A Comparison of the Physiological Responses of Brain-Damaged Alcoholics, Nonbrain-Damaged Alcoholics, and Social Drinkers to the Smell of Alcohol.

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A comparison of the physiological responses of brain-damaged alcoholics, nonbrain-damaged alcoholics, and social drinkers to the smell of alcohol

Klug, Fredric D., Ph.D.
The Louisiana State University and Agricultural and Mechanical Col., 1992

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OF BRAIN-DAMAGED ALCOHOLICS, NONBRAIN-DAMAGED ALCOHOLICS,
AND SOCIAL DRINKERS TO THE SMELL OF
ALCOHOL

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by
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Abstract

This study reviews the relapse prevention and neuropsychological literature relevant to alcoholism. The argument was made that impaired neuropsychological functioning may be an important determinant of relapse. Specifically, neuropsychological impairment was expected to affect the manner in which individuals respond to conditioned alcohol cues so as to increase their attention to the cues and increase their desire to drink, a process which might adversely affect recovery. In order to study the impact of conditioned alcohol cues, subjective, objective, and psychophysiological responses of brain-damaged alcoholics, nonbrain-damaged alcoholics, and nonbrain-damaged social drinkers were compared on their responses to alcohol and to water. The results revealed that the presence of neuropsychological deficits was differentially associated with how alcoholics responded to the two types of stimuli. It was suggested that alcoholics with neuropsychological deficits exhibited some sort of selective attentional process for alcohol that differentially reduced the attention paid to the competing stimuli. A model was proposed to illustrate this effect. It was concluded that the study has important research and clinical implications. Most importantly, alcoholics should be assessed for brain damage and any attentional deficits be rehabilitated in much the same manner as brain injured patients.
Introduction

The present project proposes to compare how quickly alcoholics with and without neuropsychological deficits habituate to naturally occurring drinking cues, such as the smell of alcohol. For the purposes of this study, and by convention, the label neuropsychological deficits is used to imply the presence of brain-damage, even though there may not be anatomical confirmation of the presence of structural lesions to the brain. The basis of this proposal is derived from the literature on relapse prevention and the neuropsychological aspects of alcoholism.

Relapse Prevention Literature

In the field of alcoholism there are three major models to explain relapse, and craving is an important factor in each model (Ludwig & Wikler, 1974; Marlatt & Gordon, 1985; Nace, 1987). Craving has been a controversial term beset by problems of measurement and definition, but there is a general consensus that it is a strong desire to experience the effects resulting from engaging in some behavioral act, such as drinking. Furthermore, a high percentage (up to 95%) of alcoholics report experiencing craving, especially in the initial stages of abstinence. In fact, the intensity of craving is negatively correlated with length of sobriety, but intensity of craving is not related to length of alcoholism (Isbell, 1955; Mathew, Claghorn, & Largen, 1979). Thus, craving exerts its greatest effect in the early stages
of recovery and affects relatively recently afflicted and chronic alcoholics alike. Among active alcoholics, cravers have been shown to have episodes of drinking on a daily basis more often than non-craving alcoholics have; they have reported more physical discomfort after a night of drinking, as well as more frequent episodes of morning drinking after a night of drinking. Based on the Alcohol Use Inventory (AUI; Horn, Wanberg, & Foster, 1974; Wanberg & Horn, 1983; Wanberg, Horn, & Foster, 1977), cravers have reported more anxiety over their drinking, and they have characterized their drinking as more obsessive-compulsive than do non-cravers (Tarter & Sugerman, 1977). Studies that have investigated different aspects of craving have studied craving in terms of cognition, physiology, and behavior.

**Cognitive Aspects of Craving**

This discussion examines the elicitation of craving and the relationship of craving to field-dependence and to expectancies. Craving can be elicited by cues which have been associated with drinking experiences; thus, the cues may be viewed as classically conditioned stimuli (CS). For example, after explaining the concept of Pavlovian conditioning to a group of alcoholics (N = 150), Ludwig (1986) found that nearly 93 percent of them could identify at least one cue that triggered craving. When faced with one of these CSs, an alcoholic may experience craving which could lead to drinking (Mathew et al. 1979).
The concept of field-dependence/field-independence is derived from the theories on cognitive style which are considered to be "characteristic, self-consistent modes of functioning which individuals show in their perceptual and intellectual activities" (Witkin, Oltman, Raskin, & Karp, 1971). In studies of field-dependence, alcoholics have been shown to be more field-dependent than non-alcoholics (Bailey, Hustmyer, & Kristoffersen, 1961; Goldstein & Chotlos, 1965), and among alcoholics, cravers have been found to be more field-dependent than non-cravers as measured by the Rod and Frame Test (Witkin, Karp, & Goodenough, 1959). Thus, these findings may reflect a continuum in which field-dependency and craving are associated in an additive manner such that those individuals exhibiting high field-dependency and high craving are at the greatest risk for relapse. However, those studies that reported greater field-dependence among alcoholics versus non-alcoholics did not categorize the alcoholics as cravers or non-cravers; thus, the findings may simply reflect that alcoholics who reported more craving were included in the alcoholic groups, rather than a general tendency for all alcoholics to show field-dependence.

At least one author (Goldstein, 1987) has suggested that alcoholics may have antecedent neuropsychological deficits which predispose the individual to develop alcoholism, and field-dependence may be one of those
deficits. Along these lines, Berent (1981) has shown that field-dependent individuals perform more poorly on tasks such as verbal paired-associate learning, writing, and calculating. Furthermore, Culver, Cohen, Silverman, and Shmavonian (1964) have found that field-dependence is closely related to poor laterality orientation or the ability to identify the sidedness of body parts. Thus, field-dependent individuals perform, at least in these cases, in a manner consistent with that of brain-damaged individuals.

Although it remains debatable whether or not high field-dependence is a neuropsychological deficit, field-dependence has been shown to correlate with neuropsychological impairments in alcoholism (Miller & Saucedo, 1983). Furthermore, both of these problems have been associated with poor treatment outcome (e.g., Karp, Kissin, & Hustmyer, 1970; O'Leary, Donovan, Chaney, & Walker, 1979).

The relationship between cues that elicit craving (i.e., CS) and field-dependence can be appreciated by the finding that field-dependent individuals have difficulty solving problems that require the individual to separate an essential element of a problem from the context in which it is presented and then using it in a different context (Witkin, et al., 1971). Furthermore, they have difficulty keeping separate their perception of external stimuli and
the interoceptive stimuli associated with the external stimuli (Tarter & Sugerman, 1977). In other words, when a drinking cue is encountered, a field-dependent person would have difficulty separating the perception of the cue from its context and from the internal stimuli that it elicits, and he or she would be more likely to experience the stimulus complex as too compelling to respond in alternative, more adaptive, ways. Thus, new learning would be inhibited, and the field-dependent person would respond in the same manner that had been established by their past experience with the stimuli, that is by imbibing.

In another area of cognitive functioning, several studies have investigated the drinker's expectancies regarding the anticipated effects of drinking alcohol, especially the expectancy that drinking will result in positive outcomes. In general, it has been shown that the expectancy of receiving alcohol had a greater enhancing effect on the desire for alcohol (i.e., craving) than the effect from actually consuming alcohol. The expectancy effect has been studied using the balanced-placebo design (BPD) which manipulates two factors independently: the actual substance administered, and the information given to the subject regarding the nature of the substance given. Thus, the design can be conceptualized as a $2 \times 2$ matrix where the subject is administered the active substance (e.g., alcohol) or a placebo, and the subject is told that
he or she is being given the active substance or a placebo. The nature of the design permits the researcher to determine whether the subjects' behavioral changes are due to the pharmacological properties of the substance administered or to the subject's expectations about the substance he or she thinks is being administered.

The expectancy effect has been shown to be positively correlated with the severity of the individual's degree of dependence on alcohol (e.g., Engle & Williams, 1972; Laberg, 1986). In other words, the greater the individual's dependence on alcohol the more the individual experiences an expectancy effect which increases the person's desire for alcohol. There are also data showing that increased alcohol dependence is associated with an increased probability of an individual having neuropsychological deficits (Parsons, 1987). Thus, information about alcohol in the form of a CS triggers the expectancy effect in alcoholics, who respond by craving, and this might be greatest for those with neuropsychological deficits. The importance of the individual's expectations for consuming alcohol has been documented in several studies. For example, several researchers have found that the intensity of alcohol expectancies can vary as a function of the amount of alcohol consumed (Connors, O'Farrell, Cutter, & Thompson, 1987; Southwick, Steele, Marlatt, & Lindell, 1981); others have found that the severity of problem drinking is positively
correlated with alcohol expectancies (Brown, Goldman, & Christiansen, 1985; Connors, O'Farrell, Cutter, & Thompson, 1986). Furthermore, alcohol expectancies have been found to predict post-treatment functioning and relapse (Brown, 1985; Eastman & Norris, 1982).

To summarize, cues related to the consumption of alcohol become CS that elicit desires to drink, and this might lead to drinking. In addition, alcoholics are more field-dependent than nonalcoholics, and those alcoholics who experience craving are more field-dependent than are alcoholics who do not experience craving. Field-dependence and neuropsychological deficits are correlated in alcoholics, and both are inversely related to treatment outcome. Field-dependent individuals have difficulty separating out the perception of conditioned alcohol cues, the context in which it is presented, and the interoceptive stimuli associated with them. Thus, alcoholics who experience high craving and high field-dependence might be at greater risk to relapse because they are more susceptible to conditioned alcohol cues. Furthermore, the desire for alcohol is affected more by the expectancy of drinking alcohol than the actual consumption of it, and the expectancy is positively correlated with the degree of dependency on alcohol, which in itself is associated with neuropsychological deficits. Thus, increased alcohol dependency and neuropsychological deficits are associated
with a greater expectancy effect, which would produce increased desires to drink.

Therefore, conditioned alcohol cues would elicit an increased expectancy effect, and the effect of both of these would be greatest for those persons with neuropsychological deficits. The result of these conditions would be to produce greater desires to drink compared to the effect on nonbrain-damaged alcoholics, and this would lead to increased probability of relapse.

Physiological Aspects of Craving

Several researchers have reported that cravings (desires to drink) are correlated with physiological measures, including salivation, heart rate, skin conductance, and hand tremor (e.g., Cooney, Baker, Pomerleau, & Josephy, 1984; Kaplan, Cooney, Baker, Gillespie, Meyer, & Pomerleau, 1985; Labert & Ellertsen, 1987; Monti et al. 1987; Pomerleau, Fertig, Baker, & Cooney, 1983; Rankin & Hodgson, 1977). However, finding a reliable physiological correlate of craving has been problematic.

Salivation. The major thrust of these efforts has focused on the use of salivation as a correlate, but its usefulness as a reliable measure has been questioned. Pomerleau and her associates found conflicting results when measuring salivation. In one study, Pomerleau et al. (1983) found that alcoholics displayed greater salivation and craving than nonalcoholics did. However, in a subsequent
study (Cooney et al., 1984), they did not find a correlation between salivation and desire-to-drink ratings. In another investigation into the relationship between salivation and craving, Monti et al. (1987) found that alcoholics compared to nonalcoholics salivated more to alcohol cues when salivation was collected using dental rolls, but there was no difference between groups on urges to drink alcohol, which were increased for both groups.

The discrepancy among these studies may be the result of several methodological problems, including the manner in which salivation was determined, the subjects used, the manipulation of data, and the use of different stimuli for determining the baseline measurements to which the responses to alcohol were compared. Thus, the use of salivation as a reliable correlate of craving is not justified for this study. Furthermore, the desirability of using salivation in this study is even further reduced by the possibility of exposing the subjects and experimenters to disease.

Heart rate and skin conductance. Studies of heart rate (HR) and skin conductance have revealed some promising results. Pomerleau et al. (1983) found that HR and galvanic skin response (GSR) were elevated along with reports of craving when alcoholics were exposed to alcohol, but the findings were not significant. Kaplan et al. (1983), on the other hand, found a significant correlation between skin conductance response (SCR) and increased desire to drink in
a group of alcoholics but not in controls; unfortunately, heart rate did not distinguish the groups. Therefore SCR, but not HR, might be a reliable correlate of craving.

Hand tremor. In an investigation into the role of hand tremor as a physiological measure of craving, Rankin and Hodgson (1977) found that tremor was significantly correlated with reports of craving 10 hours after alcoholics were given a high dose of alcohol. According to the authors, this finding suggests that these correlates may be components of the same physiological and/or psychological state; on the other hand, tremor may act as a cue that triggers or influences craving. However, administering alcohol to alcoholics is not without ethical and methodological problems. Unfortunately no studies have reported the use of hand tremor without administering alcohol, which would limit its usefulness for this study.

Vasomotor response - plethysmography (VMRP). No studies that have investigated the relationship of craving and VMRP have been found. However, Rosenberg (1970) has noted that several studies have found that emotionally charged stimuli increase the VMRP. Rosenberg found that alcoholics' VMRP to electric shock did not habituate significantly different than controls did. However, the alcoholics responded with significantly greater VMRP, than controls, on a mental arithmetic task from which the alcoholics tried to avoid participating by using such
tactics as making irrelevant statements about their ability to solve the problems. The greater the VMRP and avoidance strategies of the alcoholic group suggest that they experienced greater arousal than the control group. It was concluded that the VMRP depends on the sensory aspects of the stimulus and the complex "psychical" state that it induces.

Thus, the most useful physiological measure associated with craving appears to be the skin conductance response. However, due to the technical risk of relying on only one type of psychophysiological measure and considering the promising association of the VMRP to emotional stimuli reported by Rosenberg (1970), the VMRP appears to be a promising response to use in this study.

Behavioral Aspects of Craving

Studies of the relationship between behavior and craving have shown a direct relationship of craving to alcohol acquisition behavior (e.g., Ludwig, Cain, Wikler, Taylor, & Bendfeldt, 1977; Ludwig, Wikler, & Stark, 1974). Tarter and Sugerman (1977) found that almost twice as many cravers drank daily or continuously compared to non-cravers, who were more often binge drinkers.

Summary

The cognitive, physiological, and behavioral findings cited above have led several researchers to propose that the construct of craving is a motivational state composed of
cognitive, physiological, and behavioral components in much the same way as the construct of fear is conceptualized (Marlatt, 1985). Whether one chooses to consider the complex set of effects described above as craving or to view them as separate, but related, events they appear to play a significant role in relapse. Specifically, it has been suggested that craving or a combination of the components discussed above is a necessary but not sufficient condition for relapse (Marlatt & Gordon, 1985). It is proposed in this study that neuropsychological deficits interfere with the habituation process to CS, and this may affect the individual's motivational state and may increase the risk of relapse.

Neuropsychological Literature

From another body of research, it has been found that a significant number of alcoholics develop subtle neuropsychological deficits, and these individuals have been shown to have higher relapse rates than do alcoholics without deficits (e.g., O'Leary et al., 1979). The most frequent findings are deficits in abstract reasoning, perceptual organization, new learning, visual conceptual abilities, and visuomotor tracking abilities. Moreover, the cognitive functioning of alcoholics, compared to non-alcoholics, is distinguished by more perseveration, more short-term memory deficits, and more deficits in the ability
to integrate information (Parsons, 1987; Ryan & Butters, 1986). They have also found that alcoholics typically have difficulty shifting strategies when solving problems; alcoholics also typically exhibit deficits in using feedback from incorrect results as well as impairment in their hypothesis testing ability. In fact, Miller and Saucedo (1983) have recommended that treatment centers should routinely screen for neuropsychological and cognitive deficits because the incidence of these problems is so high among alcoholics.

Despite these findings and recommendations, most alcoholism treatment programs do not test for such deficits. Perhaps this is because of the scarcity of studies that have investigated the clinical significance of such deficits, and the fact that even less is known about their remediation. For example, Walker, Donovan, Kivlahan, and O'Leary (1983) reported that increasing the length and intensity of treatment was no more beneficial for patients with cognitive deficits than were standard length treatments. In any event, it may be important to detect such deficits in order to investigate their impact on rehabilitation efforts and to design treatment plans that better address the individual needs of each patient.

In the area of cognitive functioning, as mentioned above, craving can be elicited by cues that have been classically conditioned to the effects of alcohol. Of
significance in this regard, Wikler (1973) found that cognitive processes can alter classically conditioned physiological and subjective responses; thus, impaired cognitive functioning may interfere with an individual's ability to alter his/her responses to a CS.

The findings from primate studies regarding the effects that neuropsychological deficits have on conditioning and habituation, especially those studies that have investigated the frontolimbic area, have revealed some interesting results. These will be reviewed in the following section.

Primate Studies

In primates, lesions of the frontolimbic region result in several impairments, including reduced short-term memory, and alterations in conditioned avoidance behavior, classical conditioning, and the orienting GSR. Lesioned primates also have difficulty organizing material temporally, which affects the ability to identify a situation as familiar or novel, as well as the ability to match information to some known context. This apparently involves a disruption in the registration and consolidation processes. In addition, they learn partly from their mistakes in operant conditioning situations. Furthermore, frontal subjects are less influenced by the consequences of their behavior whether it is rewarded or nonrewarded; it is as if reinforcements and/or their expectations exert little influence on their behavior. Several studies have also found that frontally
Lesioned monkeys show defective transfer of what has been learned in one situation to another similar situation (Pribram, 1986). Such conditions might facilitate continued arousal and attention to a cue while fostering resistance to habituation. Sustained arousal and attention might lead to behavioral interaction with the source of the arousal and attention.

Of course, all of these results have been found in primates, and humans may not be affected in the same manner. Nevertheless, it is interesting to note that the most frequent deficits, when they are present in alcoholics, closely parallel the results found in the primate studies cited above. Furthermore, it is worth repeating that these deficits when found in alcoholics are significantly associated with relapse.

If the results from the primate studies could be extended to humans, it could be predicted that if an alcoholic with frontolimbic deficits was presented with a drinking cue, the individual would be faced with the choice to either drink or not drink. The impaired individual would orient towards such cues more readily and with greater intensity than a non-impaired alcoholic, and this orientation might maintain attention and arousal, thereby increasing the salience of the cue and the possibility that the individual would choose to drink.
Moreover, the impairment in the ability to temporally organize material may affect the ability to distinguish the individual's remote experience with the situation from his or her recent experiences with it. Thus, a situation that has been associated with pleasure in the distant past but has been associated with recent displeasure may only be associated with the pleasurable experience if the ability to temporally organize information is impaired. To the impaired alcoholic, the cue would not be associated with the individual's most recent past experience with alcohol which would include adverse consequences, but the individual would associate the cue with his or her early experiences with alcohol instead, which would have occurred prior to his or her impairment and consisted of feelings of euphoria and pleasurable activities.

In this regard, it is noteworthy to mention that the available research on memory deficits in alcoholics supports such an explanation. Namely, chronic alcoholism severely impairs the consolidation of newly learned information from short-term memory into long-term memory; thus, the memory consolidation process of recent, adverse experiences with alcohol might be disrupted. On the other hand, both immediate memory (i.e., STM), which is the ability to retain information in immediate awareness; and long-term storage (i.e., remote memory) are only mildly affected by chronic alcoholism (Russell, 1981).
Human Studies

The number of human studies that have investigated the relationship of brain-damage to psychophysiological functioning is quite sparse. The available literature has been reviewed by Holloway and Parsons (1980) and Stern and Jaynes (1973). The results of the systematic studies are often contradictory. For example, there is no support that brain-damaged individuals experience a generalized hyper- or hypo- activation of the ANS. There is, however, evidence that various brain-damaged individuals experience some disruption in the normal control of some ANS responses, but exact predictions can not be made from the available research.

The difficulty in drawing reliable conclusions is, no doubt, due in part to the tendency to group together individuals with heterogeneous brain-damage in the research, including patients with cortical and subcortical damage in some groups.

In the few studies (Callan, Holloway, & Bruhn, 1972; Lovallo, Parsons, & Holloway, 1973; Oscar-Berman & Gade, 1979) that have investigated the psychophysiological (including SCR, HR, and VMRP) responding of alcoholics, very little information has been uncovered that would be useful in this study. In the first two studies, alcoholics were grouped together and compared to controls and heterogeneous brain-damaged patients. Callan et al. examined
distractibility using repeated presentation of a distracting tone in a visual reaction time task. The alcoholics habituated to the distracting stimuli in a manner similar to the controls resulting in better reaction time performance for both of these groups compared to brain-damaged patients. Lovallo et al. found that alcoholics exhibited similar initial vasomotor responses to a cold-pressor test as did brain-damaged patients and controls, but the alcoholics were more similar to the controls, than to the brain-damaged patients, on the recovery of cardiovascular responsiveness. Thus, the alcoholics performed more similarly to controls than to brain-damaged groups. However, no specifications about brain-damage in the alcoholic groups were made or of alcoholism in the brain-damaged subjects; thus, the effect of brain-damage on alcoholics can not be ascertained from these studies. In the third study, Oscar-Berman and Gade compared Korsakoff patients to normal controls and three other brain-damaged patient groups. Korsakoff and Huntington chorea patients showed decreased spontaneous SCR, decreased SCR to stimuli, and decreased habituation rate to an auditory tone. While these findings may seem counter to the hypothesis proposed in this study; it should be noted that their study used a neutral auditory stimulus which can be viewed as an irrelevant stimulus in the context of the CSs which this study proposes are essential variables influencing the risk of relapse. In addition, Korsakoff
patients have more severe brain-damage than the subtle deficits proposed to affect the responding of subjects used in this study.

Specific human studies that have investigated the orienting response and habituation in brain-damaged individuals (Davidoff & McDonald, 1964; Hattangai, 1969; Holloway & Parsons, 1971; Parsons & Chandler, 1969; Parsons, Messenger, & Holloway, 1973) have also revealed contradictory findings across studies and across responses. Of particular interest to this study are the results that have revealed that brain-damaged individuals have been shown to exhibit decreased, increased, or no different response magnitude, as well as slower, faster, and no different habituation rates of skin resistance responses (SRR) or SCR. However, these studies used neutral, auditory stimuli which, as mentioned above, are irrelevant stimuli in the context of the CSs that elicit craving.

Based on this research, Holloway and Parsons (1980) have hypothesized that brain-damaged individuals may experience disruptions in their performance and/or exacerbations of their cognitive or motor deficits. They proposed that some unspecified parts associated with the reactivity of their ANS may not be adequately coordinated to enable the individual to adequately prepare to interact with a stimulus in a manner that would facilitate performance. Nevertheless, no firm conclusions can be drawn from the
available human studies to guide the hypothesis of this study, except that habituation is altered in some types of brain-damage. One possible contribution to the rather inconclusive status of this research area is that, in all of the studies of craving, the researchers either used subjects presumptively without brain-damage or they did not screen for it.

Summary

A graphic model of the relationship between CS, orienting response, craving, brain-damage, and relapse is presented in Figure 1. It is apparent that craving consists of cognitive, physiological, and behavioral components; furthermore, CSs appear to be able to elicit a craving response which may be the CR, or at least the cognitive and motivational components of it, and the orienting response (OR), which can be considered to be a multifactored reaction consisting of overt and covert physical changes to a novel stimulus. The OR, CR, and craving may be interrelated and form a response complex associated with an alcohol cue. Habituation of the orienting response, and/or this response complex, is involved in selective attention such that it decreases attention to irrelevant stimuli and allows us to shift our attention to relevant stimuli (Waters, McDonald, & Koresko, 1977). Thus, an impairment in the habituation
process would tend to sustain attention to the stimuli creating conditions favorable to the enhancement of craving. Thus, neuropsychological-cognitive deficits could affect responses to a CS by interfering with the hypothesized components of craving, one of which is the process of habituation. Therefore, the manner in which an individual processes and habituates to a cue may play an important role in how that individual responds to that cue.

Based on a tripartite model of craving and the literature described above it seems reasonable to expect that neuropsychological deficits might interfere with the process of habituation to a CS as well as interfering with the components of craving by hampering the learning of new material, which would include information that the substance (alcohol) is adversely affecting the individual. Thus, it is hypothesized that individuals with neuropsychological deficits have difficulty habituating to alcohol as a CS, and this may serve to prolong craving, leading to an increased risk for relapse. In order to test this hypothesis, the habituation process of three groups of subjects will be compared: (a) alcoholics with brain-damage (BDA), (b) alcoholics without brain-damage (NBDA), and (c) a control group of social drinkers (SD).

The major hypothesis, that neuropsychological deficits interfere with the habituation process, supposedly would be
supported by confirmation of eight minor predictions. The minor predictions can be briefly stated as follows:

1. On the severity of alcohol abuse/dependence as measured by the Michigan Alcoholism Screening Test (MAST) and the Severity of Alcohol Dependence Questionnaire (SADQ), the BDA and NBDA groups would score higher than the SD group.

2. On the number of trials to habituate to alcohol (alcohol habituation rate) the BDA group would score higher than the NBDA and SD groups.

3. On the mean response amplitudes to alcohol or alcohol responses, the BDA groups would respond more intensely than the NBDA and SD groups.

4. On the alcohol habituation rate and alcohol responses, the NBDA group would fall between the BDA and SD groups.

5. On the number of water trials to habituate or water habituation rate and the response amplitudes to water (water responses) there would be no differences among groups.

6. The BDA and NBDA groups but not the SD group will show within group differences between alcohol habituation rate compared to the water habituation rate, as well as differences in response amplitudes to the different stimuli.
7. For the desire to drink ratings (desire ratings), the post-test ratings would be greater than the pre-test ratings, and the post-test ratings of the BDA and NBDA groups would score higher than the SD group with the NBDA falling in between the two other groups.

8. For the estimates of the number of times the stimulus was presented (stimulus estimations), the BDA group would score higher than the SD group, with the NBDA group falling in between.

The importance of this study can be appreciated by looking at the implications if the findings support either the alternative or the null hypothesis. If the hypothesis is true, then the study would provide concrete evidence that alcoholic patients should be routinely screened for neuropsychological deficits; additionally, more intensive relapse-prevention training and research would be needed to develop more effective or different methods to train patients in relapse-prevention. The problem of proving the null hypothesis is always present, but if the hypothesis is not supported, one possible explanation of the results would be that neuropsychological deficits do not interfere with the processing of alcohol cues, at least as measured in this study, and therefore neuropsychological deficits may affect relapse in some other manner.
Method

Subjects

The number of subjects required for this study was determined by a compromise between the need for adequate power and the practical considerations of obtaining enough subjects who met the criteria for inclusion in the study. Based on the expectation of a large effect size, a power analysis (Cohen, 1977) was performed, and it was determined that fifteen subjects in each group produced an acceptable compromise. At an alpha level of 0.05 this size sample yields a power level of 0.64, and at an alpha level of 0.10 the power level is 0.76, which means that at alpha = 0.10 there is approximately three chances in four of finding significant differences when in fact there are true significant differences. The decision to perform the power analysis using a large effect size is based on the findings by Griffin (1963) who found a large effect size (4.8) for trials to habituation of a stimulus when comparing brain-damaged patients and nonbrain-damaged controls.

Prospective subjects with a history of schizophrenia or a history of significant (greater than 10%) polysubstance abuse were not used. Prospective subjects taking medication that would interfere with physiological responding (e.g., anticholinergic agents) or with a medical history that might interfere with performance, as well as those who had experienced significant head injuries, were also excluded.
The number of smokers and non-smokers were nearly equivalent across groups. No subject had eaten during the 2 hours before their physiological responses were measured.

Fifty-nine male subjects were recruited from the Salvation Army, local AA groups, and the community to participate in this study (39 alcoholics and 20 controls). All subjects were offered a compensation of $5.00 for participating in the study, and it was given to those subjects who wanted it. Twelve subjects in the BDA group, six in the NBDA group, and none of the SD subjects received compensation. Alcoholic subjects were so classified if they identified themselves as alcoholic and scored 5 or more on the MAST; social drinkers consisted of individuals who reported drinking 6 or less drinks per month and scored 3 or less on the MAST. Of the original 59 subjects, 14 were dropped (9 alcoholics and 5 controls) for the following reasons: (a) 5 subjects produced no physiological responses, (b) 8 subjects scored 3 on the Impairment Index, and (c) one control subject was discovered to exceed the criteria for social drinking. Of the remaining forty-five subjects each was assigned to one of three groups (n = 15): (a) Brain-damaged alcoholics (BDA), (b) Nonbrain-damaged alcoholics (NBDA), and (c) nonbrain-damaged social drinkers (SD).

In order to control for the possible effects of the length of abstinence, the groups of subjects were matched, as nearly as possible, on the time since their last drink.
However, no subjects were used who required detoxification. Due to the wide variation (1 day to 53 years) of this variable, the subjects were classified into one of four abstinence categories: A - 1 to 30 days; B - 31 to 180 days; C - 180 to 365 days; and D - greater than 365 days. The breakdown of the subjects by groups is listed in Table 1. A Chi square analysis revealed that the distribution of the groups into categories was not significantly different than what would be expected by chance ($X^2 = 6.31, p = 0.39, df = 6$).

Subjects in the NBDA and SD groups were matched by groups to the subjects in the BDA group based on the matching measures listed in the Measures section below and the information obtained on the Demographic and Background Information Form which also served as a recording form for each subject (see Appendix 1). There were no significant differences found between groups on the matching measures (see Table 2). Severity of alcohol dependence was assessed with the MAST and the SADQ; copies of each of these measures appear in Appendix 4. The ethical dilemma of exposing alcoholics to drinking cues is, to some extent, offset by the importance of this study. Nevertheless, the participants and any individuals responsible for them, were informed about the risk that they may experience increased craving as a result of their participation. No subjects requested to withdraw, and safeguards were preplanned in the event a
### Table 1

**Comparison of groups by categorical lengths of abstinence**

<table>
<thead>
<tr>
<th>Category</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (1-30 days)</td>
<td>5</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>B (31-180 days)</td>
<td>6</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>C (181-365 days)</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>D (＞365 days)</td>
<td>2</td>
<td>6</td>
<td>2</td>
</tr>
</tbody>
</table>

$X^2 = 6.31$, p value $= 0.39$, df $= 6$
Table 2

**Matching Measures: Means, F Values, and P Values Between Groups**

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Education</th>
<th>Vocabulary</th>
<th>Digits Forward</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDA</td>
<td>46.67</td>
<td>12.80</td>
<td>10.13</td>
<td>7.97</td>
</tr>
<tr>
<td>NBDA</td>
<td>47.53</td>
<td>13.87</td>
<td>10.00</td>
<td>9.00</td>
</tr>
<tr>
<td>SD</td>
<td>45.33</td>
<td>13.67</td>
<td>10.93</td>
<td>8.80</td>
</tr>
</tbody>
</table>

**F values**
- 0.22
- 0.64
- 0.73
- 0.98

**p values**
- 0.802
- 0.533
- 0.489
- 0.382

*df = 2, 43 for each analysis*

*No significant differences between groups*
subject reported distress or intolerable levels of craving induced by the experimental procedure. Furthermore, all of the subjects who were under the care of others were delivered back to their caregivers. Provisions for treatment or relapse were available either with their caregivers or through the author in conjunction with the Baton Rouge Substance Abuse Clinic. Each subject, and caregiver where appropriate, read and signed a consent form that explained all of these provisions (See Appendices 2 & 3).

Measures

Subject matching measures.

1. WAIS-R Digit Span subtest -- A test of short-term memory and attention which consists of repeating a list of numbers either in the order presented or in the reverse order. The test is sensitive to diffuse brain-damage that occurs with many dementing processes or severe brain injury, but it may not be affected by alcohol related deficits. However, Lezak (1983) has noted that poor performance on this subtest may be associated with brain-damage, but the results reveal that this is due to decreases in the Digits Backwards component of the subtest, which is more susceptible to brain-damage, and not the Digits Forward, which is more a measure of attentional efficiency than memory (Spitz, 1972). Therefore, the groups of subjects were matched on the means of their Digits Forward raw scores.
2. WAIS-R Vocabulary subtest -- A 40-item test of the meaning of words. It is relatively unaffected by deficits associated with alcoholism. It is highly correlated with the full scale IQ and is the best single, short test of full scale IQ (Lezak, 1983); thus, it is a good indicator of premorbid functioning. The groups were matched on the means of their scale scores.

Brain-damage screening measures.

1. WAIS-R Digit Symbol subtest -- A timed test that involves the substitution of symbols and taps a variety of factors that affect performance, including the ability to learn an unfamiliar task; the results are sensitive to the presence of brain-damage (Long & Gouvier, 1982).

2. WAIS-R Block Design subtest -- A timed construction test that measures visuospatial organizational ability; performance requires that logic and reasoning be used to solve problems of spatial relationships. It is quite sensitive to brain-damage (Lezak, 1983).

3. WAIS-R Object Assembly subtest -- A timed test that requires the individual to assemble pieces of a jig-saw puzzle into a familiar object; it is a test of synthesis and of perceptual, organizational ability. It is quite sensitive to brain-damage (Lezak, 1983).

4. Trail Making Test - Part B (TMT-B) -- A timed test that requires the individual to connect consecutive
letters and numbers while alternating between them. It is sensitive to the presence of brain-damage (Lezak, 1983).

5. Short Category Test - Booklet Form (SCT) -- A test of abstracting ability that taps several factors; it is sensitive to brain-damage (Wetzel & Boll, 1987).

6. An Impairment Index was developed which consisted of assigning 2 points for an impaired score on the Short Category Test (SCT) and 1 point for each of the other four screening measures. A WAIS-R subtest score, used in the brain-damage screening procedure, was considered to be impaired if it was three or more scaled-score points below the subject's score on the WAIS-R Vocabulary subtest. This difference is considered to be statistically significant at the 15% level of confidence which means that the chances are about 85 out of 100 that the difference represents a real difference in ability on the two tests.

The possible scores for the Impairment Index range from 0 to 6 with 6 representing maximum impairment. Subjects who scored 4 or more were placed in the BDA group, while subjects in the NBDA and SD groups were required to score 2 or less. In order to maximize the difference between the BDA group and the nonbrain-damaged groups, no subjects who scored on the boundary of the cutoff score (i.e., 3) were used.
Other measures.

1. Michigan Alcohol Screening Test (MAST) -- A 25-item instrument to detect alcoholism, with higher scores indicating more problems associated with drinking. It has well established reliability and validity (Selzer, 1971).

2. Severity of Alcohol Dependence Questionnaire (SADQ) -- A 20-item instrument designed to assess the central features and severity of a subject's alcohol dependence, including physical and affective symptoms of withdrawal, craving and withdrawal relief drinking, typical daily consumption, and rapidity of symptom reinstatement after a period of abstinence. It has good reliability and validity (Stockwell, et al., 1979; Stockwell, Murphy, & Hodgson, 1983).

3. Pre-test and Post-test Likert ratings (0 - 9) for Desires for Alcohol.

4. Post-test estimation of the number of times the atomized aromatic was presented.

Psychophysiological measures.

1. Skin-conductance response (SCR) -- This is a short-term response of certain eccrine sweat glands to external stimuli and stress. It is correlated with CS induced craving and the orienting response.

2. Vasomotor response (VMRP) -- A measure of blood volume in the periphery; it is a useful indicator of the
orienting response and sympathetic nervous system (SNS) activation.

**Apparatus**

The physiological responses were measured using a Grass Model 7D Polygraph (Grass Instrument Co., Quincy, Mass). Specifically, the SCR were measured on a Grass Low Level D.C. Amplifier/Preamplifier Model 7P122B; the VMRP, were measured using a Grass EEG Amplifier/Preamplifier Model 7P511H. All electrodes were 16mm silver-silver chloride and attached to the subject using Beckman Electrode Electrolyte paste. The electrodes and the electrode paste were manufactured by Beckman Instrument, Inc., Schiller Park, Ill. The VMRP responses were recorded using a custom built sensor consisting of a 12 volt, 0.025 ampere Archer miniature lamp source (Catalogue # 272-1141) and an Archer cadmium sulfide photocell (Catalogue # 276-118).

**Stimuli.** It was recognized that differences in the subjects' preferred drinks could possibly affect the results; specifically a wide variation in the types of preferred drinks between groups and a wide difference in the intensity of the smell between types of drinks are two possible confounds. In order to control for the first possibility, the plan was to limit the variety of preferred drinks to beer, wine, bourbon/whiskey, and gin; in addition, the groups were to be matched as closely as possible on the variety of these alcoholic beverages.
Differences in the beverages' intensity of smell were equated in pilot studies before using them in the experimental conditions according to the following procedure. Twenty nonbrain-damaged, middle-aged, males rated each of 3 dilutions (25%, 50%, 75%) plus a full strength sample of the types of drinks to be used. Each sample was diluted with distilled water. The samples were placed in individual, opaque containers in order to prevent identification. The raters were presented with the dilutions and beverages in a random order to control for order effects, and each rater was asked to rate the intensity of the smell on an 1 - 7 scale with 1 representing little or no smell and 7 representing an extremely strong smell. After all of the ratings were obtained, the diluted samples were matched on intensity so that the actual samples selected for the experimental conditions had nearly equal ratings across beverages, and these selections were statistically analyzed to ensure no statistical differences existed between smell intensities. The results are presented in the Results section.

Procedure

Upon arrival at the psychophysiological laboratory, each subject was rechecked for continued abstinence, medication status, NPO status, and negative dysosmia. Screening for dysosmia consisted of having the subject correctly identify at least 2 of four common smells:
perfume, aftershave lotion, vinegar, and isopropyl alcohol (see Appendix 1). Then they were asked to rate their desire to drink on a scale of 0 - 9 with 0 signifying no desire and 9 indicating that they were able to resist drinking only with great difficulty.

Each subject was tested while sitting in an upholstered, recliner chair positioned in the upright position. The subject was in a sound-insulated room, and the recording and control apparatus were in an adjoining room out of sight and hearing of the subject.

Prior to being hooked up to the polygraph equipment, the subject was given a detailed explanation of the procedure (see Appendix 2) and the consent form was signed (see Appendix 3).

Once the stimulus samples were equated for smell intensity, the active stimuli consisted of placing 2 cc's of the subject's preferred alcoholic beverage in a Airlife Misty Nebulizer (catalogue number 002010) manufactured by Airlife, Inc. a subsidiary of the American Hospital Supply Corporation of Montclair, CA. The nebulizer was fitted to a Oxygen mask model 64041 manufactured by B & F Medical (hereafter referred to as an air mask).

The inactive stimuli, water, was contained in another Airlife nebulizer which was also attached to the air mask. The mask was connected to a Sears 3/4 HP air compressor to provide constant airflow into the mask at 10 psi. Each
nebulizer was powered by an Aerosol Two compressor manufactured by Medical Industries America, Inc., of Adel, IA. All the compressors were set up outside the sound conditioned room in which the subject was seated. Each subject was fitted with sound suppressing earmuffs as an additional safeguard to further reduce the sound of the compressors.

The alcohol and water were presented to the subject in 2 second bursts of the nebulizer twenty times each by the experimenter according to the following schedule which is based on Gellermann's (1933) table of alternating stimuli: 


The experimenter was instructed on which stimulus to present and when to present it by a pre-recorded audiotape to which he was listening through a set of headphones. The pre-recorded message was designed to administer the stimuli at the average rate of one per 30 seconds, with the interstimulus interval ranging from 15 to 45 seconds. At the instant the experimenter presented the stimulus, he also triggered the appropriate event recorder button on the polygraph to identify the type of stimulus being administered. Upon completion of the 40 trials, the subject was asked to rate his desire to drink on a scale of 0 - 9.
Results

Pilot study

Twenty subjects rated the 16 alcohol samples on a Likert scale of 1 to 7. The samples with the closest average intensity to each other included: 100% beer (4.7), 100% gin (3.4), 25% bourbon/whiskey (5.1), and 50% wine (4.6). One-way ANOVA of these data revealed significant differences among the samples ($F(3, 76) = 3.15; p = .0298$). Scheffe's post hoc analysis revealed that the gin sample was significantly different from the bourbon/whiskey; therefore, gin was dropped from the experimental portion of the study, and no subjects who preferred gin were used. In addition, it was found during training of the assistants that the wine sample, when it was atomized by the nebulizer, did not smell like the wine in its original container, so wine drinkers were not used in the study either. Thus, 100% beer was used for subjects who preferred beer, and a 25% distilled water dilution of bourbon/whiskey was used for those subjects who preferred bourbon/whiskey. Eight subjects in the BDA group smelled beer while 7 smelled bourbon/whiskey. In the NBDA group, 9 smelled beer and 7 smelled bourbon/whiskey, and in the SD group 5 subjects smelled beer and 9 smelled bourbon/whiskey.

Experimental Study

Subject matching measures. One-way ANOVA of the variables age, education, Vocabulary subtest, and Digits
Forward scores revealed no significant differences among groups, which complies with the basic design of the study as set forth in the method section. Means, F values, and P values are listed in Table 2 (p. 28). Correlational analyses revealed that these matching measures did not correlate significantly with the Impairment Index: Age (r = 0.04, p = .78), education (r = -0.14, p = .34), Vocabulary subtest (r = 0.002, p = .99), and Digits Forward (r = -0.13, p = .40). Thus, these measures were independent of the Impairment Index. Interestingly, the decision to use only the Digits Forward portion of the WAIS-R Digits Span subtest was supported by the finding that the total Digit Span scaled scores (using Digits Forward plus Digits Backward) were negatively correlated with the Impairment Index (r = -0.31, p = .036).

**Brain-damage screening measures.** One-way ANOVA revealed that the BDA group performed significantly worse than the NBDA and the SD groups on each of the screening measures (see Table 3). On the Impairment Index the mean of the BDA group was 4.60 compared to 1.067 for the NBDA group and 1.000 for the SD group (F (2, 43) = 77.38, p = .0001). The NBDA and SD groups were significantly different from each other only on the Digit Symbol subtest; however, these two groups did not differ from each other on any of the other brain-damage screening measures including the Impairment Index. But the scores of the NBDA group on each
Table 3

Brain-damage Screening Measures: Means, F Values, and P Values Between Groups

<table>
<thead>
<tr>
<th></th>
<th>BD</th>
<th>OA</th>
<th>DS</th>
<th>TB</th>
<th>SCT</th>
<th>II</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDA</td>
<td>7.067b</td>
<td>6.200b</td>
<td>6.200c</td>
<td>110.47b</td>
<td>85.27b</td>
<td>4.60b</td>
</tr>
<tr>
<td>NBDA</td>
<td>9.800a</td>
<td>9.267a</td>
<td>8.200a</td>
<td>77.07a</td>
<td>56.93a</td>
<td>1.07a</td>
</tr>
<tr>
<td>SD</td>
<td>11.267a</td>
<td>10.333a</td>
<td>10.200b</td>
<td>66.33a</td>
<td>57.87a</td>
<td>1.00a</td>
</tr>
<tr>
<td>F values</td>
<td>10.14</td>
<td>9.91</td>
<td>13.32</td>
<td>16.81</td>
<td>4.74</td>
<td>77.38</td>
</tr>
<tr>
<td>p values</td>
<td>.0003</td>
<td>.0003</td>
<td>.0001</td>
<td>.0001</td>
<td>.0139</td>
<td>.0001</td>
</tr>
</tbody>
</table>

BD = Block Design, OA = Object Assembly, DS = Digit Symbol, TB = Trails B, SCT = Short Category Test, II = Impairment Index

df = 2, 43 for each analysis

Column means with the same letter are not significantly different
of the subtests were more impaired than the scores of the SD group.

**Other measures.** A one-way ANOVA was performed on the MAST scores and the SADQ scores. It was predicted that the scores for the BDA and NBDA groups would be significantly greater than the scores for the SD group. As expected, both alcoholic groups scored significantly higher than the SD group on the MAST ($F (2, 43) = 55.35, p = .0001$) and the SADQ ($F (2, 43) = 20.87, p = .0001$), but they did not differ significantly from each other; although the mean of the MAST for the BDA group (46.13) was 31% greater than for the NBDA group (35.13), and the SADQ mean was 21% greater (48.53 vs 40.07; see Table 4).

**Psychophysiological measures.** The following data was obtained for each of the psychophysiological measures (SCR & VMRP): (a) number of trials to habituation for the alcohol and the water stimuli, and (b) each individual's mean response for alcohol trials and for water trials. Habituation was defined as the number of trials before the subject failed to respond 3 times consecutively to the stimulus. For an SCR to be scored, it had to have an amplitude of at least one percent of the pre-stimulus baseline and reached peak amplitude within 5 seconds of the stimulus onset; all other SCR were considered to be spontaneous SCR and were discarded. The responses for VMRP were computed by subtracting the post-stimulus amplitude
Table 4

Alcoholism Severity Measures, F Values, and P Values Between Groups

<table>
<thead>
<tr>
<th></th>
<th>MAST</th>
<th>SADO</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDA</td>
<td>46.13a</td>
<td>48.53a</td>
</tr>
<tr>
<td>NBDA</td>
<td>35.13a</td>
<td>40.07a</td>
</tr>
<tr>
<td>SD</td>
<td>0.80b</td>
<td>20.87b</td>
</tr>
<tr>
<td>F values</td>
<td>55.35</td>
<td>20.87</td>
</tr>
<tr>
<td>p values</td>
<td>.0001</td>
<td>.0001</td>
</tr>
</tbody>
</table>

df = 2, 43 for all analyses

Column means with the same letter are not significantly different
from the pre-stimulus amplitude; this difference was divided by the pre-stimulus amplitude, and the results were multiplied by 100 to give the percent decrease in amplitude (i.e., a baseline controlled measure of vasoconstriction).

The pre-stimulus amplitude was computed by averaging the amplitude for the 5 beats preceding the stimulus onset. The post-stimulus amplitude was computed by first identifying the smallest beat in the 8 second interval after stimulus offset; then computing the average amplitude of that beat and the 2 beats on each side of it.

These dependent measures were analyzed according to a two-way ANOVA (group X stimulus). It was predicted that the number of trials to habituation and the mean response to alcohol trials would be significantly greater for the BDA group compared to the NBDA and SD group. It was further predicted that the alcohol trial scores for the NBDA group would fall between the BDA and SD groups. For the water trials, it was predicted that no differences would be found among groups. In addition, it was predicted that the differences between the responses to alcohol and water would be significantly different within groups for the BDA and NBDA groups but not for the SD group. A graph of these predictions is presented in Figure 2.

For the SCR, the results (Table 5 & Figure 3) revealed no differences between any of the groups on the number of trials to habituation to alcohol $(F(2,31) = 0.37,$
**Figure 2**

Pre-experimental predictions for psychophysiological measures

---

- solid line = alcohol
- dashed line = water
Table 5  
SCR Measures Related to Habituation: Means, F and t Values, and P Values Between Groups (rows) and Within Groups (columns)

<table>
<thead>
<tr>
<th>Measures</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
<th>F</th>
<th>P&gt;F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASNH</td>
<td>5.22a</td>
<td>4.54a</td>
<td>5.91a</td>
<td>0.37</td>
<td>0.694</td>
<td>2.31</td>
</tr>
<tr>
<td>WSNH</td>
<td>2.11a</td>
<td>2.77a</td>
<td>4.36a</td>
<td>1.75</td>
<td>0.191</td>
<td>2.31</td>
</tr>
<tr>
<td>A-WNH</td>
<td>3.11a</td>
<td>1.77a</td>
<td>1.55a</td>
<td>0.91</td>
<td>0.574</td>
<td>2.31</td>
</tr>
<tr>
<td>t Value</td>
<td>2.06</td>
<td>1.87</td>
<td>0.82</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
<tr>
<td>p values</td>
<td>0.028</td>
<td>0.037</td>
<td>0.210</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
<tr>
<td>ASMH</td>
<td>0.267a</td>
<td>0.377a</td>
<td>0.397a</td>
<td>0.57</td>
<td>0.574</td>
<td>2.31</td>
</tr>
<tr>
<td>WSMH</td>
<td>0.171a</td>
<td>0.317a</td>
<td>0.367a</td>
<td>1.78</td>
<td>0.185</td>
<td>2.31</td>
</tr>
<tr>
<td>A-WMH</td>
<td>0.096a</td>
<td>0.060a</td>
<td>0.030a</td>
<td>0.39</td>
<td>0.679</td>
<td>2.31</td>
</tr>
<tr>
<td>t Value</td>
<td>0.174</td>
<td>0.547</td>
<td>0.290</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
<tr>
<td>p values</td>
<td>0.432</td>
<td>0.295</td>
<td>0.387</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
<tr>
<td>df</td>
<td>1.16</td>
<td>1.24</td>
<td>1.20</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
</tbody>
</table>

ASNH = mean number of trials for SCR to habituate to alcohol  
WSNH = mean number of trials for SCR to habituate to water  
A-WNH = difference between ASNH and WSNH  
ASMH = mean amplitude of SCR trials to habituate to alcohol  
WSMH = mean amplitude of SCR trials to habituate to water  
A-WNH = difference between ASMH and WSMH  
Row means with the same letter are not significantly different. All t-tests are one-tailed.
Figure 3

SCR mean trials to habituate to alcohol and water by groups

--- = alcohol
---- = water
p = .694) or to water ($F (2, 31) = 1.75, p = .191$). In addition, no differences were found between groups on the mean response amplitudes to either alcohol ($F (2, 31) = 0.57, p = .574$) or water ($F (2, 31) = 1.78, p = .185$). Within group comparisons, using one-tailed $t$-tests, of the performance differences between alcohol and water revealed significant differences for the number of trials to habituation for the BDA group ($t (1, 16) = 2.06, p = .028$) and for the NBDA group ($t (1, 24) = 1.87, p = .037$) but not for the SD group ($t (1, 20) = 0.82, p = .211$). Thus, the prediction that the SCR of the alcoholic group would habituate differently to the two stimuli while the SD group would not was confirmed. However, for the mean SCR amplitudes no within group differences were found between the responses to alcohol compared to water for any of the groups (BDA: $t (1, 16) = 0.174, p = .432$; NBDA: $t (1, 24) = 0.547, p = .295$; SD: $t (1, 20) = 0.290, p = .387$).

For the VMRP, the results (Table 6 & Figure 4) revealed no differences between any of the groups for the number of trials to habituation to alcohol ($F (2, 40) = 0.01, p = .988$) or of the trials to habituation to water ($F (2, 40) = 2.07, p = .140$). As with the SCR, the VMRP within group comparisons (using one-tailed $t$-tests) between alcohol and water revealed significant differences for the BDA group ($t (1, 27) = 3.44, p = .001$) and the NBDA group ($t (1, 24) = 2.66, p = .007$) but not for the SD group ($t (1, 26) = 1.09,$
Table 6

**VMRP Measures Related to Habituation: Means, F and t Values, and P Values Between Groups (rows) and Within Groups (columns)**

<table>
<thead>
<tr>
<th>Measures</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
<th>F</th>
<th>P&gt;F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVNH</td>
<td>8.57a</td>
<td>8.31a</td>
<td>8.50a</td>
<td>0.01</td>
<td>0.988</td>
<td>2,40</td>
</tr>
<tr>
<td>WVNH</td>
<td>3.43a</td>
<td>4.31a</td>
<td>6.43a</td>
<td>2.07</td>
<td>0.140</td>
<td>2,40</td>
</tr>
<tr>
<td>A-WNH</td>
<td>5.14a</td>
<td>4.00a</td>
<td>2.07a</td>
<td>2.74</td>
<td>0.077</td>
<td>2,40</td>
</tr>
<tr>
<td>t Value</td>
<td>3.44</td>
<td>2.66</td>
<td>1.09</td>
<td>-----</td>
<td>------</td>
<td>----</td>
</tr>
<tr>
<td>p values</td>
<td>0.001</td>
<td>0.007</td>
<td>0.143</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Measures</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
<th>F</th>
<th>P&gt;F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVMH</td>
<td>0.272a</td>
<td>0.245a</td>
<td>0.286a</td>
<td>0.60</td>
<td>0.551</td>
<td>2,40</td>
</tr>
<tr>
<td>WVMH</td>
<td>0.258a</td>
<td>0.213a</td>
<td>0.245a</td>
<td>0.36</td>
<td>0.699</td>
<td>2,40</td>
</tr>
<tr>
<td>A-WMH</td>
<td>0.014a</td>
<td>0.032a</td>
<td>0.041a</td>
<td>0.15</td>
<td>0.863</td>
<td>2,40</td>
</tr>
<tr>
<td>t Value</td>
<td>0.36</td>
<td>0.94</td>
<td>1.94</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
<tr>
<td>p values</td>
<td>0.362</td>
<td>0.178</td>
<td>0.122</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
<tr>
<td>df</td>
<td>1,27</td>
<td>1,24</td>
<td>1,26</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
</tbody>
</table>

AVNH = mean number of trials for VMRP to habituate to alcohol
WVNH = mean number of trials for VMRP to habituate to water,
A-WNH = difference AVNH and WVNH,
AVMH = mean amplitude of VMRP trials to habituate to alcohol
WVMH = mean amplitude of VMRP trials to habituate to water,
A-WMH = difference between AVMH and WVMH

Row means with the same letter are not significantly different. All t-tests are one-tailed.
Figure 4

VMRP mean trials to habituate to alcohol and water by groups

--- = alcohol
----- = water
p = .143). Thus, the prediction that the alcoholic groups' VMRP would habituate differently to the stimuli while the SD group would not was confirmed. However, for the mean VMRP amplitude no within group differences were found between the responses to alcohol compared to water for any of the groups (BDA: \( t(1, 27) = 0.36, p = .362 \); NBDA: \( t(1, 24) = 0.94, p = .174 \); SD: \( t(1, 26) = 1.94, p = .122 \)).

Because the degrees of freedom was reduced for the separate SCR and VMRP analyses due to the failure of some subjects to respond in both psychophysiological channels, an Overall Habituation Index was developed to include all 45 subjects. The index score was derived in the following manner: For those subjects who responded in both channels (SCR & VMRP) the average of the habituation trials of the channels was used, for those subjects who responded in only one channel the trials to habituation for the responding channel was used. A two-way ANOVA (group X stimulus) was performed on this data, and the results (Table 7 & Figure 5) paralleled those found in the SCR and the VMRP analyses with one important exception. Specifically, no between group differences were found on the Overall Habituation Index in response to alcohol \( (F(2, 43) = 0.39, p = .678) \). However, unlike the findings of the habituation of the SCR and VMRP to water, differences were found between groups on the Overall Habituation Index in response to water \( (F(2, 43) = 4.88, p = .012) \). Scheffe's post hoc analysis revealed that
Table 7

**Overall Habituation Indices:** Means, F and t Values, and P Values Between Groups (rows) and Within Groups (columns)

<table>
<thead>
<tr>
<th>Measures</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
<th>F</th>
<th>P&gt;F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>AOHI</td>
<td>7.9a</td>
<td>6.9a</td>
<td>7.1a</td>
<td>0.39</td>
<td>0.678</td>
<td>2,43</td>
</tr>
<tr>
<td>WOHI</td>
<td>2.9a</td>
<td>3.7ab</td>
<td>5.8b</td>
<td>4.88</td>
<td>0.012</td>
<td>2,43</td>
</tr>
<tr>
<td>A-WOHI</td>
<td>5.0a</td>
<td>3.20ab</td>
<td>1.3b</td>
<td>8.75</td>
<td>0.0007</td>
<td>2,43</td>
</tr>
<tr>
<td>t Value</td>
<td>4.45</td>
<td>3.52</td>
<td>1.35</td>
<td>-----</td>
<td>-----</td>
<td>----</td>
</tr>
<tr>
<td>p values</td>
<td>0.00005</td>
<td>0.0005</td>
<td>0.094</td>
<td>-----</td>
<td>-----</td>
<td>----</td>
</tr>
<tr>
<td>df</td>
<td>1,28</td>
<td>1,28</td>
<td>1,28</td>
<td>-----</td>
<td>-----</td>
<td>----</td>
</tr>
</tbody>
</table>

AOHI = overall habituation index to alcohol
WOHI = overall habituation index to water
A-WOHI = difference between alcohol and water overall habituation indices

Row means with the same letter are not significantly different.
All t-tests are one-tailed.
Figure 5

Overall habituation indices by groups

---

= alcohol

= water
the BDA group (mean = 2.9 trials) habituated significantly faster than the SD group (mean = 5.8 trials). Thus, the length of habituation of the SD group was twice as long as that of the BDA group. There were no significant differences between the BDA and NBDA groups and no differences between the NBDA and SD groups.

Within group analysis of the Overall Habituation Indices comparing the rate of habituation of alcohol to water revealed significant differences for the BDA group ($t (1, 28) = 4.45, p = .00005$) and the NBDA group ($t (1, 28) = 3.52, p = .0005$) but not for the SD group ($t (1, 28) = 1.35, p = .188$). Thus, the alcoholic groups habituated to water significantly faster than they did to alcohol while there was no significant differences in the comparison of the Overall Habituation Index of alcohol to water for the SD group.

Desire ratings. A two-way ANOVA (group X pre-test/post-test desire ratings) between groups was performed on the Likert ratings of the desire to drink alcohol. It was predicted that the post-test ratings would be greater than the pre-test ratings, and the post-test ratings for the BDA and NBDA groups would be significantly greater than the SD ratings. As with the psychophysiological measures, it was expected that the NBDA ratings would fall between the BDA and the SD groups. Because none of the subjects were exposed to alcohol before the experiment, it was expected that the
pre-test Likert ratings of desire for alcohol would not differ significantly between groups; however, it was considered conceivable that the alcoholic groups could score higher than the SD group simply because of their tendency to be preoccupied with alcohol. The results (Table 8 & Figure 6) were generally consistent with these predictions. Specifically, no significant differences were found between groups on the pre-test desire ratings ($F(2, 43) = 1.26, p = .293$). As expected, differences between groups on the post-test desire ratings were found ($F(2, 43) = 3.53, p = .038$). Post hoc analysis revealed that the post-test ratings of the BDA (mean 3.00) and the NBDA (mean 3.20) groups were significantly greater than the SD group (mean 0.87), but the alcoholic groups (BDA & NBDA) were not significantly different from each other.

Within group analysis between pre-test and post-test desire ratings revealed significant differences for the BDA group ($t(1, 28) = 1.95, p = .031$), and the NBDA group ($t(1, 28) = 3.68, p = .005$) but not for the SD group ($t(1, 28) = 0.84, p = .204$). Thus, exposure to alcohol increased desire ratings of both groups of alcoholics, but not for non-alcoholics.

**Estimate ratings.** A one-way ANOVA was performed on the post-test estimates of the number of times alcohol was presented. It was expected that the BDA group would have significantly higher estimates than the SD group, and the
Table 8

**Desire and Estimate Ratings: Means, F and t Values, and P Values Between Groups (rows) and Within Groups (columns)**

<table>
<thead>
<tr>
<th>Measures</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
<th>F</th>
<th>P&gt;F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Desire</td>
<td>1.13a</td>
<td>0.33a</td>
<td>0.33a</td>
<td>1.26</td>
<td>0.293</td>
<td>2,43</td>
</tr>
<tr>
<td>Post-Desire</td>
<td>3.00a</td>
<td>3.20a</td>
<td>0.87b</td>
<td>3.53</td>
<td>0.038</td>
<td>2,43</td>
</tr>
<tr>
<td>Post - Pre</td>
<td>1.87ab</td>
<td>2.87a</td>
<td>0.54b</td>
<td>5.42</td>
<td>0.017</td>
<td>2,43</td>
</tr>
<tr>
<td>t Values</td>
<td>1.95</td>
<td>3.68</td>
<td>0.84</td>
<td>-----</td>
<td>------</td>
<td>-----</td>
</tr>
<tr>
<td>P &gt; t</td>
<td>0.030</td>
<td>0.0005</td>
<td>0.204</td>
<td>-----</td>
<td>------</td>
<td>-----</td>
</tr>
<tr>
<td>df</td>
<td>1.28</td>
<td>1.28</td>
<td>1.28</td>
<td>-----</td>
<td>------</td>
<td>-----</td>
</tr>
</tbody>
</table>

| Stim-Est      | 12.8a  | 14.5a  | 10.7a | 1.66  | 0.202 | 2,44 |

Pre-Desire = pretest Likert desire ratings to have a drink
Post-Desire = posttest Likert desire ratings to have a drink
Stim-Est = subject's estimate of the number of times he received the alcohol stimulus
Post - Pre = the difference between the subject's posttest and pretest Likert desire ratings

Row means with the same letter are not significantly different.
All t-tests are one-tailed.
Figure 6
Mean desire ratings by groups

---

= post-test

= pre-test
NBDA group would fall between these two groups. However, the results (Table 8, p. 55) revealed no significant differences between groups ($F (2, 43) = 1.66, p = .202$).

Discussion

Based on the results, it can be seen that two predictions were confirmed: (a) Analysis of the severity of alcohol abuse/dependence data revealed that the BDA and NBDA scored higher than the SD group (prediction #1, p. 23), and (b) The post-test desire to drink ratings were greater than the pre-test desire ratings for the BDA and the NBDA group, but not for the SD group (prediction #7, p. 24). The pre-test desires were not significantly different between groups. While the post-test desires were greater for the BDA and the NBDA groups compared to the SD, it is not clear why the NBDA group did not fall between the other two groups. In fact, the increase in desire ratings from pre-test to post-test was much greater for the NBDA group (0.33 to 3.20) compared to the BDA group (1.13 to 3.00). In other words, the NBDA group's ratings increased 870 per cent after exposure to alcohol while the BDA group increased only 165 per cent or 5.3 times smaller than the NBDA group's ratings. On the surface, it appears that exposure to alcohol had a much greater effect on the NBDA subjects than it did on the BDA subjects; a finding that is contrary to the hypothesis that brain-damage interacts with the CS (alcohol) to
increase the attention to and desire for the CS. The results are even more baffling, considering that more BDA subjects (5) were in the shortest (1 - 30 days) abstinence category than NBDA subjects (2), and the opposite trend occurred in the longest abstinence category ( > 365 days) which had 6 NBDA subjects and 2 BDA subjects (Table 1, p.28). As noted earlier, the length of sobriety is negatively correlated with craving (Isbell, 1955; Mathew et al., 1979); thus, the BDA group presumably would have experienced the greatest desire based on abstinence because they had the least amount of sobriety. In addition, the severity ratings, even though not statistically significant, of the BDA group were greater than the NBDA group (46.13 vs 35.13 on the MAST, 48.53 vs 40.07 on the SADQ; Table 4, p. 42). As noted above (Engle & Williams, 1972; Labert, 1986), the severity of alcohol dependence is positively correlated with the expectancy effect and craving; thus, once again it would be expected that the BDA group would have experienced greater desire than the NBDA group. These explanations are supported by the findings that the post-test desire ratings were positively correlated with the MAST ($r = 0.47, p = .001, N = 45$) and with the SADQ ($r = 0.35, p = .017, N = 45$). Thus, post-test desire ratings were positively correlated with severity of alcohol dependence, which is consistent with the research cited above.
The discrepancy between these expectations and the results may be in what the subjects experienced compared to what they reported. All of the alcoholic subjects either lived in a facility that required expulsion for any drinking behavior, or they were participants in a recovery program, such as Alcoholics Anonymous. Thus, even though the subjects were guaranteed confidentiality, they may have been reluctant to admit experiencing any undue desire to drink for fear of negative consequences, such as loss of bed and board, or fear of negative evaluation by the experimental team. The idea that the desire rating results were biased by a demand characteristic is creditable considering that the possible range of desire ratings was 0 to 9, and the alcoholic groups' averages (3.00 & 3.20) were only about 40 percent of the possible maximum. It seems reasonable to think that the report of a dieting person's desire for a chocolate bar would be lessened when exposed to the chocolate in the presence of another; in much the same way, it seems reasonable that the alcoholics' reports of their desire ratings were influenced by outside factors. Thus, the results of the desire ratings are questionable as indicators of how much attention was paid to the cue.

Four predictions were not confirmed: (a) Hypothesis number two (p. 23) predicted that the BDA group would take longer to habituate to the alcohol stimulus than the NBDA and the SD groups. However, the results revealed that the
alcohol habituation rate was not different across groups for the SCR habituation rate, the VMRP habituation rate, or for the Overall Habituation Indices; (b) The third hypothesis (p. 23) predicted that the BDA group would produce greater response amplitudes to the alcohol stimulus than the NBDA and SD groups. Instead, the results revealed that the response amplitudes to alcohol were not different across groups as measured by the SCR amplitudes, or as measured by the VMRP amplitudes; (c) The fourth hypothesis (p. 23) predicted that the NBDA group would fall between the other two on their habituation rate and response amplitudes to alcohol. However, the results revealed that the NBDA group did perform between the BDA and SD groups, but the differences were not statistically significant; and finally (d) The eighth hypothesis (p. 24) predicted that the stimulus estimations would be highest for the BDA group followed by the NBDA group and then the SD group. The results revealed no significant differences between the groups.

The data produced mixed results for two of the predictions. The first prediction (#5, p. 23) was supported when the SCR and VMRP data were analyzed and no differences were found between groups in their habituation to water. This also held true for the SCR amplitude to water and the VMRP amplitude to water. However, the failure to find differences between groups on the habituation rate may have
been due to the lack of power (rather than true "no differences" between groups) because the number of subjects in each group was reduced by the non-responders. In fact, analysis using the Overall Habituation Index described above (using data from all 45 subjects) revealed significant differences in the habituation rate to water between groups with the BDA group obtaining a significantly faster rate than the SD group rate. Thus it seems that, given an adequate sample size, the groups differed on the habituation rate to water, which is contrary to the original prediction.

The other prediction (#6, p. 23) that produced mixed results was the expected within group differences between the alcohol and water habituation rates as well as the within group differences between the alcohol and water response amplitudes. On the one hand, the prediction was not confirmed by the alcohol and water response amplitude data from each of the groups. These data revealed no differences between the alcohol and water as measured by the SCR or by the VMRP. However, the prediction was supported by the within group t-tests looking at differences between alcohol and water SCR and VMRP habituation rates as well as the Overall Habituation Index. The results revealed that both alcoholic groups (BDA & NBDA) habituated faster to water than to alcohol, while the SD group showed no significant differences on habituation rate to either stimulus.
Briefly, the major hypothesis can be stated that neuropsychological deficits interfere with the habituation process, at least as it's related to the smell of alcohol, and this serves to increase the attention paid to the stimulus. This results in increased desire for the stimulus, and this may lead to an increased risk for relapse.

Based on the accuracy of the predictions, the major hypothesis was not supported. However, in the original hypothesis, it was assumed that increased attention to a stimulus would be exhibited by an increase in the number and the intensity of responses to the alcohol stimulus. Alternatively, increased attention to a stimulus can be conceptualized as decreased responding to competing stimuli in comparison to responses to the target stimulus. This is exemplified by the significant Water-Overall Habituation Index (WOHI) effect found in the Overall Habituation Indices shown in Table 7 (p. 51). Inspection of Table 7 and Figure 5 (p. 52) reveals that the BDA group and, to a lesser extent, the NBDA group responded significantly less to the competing water stimulus than they did to alcohol (BDA: 2.9 vs 7.9; NBDA: 3.7 vs 6.9 responses). Specifically, the BDA group habituated 2.7 times faster to water than they did to alcohol, while the NBDA group habituated 1.9 times faster to water compared to alcohol.

While these findings are intriguing, it is possible that the group differences in the water habituation rates
may be explained by the process of dishabituation. Dishabituation is the cancellation or removal of habituation. This often occurs when a different stimulus from the original habituation stimulus is presented, resulting in restoration of the habituated response. It is generally assumed that response restoration indicates the presence of dishabituation. Thus, if the response to water was restored via dishabituation, the habituation rate of the SD may have been artificially increased.

As cited in Graham (1973), Zimny and associates (Zimny & Kienstra, 1967; Zimny & Schwabe, 1966) have concluded that dishabituation occurs to a greater extent as the difference between the dishabituating stimulus and the habituating stimulus becomes greater. Thus, in this study the first three stimulus presentations are water and the alcohol stimulus could act as a dishabituating stimulus on the water habituation process. From Zimny and associates' findings, the greater the difference between the subject's perception of the two stimuli, the greater will be the dishabituation. So if a subject perceives a greater difference between alcohol and water than a second subject does, then dishabituation will occur with the first subject to a greater extent than with the second subject.

In order to test if the dishabituation process explains the results of this study better than the effect of brain-damage does, it is necessary to determine whether the
alcohol acted as a dishabituating stimulus differentially across groups. It can be concluded from the findings of Zimny and Kienstra (1967) that the greater the restoration of the response to water after it has habituated, the greater the subject perceives the difference between the stimulus properties of water and alcohol. This begs the question: Which group (BDA, NBDA, SD) of subjects would be expected to perceive the greatest difference between alcohol and water? It seems reasonable to think that the alcoholic subjects would perceive the greatest difference between the two stimuli primarily because of their extensive history of using alcohol. Thus, they would dishabituate to water to a greater extent than the non-alcoholics, resulting in a restoration of the response to water; this would slow the habituation process to water. But, in fact, both alcoholic groups' habituation to water was faster than the non-alcoholics' habituation, which is contrary to empirical studies of dishabituation.

Therefore, in order to explain the results found in this study (namely, the group differences in habituation rate to water) according to a dishabituation process, it follows that the "true" habituation to water had to have been the same for all three groups as made in the original proposal. However, the results revealed a longer habituation rate for the SD group, which had to have been artificially lengthened because the SD group experienced a dishabituating
effect produced by the alcohol stimulus. In order for this to occur, the SD subjects would have had to experience a greater difference between the stimuli than the other groups did. Thus, the SD subjects would have perceived the relationship between alcohol and water much more differently than how the alcoholics' perceived the difference. Such an expectation seems counterintuitive considering the extensive familiarity alcoholics have with alcohol as a stimulus. Increased familiarity with a stimulus suggests that an individual would be able to detect the familiar stimulus more readily and identify deviations from it much more readily than individuals less familiar with the stimulus. Thus, it does not seem reasonable to expect the SD group to be able to detect a greater difference between the two stimuli than the alcoholic groups could.

However, this notion as it applies to this study assumes that the subjects can detect the difference in smell between alcohol and water, but in fact water has no smell. In this experiment the properties of both stimuli consisted of tactile stimulation resulting from the atomization of the liquid, while only the alcohol added a smell stimulus. It is reasonable to think that none of the subjects had any extensive tactile experience with an atomized liquid -- be it water, alcohol or any other liquid; thus, the tactile stimulation from each stimulus condition would be constant for all subjects. Subjects would not perceive any difference
between the tactile stimulation component of water and from alcohol; thus, the tactile component of each type of stimulus would not interfere with the habituation process via dishabituation.

On the other hand, the smell of alcohol is quite different from the tactile stimulation of atomized water, and this difference could conceivably dishabituate the habituation process to water. Assuming, for a moment, that these processes are influencing the obtained results (either habituation to water is reduced for the BDA group or the habituation to water is increased for the SD group) then one of two possible mechanisms is affecting the results. First, if the BDA group truly habituated more rapidly to water compared to the other groups, then dishabituation must not have taken place. In order for this condition to occur, the alcoholics must have perceived less of a difference between the smell of alcohol and the tactile stimulation of atomized water than the SD subjects did resulting in no dishabituation. On the other hand, both groups could have perceived similar differences between the stimuli, which suggests that brain-damage interfered with the habituation process. The second possibility to explain the results from a dishabituation perspective could occur if the SD subjects' habituation is artificially prolonged (compared to the other groups) by dishabituation. In order for this to occur, the SD subjects' must have experienced a greater difference
between the smell of alcohol and the tactile sensation of atomized water compared to the alcoholic subjects.

Upon reflection, it can be seen that these two explanations are really the same. Saying that alcoholics perceived less difference between the stimuli is the same as saying that the non-alcoholics perceived more difference between the stimuli. As a result, dishabituation would not interfere with the habituation process of alcoholics resulting in faster habituation, but it would for the non-alcoholics resulting in slower habituation. In order to accept this logic, one must assume that each group of subjects perceived the difference between atomized water and the smell of atomized alcohol differently and in a direction that is counterintuitive considering the extensive familiarity that alcoholics have with alcohol. While such an assumption may be testable, the notion that such differences would exist based on group membership seems highly implausible.

Nevertheless to test this, two statistical analyses were performed. In the first, group comparisons of a Dishabituation Index were performed. Dishabituation was operationalized as the restoration of two responses to water after failing to respond (i.e., habituation) to water for three consecutive water stimulus presentations. One-way ANOVA of the Dishabituation Index to water revealed no differences between groups for the SCR data ($F (2, 30) = \ldots$
0.90, \( p = .416 \) or for the VMRP data (\( F(2, 38) = 1.06, p = .356 \)). The second analysis consisted of group comparison of the first response to water after the alcohol stimulus was presented (i.e., comparisons of the 6th response in the stimulus presentation schedule). If dishabituation accounted for the prolonged habituation for the SD group, then it would be expected that the first post-alcohol water response would be greater for the SD group than the other groups, especially the BDA group. However, one-way ANOVA revealed no significant differences between groups for either the skin conductance data (\( F(2, 30) = 0.222, p = .802 \)) or the vasomotor response data (\( F(2, 38) = 0.639, p = .534 \)).

As mentioned above, the obtained group differences in the water habituation rates could have resulted from a dishabituation process. However, based on the discussion of the process of dishabituation and the statistical analyses, it is not reasonable to think that the group differences in the water habituation rates is due to dishabituation. A more parsimonious explanation would be that the BDA group, compared to the other groups, attended differently to the two stimuli.

As noted above, attending more to one stimulus compared to another can be conceptualized as increased attention to the former at the expense of decreased attention to the latter. However, relatively increased attention to one stimulus by a reduction of attention to a competing stimulus
does not necessarily mean that the habituation process is impaired, or that some kind of interference has taken place. Indeed, the habituation process of the BDA group, at least in this experiment, is the same as it is for the nonbrain-damaged alcoholics and non-alcoholics alike. This is further supported by the additional finding that the Impairment Index was not significantly correlated with any of the psychophysiological responses to alcohol as shown in Table 9. Thus, in responding to alcohol, the habituation process of each group is independent of the individual measures of brain-damage, and habituation to alcohol is independent of brain-damage itself if the measures are considered to be valid indicators of brain-damage.

On the other hand, the groups habituated differentially to water as indicated by the findings that the BDA group habituated significantly faster to water than the SD group did, and this difference does not result from dishabituation. This is further supported by the significant finding that the Impairment Index was negatively correlated with the VMRP water habituation rate ($r = -0.33, p = .037, N = 41$) and with the Overall Water Habituation Index ($r = -0.38, p = .009, N = 45$). It is not clear why a significant relationship was not found between the SCR water habituation rate and the Impairment Index, but the total number of subjects who responded with SCR's was only 33 which may have reduced the likelihood of finding a significant correlation.
Table 9  

Correlation of the Impairment Index (II) with Psychophysiologial Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Correlation Coefficient</th>
<th>Significance</th>
<th>Sample Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASNH</td>
<td>( r = 0.06 )</td>
<td>( p = 0.79 ), ( N = 33 )</td>
<td></td>
</tr>
<tr>
<td>ASMH</td>
<td>( r = -0.06 )</td>
<td>( p = 0.75 ), ( N = 33 )</td>
<td></td>
</tr>
<tr>
<td>AVNH</td>
<td>( r = 0.11 )</td>
<td>( p = 0.51 ), ( N = 41 )</td>
<td></td>
</tr>
<tr>
<td>AVMH</td>
<td>( r = 0.06 )</td>
<td>( p = 0.69 ), ( N = 41 )</td>
<td></td>
</tr>
<tr>
<td>WSNH</td>
<td>( r = -0.20 )</td>
<td>( p = 0.25 ), ( N = 33 )</td>
<td></td>
</tr>
<tr>
<td>WSMH</td>
<td>( r = -0.22 )</td>
<td>( p = 0.22 ), ( N = 33 )</td>
<td></td>
</tr>
<tr>
<td>WVNH</td>
<td>( r = -0.33 )</td>
<td>( p = 0.04 ), ( N = 41 ) significant</td>
<td></td>
</tr>
<tr>
<td>WVMH</td>
<td>( r = 0.05 )</td>
<td>( p = 0.74 ), ( N = 41 )</td>
<td></td>
</tr>
<tr>
<td>AOHI</td>
<td>( r = 0.21 )</td>
<td>( p = 0.16 ), ( N = 45 )</td>
<td></td>
</tr>
<tr>
<td>WOHI</td>
<td>( r = -0.38 )</td>
<td>( p = 0.01 ), ( N = 45 ) significant</td>
<td></td>
</tr>
</tbody>
</table>

ASNH = mean number of trials for SCR to habituate to alcohol  
WSNH = mean number of trials for SCR to habituate to water  
ASMH = mean amplitude of SCR trials to habituate to alcohol  
WSMH = mean amplitude of SCR trials to habituate to water  
AVNH = mean number of trials for VMRP to habituate to alcohol  
WVNH = mean number of trials for VMRP to habituate to water  
AVMH = mean amplitude of VMRP trials to habituate to alcohol  
WVMH = mean amplitude of VMRP trials to habituate to water  
AOHI = overall habituation index to alcohol  
WOHI = overall habituation index to water
Thus, the significant relationship between the water habituation indices and the Impairment Index indicates that the more severe the brain-damage, the faster the subject habituated to the water stimulus. Considering that the first 3 stimulus trials were water and the mean trials to habituation to water for the BDA group was 2.9 (Table 7, p. 51), it can be seen that on the average, the BDA group essentially stopped responding to water once the alcohol was presented.

Thus, it seems reasonable to conclude that the presence of neuropsychological deficits, at least in this sample of alcoholics, is differentially associated with how the groups responded to the two types of stimuli. This suggests that brain-damage may not interfere with the habituation process to an individual stimulus; but in the presence of a competing stimulus, brain-damage interferes with the subject’s capacity to respond to the irrelevant, competing stimulus.

This suggests that the BDA subjects exhibited some sort of selective attentional process for alcohol that excluded the attention paid to the competing stimulus. Such an explanation is consistent with the neuropsychological problems that are frequently found among alcoholics and which were cited above; namely, they exhibit perseveration, deficits in the ability to integrate information, deficits in using feedback from incorrect results, and failure to
shift-strategies when solving problems (Parsons, 1987; Ryan & Butters, 1986).

Such an explanation can fit the data obtained from the habituation rates; however, it is not clear why the same pattern of results was not found for the response amplitude measures. As mentioned above the first three stimulus trials for every subject were water, so it is reasonable to expect that the first response may have consisted of the response to the stimulus plus an anticipatory response to the procedure and the laboratory setting. This may have resulted in an exaggerated response which could have been large enough to artificially elevate the mean of the responses to that type (water) of stimulus. Therefore, the data were reanalyzed after parceling out the first response by refiguring the mean of the water trials to habituation for the SCR and VMRP data without the first response. The revised SCR results revealed that the mean amplitude of the responses to water decreased from 0.171 to 0.118 for the BDA group, from 0.317 to 0.235 for the NBDA, and from 0.367 to 0.347 for the SD group.

As shown in Table 10 and illustrated in Figure 7, a one-way ANOVA of the revised water SCR data was not statistically significant ($F(2, 31) = 2.38, p = .110$). The SCR within group differences between the alcohol and water responses were not different for any of the groups. Thus, the revised SCR analysis did not reveal any new information.
Table 10

Revised SCR Amplitudes Related to Habituation: Means, F and t Values, and P Values Between Groups (rows) and Within Groups (columns)

<table>
<thead>
<tr>
<th>Measures</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
<th>F</th>
<th>P&gt;F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASMH</td>
<td>0.267a</td>
<td>0.377a</td>
<td>0.397a</td>
<td>0.57</td>
<td>0.574</td>
<td>2.31</td>
</tr>
<tr>
<td>RWSMH</td>
<td>0.118a</td>
<td>0.235a</td>
<td>0.347a</td>
<td>2.38</td>
<td>0.110</td>
<td>2.31</td>
</tr>
<tr>
<td>A-RWMH</td>
<td>0.149</td>
<td>0.142</td>
<td>0.050</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>t Value</td>
<td>1.228</td>
<td>1.278</td>
<td>0.479</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>p values</td>
<td>0.118</td>
<td>0.106</td>
<td>0.318</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>df</td>
<td>1,16</td>
<td>1,24</td>
<td>1,20</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

ASMH = mean amplitude of SCR trials to habituate to alcohol
RWSMH = revised mean amplitude of SCR trials to habituate to water
A-RWNH = difference between ASMH and RWSMH

Row means with the same letter are not significantly different.
All t-tests are one-tailed.
Figure 7
Revised SCR mean amplitude of trials to habituate to alcohol and water by groups

--- = alcohol
---------- = water
Analysis of the revised VMRP amplitude to water was also performed. More subjects responded in the VMRP channel SCR, and it would thus be a more powerful analysis. For the BDA group, the mean response decreased from 0.258 to 0.162, from 0.213 to 0.163 for the NBDA, and from 0.245 to 0.210 for the SD group (Tables 6 & 11). There were no significant differences between groups in the revised VMRP water amplitude data ($F(2, 40) = 0.47, p = .628$) as shown in Table 11 and illustrated in Figure 8. However, the within group comparisons between the types of stimuli revealed significant differences for all of the groups using a one-tailed $t$ test and an alpha level of 0.05 as shown in Table 11 (BDA: $t(1, 28) = 1.78, p = .043$; NBDA: $t(1, 24) = 2.02, p = .028$; SD: $t(1, 26) = 1.90, p = .034$). Thus, each group responded more intensely to the alcohol than to the water as measured by the revised VMRP.

While the significant within group differences between the two stimuli found in the revised VMRP analyses support the idea that the no differences found for the revised SCR data was due to lack of power, the revised VMRP within group findings do not exactly parallel the pattern of findings found with the habituation rate data. That data revealed differences for the BDA and the NBDA groups but not for the SD group. On the surface, these findings appear contradictory; however, considering that every group responded more intensely to the alcohol stimulus as measured
### Table 11

**Revised VMRP Amplitudes Related to Habituation: Means, F and t Values, and P Values Between Groups (rows) and Within Groups (columns)**

<table>
<thead>
<tr>
<th>Measures</th>
<th>BDA</th>
<th>NBDA</th>
<th>SD</th>
<th>F</th>
<th>P&gt;F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVMH</td>
<td>0.272a</td>
<td>0.245a</td>
<td>0.286a</td>
<td>0.60</td>
<td>0.551</td>
<td>2,40</td>
</tr>
<tr>
<td>RWVMH</td>
<td>0.162a</td>
<td>0.163a</td>
<td>0.210a</td>
<td>0.47</td>
<td>0.628</td>
<td>2,40</td>
</tr>
<tr>
<td>A-RWVMH</td>
<td>0.110a</td>
<td>0.082a</td>
<td>0.076a</td>
<td>----</td>
<td>------</td>
<td>----</td>
</tr>
</tbody>
</table>

| t Value    | 1.78  | 2.02  | 1.90 | ---- | ------| ----|
| p values   | 0.042 | 0.028 | 0.034| ---- | ------| ----|
| df         | 1,28  | 1,24  | 1,26 | ---- | ------| ----|

AVMH = mean amplitude of VMRP trials to habituate to alcohol
RWVMH = revised mean amplitude of VMRP trials to habituate to water
A-RWVMH = difference between AVMH and RWVMH

Row means with the same letter are not significantly different.

All t-tests are one-tailed.
Figure 8
Revised VMRP mean amplitudes to habituate to alcohol and water by groups

--- = alcohol
---------- = water
by the revised VMRP, it seems reasonable to think that all
of the subjects exhibited greater arousal to alcohol than to
water. Such a result would not necessarily contradict the
interpretation that the BDA exhibited selective attention to
alcohol versus water.

In order to have more confidence in the suggestion that
brain-damage interfered with the BDA subjects' capacity to
respond/attend to the irrelevant, non-alcohol stimuli, it
would have been more powerful if the NBDA group had not
responded to water significantly differently than they did
to alcohol. Such an expectation would be logical if brain-
damage were an all-or-none phenomena instead of a continuum.
Comparisons of the brain-damage screening measures (Table 3.
p. 40) revealed that the NBDA group's performance was not
statistically different than the SD group's on the Block-
Design, Object Assembly, Trails B, and Short Category Test.
However, even though the differences were not statistically
significant, the NBDA group's performance was more impaired
than the SD group on all of the tests except the Short-
Category Test. Furthermore, the NBDA group's performance was
significantly more impaired than the SD group on the Digit
Symbol subtest. Among other things, this test measures the
ability to sustain attention, and it does so more than any
of the other tests used. Thus, the impairments could be
characterized as severe for the BDA, mild-moderate for the
NBDA, and none for the SD group. Therefore, it is suggested
that these impairments had progressively more effect on responding/attending to the neutral stimulus as they became more severe.

According to Posner and Rafal (1987), there is no standard definition of attention, but experts generally agree that there are three "senses" of the term: (a) Alertness or generalized arousal of the physical and mental state, (b) Selective attention which involves the selection of specific information from the environment or from internal stimuli for conscious processing, and (c) Vigilance or sustained concentration to a stimulus. Selective attention appears to be the most likely sense affected in the BDA group.

Bernstein and associates, (1973, 1979; Bernstein, Taylor, & Weinstein, 1975) and Maltzman (1979) have suggested that the orienting response is related to selective attention, because it can be elicited either by a novel stimulus or when one encounters an important stimulus. As the individual habituates to the stimulus, attention is no longer focused on it, and the individual is free to attend to a different stimulus.

Posner and Rafal (1987) conceptualize the orienting process as several components consisting of phasic arousal with physiological changes, and this is followed by the selective component. The selective component consists of overt and covert responses. Overt responses consist of
movement towards a stimulus, such as turning towards the stimulus. The covert responses are quite complex, characterized by mental shifting to the stimulus. In order to select a stimulus, the individual must disengage from one stimulus, move or shift attention to a different stimulus, and then engage the new stimulus.

According to the experimental paradigm of this study, it can be seen that the subjects were required to go through this process repeatedly as they were exposed to the different stimuli. It can be appreciated that the subjects in the SD group were relatively more efficient than the NBDA group at shifting attention from one stimulus to the other. The BDA subjects were the least able to shift from one stimulus to the other. The findings suggest that all of the groups were able to engage and shift between the stimuli initially; however, the BDA subjects, and to a lesser extent the NBDA subjects, soon found themselves less able to disengage from the alcohol stimulus and shift their attention back to the water. On the other hand, the SD subjects shifted back and forth with relative ease.

Most of the work on attentional shifting has been done with visual-spatial tasks, and the results have indicated that the impaired ability to disengage from a stimulus is due to some defect(s) in the parietal lobes (Posner & Rafal, 1987). While there is no evidence to support the notion that the disengagement mechanism is located in the parietal lobes
for other sensory modalities, Posner and Rafal have noted that such an idea is appealing and may explain the illogical thought processes found in many parietal lobe patients. These patients have difficulty disengaging from one idea and shifting to another. And, as mentioned above, this is a finding frequently found in alcoholics.

The entire process may be likened to visual-spatial neglect where the individual does not attend to certain aspects of the visual field. Thus, the effect on the BDA group can be conceptualized as a perceptual inattention or neglect to novel or less familiar stimuli when presented with the highly familiar stimulus of alcohol. This is consistent with Heilman and Watson's (1977) theory of neglect. They have proposed that neglect is "manifested by a defect in orienting to stimuli (and) the defect results from disruption of a system whose function is to 'arouse' the individual when new sensory stimulation is present". Similarly, the BDA subjects may not have been sufficiently "aroused" by the sensory information of the water stimulus to "capture" the individual's attention once the alcohol stimulus was present to compete for the individual's attention.

As mentioned above, disengaging from a stimulus appears to be a function of the parietal lobes, and the parietal lobes project sensory input to the frontal lobes, which does not receive any direct sensory input. Filskov, Grimm, and
Lewis (1981) have noted that prefrontal damage disrupts the regulation of attention so that the individual appears to lose interest in a task resulting in a decreased rate of behavior or lessened spontaneity. The lessened spontaneity may result from increased focus to some relevant stimulus (such as alcohol) and when an irrelevant, non-alcohol stimulus is presented to a brain-damaged alcoholic, the individual quickly habituates to it. As a result, the individual's attention is not diverted from the alcohol cue.

In addition, individuals with frontal lobe lesions have deficits in associative learning tasks, and this suggests that the individual's ability to use external cues to guide behavior is impaired; thus, behavior appears inflexible and disordered (Kolb & Whishaw, 1985). Thus, if attention is focused on alcohol cues at the expense of attending to non-alcohol stimuli, behavior may be guided by the alcohol cues, resulting in relapse. Moreover, the deficits in learning new associations combined with the inability to attend to competing, non-alcohol stimuli when in the presence of alcohol stimuli suggest that the brain-damaged alcoholic have difficulty learning non-drinking behavior.

The results obtained in this study suggest a modification of the original hypothesized model depicted in Figure 1 (p. 21). In the revised model (Figure 9), it is hypothesized that brain-damage affects the attentional process when the individual is faced with multiple cues.
Revised model of the relationships: Cues, B-D, attention, and relapse

Figure 9

- F-D: ability to integrate information
- B-D: perceptual organizational ability

Alcohol Cue -> Processing
- Attention to Alcohol Cue
- Failure to shift attention
- Inattention to neutral cue

Preoccupation with Alcohol Cue
- >> Craving
- >> Motivation to Drink

Choice: Drink or not Drink -> Probability of Relapse

P-D
- Pz analyzing & structuring cues
- Pz processing info. in ambiguous situations
- Stress leads to F-D

P-O: alcohol is wonderful

F-D
- F-D: alcohol dependence
- P-O: positive outcome expectations
- P-D: problem
- > more increased/greater

b-D: decreased/small
> more increased/greater
Namely, the results indicate that the brain-damaged individual's ability to shift attention from the alcohol cue to the irrelevant, non-alcohol cue is impaired. As mentioned above, irrelevant stimuli are those stimuli that do not serve as CSs which this study proposes are essential variables that influence the risk of relapse. This results in increased processing of, and sustained attention to, the alcohol cue. This interacts with the positive outcome expectancies at the choice point (drink or not drink) to increase the probability that the individual will drink or relapse. In contrast, the SD group was more able to shift attention back and forth between the stimuli suggesting that the non-alcoholic controls were more able to select from a wider range of stimuli from which to process and attend, and thereby guide their behavior. Thus, it can be appreciated that their choices would be less limiting than the choices of the BDA group.

As mentioned, the interaction of brain-damage and positive outcome expectancies would increase the probability of relapse. This would increase the severity of the individual's brain-damage, which would affect how information is processed in the future. It seems reasonable that under controlled laboratory conditions a brain-damaged alcoholic would stop responding to an unvarying cue; however, outside the laboratory the individual is literally bombarded with salient cues that vary (e.g., in exposure
time or intensity), and such cues are more resistant to habituation and/or inattention. Thus, it can be appreciated that when subjected to a virtual unlimited supply of cues, the individual who experiences selective attention, by blocking out competing stimuli or by failing to shift attention, would be at great risk to maintain attention to alcohol cues leading to an increased probability of interacting with that cue (i.e., relapse).

A more direct test of this impairment in attention appears appropriate. Such a test might instruct the subjects to attend to a non-alcohol, aromatic stimulus (e.g., count how many times the stimulus is presented) while being exposed to the neutral stimulus and an alcohol cue. The dependent measures would be used to determine which group, if any, was distracted more by the alcohol stimulus. The hypothesis would be that a BDA group's attention would be drawn to the alcohol stimulus more than a NBDA or SD group, and the BDA group's performance of keeping track of the number of times the non-alcohol stimulus was presented would be less accurate.

In addition, the findings of the present study raise other issues which need to be addressed. The first of these involves the generalizability of the results to other subjects. Obviously a replication of the study using procedural refinements, such as prescreening subjects to include only those who responded to the psychophysiological
measures of interest would enhance the generalizability of these findings, especially if it included groups of subjects of different races and gender. A second question needing to be addressed concerns whether the results found in this study generalize to other alcohol related cues, such as handling old drinking paraphernalia or seeing old haunts. It is conceivable that because this stimulus tapped the sense of smell, other stimuli that tap other sensory modalities, such as touch and sight, may not produce the same results. Given that some interesting results were obtained, it is reasonable to suggest that further research be undertaken with different alcohol related cues to see if the brain-damaged alcoholic's failure to shift attention from one stimulus to another is applicable with other sensory modalities.

Finally, the importance of this study can be appreciated by considering its effect as a guide to future research, and its implications for the clinical aspects of alcoholism. As mentioned previously, there have been only a few studies that have investigated the psychophysiological responding of alcoholics and/or brain-damaged individuals (e.g., Callan, Holloway, & Bruhn, 1972; Davidoff & McDonald, 1964; Hattangai, 1969; Holloway & Parsons, 1971; Lovallo, Parsons, & Holloway, 1973; Oscar-Berman & Gade, 1979; Parsons & Chandler, 1969; Parsons, Messenger, & Holloway,
1973), resulting in contradictory findings. In contrast to those studies, the paradigm of using a relevant target stimulus (smell of alcohol) instead of an irrelevant target stimulus (auditory tone) suggests an important new experimental tool to study such relationships. Using such a tool would increase the confidence in, and the generalizability of the findings and the conclusions.

In addition, it was noted that a significant number of alcoholics develop subtle neuropsychological deficits. Except by standardized testing, these deficits are difficult to detect, and the results of this study indicate that such deficits have serious implications for the assessment and treatment of alcoholics. Specifically, brain-damaged alcoholics should be identified and, where necessary, trained in techniques that enable the individual to consider other aspects of a stimulus besides their initial reaction to it in much the same way that cognitive rehabilitation of attention is done with brain injured patients (e.g., Ben-Yishay, Piasetsky, & Rattok, 1987; Sohlberg & Mateer, 1989).
References


component of the orienting response in brain-damaged patients. *Physiological Psychology*, 1, 37-40


mechanism subserving selective attention. 
*Psychophysiology, 14,* 228-236.

*Booklet Format.* Los Angeles: Western Psychological Services


Appendix 1

Background and Demographic Form

Group assignment/subject no. ________

1. Age/sex/race
2. Educational level
3. Trail Making - B
4. WAIS-R Block Design
5. WAIS-R Object Assembly
6. WAIS-R Digit Symbol
7. SCT (r.s./T-score/¢tile)
8. WAIS-R Vocabulary Subtest scaled score
9. WAIS-R Digits Forward score
10. Number of days abstinent
11. Preferred alcoholic beverage
12. Screening for dysosmia
   a. perfume  Yes  No
   b. aftershave  Yes  No
   c. vinegar  Yes  No
   d. isopropyl alcohol  Yes  No
13. Negative history of polysubstance abuse
    (< 10% of drugs other than alcohol)  Yes  No
14. Free of prescription medication  Yes  No
15. Smoking history  Yes  No
16. Negative history of schizophrenia  Yes  No
17. NPO two hours pre-experiment  Yes  No
18. Negative head injury history  Yes  No
19. Unremarkable medical history  Yes  No
20. Adequate nutrition  Yes  No

MAST _______  SADQ _______

Desire for Alcohol:  Pre-test ______  Post-test _____

Post-test stimulus ratings:
   No. of Presentations ______
Appendix 2

Instructions to Subjects Form

Thank you for participating in our study. We are trying to see if alcoholics respond differently than non-alcoholics to different things, such as smells and sounds. In order to do this we will hook you up to some recording equipment similar to an elaborate EKG machine. This equipment will measure some of your body's responses to smells and sounds that we will present to you. We'll be measuring such things as heart rate and the activity of your sweat glands. The entire procedure will take about 25 minutes.

We are going to have you wear this air mask (demonstrate) which is attached to an air supply, so you'll feel a constant flow of air through the mask. It may feel strange at first, but just breathe normally and you'll get used to it in no time. In addition, these two nebulizers (point to them) are attached to the mask. These allow us to atomize the liquids inside of them, so that you can smell them. We will be giving you brief smells of each of them in a random order. It is not necessary for you to inhale deeply in order to identify the smell; just breathe normally. At the end of the experiment we'll ask you some questions about the smells.

It's important that you know that the smells are not a threat to your health. I assure you that nothing will be done to you that will harm you, but sometimes some people get a little nervous when they wear an air mask; we will be seated in the next room, and we will make sure that no harm comes to you. If you do feel extremely nervous, you may ask to stop the procedure, and we will remove the air mask.

We want you to relax and to remain as still as possible. Please try not to talk or try to identify the stimulus while the procedure is in progress because that would change the recordings.

Each stimulus will be presented separately, and they will be presented for different lengths of time and in different orders so pay attention. You may not be able to detect all of the stimuli because some of them will be very brief and faint; therefore, don't get frustrated if you experience some gaps between stimuli.
Appendix 3

Consent Form

I, ___________________________, agree to volunteer for this study which will measure my physiological responses to different smells. I understand that none of the procedures, including the substances used to generate the smells, will harm me. Nevertheless, one of the smells might be alcohol, so I may experience some discomfort related to that, but not enough of the substance will be used to change my sobriety or functioning.

I further understand that this study is important from a scientific standpoint, and I will make every effort to complete it. However, I understand that I will be allowed to terminate the procedure at anytime without any adverse consequences.

Furthermore, I understand that none of the information gathered from me will be shared with anyone except those directly involved in the research or to those individuals to whom I may authorize such information.

Upon completion of the project, I understand that I will be given a full explanation of the research, and any of my questions will be answered.

By my signature, I agree that I have read and understand the conditions set forth above; I also agree that if I was picked up by the research team, I will remain with them until they deliver me back to the place from which I was picked up. By my signature, I also indicate that I am not a minor.

Volunteer ___________________________

Witness ___________________________

Caregiver ___________________________
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VITA

Fredric D. Klug was born and brought up in Green Bay, Wisconsin where he lived until he was 23. He graduated from East High School in Green Bay and attended the University of Wisconsin. After a tour of duty in the U.S. Army, he moved to Baton Rouge, Louisiana and enrolled at Louisiana State University where he earned Bachelor of General Studies and Master of Arts degrees.

Mr. Klug is married to the former Brenda Lee Landry of Erwinville, Louisiana, and they have two children - Arlis aged 19 and Thomas aged 16.

Prior to entering Graduate School at Louisiana State University, Mr. Klug was in the insurance business. He owned and operated his own independent agency for 12 years. Mr. Klug also worked in the field of medical technology for approximately 8 years. Thus, earning a doctorate in clinical psychology represents his third career.

Mr. Klug has availed himself of a variety of clinical experiences while earning his Ph.D., including forensic, substance abuse, outpatient, and inpatient work. He completed his predoctoral internship training at Broughton Hospital in Morganton, North Carolina.
Candidate: Fredric Dale Klug

Major Field: Psychology

Title of Dissertation: A Comparison of the Physiological Responses of Brain-Damaged Alcoholics, Non Brain-Damaged Alcoholics, and Social Drinkers to the Smell of Alcohol

Approved

[Signature]
Major Professor and Chairman

[Signature]
Dean of the Graduate School

EXAMINING COMMITTEE

[Signature]
[Signature]
[Signature]

Date of Examination: June 20, 1991