

When groups are compared on the tendency to shift sub-item poles, more shifts of both types took place in the nonalcoholic group, as described above. Across groups the difference between negative shifts and positive shifts was small and nonsignificant, $t(43) = .12$, NS. The nonalcoholic group had more shifts to negative poles than to positive poles, and the alcoholic group had more shifts to positive poles than to negative poles. Therefore, contrary to the hypothesis, the dominant type of shift was different for each group. As stated under the results for Hypothesis 13, the significant differences on use of the positive pole and negative pole were not significantly attributable to depression. The hypothesis was not supported.

To summarize the findings on strategic processing: nonalcoholic subjects demonstrated significantly more strategic processing than alcoholics, as predicted. Some

significant correlations were found between strategic processing variables and depression variables. However, contrary to the assumptions of these hypotheses, group differences in strategic processing were not significantly attributable to depression. Some evidence supported the assumption that order of sub-item performance was related to accuracy in general and specifically to underestimation of the first sub-item performed. Nonalcoholic subjects demonstrated significantly more shifts to both negative sub-items and to positive sub-items than did alcoholic subjects, although only the higher number of shifts to positive sub-items was predicted. Contrary to prediction, the type of shift made did not vary with either the kind of depression or with group membership.

CHAPTER VI

DISCUSSION

The Effects of State and Trait Depression on Affective Memory

Understanding the relationship between the two measures of depression used in the present study is key in understanding the results for all three sets of hypotheses. As was expected, the correlation between the two measures was positive, moderate in magnitude ($r = .351$), and comparable to correlations found in other studies. However, several of the hypotheses tested assumed different and opposing effects on memory of trait and state depression. Trait depression was assumed to cause decreased self-protective bias due to depressive realism, increased inaccuracy of memory related to cognitive inefficiency, and decreased strategic processing related to depression-related declines in initiative, motivation, and capacity. State depression was assumed to cause increased depressogenic bias and to guide the direction of strategic processing by decreasing the use of the positive pole as the first item performed among sub-items.

In the case of hypotheses based on these assumptions about state depression, significant effects, when observed, were found to be stronger for the trait measure than for the

state measure, suggesting that the two kinds of depression at times have similar, not opposing effects, and that the two measures could be considered measures of the same construct, with the MMPI Scale 2 being the more sensitive, or reliable measure.

The finding of low levels of state depression in both groups also challenges the assumption that two separate depressive processes were influencing memory in the present study. From the conceptualization of diagnostic severity and current mood introduced earlier, one would expect individuals who were trait depressed to also be more likely to be in a depressed mood at the time of the memory task. Thus it was predicted that the alcoholic group and the nonalcoholic group would differ in levels of both kinds of depression. However, the level of state depression in both groups was surprisingly low. Even in the alcoholic group, which had 8 individuals with clinical levels of trait depression, only 3 persons were state depressed by the less stringent non-patient norms. Perhaps stronger effects for state depression would have been observed in samples with higher levels of state depression, or larger samples with a greater range of state depression.

Several possibilities exist to account for for this low level of state depression. It could be argued that the SCL 90 is not a sensitive measure of state depression, since it is a measure of general distress over two weeks, and not a measure of a specific affect at the moment of testing. Also

it is possible the administration of the memory task at the end of two weeks of beeper carrying and self-reporting, accompanied by the attention given to the subject's experiences by the experimenters served as a positive mood induction that influenced the entire experimental situation, including memory estimates, SCL 90 scores, and strategic processing of sub-items (both the SCL 90 and the Memory Task were performed in the presence of the experimenters). The SCL 90 and other self-report measures of depression have been shown to be highly correlated with measures of social desirability response sets (Tanaka-Matsumi & Kameoka, 1986).

Some evidence for two separate depressive processes related to memory was garnered from the data, despite the low level of state depression. Two exceptions to the generalization that trait depression effects are stronger than and parallel to state depression effects were found in the present study; both were consistent with the assumption of two separate depression processes. The first exception was that state depression was more strongly related to strategic processing than was trait depression. The second exception was found among correlations between the depression measures and the components of depression bias scores and self-protective bias scores. Whenever the correlations were of any interpretable magnitude, the two types of depression had relationships in the same direction, but the type of depression with the higher correlation varied among the

measures. Although it is possible to attribute these differences to sampling error, they are consistent with theories premising two separated depressive processes. (These exceptions will be discussed in more detail later and are cited here only as partial evidence of separate depressive processes).

Given the caveats concerning the state depression measure and the low levels of state depression observed, the one significant effect for state depression different for that of trait depression and several situations where state depression had stronger, albeit nonsignificant, correlations with relevant variables are viewed here as fairly persuasive evidence for two separate depressive processes. Therefore, the effects of each type of depression on memory and cognitive strategy will be discussed separately before an attempt will be made to understand their combined effects.

In the present study, the effects of trait depression on memory were assumed to be stronger than those for state depression, and more hypotheses related to trait depression were tested. To briefly review the results related to these hypotheses, as predicted, the groups differed significantly in the level of trait depression. However, not all of the differences found between groups on negative affect measures, record variability measures, and memory were related to trait depression. Memory accuracy for negative and positive moods was related differently to trait depression, which increased

accuracy on positive moods and decreased accuracy on negative moods. When depression was controlled, negative affect differences were more pronounced between alcoholic and nonalcoholic subjects. Memory estimates by alcoholics were less sensitive to extremes of mood state occurrences, although they had experienced a higher percent of extreme responses.

These results suggest that trait depression is related to negative affective experience, but that alcoholics when compared to nonalcoholics experience significantly more additional negative affect that is unrelated to depression. Trait depression is related to reduced variability in emotional experiences, as well as to increased emotional extremes; these extremes subsequently have less of a distorting impact on memory in trait depressed alcoholic subjects than is the case with the less depressed non-alcoholic subjects. The later finding is consistent with the depressive realism prediction that alcoholics, being more depressed, will recall experiences (especially negative experiences) with less self-protective bias than controls.

Despite the significant effects of depression related to differences between the alcoholic and nonalcoholic groups, controls were not found to have significantly higher self-protective bias scores, as would have been consistent with the depressive realism view that nondepressed persons distort memories in order to protect themselves, whereas depressed

alcoholics do not. A cognitive deficit view also does not account for the findings, since accuracy varied by item type, with the two groups being significantly different only on items with negative content. The findings on trait depression are more consistent with the view that alcoholics have a depressogenic bias: alcoholics overestimate negative events more and overestimate positive events less than do nonalcoholic subjects. Note that this pattern remains even when the frequency of negative mood state occurrence is statistically controlled.

On the other hand, contrary to predictions following from the assumption of depressogenic cognitive schema in alcoholics, the alcoholics underestimate negative mood items more than nonalcoholic subjects. The fact that the two groups report negative affective memories with different degrees of accuracy, with the alcoholic group both overestimating and underestimating negative affects more than nonalcoholics is consistent with a psychodynamic interpretation that the alcoholic subjects have powerful defenses against negative affect which involve alternately avoiding and immersing themselves in negative affects, especially depression (Khantzian, 1980).

Full investigation of this possibility would involve item by item analysis of the data to find which items were contributing most to these differences, with apriori hypotheses made about each item in regard its relevance to

the various spheres of conflict typical of alcoholics. Such an investigation would also have to take into consideration that the alcoholics treated in an Alcoholics Anonymous oriented facility (as was the case with those in the present study) are engaged in daily ideological training related to the relative value and dangers of mood states and the importance of the "owning" of various negative emotions. If such training is effective, a new basis of self-esteem based on perceiving one's self as either consistent or inconsistent with the treatment ideology may influence emotional experience, self-reports of that experience, and ultimately may influence memory estimates. In essence, an artificial source of cognitive bias related to affect and affective memories, an artificial defense, may be provided by Alcoholics Anonymous oriented treatment to alcoholic subjects and not to the nonalcoholic subjects. Additional conflict about negative moods might be created by such treatment if the new treatment related cognitive biases are not compatible with the alcoholic's previous cognitive predispositions.

Beyond the effects of indoctrination and attitude change, such treatment may also involve increased rehearsal of various moods, both covertly and overtly. Lynn Hasher (personal communication, November, 1986) has suggested the group differences in accuracy by emotional content found in the preliminary study might be due to different levels of

memory rehearsal between groups. Such rehearsal could be generated by affective processes, naturally occurring cognitive structures, or the directed rehearsal provided in A.A. group activities. Clearly, however, confirming or refuting these speculations remains beyond the scope of the present study.

Little remains to be discussed about the effects of state depression as independent of trait depression. Although the two groups did not differ significantly in the level of state depression, evidence was found to support the existence of increased depressive bias in alcoholics, with this bias consisting primarily in the overestimation of negative emotional events. Depressive bias differences were not significantly attributable to state depression, but across groups depressive bias was significantly correlated with both forms of depression, with this relationship being stronger for trait depression than state depression. These findings appear to be consistent with Beck's view of a depressogenic cognitive stance wherein cognitive structures influence perceptions, affects, and memories, which in turn interactively influence mood predispositions. The observation of depressive bias in alcoholics in the absence of high levels of state depression favors Beck's cognitive schema view, which provides the mediation of memory bias by a depressogenic cognitive structure that is not dependent on current depressed affect. These findings do not support

Bower's original formulation of mood selectivity theory, however, which states that it is the current mood, not longstanding cognitive or affective predispositions, that influences availability of mood relevant memories.

The present study's procedures and hypotheses have assumed that it is the combined effects of state and trait depression that accounts for accuracy and bias differences between alcoholics and nonalcoholic subjects. As mentioned earlier, the exceptions to the general rule of trait depression having a stronger influence on accuracy and bias than state depression are an important place to begin formulating what those combined effects might be. When the correlations of state and trait depression with the components of self-protective bias and depressive bias are examined, a plausible model for the combined effects of two separate depressive processes can be constructed based on their selective impacts on positive and negative affective memories. Increases in state depression were significantly related to decreases in overestimation of positive items, whereas the relationship of this variable with trait depression was not significant. The correlation of underestimation of positive moods with state depression was higher than that of trait depression with this measure. Both types of depression had negligible correlations with the underestimation of negative moods. Both types of depression had significant positive correlations with the overestimation

of negative moods, with that with trait depression being appreciably higher.

These correlations may be the result of two depressive processes, which can be distinguished from each other by their different magnitudes of impact on positive and negative affective memories. In turn, these different impacts on affective memories can be viewed as related to self-protective bias and depressive bias. State depression may be the primary source of self protective bias deficits which it causes by inhibiting an otherwise dominant trend toward maximizing positive events in memory. Trait depression may be the primary source of increased depressive bias by maximizing negative events in memory, perhaps in part via increased covert rehearsal of negative moods.

This view combines Alloy and Abramson's (1981) construct of the absence of self-protective bias in depressives with the depressogenic bias of Beck (1974) and other cognitive theorists. The fact that in the present study, state depression correlated with positive memory content more than did trait depression is also consistent with reports in the literature of asymmetrical mood selectivity effects, that is that congruity of mood and stimuli increases recall and retrieval for positive stimuli, but the same effect is less powerful for negative mood and negative stimuli (Isen, Shalcker, Clark, & Karp, 1978). Asymmetry of mood enhances integrative theories such as Johnson and Magraro's (1987).

Strategic Processing, Memory, and Affect

The order of sub-item performance and accuracy by sub-item performance were used in the present study as behavioral indices of cognitive strategies involving both affective and cognitive components. Weighing the relative strengths and directions among hypothetical affective and cognitive processes to predict outcomes on a previously unexplored behavioral measure is clearly a risky endeavor. Nevertheless, significant differences between groups on these behavioral indices were found, accompanied by significant relationships with cognitive and affective measures. Unfortunately, the significant relationships among these sets of variables were not consistently those predicted.

As predicted, significant relationships were found between behavioral measures of cognitive strategy and measures of cognitive output: accuracy and bias measures. Due to differences in strategic processing, the two groups had different typical outcomes on the first sub-item performed. For the alcoholic group, the first sub-item performed was most likely to be the first sub-item presented on the memory task form, and to be less accurate than the two subsequent sub-item judgments. For the nonalcoholic group, the first sub-item performed was not likely to be the one presented on the memory form, and was not likely to be the most inaccurate of the three sub-item judgments. For both groups the first sub-item performed was on the average the most overestimated among the three sub-items.

Evidence was provided for the assumption that accuracy and bias were related to the order of sub-item performance. As strategic processing increased (as measured by the number of sub-items performed in an order other than that presented on the memory task form), self-protective bias scores increased significantly, as did accuracy on positive mood items. The number of shifts to negative sub-items was significantly correlated with increased accuracy on the first sub-item performed. The number of shifts to positive items was significantly correlated with less underestimation of the first sub-item performed, and with reduced error on subsequent sub-items. Taken together these findings suggest that increased strategic processing as defined in the present study is related to increased accuracy and increased self-protective bias.

The nonalcoholic group made significantly more shifts to both positive and negative mood poles than did the alcoholic group. Contrary to earlier predictions, this significant difference between alcoholic and nonalcoholic subjects in strategic processing was not significantly attributable to depression. However, the negative correlation of state depression with items performed as presented approached significance. This suggests that the moderate levels of state depression observed in the present study may increase strategic processing, whereas (again contrary to the prediction made in the present study) trait

depression has less impact on strategic processing of this kind of task.

Stronger evidence linked the use of strategic processing to affect generally, rather than to specific measures of depression. Strategic processing was found to increase significantly with positive affect over the two week recording interval. The number of shifts to positive poles increased significantly with the frequency of positive affect over two weeks and decreased significantly with the frequency of negative affect over two weeks. Given these significant relationships of strategic processing variables with occurrences of affect in ways consistent with a process that would be expected to decrease with depression, it is surprising that no significant relationship with depression was detected, and that the relationship that approached significance suggested an increase in strategic processing with an increase in state depression.

Several explanations of these findings may be advanced. A simple explanation of the failure to find significant relationships between strategic processing and depression is that the absence of the expected relationship is an artifact of the depression measures. Specifically, the limitations of the state depression measure and the unexpectedly restricted range in state depression described previously may have obscured the relationship between state depression and strategic processing. A similar argument to account for the

failure to find a relationship between trait depression and strategic processing is less convincing, however, since high levels of trait depression and significant differences in trait depression scores were found between alcoholics and nonalcoholics.

Perhaps, as was assumed in the hypotheses of this study, the amount and direction of strategic processing are controlled by different kinds of depression. Trait depression may determine the amount of strategic processing, while state depression may determine the choice of either the positive or negative mood pole when it does occur. This would explain the higher strategic processing by nonalcoholic subjects, who had significantly lower trait depression, and the absence of any clear difference in the direction of processing either within or between the groups, due to the low level of state depression in both groups. However, two facts argue powerfully against this interpretation: the differences observed between groups were not significantly attributable to depression, and the trait depression measure (on which the groups were significantly different) was only weakly correlated with the amount of strategic processing.

Another possibility is also consistent with the assumption of two separate depressive processes which may interact. Trait depression may inhibit strategic processing, whereas state depression may increase it. In such a scenario, individuals with low trait depression and moderate

state depression would be most likely to exhibit strategic processing. These individuals would most likely occur in the control group, which had significantly lower trait depression and roughly equivalent state depression when compared to the alcoholic group. A similar explanation involves interactions between levels of depression rather than interactions between types of depression, and is based on the hypothesis that the relationship between any form of depression and strategic processing is not linear. For example, high levels of depression may inhibit strategic processing, moderate levels may motivate increased strategic processing, and low levels may not provide the requisite motivation.

To the extent that the difference between groups in strategic processing may be attributed to some depressive process, or interaction of depressive processes, the relative paucity of strategic processing in alcoholic subjects appears to argue against both the earlier proposed psychodynamic view of defensive responding in alcoholics and Beck's depressogenic world view theory (Beck, 1976), but is consistent with Schwartz's interpretation of depressive realism as related to the absence of cognitive strategies and the general failure of the defenses in depression (Schwartz, 1981a; 1981b).

Personality traits other than the predisposition toward depression are also probably reflected in the strategic processing measures. Subjects were told they may perform

sub-items however they may choose, but were not otherwise encouraged to develop any strategy. Passivity, compliance, and oppositionality are traits that may be relevant in such a context, as well as creativity and field dependence/independence.

Explanations of these group differences based on factors other than personality may be more convincing. Perhaps cognitive capacity differences between groups is a relevant factor in determining strategic processing differences. The fact that the groups have roughly equivalent memory accuracy performances when item content is not considered makes this interpretation less plausible upon first examination. However, memory for frequency of occurrences is conceptualized as a low capacity demand cognitive function, has been typified as automatic, or effortless. The memory task is a test of incidental learning. Cognitive functions other than incidental learning and are involved in the strategic processing measures. The strategic processing of a cognitive task is conceptualized as a relatively effortful, high capacity demand cognitive function, requiring more mental faculties, and involving intention, initiative, and probably the absence of apathy toward the task. The alcoholics might have been less motivated to perform the task creatively, or less apt to use available cognitive strategies while maintaining the ability to do so. This passive, less initiating tendency is strikingly similar in some ways

to what one might expect from depressed subjects, but may have sources other than depression in alcoholic subjects. These other sources may also account for the significant relationship between gender and strategic processing, such that males performed significantly more items as presented, as did alcoholic subjects. Since male alcoholics typically have more severe courses than female alcoholics, according to some researchers, the observed differences in strategic processing may be related to a severity factor that reduces effortful processing, or the spontaneous initiation of effortful processes.

Limitations of the Present Study

The present study would have been improved considerably by increasing the number of subjects in each group, providing multiple measures of both trait and state depression, and measuring the severity of both cognitive deficits and alcoholism symptoms. In addition, the concept of bias and how it is measured might have been refined.

Several caveats have already been advanced about the use of the SCL 90 as a measure of state depression, and the possibility that a positive mood induction was unintentionally included in the administration of the memory task and state depression measure. The SCL 90 has been used for this purpose in other studies, and despite its limitations, it nonetheless remains appropriate in studies involving clinical subjects. Although depression was operationally

measured in two ways (the SCL 90 DEP and the MMPI Scale 2) two separate constructs were assumed. A separate measure of state depression specific to the moment that the memory task was performed would have been a valuable addition to this experiment, which for other constructs (accuracy, bias, strategic processing) utilized more than one operational measure.

A ESF self-report of mood at the time of memory task performance might have provided a suitable second measure of state depression. This would have provided both a second measure of state depression, and a means to investigate the possibility that the experimental procedures themselves contained a mood induction. ESF reports for subjects could be obtained after systematically controlling for the presence or absence of two weeks of self reporting, interviews, and memory task. Following the same logic for a second measure of trait depression, perhaps the BDI or other trait measure could have been used for a second measure of this construct.

The use of relatively small numbers of subjects in each group is both a strength and a weakness of the present study. Significant differences and correlations were found even with these modest samples; the use of larger samples might have made less ambiguous those situations in which one-tailed tests were required to reach significance, or where statistical tests approached significance.

Ideally, the present study would include a measure of

cognitive efficiency separate from the memory accuracy and bias measures related to ESF records. Scores from subtest of the WAIS-R or other relevant tests sensitive to cognitive deficits might have been used. The Wechsler Memory Scale paired associated learning subtest might have provided a measure of affectively neutral verbal memory and learning. Such measures could be used to investigate the possibility that cognitive efficiency or capacity differences mediated some of the differences between groups that are not attributable to depression.

Related to the criticism that no external measure of cognitive efficiency or capacity was used in the present study is the criticism that a severity measure for alcoholic course would be needed to understand any between group differences that might emerge on any of the other measures. It is notoriously difficult to equate courses and severity of alcohol abuse, weighing years of use, periods of abstinence or unproblematic use, binge using, dose per episode, average intake per interval of use, and the presence and frequency of pathognomic symptoms such as hallucinations, black outs, and physical complications. The alcoholics in the present study were equated only by their recent treatment. This implies only a degree of alcohol related symptoms severe enough to result in treatment, and the existence of a period of recuperation, abstinence, and recovery deemed sufficient enough by the treatment facility to merit discharge. Clearly

both depression and cognitive deficits may be related to severity of use, and a measure of what probably was a wide range in severity of alcoholism would have been useful in investigating these relationships.

Finally, the concept of bias is limited in the present study. Bias in regard to affective memory may exist in at least three forms. Bias may exist not only in the degree of overestimation or underestimation, as was the emphasis in the present study, but also bias may be reflected in the tendency to overestimate or underestimate generally. It may also be reflected in both more overestimation and more underestimation on the same category of mood item, as was the case with alcoholics in the present study who overestimated and underestimated negative moods more often than controls, yet had higher correlations of their estimates of negative moods with their reported negative moods, thus suggesting that the greater error was not simply inaccuracy, but bias. As suggested earlier item by item analysis of this third type of bias is required. Any of the three forms of bias discussed here may be mediated by depression or other affective factors.

Conclusions and Implications for Future Research

The present study made several methodological contributions to the understanding affect and cognition in alcoholics. By using both state and trait measures of depression and clinical subjects, it provided an example of

operationalizing recent theoretical formulations about the effects of current mood and affective psychopathology on memory. To the author's knowledge, it is the first study to introduce both state and trait depression measures in research on memory biases related to depression. This is also the first study to use the ESM to investigate memory for rate of mood state occurrence. Another methodological contribution was the development of behavioral indices of cognitive processes. Based on order of item choice, these behavioral measures consistently resulted in significant differences between alcoholics and nonalcoholics, suggesting that such indices may be useful for investigating variables related to alcoholism other than depression.

The present study's findings lend support to Beck's (1976) theory of depressive biases in the cognitive functioning of depressives, specifically for affective memory functioning. It was demonstrated that depression is significantly related to the tendency to make more error on items with negative emotional content than on other items, with the additional error primarily being due to the overestimation of rates of reported negative mood states. This finding, among others in the present study, adds to the growing body of research that points to an asymmetry of the selectivity effects of mood on memories for positive versus negative content (Isen, et al., 1978).

The present study suggests several directions for

future research. Research using the ESM is growing, but the use of that method to investigate memory for frequency of mood occurrence is unique to the present study. It is hoped that other studies will be conducted investigating memory for mood in a wide range of clinical and nonclinical populations. The role of affective predispositions, current affect, and cognitive structures relevant to affect would remain variables of interest in such studies regardless of the population under investigation.

The critique above provides more specific directions for future research. A study similar to the present study could be conducted, but with a larger number of subjects, added depression measures, external measures of cognitive efficiency, a severity index for alcoholism, and an improved approach to bias.

Studies of memory bias before and after in vivo ingestion of alcohol might be incorporated in a programmatic investigation of some of the issues raised in the present study. Intentional mood induction before the memory task could also be used to investigate the relationship between state and trait depression in alcoholics and normals, with induction of various moods combined with alcohol ingestion in some trials to investigate the effects of alcohol on memory accuracy and bias under varying conditions of current mood. Memory for affects in controlled stimuli other than the self might also be investigated under the same conditions, such as

memory for the affect expressed by characters in a brief story or film.

The attempt to track cognitive strategies by means of measures formed from simple behavioral observations could produce several fruitful lines of research. Depression did not account for most of the observed differences between groups, leaving much room for personality and cognitive variables to be investigated. Perhaps the simple method of observing order of performance of a limited range of choices could be used effectively for investigating functions other than memory. These functions might include those related to decision making and problem solving: for example, information collection, abstraction, and the way people divide whole tasks into smaller parts.

In general, any research conducted with the goal of delineating further the role of various affective processes on memory in alcoholics, that may not be symmetrical for nonalcoholics would be a useful contribution to following up on some of the findings in the present study, and clarifying some of the many issues it leaves unresolved. Although the scope of such studies might be more focused than that of the present study, and more tied to a specific theoretical perspective, it is hoped that this attempt to combine affective, cognitive, and behavioral aspects of a clinically relevant situation will serve as encouragement to other researchers to also avoid isolating these aspects in future studies.

REFERENCES

- Abramson, L., & Alloy, L. (1981). Depression, nondepression, and cognitive illusions: Reply to Schwartz. Journal of Experimental Psychology:General, 110, 436-447.
- Abramson, L., Seligman, M., & Teasdale, J. (1978). Learned helplessness in humans. Critique and reformulation. Journal of Abnormal Psychology, 87, 49-74.
- Alloy, L. & Abramson, L. (1979). Judgment of contingency in depressed and nondepressed students: Sadder but wiser. Journal of Experimental Psychology: General, 108, 441-485.
- Beck, A. (1967). Depression: Clinical, experiential, and theoretical aspects. New York: Harper & Row.
- Beck, A. (1976). Cognitive Therapy and the Emotional Disorders. International Universities Press, N.Y.
- Beck A., Steer, R. & McElroy, M. (1982). Self-reported preoccupations of depression in alcoholism. Drug and Alcohol Dependence, 10, 185-190.
- Beck, A., Ward, C., Mendelson, M., Hock, J. & Erbaugh, J. (1961). An inventory for measuring depression. Archives of General Psychiatry, 4, 561-571.
- Becker, J., & Jaffe, J. (1984). Impaired memory for treatment-relevant information in inpatient men alcoholics. Journal of Studies in Alcohol, 45, 339-343.
- Becker, J. & Kaplan, R. (1986). Neurophysiological and neuropsychological comcomitants of brain dysfunction in alcoholics. In Psychopathology and addictive disorders. Roger E. Meyer (Ed.) Guilford Press, NY:NY.
- Bohman, M., Cloniger, R., von Knorring, A., & Signordsson, S. (1984). A genetic study of somataform disorders: III. Cross-fostering analyses and genetic predispositions to alcoholism and criminality. Archives of General Psychiatry, 41, 872-896.
- Bower, G. (1981). Mood and memory. American Psychologist, 36, 129-148.

- Bower, G., & Mayer, M. (1985b) Failure to replicate mood-dependent retrieval. Bulletin of the Psychonomic Society, 23, 39-42.
- Bower, G., & Mayer, M. (1985b). Naturally occurring mood and learning: Comment on Hasher, Rose, Zacks, Sanft, and Doren. Journal of Experimental Psychology: General, 114, 396-403.
- Bower, G., Gilligan, S. (1979). Remembering information related to one's self. Journal of Research in Personality, 13, 420-432.
- Bower, G., Monteiro, K., & Gilligan, S. (1978). Emotional mood as context of learning and recall. Journal of Verbal Learning and Verbal Behavior, 17, 573-585.
- Brandt, J., Butters, N., Ryan, C., & Bayog, R. (1983). Cognitive loss and recovery in long-term alcohol abuser. Archives of General Psychiatry, 40, 435-438.
- Bibring, E. (1953). The mechanism of depression. In P. Greenacre. (Ed.), Affective disorders. New York: International Universities Press.
- Buchwald, A. (1977). Learning theory and behavior therapy. In W.K Estes (Ed.), Handbook of learning and cognitive processes (Vol. 3). Hillsdale, N.J.:Erlbaum.
- Buchwald, A., Coyne, J., & Cole, C. (1978). A critical evaluation of the learned helplessness model of depression. Journal of Abnormal Psychology, 87, 180-193.
- Butters, N., & Cermak, L. (1980). Alcoholic Korsakoff's syndrome: An information processing approach to amnesia. New York: Academic Press.
- Cadore, R., O'Gorman, T., Troughton, E., & Heywood, E. (1985). Alcoholism and antisocial personality. Archives of General Psychiatry, 42, 161-167.
- Clark, D., & Teasdale, J. (1982). Diurnal variation in clinical depression and accessibility of memories of positive and negative experiences. Journal of Abnormal Psychology, 91, 87-95.
- Cadore, R., Troughton, E., & Widmer, R. (1984). Clinical differences between antisocial and primary alcoholics. Comprehensive Psychiatry, 25, 1-8.
- Cohen, R., Weingartner, H., Smallberg, S., Pickar, D., & Murphy, D. (1982). Effort and cognition in depression.

Archives of General Psychiatry, 39, 593-597.

- Cowan, J. (1983). Testing the escape hypothesis: Alcohol helps users to forget their feelings. Journal of Nervous and Mental Disease, 171, 40-47.
- Coyne, J., & Gotlib, I. (1983). The role of cognition in depression: A critical appraisal. Psychological Bulletin, 94, 87-95.
- Csikszentmihalyi, M., & Larson, R. (1984). Being adolescent: Conflict and growth in the teenage years. New York: Basic Books.
- Csikszentmihalyi, M. & Larson, R. (1985). The experience sampling method: Towards a systematic phenomenology. Unpublished Manuscript, University of Chicago, Chicago, Illinois.
- Curt, L. (1982). The effect of depressive affect on judgments of frequency of occurrences. Unpublished Masters Thesis, Loyola University of Chicago, Chicago, Illinois.
- Cyr, J., McKenna-Foley, J., & Peacock, E. (1979). Factor structure of the SCL-90: Is there one? Journal of Personality Assessment, 49, 571-577.
- Derogatis, L. (1977a). A confirmation of the dimensional structure of the SCL 90: A study in construct validation. Journal of Clinical Psychology, 33, 981-989.
- Derogatis, L. (1977b). SCL-90 administrations, scoring and procedures manual-I. Johns-Hopkins, Baltimore: Md.
- Derogatis, L., Rickels, K., & Rock, A. (1976). The SCL-90 and the MMPI: A step in the validation of a new self-report scale. British Journal of Psychiatry, 128, 280-289.
- Diener, E., & Larsen, R. (1984). Temporal stability and cross-situational consistency of affective, behavioral, and cognitive responses. Journal of Personality and Social Psychology, 47, 871-883.
- Diener, E., Larson, R., & Emmons, R. (1984). Person X situation interactions: Choices of situations and congruence of response models. Journal of Personality and Social Psychology, 47, 580-592.
- Dinning, W. & Evans, R. (1977). Discriminant and convergent validity of the SCL-90 in psychiatric inpa-

- tients. Journal of Personality Assessment, 41, 304-310.
- Dobson, D. & Dobson, K. (1981). Problem solving strategies in depressed and non-depressed college students. Cognitive Therapy and Research, 5, 237-249.
- Dobson, K. & Shaw, B. (1987). Specificity and stability of self-referent encoding in clinical depression. Journal of Abnormal Psychology, 97, 34-40.
- Donat, D. (1986). Semantic and visual memory after alcohol abuse. Journal of Clinical Psychology, 42, 537, 539.
- Ellis, H. (1985). On the importance of mood intensity and encoding demands in memory: Commentary on Hasher, Rose, Zacks, Sanft, and Doren. Journal of Experimental Psychology: General, 114, 393-395.
- Ellis, H., Thomas, R., McFarland, A., & Lane, W. (1985). Emotional mood states and retrieval in episodic memory. Journal of Experimental Psychology: Learning, Memory and Cognition, 11, 363, 370.
- Evenson, R., Holland, R., Metha, S., & Yasif, F. (1980). Factor structure of the SCL 90 in a psychiatric population. Journal of Consulting and Clinical Psychology, 46, 695-699.
- Filstead, W., Reich, W., Parrella, D., & Rossi, J. (1985). Using electronic pagers to monitor the process of recovery in alcoholics and drug abusers. Paper presented at the 34th International Congress on Alcohol, Drug Abuse and Tobacco, Calgary.
- Fitch, G. (1970). Effects of self-esteem, perceived performance, and choice on causal attributions. Journal of Personality and Social Psychology 4, 47, 311-315.
- Flexner, J. & Bower, G. (1975). Further evidence regarding instructional effects on frequency judgments. Bulletin of the Psychonomic Society, 6, 321-324.
- Franzoi, S., & Brewer, L. (1984). The experience of self-awareness and its relation to level of self-consciousness: An experiential sampling study. Journal of Research in Personality, 48, 768-780.
- Freed, E. (1978). Alcohol and mood: An updated review. International Journal of the Addictions, 13, 173-200.
- Freeman, M., Csikszentmihalyi, M., & Larson, R. (in press)

Adolescence and its recollection: Towards a interpretive model of development. Merrill-Palmer Quarterly of Behavior and Development.

- Gass, C. & Russell, E. (1986). Differential impact of brain damage and depression on memory test performance. Journal of Consulting and Clinical Psychology, 54, 261-263.
- Gianinno, S., Graef, R., & Csikszentmihalyi, M. (1979). Well-being and the perceived balance between opportunities and capabilities. Paper presented at the 87th American Psychological Association Convention, New York City.
- Gibson, S. & Becker, J. (1973). Alcoholism and depression: The factor structure of alcoholics' responses to depression inventories. Quarterly Journal of Studies on Alcohol, 85, 400-408.
- Goldman, M. (1985). Cognitive impairment in chronic alcoholics: Some cause for optimism. American Psychologist, 38, 1045-1054.
- Greene, R. (1986). Effects of intentionality and strategy on memory for frequency. Journal of Experimental Psychology: Learning, Memory and Cognition, 12, 489-495.
- Hamilton, M. (1960). A rating scale for depression. Journal of Neurology, Neurosurgery, and Psychiatry, 23, 56-62.
- Hamilton, J., Haier, R., & Buchsbaum, M. (1984). Intrinsic enjoyment and boredom coping scales: Validation with personality, evoked potential and attention measures. Personality and Individual Differences, 5, 183-193.
- Hasher, L., Rose, K., Zacks, R., Sanft, H., & Doren, B. (1985). Mood, recall, and selectivity effects in normal college students. Journal of Experimental Psychology: General, 114, 104-118.
- Hasher, L., & Zacks, R. (1979). Automatic and effortful processes in memory. Journal of Experimental Psychology: General, 109, 356-388.
- Hasher, L., & Zacks, R. (1984). Automatic processing of high priority information: The case of frequency of occurrence. American Psychologist, 57, 1372-1388.
- Hathaway, S., & McKinley, J. (1951). Minnesota Multiphasic Personality Inventory Manual, Rev. Edition, New York: Psychological Corp.
- Hays, W. (1973). Statistics for the social sciences. New

York: Holt, Rinehart and Winston, Inc.

- Hesserlbrock, M., Hesselbrock, V., Tennen, H., Meyer, R., & Workman, K. (1983). Methodological considerations in the assessment of depression in alcoholics. Journal of Consulting and Clinical Psychology, 51, 399-405.
- Hightower, M., & Anderson, R. (1986). Memory evaluation of alcoholics with Russell's revised Wechsler Memory Scale. Journal of Clinical Psychology, 42, 1000-1005.
- Hoffman, N., & Overall, P. (1978). Factor structure of the Symptom Checklist-90. Journal of Consulting and Clinical Psychology, 46, 1187-1191.
- Holcomb, W., Adams, N., & Ponder, H. (1983). Factor structure of the symptom checklist-90 with acute psychiatric inpatients. Journal of Consulting and Clinical Psychology, 51, 535-538.
- Hurlburt, R. (1979). Random sampling of cognitions and behavior. Journal of Research in Personality, 13, 103-111.
- Ickes, W., & Layden, M. (1978). Attributional styles. In J. Harvey (Ed.), New directions in attribution research (Vol. 2, pp. 119-152).
- Isen, A. (1984). Toward understanding the role of affect in cognition. In R.S. Wyer & T.K. Srull (Eds.), Handbook of social cognition, (pp 179-236). Hillsdale, NJ: Erlbaum.
- Isen, A. (1985). Asymmetry of happiness and sadness in effects on memory in normal college students: Comment on Hasher, Rose, Zacks, Sanft, and Doren. Journal of Experimental Psychology: General, 114, 388-391.
- Isen, A., Shalke, T., Clark, M., & Karp, L. (1978). Affect, Accessibility of material in memory, and behavior: A cognitive loop? Journal of Personality and Social Psychology, 36, 1-12.
- Jaffe, J. & Ciraulo, D. (1986). Alcoholism and depression. In: Psychopathology and addictive disorders. Roger Meyer (Ed.). Guilford Press, NY:NY.
- Johnson, M., Kim, J., & Risse, G. (1985). Do alcoholic Korsakoff's syndrome patients acquire affective reactions. Journal of Experimental Psychology: Learning, Memory and Cognition, 11, 22-36.

- Johnson, M. & Magaro, P. (1987). Effects of mood and severity on memory processes in depression and mania. Psychological Bulletin, 101, 28-40.
- Jones, M. (1968). Personality correlates and drinking patterns in men. Journal of Consulting and Clinical Psychology, 32, 2-12.
- Jones, M. (1971). Personality correlates and drinking patterns in women. Journal of Consulting and Clinical Psychology, 36, 61-69.
- Kahneman, D. (1973). Attention and effort. Englewood Cliffs, NJ:Prentice-Hall.
- Keeler, M., Taylor, C., & Miller, W. (1979). Are all recently detoxified alcoholics depressed? American Journal of Psychiatry, 136, 386-588.
- Khantzian, E. The Alcoholic patient: An overview and perspective. American Journal of Psychotherapy, 34, 4-17.
- Klein, D. & Seligman, M. (1976). Reversal of performance deficits and perceptual deficits in learned helplessness and depression. Journal of Abnormal Psychology, 86, 700-707.
- Klinger, E. (1978). Dimensions of thought and imagery in normal waking states. Journal of States of Consciousness, 4, 97-113.
- Kubey, R. (1984). Leisure, television, and subjective experience. Unpublished doctoral dissertation, The University of Chicago.
- Kuiper, N. (1978). Depression and causal attributions for success and failure. Journal of Personality and Social Psychology, 36, 236-246.
- Kuiper, N., Mac Donald, M., & Derry, P. (1983). Parameters of a depressive self-schema. In J. Suls & A. Greenwald (Eds.), Psychological perspectives on the self. (Vol. 2, pp. 191-217). Hillsdale, NJ: Erlbaum.
- Laird, J., Wagener, J., Halal, M., & Szegda, M. (1982). Remembering what you feel: Effects of emotion on memory. Journal of Personality and Social Psychology, 42, 646-657.
- Larson, R. (1979). The significance of time alone in adolescents' lives. Unpublished doctoral dissertation,

The University of Chicago.

- Larson, R., & Csikszentmihalyi, M. (1980). The experience sampling method. In H. Reis (Ed.), New Directions for Naturalistic Methods in the Behavioral Sciences. San Francisco: Jossey Bass.
- Larson, R., & Johnson, C. (1985). Bulimia: Disturbed patterns of solitude. Addictive Behaviors, 10, 281-290.
- Lazarus, R. (1979). Positive denial: The case of not facing reality. Psychology Today, 13, 44-60.
- Lewinsohn, P. (1974). A behavioral approach to depression. In R.J. Friedman & M.M. Katz (Eds.), The psychology of depression: Contemporary theory and research. Washington, D.C.: Winston/Wiley.
- Lewinsohn, P., Steinmetz, J. Larson, D., & Franklin, J. (1981). Depression-related cognitions. Antecedent or consequence? Journal of Abnormal Psychology, 90, 213-219.
- Loranger, A. & Tulis, E. (1985). Family history of alcoholism in borderline personality disorder. Archives of General Psychiatry, 42, 153-157.
- Maier, S. & Seligman, M. (1976). Learned helplessness: Theory and evidence. Journal of Experimental Psychology: General, 105, 3-46.
- Markowitsch, H., Kessler, J., & Bast-Kessler, C., & Riess, R. (1984). Different emotional tones significantly affect recognition performance in patients with Korsakoff psychosis. International Journal of Neuroscience, 25, 145-159.
- Markowitsch, H., Kessler, J. & Denzler, P. (1986). Recognition memory and psychophysiological responses to stimuli with neutral or emotional content: A study of Korsakoff patients and recently detoxified and longterm abstinent alcoholics. International Journal of Neuroscience, 29, 1-35.
- McAdams, D., & Constantian, C. (1983). Intimacy and affiliation motives in daily living: An experience sampling analysis. Journal of Personality and Social Psychology, 45 851-861.
- McMahon, R. & Davidson, R. (in press). Transient versus enduring depression among alcoholics in inpatient treatment. Journal of Psychopathology and Behavioral Assessment.

- McMahon, R. & Davidson, R. (1986). An examination of depressed vs. nondepressed alcoholics in inpatient treatment. Journal of Clinical Psychology, 42, 177-184.
- McNair, D., Lorr, M., Droppleman, L. (1971). Profile of mood states manual San Diego, CA: Educational and Industrial Testing Service.
- Merikangas, K., Weissman, M., Prusoff, B., Pauls, D., & Leckman, J. (1985). Depressives with secondary alcoholism: Psychiatric disorders in offspring. Journal of Studies in Alcoholism, 46, 199-204.
- Midanik, L. (1983). Alcohol problems and depressive symptoms in a national survey. In B. Stimmel (Ed.), Psychosocial constructs of alcoholism and substance abuse. (pp. 6-28). New York: Haworth Press.
- Miller, D., Rosellini, R., & Seligman, M. (1977). Learned helplessness in depression. In J. D. Maser & M. Seligman (Eds.), Psychopathology: Experimental models. San Francisco: Freedman.
- Miller, W., & Seligman, M. (1973). Depression and the perception of reinforcement. Journal of Abnormal Psychology, 82, 62-73.
- Mischel, W., & Ebbesen, E. (1976). Determinants of selective memory about the self. Journal of Consulting and Clinical Psychology, 44, 92-103.
- Mueller, C. & Klajner, F. (1984). The effect of alcohol on memory for feelings: Does it really help users to forget? The Journal of Nervous and Mental Disease, 172, 225-227.
- Nasby, W., Yando, R. (1982). Selective encoding and retrieval of two cognitive consequences of children's mood states. Journal of Personality and Social Psychology, 43, 1244-1253.
- Natale, M. & Hantas, M. (1982). Effect of temporary mood states on selective memory about the self. Journal of Personality and Social Psychology, 42, 927-934.
- Naveh-Benjamin, M., & Jonides, J. (1986). On the automaticity of frequency encoding: Effects of competing task load, encoding strategy, and intention. Journal of Experimental Psychology: Learning, Memory and Cognition, 12, 378-386.
- Nelson, R. & Craighead, W. (1977). Selective recall of

- positive and negative feedback, self-control behaviors and failure. Journal of Abnormal Psychology, 86, 379-388.
- Nerviano, V. (1981). Personality patterns of alcoholics revisited: deliniation against the MMPI and clinical implication. Journal of the Addictions, 16, 723-729.
- Nerviano, B., McCarthy, D., & McCarthy, S. (1980). MMPI profile patterns of men alcoholic in two contrasting settings. Journal of Studies on Alcohol, 41, 1143-1152.
- O'Leary, M., Donovan, D. Cysewski, B., & Chaney, E. (1977). Perceived locus of control, experienced control, and depression: A trait description of the learned helplessness model of depression. Journal of Clinical Psychology, 33, 164-168.
- O'Leary, M., Donovan, D., Kruger, D., & Cysewski, B. (1978). Depression and perception of reinforcement: Lack of differences in expectancy change among alcoholics. Journal of Abnormal Psychology, 87, 110-112.
- O'Sullivan, K., Daley, M., Carroll, B., Clare, A., & Cooney, J. (1979). Alcoholism and affective disorder among patients in a Dublin hospital. Journal of Studies in Alcohol, 40, 1014-1022.
- Parker, E., Birnbaum, I., Weingartner, H. (1981). Retrograde enhancement of human memory with alcohol. Psychopharmacology (Berlin), 69, 219-222.
- Parson, O., & Farr, S. (1981). The neuropsychology of alcohol and drug use. In S. Filskov & T. Boll (Eds.). Handbook of Clinical Neuropsychology. Wiley, NY:NY.
- Price, K., Tryon, W., & Raps, C. (1978). Learned helplessness and depression in a clinical population: A test of two behavioral hypotheses. Journal of Abnormal Psychology, 87, 113-121.
- Query, W. & Megran, J. (1984). Influence of depression and alcoholism on learning, recall and recognition. Journal of Clinical Psychology, 40, 1094-1100.
- Raps, C., Peterson, C., Reinhard, K., Abramson, L., & Seligman, M. (1982). Attributional style among depressed patients. Journal of Abnormal Psychology, 91, 102-108.
- Raskin, A., Schulterbrandt, J., Reatig, N., & McKeon, J. (1969). Replication of factors of psychopathology in interview, ward behavior and self-report ratings of

- hospitalized depressives. Journal of Nervous Disorders and Mental Disease, 148, 87-98.
- Reber, A. (1967). Implicit learning of artificial grammars. Journal of Verbal Learning and Verbal Behavior, 6, 855-863.
- Reber, A. (1969). Transfer of syntactic structure in synthetic languages. Journal of Experimental Psychology, 81, 115-119.
- Reber, A. (1976). Implicit learning of synthetic languages: The role of instructional set. Journal of Experimental Psychology: Human Learning and Memory, 2, 88-94.
- Rholes, W., Riskind, J., & Lane, J. (1987). Emotional states and memory biases: Effects of cognitive priming and mood. Journal of Personality and Social Psychology, 52, 91-99.
- Richards, H. (1986). Memory for rate of mood occurrences in recovering alcoholics. Unpublished master's thesis, Loyola University of Chicago, Chicago, IL.
- Riskind, H., Rholes, W., & Eggers, J. (1982). The Velten mood induction procedure: Effects on mood and memory. Journal of Consulting and Clinical Psychology, 50, 146-147.
- Robinson, R. (1985). The daily experience of giftedness in adolescence: Sex differences and achievement. Paper presented at the Biannual meeting of the Society for Research on Child Development, Toronto.
- Rogers A., Kuiper, M., & Kirker, W. (1977). Self-reference and the encoding of personal information. Journal of Personality and Social Psychology, 35, 677-688.
- Roundsaville, B., Weissman, M., Rosenberger, P., Wilber, C., & Kleber, H. (1979). Detecting depressive disorders in drug abusers: A comparison of screening instruments. American Journal of Affective Disorders, 1, 255-267.
- Ryan E. & Butters, N. (1983). Cognitive deficits in alcoholics. In B. Kissin & H. Begleiter (Eds.), The pathogenesis of alcoholism. New York: Plenum Press.
- Savin-Williams, R., & Demo, D. (1983). Situational and transsituational determinants of adolescents' self-feelings. Journal of Personality and Social Psychology, 44, 824-833.
- Schare, M., Lisman, S., & Spear, N. (1984). The effects

- of mood variation on state-dependent retention. Cognitive Therapy and Research, 8, 387-408.
- Schuckit, M. (1979). Alcoholism and affective disorder: Diagnostic confusion. In D.W. Goodwin & C.K. Erikson (Eds.), Psychosocial constructs of alcoholism and substance abuse (pp. 9-28). New York: Spectrum.
- Schuckit, T. (1983). Alcoholics with secondary depression. American Journal of Psychology. 140, 711-714.
- Schwartz, B. (1981a). Does helplessness cause depression, or do only depressed people become helpless? Comment on Alloy and Abramson. Journal of Experimental Psychology: General, 110, 429-435.
- Schwartz, B. (1981b). Helplessness, illusions, and depression: Final comment. Journal of Experimental Psychology: General, 110, 448-449.
- Seligman, M. (1975a). Depression and learned helplessness. In R.J. Friedman & M. M. Katz (Eds.), The psychology of depression; Contemporary theory and research. New York: Wiley.
- Seligman, M. (1975b). Helplessness: On depression, development, and death. San Francisco: Freeman.
- Seligman, M. (1978). Comment and integration. Journal of Abnormal Psychology, 87, 165-179.
- Seligman, M., Abramson, L., Semmel, A., & von Baeyer, C. (1979). Depressive attributional style. Journal of Abnormal Psychology, 88, 242-247.
- Shaw, J., Donley, P., Morgan, D., & Robinson, J. (1975). Treatment of depression in alcoholics. American Journal of Psychology. 132, 641-644.
- Spitzer, R., Endicott, J., & Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. Archives of General Psychiatry, 35, 773-382.
- Sussman, S., Rychtarik, R., Mlueser, K., Glynn, S., & Prue, D. (1986). Ecological relevance of memory tests and predictions of relapse in alcoholics. Journal of Studies in Alcohol, 47, 305-310.
- Tanaka-Matsumi, J., & Kameoka, V. (1986). Reliabilities and concurrent validities of popular self-report measures of depression, anxiety, and social desirability. Journal of Consulting and Clinical Psychology, 54, 328-333.

- Teasdale, J. (1986). Negative thinking in depression: Cause, effect or reciprocal relationship? In L. Joyce-Money, F. Kiwem & O. Higson (Eds.), Theoretical issues in cognitive-behavioral therapy. New York: Plenum Press.
- Teasdale, J., & Russel, M. (1983). Differential effects of induced mood on the recall of positive, negative and neutral words. British Journal of Clinical Psychology, 22, 163-72.
- Teasdale, J., Taylor, R., & Fogarty, S. (1980). Effects of induced elation-depression on the accessibility of memories of happy and unhappy experiences. Behavioral Research and Therapy, 18, 339-346.
- Tennen, H. & Herzberger, S. (1987). Depression, self-esteem, and the absence of self-protective attributional biases. Journal of Personality and Social Psychology, 52, 72-80.
- Tennen, H., Herzberger, S., & Nelson, H. (1986). Depressive attributional style: The role of self-esteem. Manuscript submitted for publication.
- Tversky, A. & Kahneman, D. (1973). Availability: A heuristic for judging frequency and probability. Cognitive Psychology, 5, 285-232.
- von Knorring, A., Cloninger, C., Bohman, M., & Sigvardson, S. (1983). An adoption study of depressive disorders and substance abuse. Archives of General Psychiatry, 40, 943-950.
- Warrington, E. (1986). Memory of facts and memory for events. British Journal of Clinical Psychology, 25, 1-12.
- Watson, D., & Clark, L. (1984). Negative Affectivity: The disposition to experience aversive emotional states. Psychological Bulletin, 96, 465-490.
- Weingartener, H., Cohen, R., Murphy, P., Martello, J., & Gerdt, C. (1981). Cognitive processes in depression. Archives of General Psychiatry, 38, 42-47.
- Weingold, H., Bell, A., & Coxe, R. (1968). Depression as symptom of alcoholism: Search for a phenomenon. Journal of Psychiatric Research, 4, 37-50.
- Weissman, M. & Myers, J. (1980). Clinical depression in alcoholism. American Journal of Psychiatry. 1, 37, 372-373.
- Well, A. (1985). Variations in self-esteem in the daily

life of mothers. Unpublished doctoral dissertation, The University of Chicago.

- Werner, A., & Rehm, I. (1975). Depressive affect. A test of behavioral hypotheses. Journal of Abnormal Psychology, 84, 221-227.
- Wiggins, J. (1966). Substantive dimensions of self-report in the MMPI item pool. Psychological Monographs, 80, 1-22.
- Wiggins, J. (1969). Content dimensions in the MMPI. In J. Butcher (Ed.), MMPI: Research Developments and Clinical Applications, New York: McGraw-Hill.
- Wiggins, J., Goldberg, L., & Applebaum, M. (1971). MMPI content scales, interpretive norms and correlations with other scales. Consulting and Clinical Psychology, 37, 403-410.
- Wikler, A. (1973). Dynamics of drug dependence: Implications of a conditioning theory for research and treatment. Archives of General Psychiatry, 28, 611-616.
- Willenbring, M. (1986). Measurement of depression in alcoholics. Journal of Studies in Alcohol, 47, 367-372.
- Winokur, G., Rimmer, J., & Reich, T. (1971). Alcoholism: IV. Is there more than one type of alcoholism? British Journal of Psychiatry, 118, 525-531.
- Woodruff, R., Guze, S., Clayton, J., & Carr, D. (1973). Alcoholism and depression. Archives of General Psychiatry, 28, 97-100.
- Yesavage, J. Brink, T., Rose, T., Lum, O., Huang, V., Adey, M., & Leirer, V. (1983). Development and validation of a geriatric depression screening scale: A preliminary report. Journal of Psychiatric Research, 17, 37-49.
- Zechmeister, E., & Nyberg, S. (1982). Human memory: An introduction to research and theory. Monterey, CA: Brooks Cole Publishing Co.
- Zuckerman, M. (1979). Attribution of success and failure revisited, or: The motivational bias is alive and well in attribution theory. Journal of Personality, 47, 245-287.
- Zuckerman, M., & Lubin, B. (1965). Manual for the Multiple Affect Adjective Checklist. San Diego: Educational and Industrial Testing.

Zung, W. (1965). A self-rating depression scale. Archives of General Psychiatry, 12, 63-70.

Zuroff, D. & Mongrain, M. (1987). Dependency and self-criticism: Vulnerability factors for depressive affective states. Journal of Abnormal Psychology, 96, 14-22.

APPENDIX A

MEMORY TASK INSTRUCTIONS

NOTE: Use this answer sheet and a blank booklet to get the person oriented to the task.

KEY POINTS:

1. Want to get the subject to think about how he/she filled out the booklet, not how they felt then or now about the items. Many subjects may use their recollection of feelings to "jog" their memories as to how they filled out the book.
2. These responses are in terms of percentages of 100%.
3. After you explain the task, see if they can tell you what they are going to be doing.
4. "General Explanation": We are trying to understand how people remember and what ways people may or may not use to remember things.

What we'd like you to do is help us in the memory test. There are no right or wrong answers. All we will ask you to do is remember some aspects of what you have been doing in regards to the patient workbook.

We are going to concentrate on trying to find out how you filled out ("marked") the book; not how you were feeling. This memory task is only related to how you filled out the questions.

5. Under the heading of General Questions:

The first two (#1 and #2) refer to a special dimension of memory. All these questions are getting at is how often the mark was to the right or left of the page.

Question #2 and #3 are related to the positive and/or negative dimension of the item. This is the emotional/feeling aspect of the task.

Help the subjects understand these two related, but by very distinct tasks. Repeat it or have them repeat it before they do the task. You can refer to the mood rating scale on the page itself or to the unanswered page in the booklet.

6. When the subject actually gets to the mood items that are scaled like the booklet, make it clear that the (brackets) over the various responses are calling for a summary of those marks.

The total response should equal 100%. They can answer the questions any way they choose (e.g., figuring out % positive, then neutral, and then negative or whatever sequence they choose).

7. The final four questions ask for two ratings that cut across these dimensions. Again, these are summaries of their marks and the total has to equal 100%.

Refer to the blank booklet to orient subject, if necessary.

8. Some subjects, when given the instructions, will feel it is impossible to do. Encourage them, provide extra time, suggest that whatever they can do will be helpful.

If subject persists, then excuse him/her from the task.

SCHEDULE OF SUBJECTS

1. Presently Active Subjects

Gp I	Book 6	Overall Assessment
Gp II		Overall Assessment

2. New Subjects as of 5/21/84

Gp I	Book 1	Book 6	Overall Assessment
Gp II	Book 1	X	Overall Assessment

3. Community Sample

Book 1

These forms will be located in a folder in Lil's desk (marked "Memory Study") and will be in the appropriate folders when subjects return.

A red dot will remind you that the task needs to be done on a given subject.

WJF/gj

APPENDIX B

Table B-1

Mean Frequency by Item and by Group

Item	Group				P Level	
	Positives	Nonalcoholic		Alcoholic		
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Alert		69.7	23.0	79.4	13.7	
Happy		64.3	20.1	63.8	20.2	
Strong		41.0	30.8	55.2	23.5	
Active		49.6	27.3	59.3	18.6	
Proud		33.4	28.2	49.4	26.9	
Cheerful		51.1	23.6	57.2	18.9	
Friendly		58.6	24.0	58.4	18.6	
Sociable		50.0	22.9	52.1	21.9	
Clear		61.5	33.3	63.6	21.8	
Relaxed		58.5	27.8	45.8	25.6	
Neithers	Nonalcoholic		Alcoholic		P Level	
Alert/Drowsy	4.5	5.2	3.0	4.6		
Happy/Sad	24.8	18.3	18.1	15.5		
Irritable/Cheerful	27.4	21.5	18.8	12.9		
Strong/Weak	43.1	32.4	25.8	23.4	*	
Angry/Friendly	30.8	23.4	20.9	17.5		
Active/Passive	22.5	27.7	13.4	13.7		
Lonely/Sociable	32.1	25.1	17.9	18.1	*	
Proud/Ashamed	62.7	31.4	37.1	27.5	**	
Confused/Clear	26.9	31.5	14.9	13.9		
Tense/Relaxed	19.1	19.9	12.8	19.0		

Table B-1 -Continued.

Mean Frequency by Test Item and by Group

Item	Group				P Level	
	Negatives	Nonalcoholic		Alcoholic		
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Angry		10.6	7.4	20.7	14.8	*
Irritable		21.4	10.1	23.9	15.5	
Lonely		17.9	20.3	20.0	20.6	
Confused		11.6	10.8	21.6	15.9	*
Tense		22.3	17.1	41.4	21.7	**
Drowsy		25.8	20.2	17.6	14.1	
Sad		11.0	9.2	18.1	16.9	
Weak		15.9	15.5	19.0	13.4	
Passive		27.8	17.3	27.3	18.5	
Ashamed		3.9	5.7	13.4	12.7	*
Non-Mood Items	Nonalcoholic		Alcoholic		P Level	
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>		
Preoccu. Eating	5.7	6.5	7.4	10.3		
Preoccu. Using	2.6	5.3	5.1	9.0		
Confident-Resist.	94.5	8.4	88.3	19.5		
Shared	14.0	14.7	3.3	28.3	**	

NOTE: n = 22 for all group means.

* $p < .05$.

** $p < .01$.

Table B-2

Mean Estimates by Item and by Group

Item	Group				<u>P Level</u>	
	Positives	Nonalcoholic		Alcoholic		
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Alert		69.2	24.0	69.7	25.6	
Happy		67.6	31.2	62.6	29.8	
Strong		48.2	36.2	54.3	31.5	
Active		58.5	32.4	62.1	27.2	
Proud		33.0	35.0	52.6	36.2	
Cheerful		62.6	29.9	59.1	29.7	
Friendly		70.6	29.3	61.5	28.7	
Sociable		64.1	33.09	53.0	29.4	
Clear		71.6	31.3	63.9	30.3	
Relaxed		58.1	33.4	45.8	33.8	
Neithers		Nonalcoholic		Alcoholic	<u>P Level</u>	
Alert/Drowsy		7.1	9.8	17.7	23.1	
Happy/Sad		22.6	30.8	21.2	28.7	
Irritable/Cheerful		22.5	28.0	18.7	22.7	
Strong/Weak		38.5	37.4	26.7	33.5	
Angry/Friendly		21.0	28.7	17.1	22.9	
Active/Passive		22.8	33.7	17.3	23.8	
Lonely/Sociable		22.0	29.8	18.9	29.1	
Proud/Ashamed		61.8	38.2	34.5	37.4	*
Confused/Clear		24.2	29.5	15.1	18.7	
Tense/Relaxed		20.7	19.8	41.3	32.0	

Table B-2 -Continued.

Mean Estimates by Item and by Group

Item	Group				<u>P Level</u>	
	Negatives	Nonalcoholic		Alcoholic		
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Angry		8.5	7.9	23.5	25.4	*
Irritable		14.9	15.5	22.2	25.2	
Lonely		13.9	18.4	27.6	25.0	*
Confused		8.4	11.5	21.0	22.4	*
Tense		20.7	19.8	41.3	32.0	*
Drowsy		23.5	19.6	16.3	19.2	
Sad		9.6	10.3	16.1	18.3	
Weak		13.4	14.2	19.0	16.3	
Passive		18.7	16.9	20.6	19.1	
Ashamed		2.5	4.7	12.8	18.5	*

Non-Mood Items	Group				<u>P Level</u>	
		Nonalcoholic		Alcoholic		
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Preoccu. Eating		10.2	16.2	20.2	30.0	
Preoccu. Using		2.5	4.5	16.8	27.5	*
Confident-Resist.		62.1	47.8	61.0	41.8	
Shared		26.8	26.4	43.1	31.9	

NOTE: n = 22 for all group means.

* $p < .05$.

Table B-3

Signed Difference Scores by Item and by Group

Item	Group				<u>P Level</u>
	Positives	Nonalcoholic		Alcoholic	
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Alert		0.4	16.5	9.7	21.0
Happy		3.3	22.7	1.2	18.6
Strong		0.72	24.7	1.2	18.6
Active		8.9	19.6	2.8	19.9
Proud		0.3	20.7	3.2	17.7
Cheerful		11.6	18.6	1.9	18.6
Friendly		12.1	21.8	3.0	18.2
Sociable		4.2	23.5	0.9	20.8
Clear		0.1	22.1	0.3	17.1
Relaxed		0.4	19.6	0.0	17.7
Neithers	Nonalcoholic		Alcoholic		<u>P Level</u>
Alert/Drowsy	2.6	9.4	14.6	21.8	
Happy/Sad	2.2	23.7	3.2	19.5	
Irritable/Cheerful	4.9	17.3	0.1	17.6	
Strong/Weak	4.6	23.6	0.9	17.5	
Angry/Friendly	9.8	19.8	3.7	14.8	
Active/Passive	0.3	14.2	3.8	16.3	
Lonely/Sociable	10.1	19.6	1.0	23.7	
Proud/Ashamed	0.9	26.8	2.6	20.8	
Confused/Clear	2.7	22.0	0.3	12.7	
Tense/Relaxed	2.1	24.7	0.1	13.6	

Table B-3 -Continued.

Signed Difference Scores by Item and by Group

Item	Group				
	Negatives	Nonalcoholic		Alcoholic	<u>P</u> Level
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Angry		2.1	6.7	2.7	20.0
Irritable		6.6	12.9	1.7	20.1
Lonely		4.0	13.6	2.3	14.6
Confused		3.2	11.8	0.6	14.1
Tense		1.7	16.5	0.1	21.7
Drowsy		2.4	15.0	1.3	10.4
Sad		1.3	10.5	2.0	9.1
Weak		2.6	9.8	0.0	14.2
Passive		9.1	12.3	6.6	16.4
Ashamed		1.4	4.0	.06	12.4

Non-Mood Items	Nonalcoholic		Alcoholic		<u>P</u> Level
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Preoccu. Eating	4.6	15.4	12.9	25.4	
Preoccu. Using	0.1	5.8	11.7	22.6	*
Confident-Resist.	32.4	51.6	27.2	40.0	
Shared	12.8	25.9	9.8	37.2	

NOTE: n = 22 for all group means.

* $p < .05$.

Table B-4

Absolute Difference Scores by Item and by Group

Item	Group				
	Positives	Nonalcoholic		Alcoholic	P Level
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Alert		13.0	9.7	16.4	16.1
Happy		18.0	13.5	14.9	10.7
Strong		17.8	18.31	15.2	10.1
Active		14.7	15.5	16.4	11.0
Proud		15.3	13.5	13.8	11.2
Cheerful		16.9	13.6	14.0	12.0
Friendly		19.9	14.6	13.8	11.8
Sociable		20.8	17.5	16.1	12.7
Clear		15.7	18.3	13.5	10.1
Relaxed		15.2	11.8	14.4	9.8
Neithers	Nonalcoholic		Alcoholic		P Level
Alert/Drowsy	6.0	7.6	15.4	21.2	
Happy/Sad	18.8	13.9	14.8	12.8	
Irritable/Cheerful	14.6	9.9	13.4	11.0	
Strong/Weak	17.6	16.0	12.3	12.2	
Angry/Friendly	17.7	12.8	10.1	11.3	*
Active/Passive	10.6	9.2	10.6	12.8	
Lonely/Sociable	16.7	14.1	16.4	16.7	
Proud/Ashamed	18.2	19.2	16.8	12.0	
Confused/Clear	15.5	15.5	7.9	9.8	
Tense/Relaxed	17.3	17.3	8.3	10.7	

Table B-4 -Continued.

Absolute Difference Scores by Item and by Group

Item	Group				<u>P Level</u>	
	Negatives	Nonalcoholic		Alcoholic		
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Angry		5.5	4.2	13.0	15.1	*
Irritable		11.0	9.2	13.1	15.0	
Lonely		7.6	11.9	12.3	7.6	
Confused		8.0	9.1	9.6	10.1	
Tense		12.0	11.1	16.9	13.0	
Drowsy		11.0	10.2	8.4	6.1	
Sad		7.8	6.9	7.4	5.5	
Weak		7.5	6.7	10.9	8.7	
Passive		10.6	11.0	15.1	8.7	
Ashamed		2.3	3.5	9.4	7.7	*

Non-Mood Items	Group				<u>P Level</u>	
		Nonalcoholic		Alcoholic		
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
Preoccu. Eating		7.9	13.9	14.3	24.5	
Preoccu. Using		2.9	5.0	12.5	22.2	
Confident-Resist.		40.2	45.5	28.2	39.4	
Shared		20.6	19.9	27.0	26.8	

NOTE: n = 22 for all group means.

* $p < .05$.

APPROVAL SHEET

The dissertation submitted by Henry Jay Richards has been read and approved by the following committee:

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The final copies have been examined by the director of the dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the dissertation is now given final approval by the Committee with reference to content and form.

The dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

November 16, 1987
Date

Patricia A. Rupert
Director's Signature