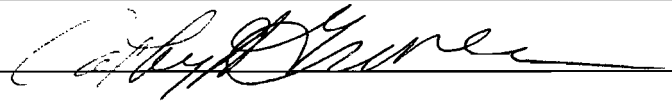
AN ABSTRACT OF THE THESIS OF

Robert L. Buford III for the Master of Science

in Psychology presented on November 7, 2003

Title: An Examination of the Effects of Chronic Alcohol Exposure on Superstitious Behavior in the Rat

Abstract approved: 

Past research has generally been supportive of the case for superstitious behavior in a laboratory setting. Experiments have been done with pigeons, rats, monkeys, and humans. The majority of the research suggests that superstitious behavior occurs when the subject experiences the illusion of control over circumstances such as reinforcement or termination of an aversive stimuli. In the case of the rat, research has demonstrated that the subject will perform a variety of behaviors prior to receiving a food reinforcement because those behaviors have been inadvertently or adventitiously reinforced. The animal behaves as if it is the behaviors that are precipitating the delivery of the reinforcement when in fact the reinforcement is not contingent on any particular behavior. Research has also supported the belief that chronic exposure to alcohol results in numerous neurological impairments and cognitive deficits, including the inability to integrate information that permits the elimination of superfluous operant behavior. The present study investigated whether chronic alcohol exposure had any effect on superstitious behavior in the laboratory rat. This study showed that there was a marginally significant increase in superstitious responding between pre and post alcohol animals on the fixed-time schedule relative to control subjects, but not for the alcohol treated rats that did not receive food reinforcement.

AN EXAMINATION OF THE EFFECTS OF CHRONIC ALCOHOL EXPOSURE ON SUPERSTITIOUS BEHAVIOR IN THE RAT

A Thesis

Presented to

The Department of Psychology and Special Education

EMPORIA STATE UNIVERSITY

In Partial Fulfillment

of the Requirements for the Degree

Master of Science

---

by

Robert L. Buford III

December 2003

Approved for the Department of
Psychology and Special Education

Approved for the Graduate Council

ACKNOWLEDGEMENTS

Sincere appreciation to Dr. Cathy Grover for reviewing my numerous drafts of this thesis and for continually challenging me to improve my research abilities. Thanks to Dr. Cooper Holmes and Dr. Michael Leftwich for having the patience to be on my committee and for offering advice that made this study more significant. Thanks also to the undergraduate students who agreed to donate their time to review hours of videotape. Lastly, sincere thanks to my family for supporting and encouraging my vision of academic achievement.

TABLE OF CONTENTS

LIST OF FIGURES

FIGURE

CHAPTER 1

INTRODUCTION

In 1948, B.F. Skinner coined the term "superstitious behavior" to account for idiosyncratic, stereotyped behavior that pigeons exhibited before, during, and after a fixed interval reinforcement of food (Davis & Hubbard, 1972). The pigeons would turn in circles, bob their heads, and make excursions to and from the food hopper between the food deliveries. There have been similar studies done on laboratory rats, which have yielded similar results with the animals behaving in idiosyncratic, stereotyped ways. There has yet to surface in the literature a study of the effects of chronic alcohol exposure on such superstitious behavior.

Human beings often exhibit superstitious behaviors. The gambler blows on his dice before throwing at the table or a bowler who stands on one foot and moves his hand as if he is able to control the path of the ball down the lane after he has already let go (Davis & Hubbard, 1972). The free throw shooter in a basketball game frequently performs a number of idiosyncratic, stereotyped behaviors during the 10 sec. before having to shoot. (Lobmeyer & Wasserman, 1986). These behaviors obviously do not control the outcome of the roll of the dice, the accuracy of the free throw, or the strike of the bowling ball, but all are believed to become stereotyped because of chance pairings with the desirable (reinforced) outcomes (Davis & Hubbard).

The purpose of this study was to determine if chronic alcohol exposure increases superstitious behavior in laboratory rats that have been placed on a fixed time schedule of food reinforcement. A superstition account of uncontrollability would have predicted that the rats would not learn that outcomes were response-independent, and that

superstitious responses would become conditioned during exposure to non-contingent reinforcement. It was hypothesized that chronic alcohol exposure rats would exhibit more repetitive, idiosyncratic behavior than control rats, but they did not. It was also hypothesized that in a repeated measures design they would exhibit more of these types of behaviors after being chronically exposed to alcohol, than during baseline testing.

A study of the effects of chronic alcohol exposure on superstitious behavior in the rat may be far from generalizing reliably to human beings but may also shed some light on the different ways that people behave when under the influence of alcohol, or those who have been using alcohol at a chronic level. Perhaps this type of research could aid the understanding of many of the psychological problems that accompany chronic alcohol use.

Review of the Literature

Defining Superstitious Behavior

Operant conditioning is based on the fundamental principle that the occurrence of a reinforcer enhances the frequency of behavioral responses that the reinforcer is contiguous with. Any behavioral response that immediately precedes a reinforcer will become more probable even though the delivery of the reinforcer does not depend on that behavior. B.F. Skinner first studied response-independent reinforcement in 1948 using pigeons (Ono, 1987). The behavior that was shaped by these non-contingent reinforcement schedules consisted of idiosyncratic and stereotyped topographies of behavior and was referred to as "superstitious behavior" (Ono). Since Skinner's experiments, many researchers have studied superstitious behavior but have reported very

little about the properties of idiosyncratic and stereotyped superstitious behavior in either humans or in animals.

Justice and Looney (1990) explained that Skinner exposed 8 pigeons to a fixed-time 15-second non-contingent schedule of food. According to these authors, Skinner reported that six of the birds developed reliable superstitious behaviors including circling, head swinging, and pecking. These behaviors, according to Skinner were operantly conditioned. Skinner proposed that this operant conditioning occurred when the bird was engaged in a behavior that preceded food delivery. The food increased the probability of the behavior's recurring and being accidentally reinforced again. Each reinforcement following the behavior increased the probability of the behavior's recurrence (Justice & Looney).

Hergenhahn and Olson (2001) explained that when we speak about operant conditioning, we are talking about contingent reinforcement. Reinforcement following a barpressing response is an example of contingent reinforcement because the reinforcement is dependent on the response. However, if we arrange the situation so that the reinforcement was only delivered now and then, independently of the animal's behavior, then according to the principles of operant conditioning, we can predict that whatever the animal is doing when the reinforcement is delivered will be reinforced, and the animal will tend to repeat the reinforced behavior. After a period of time, the reinforced behavior will reoccur when the reinforcement is delivered and the response will be strengthened. Thus, the animal is apt to develop strange ritualistic responses such as bobbing its head, or turning in circles, or standing on its hind legs or performing a series of actions according to what it was doing when the reinforcement was delivered.

This ritualistic behavior is what is referred to as superstitious behavior because the animal behaves as if what it is doing is causing the reinforcement. However, because the reinforcer is independent of the animal's behavior, it is referred to as noncontingent reinforcement.

This explanation of superstitious behavior dominated operant theory for over two decades. Speculating on the relationship between contiguity and contingency and their role in conditioning, Skinner proposed that a contiguous situation may mean only that the reinforcement follows the behavior. Skinner believed that when reinforcement is delivered, conditioning occurs, even when as in the case of a non-contingent schedule, the behavior has not caused the reinforcement. The animal behaves as if there is a causal relationship between the behavior and the reinforcement (Justice & Looney, 1990)

Reberg, Mann, and Innis (1977) indicate that in one of their experiments, pigeons developed different patterns of superstitious behaviors when deprived of food and water, and then placed in a long box response chamber. Food and water were then simultaneously presented in 15-second intervals at opposite ends of the box. The pigeons would move away from the food hopper early in the interval and then would peck at the wall in the area of the food hopper later in the interval. During the water interval, the pigeons rarely moved away from the water hopper. The most common behavior early in the water interval was a sustained thrust of the head into the hopper opening. Late in the water interval the birds usually pecked around the water hopper with a distinctive drinking motion that clearly differed from the snapping pecks seen during food intervals (Reberg et al.).

Timberlake and Lucas (1985) cast some doubt on the findings of Skinner, who tried to explain superstitious behavior as an accidental response contingency whereby such behavior is an operant that occurs under the response-independent presentation of reward because of accidental (unprogrammed) juxtapositions of reward and response, which create a positive feedback loop. Timberlake and Lucas also cast doubt on the findings of other researchers who argue that superstitious behaviors are nothing more than terminal responses elicited in anticipation of a food reward. According to these authors, the commonality of the pecking response in pigeons favored an explanation based on elicitation rather than on accidental response contingencies. In addition, some researchers suggest that the occurrence of pecking is readily explained by Pavlov's stimulus-substitution hypothesis. According to this hypothesis, conditioned responses are formed from components of the unconditioned stimulus (in this case the food). But prior to the terminal response of pecking, pigeons also engage in other interim behavior patterns apparently unrelated to food. Timberlake and Lucas showed in their studies that the consistency of these superstitious behavior patterns argued against an explanation of accidental response contingencies, and the complexity of the behaviors was incompatible with the classic stimulus-substitution account (Timberlake & Lucas). These authors instead concluded that superstitious behavior under periodic delivery of food probably develops from components of species-typical patterns of appetitive behavior related to feeding.

Liddell and Morgan (1978) measured the degree of superstitious compulsion present in a sample of undergraduate students at North East London Polytechnic in a two-part study. Initially, measurement was carried out using a questionnaire designed to

investigate the effects of age, sex, and rural or urban home area on the level of compulsion to indulge in superstitious behavior. The second part of the study utilized a questionnaire that was modified from the one used in the first part of the study and was combined with the Eysenk Personality Inventory Form A in order to investigate a relationship between the degree of neuroticism, obsessionality, and superstitious compulsions. Superstitious compulsions were shown to decrease with age. The assumption that women are more superstitious than men was found to be erroneous, and age, type of superstition, and personality factors were more important variables. Liddell and Morgan concluded that superstitious compulsion of a ritualistic type between the ages of ten and fifteen possibly predicts later levels of neuroticism and that there is an association between present obsessionality and phobic superstitions in young adults. It should be noted that the type of superstition in this experiment is not radically different from the type of superstition that a laboratory animal exhibits under the assumption that it is controlling the delivery of a reinforcement such as food. In fact, superstition can be viewed from a number of different perspectives. White and Liu (1995) defined superstition as a manifestation of unconscious mental processes, but again, and more importantly as an operantly conditioned response. It is an association between a particular, voluntary, response made by the organism and the presentation of reinforcement.

An analysis of superstitious behavior leads us to ask the more general question of which experimental conditions are necessary in order to maintain the probability of a particular behavior. A contingency may be necessary for the behavior to be maintained. However, experimental situations in which superstitious behavior has been demonstrated

have purposely prevented any contingency between the behavior and the reinforcement. It was argued that adventitious reinforcement, or the occasional contiguity between behavior and the food delivery, was sufficient to account for the increases in probability of the superstitious behavior. An analysis of superstitious behavior may help us to understand the relative roles of contingency and contiguity, as well as the relationship of these two variables in the control of behavior.

The nature of the link between operant activity and reinforcement is traditionally exemplified by one of four schedules of reinforcement (White & Liu, 1995). A fixed interval schedule is one in which the animal is reinforced for a response made only after a set interval of time. For example, only a response following a three-minute interval is reinforced. A fixed ratio schedule occurs when every *n*th response that the animal makes is reinforced. For instance, F5 means that the animal is reinforced at every fifth response. A variable interval schedule means that the animal is reinforced for responses made at the end of time intervals of variable durations. For instance, the animal is reinforced for a response made after the average of, say, every 3 min., but it may be reinforced immediately after a prior reinforcement, or after 30 sec., or after 7 min. A variable ratio schedule means that the animal is reinforced following an average of so many responses. For instance a VR5 means that the animal is reinforced on an average of every 5 responses, so it may be reinforced 2 times in a row, or it might make 10 or 15 responses without being reinforced (Hergenhahn & Olson, 2001).

Learned Helplessness, Stereotypy, and Superstitious Behavior

It has been argued that the perceived lack of control over reinforcement, results in learned helplessness rather than superstitious behavior. Learned helplessness is defined

as the belief that one can do nothing to terminate or avoid an aversive situation. It is not caused by traumatic experience but by the inability, or perceived inability to do anything about it (Hergenhahn & Olson, 2001). Matute (1994) argues that learned helplessness and superstition accounts of uncontrollability predict opposite results for subjects exposed to non-contingent reinforcement. This is especially clear when we consider that learned helplessness is the same as relinquishing control over one's circumstances, and that superstitious behavior is an attempt to exert control over one's circumstances. In her experiment, Matute (1994) showed that yoked human participants tended to exhibit superstitious behavior and illusion of control when exposed to uncontrollable noise. Matute (1994) argues that conditions of response-independent reinforcement commonly used in human research do not lead to learned helplessness, but rather to superstitious behavior and illusion of control.

Helplessness results from the individual's learned expectations that their responses are independent of desired outcomes. This is quite different from trying to control outcomes through the stereotyped response patterns characteristic of superstitious behavior. When people engage in a task, they typically know what outcomes are expected. They also have a sense of how controllable or uncontrollable that outcome is. With controllable outcomes, a one-to-one relationship exists between behavior or what a person does, and outcomes or what happens to that person. With uncontrollable outcomes, a random relationship exists between behavior and outcomes. Therefore the person will realize that they have no idea what effect, if any, their behavior will have on what happens to them (Reeve, 2001).

In addition to making the distinction between learned helplessness and superstitious behavior, the distinction should be made between stereotypy and superstitious behavior. Stereotypy develops as a result of replacing an aversive behavior with a behavior or behavioral sequence that is less aversive (Strongman, 1984). In addition, stereotypy develops as an attempt to replace information that is difficult to process with information that can be encompassed, or as an attempt to replace unpalatable beliefs or experiences or feelings with those that can be swallowed. These three possibilities can occur simultaneously or in some sequence or another (Strongman). Picture the fox in the zoo cage where the sequence might be to walk around the perimeter of the enclosure for hours on end, breaking its walk always at the same corner to turn twice through 360 degrees before continuing. This behavior will certainly not result in an extrinsic reinforcement, however, this is behavior that can be accomplished and that provides stimuli that can be processed. The aversive alternative would almost certainly involve looking through the bars of the cage at the unattainable (Strongman). However, the present experiment further distinguishes between stereotypy and superstitious behavior because the group that was exposed to alcohol but received no food reinforcement during the testing phase did not show the same behavior as the group that did receive a food reinforcement. If alcohol had merely caused an increase in stereotyped behavior or simply increased motor behavior, then both groups would have shown significant increases.

Other Indications of Superstitious Behavior

Hendry and Van Toller (1964) conducted a study whereby a positively correlated amount of reward was delivered to rats dependent on barpressing response times. If the

animal responded to a signal light by taking more time to make a barpressing response, they were reinforced with a larger amount of food. The authors of this experiment claim that this resulted in superstitious behavior as evidenced by slow response times in the experimental group compared to control animals, which responded quickly throughout the experiment and were always given a small amount of reinforcement. Actually this experiment more closely resembles ordinary learning rather than superstitious behavior.

Davis and Hubbard (1972) explain that there have been several instances where superstitious behavior has occurred under conventional schedules of reinforcement that were contingent on the response of the subject. Davis and Hubbard point to research in which superstitious behavior appeared during temporal reinforcement schedules. These studies have been conducted on rats and rhesus monkeys, as well as humans. The more recent research has involved the use of some non-contingent reinforcement schedules and has analyzed superstition in terms of the rate of a previously conditioned behavior such as barpressing or key pecking.

In their own research, Davis and Hubbard (1972) attempted to record idiosyncratic, stereotyped behavior patterns as they occurred in laboratory rats. The design of their experiment replicates Skinner's design except for the use of rats instead of pigeons, and also extends the schedules of non-contingent reinforcement to include variable as well as fixed time schedules. For eight consecutive days, laboratory rats were exposed to one-hour sessions during which non-response contingent food was delivered according to one of six different schedules. The schedules were fixed time 15 seconds, fixed time 30 seconds, fixed time 60 seconds, variable time 15 seconds, variable time 30 seconds, and variable time 60 seconds.

Davis and Hubbard (1972) indicated that in their experiment, repeated stereotyped behavior patterns were observed under each of the conditions where food was delivered on a non-response-contingent schedule. These authors explain that the continuous presence of certain behaviors in the absence of a response-reinforcement contingency reflects a "superstition," and that such a superstition is based on occasional contiguities between the behavior and the delivery of food. This superstition shows that some emitted behavior pattern becomes more probable after its proximity with a reinforcer. However, if we consider the role of consequences in maintaining behavior, we are basically implying that there is a dependency or contingent relationship between the behavior and the consequence it produces. Many researchers (e.g. Rescorla) feel that only a true contingency can maintain the probability of a conditioned behavior (Davis & Hubbard). So on the one hand we have a behavior that is maintained superstitiously and occurs in the absence of a contingency, while on the other hand a contingency might be required to maintain the probability of the behavior.

Contingency and Contiguity

Contingency is usually defined as the dependency between a response and any reinforcers that follow. As such, contingency is separate from contiguity, which is the temporal relationship between behavior and events that follow, although in most standard experimental situations the two are correlated. Contiguity often arises as a result of the interaction between behavior and the contingency, although under some circumstances contiguity alone is suggested by some (e.g. Skinner) to mimic the effects of contingency (Lattal & Shahan, 1997). Reinforcement contingencies have their effects, either directly or indirectly, as a result of the organism detecting, recognizing, or perceiving, and then

engaging the contingency. Thus, perceptions of contingencies give rise to hypotheses of behavior, which lead to actions. These actions will be maintained because of the perception that the reinforcement is contingent on the behavior.

One way to study superstitious behavior is to first condition a given response using response-dependent reinforcement, and then switch to response-independent reinforcement. For instance, the subject might first be exposed to a fixed ratio schedule, in which reinforcement is contingent upon each example of the specified behavior that occurs following the previous reinforcement, and then be switched to a fixed time schedule in which the reinforcement occurs at fixed periods of time regardless of the occurrence of the behavior. It has been found that the behavior strengthened under response-dependent reinforcement is maintained, although at a reduced level, under response-independent reinforcement (Eldridge, Pear, Torgrud, & Evers, 1988).

Likewise, there is evidence that rate of responding in variable-interval schedules where reinforcement is contingent on the first response after a varied interval of time following the previous reinforcement, is determined jointly by the rate of reinforcement, and an implicit contingency whereby certain interresponse times are differentially reinforced (Pear, 1985). Skinner's concept of superstitious behavior may also be reinforced by variable interval schedules because the behavior occurring prior to a reinforced response is unspecified by the variable interval contingency, and therefore various spatiotemporal patterns of behavior may tend to be adventitiously reinforced.

Effects of Stress and Desire for Control

Research shows that the frequency of superstitious behavior increases under conditions of stress (Keinan, 2002). A possible explanation for this finding is that stress

reduces the participant's sense of control and that to regain control the participant engages in superstitious behavior (Keinan). This is consistent with Pisacreta (1998), who defines superstitious behavior as behavior that is controlled by the belief that certain behaviors contribute to reinforcement when, in fact, they are not necessary for the reinforcement to occur. Behavior that is under the control of a superstitious rule typically develops into stereotyped response chains. When given multiple ways to solve a problem (e.g., obtaining a food reward) the subject emits the same response sequence over and over in an attempt to control the delivery of the reinforcement.

Matute (1995) indicates that research has shown superstitious behavior and the illusion of control in human participants exposed to negative reinforcement conditions such as loud and uncontrollable noise. In her own experiments, Matute obtained superstitious behavior and the illusion of control. As discussed above, in her experiments Matute explored the generality of superstition and illusion of control effects in humans exposed to uncontrollable noise under different conditions of negative reinforcement. Participants were induced to try to terminate aversive noise. However, the reinforcement (defined as quickly terminated noise) was pre-programmed and occurred independently of responding. Experimental conditions differed in the percentage and distribution of reinforcement. Matute's experiments resulted in most participants exposed to non-contingent negative reinforcement tending to behave superstitiously, and to believing that they had found a way to stop the noise, that the task was controllable, and that they were controlling it. The results of Matute's experiment are incompatible with the development of learned helplessness and question the generality of learned helplessness as a consequence of exposing humans to uncontrollable outcomes. These results suggest

instead that humans will try to gain control of negative reinforcement through the use of superstitious behavior.

Chronic Alcohol Exposure

Darbra, Pratt, Pallares, and Ferre (2002), studied the effect of chronic alcohol exposure on the development of tolerance to the depressive effects of alcohol in rats that voluntarily self-administered alcohol. Not surprisingly, these authors found that tolerance increased with increased exposure to alcohol and that these effects coincided with decreases in alcohol induced sleep time. That is, the rats that drank more, slept less. These findings are corroborated in Ehlers and Slawecki (2000). In this study, chronic alcohol exposure and withdrawal was found to produce changes in the EEG, eye movements, muscle activity, and breathing patterns during sleep. Rats that were exposed to chronic doses of alcohol slept more superficially and experienced lighter sleep according to EEG recordings.

High doses of alcohol results in impairment of the motor systems, sedation, and sleep. However, shortly after ingesting low doses of alcohol, and during the rising phase of blood alcohol concentration, stimulatory effects that are similar to those produced by stimulant drugs (e.g. amphetamine) are observed. Because alcohol can produce behavioral stimulation and then depression it's effects are said to be biphasic. Low doses of alcohol increase spontaneous motor activity while high doses decrease it. Stimulation of locomotor behavior occurs when blood alcohol concentrations are increasing. The Eighth Special Report to the U.S. Congress on Alcohol and Health from the Secretary of Health and Human Services (The Secretary, 1993) suggests that alcohol and stimulant drugs such as cocaine and amphetamine induce locomotor stimulation by a similar

mechanism, which is enhancing the neurotransmitter dopamine in the ventral tegmental area of the brain. This is an important part of the brain and dopamine is an important neurotransmitter in this system.

Alcohol is known to affect a vast number of neurochemical (e.g.,neurotransmitters, neuromodulators, and neurohormones) systems. For example, Kekki, Pentikainen, and Mustala (1974), suggests that chronic alcohol exposure causes marked changes in the metabolism of serotonin. This report concerns the urinary excretion of 5-hydroxyindoleacetic acid (5-HIAA), the main metabolite of serotonin, after chronic alcohol exposure. The study shows that the excretion of 5-HIAA increases as a result of alcohol administration, which indicates that serotonin is metabolized more rapidly in rats exposed to alcohol than in control rats that did not receive alcohol.

Roy and Pandey (2002) observed a decrease in levels of neuropeptide Y, a peptide that acts as a neurotransmitter and neuromodulator, which is very abundant in the central nervous system. The decreases were found in the central nucleus of the amygdala and are believed to play a role in promoting alcohol-drinking behavior. In addition, these authors observed changes in the levels of neuropeptide Y in hypothalamic structures during chronic alcohol exposure and withdrawal. They suggest that it is possible that these decreased protein levels may be involved in stimulating an appetite for alcohol.

Adams and Cicero (1998) imply that alcohol affects nitric oxide systems in the brain, and that nitric oxide agents affect alcohol intoxication and withdrawal. Nitric oxide appears to mediate aspects of physical dependence and preference, but it is not known if nitric oxide mediates clinically dangerous alcohol withdrawal seizures or other chronic alcohol-induced effects on brain functions. Overall, data suggests that acute and

chronic alcohol treatments change the activity of brain nitric oxide systems so that nitric oxide synthase activity decreases when alcohol is present and increases when alcohol concentrations decrease.

Madeira et al. (1997) indicate that there is evidence that chronic ethanol treatment disrupts the biological rhythms of various brain functions and behaviors. They examined the effects of chronic alcohol exposure on the main morphological features and chemoarchitecture of the suprachiasmatic nucleus in the hypothalamus. Their study revealed that chronic alcohol exposure induced a significant reduction in the total number of neurons containing vasopressin, vasoactive intestinal polypeptide, gastrin-releasing peptide, and somatostatin. An interesting feature of their research was that withdrawal from alcohol did not reduce the loss of these neurons, but rather augmented the loss.

Gianoulakis (2001) reported that there is increasing evidence supporting a link between the endogenous opioid system and excessive alcohol consumption. Acute or light alcohol consumption stimulates the release of opioid peptides in brain regions that are associated with reward and reinforcement and that mediate, at least in part, the reinforcing effects of alcohol. However, chronic heavy alcohol consumption induces a central opioid deficiency, which may be perceived as opioid withdrawal and may promote alcohol consumption through the mechanisms of negative reinforcement. The effectiveness of opioid receptor antagonists in decreasing alcohol consumption in people with an alcohol dependency and in animal models lends further support to the view that the opioid system may regulate, either directly or through interactions with other neurotransmitters, alcohol consumption.

It has been shown that impairments of learning and memory are common neuropsychological effects of chronic alcohol abuse (Obernier, White, Swartzwelder, & Crews, 2002). Chronic alcohol abuse is also associated with cortical and limbic atrophy through white matter loss and gray matter loss. Alcoholic humans often suffer from learning and memory impairments, including deficits in spatial memory and, most important to the present study, abnormal response perseveration. Rintala et al. (1997) indicate that one of the main neurological manifestations of chronic alcoholism is cerebellar atrophy. These authors explain that postmortem studies have shown that almost one half of severe alcoholics have histologically verified atrophic changes in the cerebellum. In addition to these findings, Roberto, Nelson, Ur, and Gruol (2002) reported that the hippocampal region of the brain plays a pivotal role in memory processing and is likely to be an important site of alcohol effects that lead to altered cognitive function. These authors point to recent research that shows that both acute (e.g., exposure for tens of minutes) and chronic (e.g., exposure for months) alcohol exposure blocks the induction of long-term potentiation (LTP) in the hippocampus. LTP is thought to be one important cellular mechanism of learning and memory.

Grover, Frye, and Griffith (1994) examined ethanol-induced inhibition of N-methyl-D-aspartate (NMDA) mediated synaptic activity in the CA1 region of the hippocampus, and found that ethanol does inhibit the NMDA mediated excitatory postsynaptic potentials. A reduction in ethanol-induced inhibition while still exposed to ethanol indicated acute tolerance, which did not develop to an NMDA antagonist induced inhibition. The NMDA receptor is a subtype of glutamate receptor, which is basically a glutamate-gated ion channel that is permeable to sodium, potassium, and calcium. The

CA1 region of the hippocampus is an area thought to be important for declarative memory, and spatial memory in rats.

Devenport (1979) reported that unlike normal animals, rats with hippocampal lesions behaved in an operant chamber as if a dependency existed between food delivery and their behavior, despite the fact that reinforcement was based on time, not behavior. This superstitious behavior did not result from a general inability to inhibit responding, as responding rapidly ceased when the reinforcement was discontinued. These findings suggest that the hippocampus integrates information regarding response-reinforcer relations, which in the normal rat permits superfluous operant behavior to be eliminated. So if alcohol exposure blocks the induction of long-term potentiation (e.g., integration of information) in the hippocampus and the hippocampus is an important site of alcohol effects that lead to cognitive deficits (e.g., disinhibition of superfluous operant behavior), we can expect chronic exposure to alcohol to lead to increased superstitious behavior.

Conclusion

The literature suggests that superstitious behavior is a phenomenon that results from the desire for control over the consequences of behavior such as food reinforcement. When an organism is reinforced, it naturally tries to exhibit the behavior that caused the reinforcement even if the reinforcement was not a result of the behavior. The literature also indicates that the effects of chronic alcohol exposure include cognitive deficits that are a result of neurologioal impairment. Taken together, these manifestations provided some insight into the possibility that chronic alcohol exposure would increase the superstitious behavior of an organism, which was aimed at gaining control over the amount of reinforcement it received. It was on this hypothesis that the present study was

based. Following a procedure similar to that of Davis and Hubbard (1972), the present study attempted to determine if chronic alcohol exposure does in fact cause an increase in the superstitious responding of laboratory rats.

Research Questions

Based on past research, the following research questions were developed:

Research Question 1: Does chronic alcohol exposure increase behavior?

Research Question 2: If there is an increase in behavior as a result of chronic alcohol treatment, is it superstitious behavior or just general motor behavior?

Hypotheses

The present study also investigated the following hypotheses:

Hypothesis 1: Chronic alcohol exposure causes an increase in superstitious behavior.

Hypothesis 2: Chronic alcohol exposure causes an increase in superstitious behavior rather than just increased motor activity.

CHAPTER 2

METHOD

Subjects

Twenty-one Holtzman Sprague-Dawley rats purchased from Harlan (Madison, Wisconsin) served as subjects for this experiment. These subjects were experimentally naïve and had no prior experience with the test chamber. Emporia State University Animal Care and Use Committee approval was granted, ESU ACUC # 03-004 (see Appendix A).

Design

The present experiment utilized a completely randomized multiple group design. The independent variable was treatment (combination of drinking and operant box condition). Levels of the independent variable included no alcohol/fixed time food, alcohol/fixed time food, and alcohol/no fixed time food. The dependent variable was the individualized behavior score for each subject.

Apparatus

Subjects were run in a rodent operant test chamber, Model # 84022SS, Lafayette Instruments Company, which was located in the Davis Lab in Visser Hall at Emporia State University. The dimensions of the chamber were 9 ½ inches (24.13 cm) by 8 ½ inches (21.59 cm) by 7 ½ inches (19.05 cm; *l* x *h* x *w*). The front and rear walls of the cage were constructed of metal, and both side walls and cage top were made of clear Plexiglas. The cage was equipped with a lever on the front wall. The cage floor consisted of 15 stainless steel rods, 1/8 inch (.32 cm) in diameter, set 3/8 inch (.95 cm) apart from edge to edge. The floor grid closest to the front wall was spaced 3/4 inch

(1.91 cm) from that wall. Food deliveries were programmed automatically and consisted of 45 mg dry food pellets.

Treatments

There were two actual test periods in this experiment, which included a baseline test period and an actual test period. Between the test periods, an alcoholic solution consisting of 10% ethanol was prepared and dissolved in water. Fourteen of the subjects were given free access to a bottle containing the alcoholic solution for a period of three weeks. During this time the other seven subjects were given free access to ordinary water.

Procedure

Baseline. Three days prior to testing, all subjects were placed on a food deprivation schedule. This consisted of a single feeding of 10-12 g of food at approximately 6:30 p.m. (their light cycle) each day. They were never allowed to go below 85% of their free feeding weight, and body weights were recorded daily. They remained on food deprivation throughout all phases of this study. The subjects were individually caged with drinking solution (water) continuously available. The subjects were maintained on a 12-hour light/ dark cycle. Approximately 5 hours into the light schedule, the subjects were placed in the experimental chamber and observed and video taped for one 15-minute period everyday for eight consecutive days. During this time all of the subjects were exposed to a non-contingent fixed time schedule of reinforcement, which consisted of one 45 mg food pellet every 30 seconds. The amount of fixed-time schedule induced behavior exhibited during the eighth day was used as a baseline.

Treatment. After the baseline for fixed-time behavior was obtained the 14 treatment subjects were exposed to the 10% alcohol drinking solution for three weeks while the 7 control subjects were maintained on water without alcohol. Fresh alcohol solutions were made every 3 days. All subjects remained on food deprivation during this three week period and body weights and volume of drinking solution consumed were recorded daily. During the actual test period, 7 of the subjects exposed to alcohol and the 7 water-control subjects that were not exposed to alcohol were then exposed to 15-minute sessions daily for 5 days during which non-contingent food pellets were delivered according to the fixed time schedule of 30 seconds. Food deliveries were always programmed without regard to the subjects' behavior. The remaining alcohol-treated animals were placed in the chamber and were not exposed to the food delivery. This was done in order to determine whether motor movement effects might result from alcohol exposure.

Testing. Recording of behavior during both baseline and testing sessions in the operant chamber was accomplished using a video camera, and the videotapes were then reviewed carefully by persons blind to the subjects' treatment conditions to ascertain the extent of superstitious responding. In order to get the scores, a behavior profile based on the last session of baseline was developed for each subject by myself prior to the scorers viewing of the videotapes. The profile consisted of possible behaviors such as pawing, licking, or biting the feeder cup inside the test chamber, leaving the vicinity of the feeder cup and then quickly returning to check the feeder cup for food, randomly checking the feeder cup while engaging in other behaviors, hovering over the feeder cup, deliberate barpressing behavior, and turning in circles and then checking the feeder cup. Each

subject profile consisted of four of these possible behaviors. For instance, the profile for subject #201 consisted of barpressing or nuzzling the bar, pawing, licking, or biting the feeder cup, leaving the feeder cup and then quickly returning to the feeder cup, and frequently checking the feeder cup, while the profile for subject #207 was turning in circles, hovering over the feeder cup, frequently checking the feeder cup, and barpressing. The subject was then given a point by the scorers every time one of the behaviors contained in its profile was exhibited during a particular test session.

CHAPTER 3

RESULTS

Method

Twenty-one male rats served as subjects for this experiment. Each rat was exposed to a fifteen-minute session everyday for eight days. During each session the subject was given a food pellet every 30 seconds. Fourteen of the subjects were then exposed to an alcohol solution for three weeks. The subjects were then tested again for 15 minutes everyday for five days using the same 30-second fixed-time schedule. Seven of the alcohol exposed subjects were not exposed to the food during this final five days of testing in order to determine possible increased motor effects caused by the exposure to the alcohol. All sessions were video taped and reviewed by persons who were blind to the subjects' treatment condition.

Statistical Analyses

Body weights. The means and standard deviations were calculated for each of the three groups of subjects for body weights (see Figure 1). For the week of baseline, neither the main effect of Group, $F(2,18) = 1.43, p = .26$, nor the interaction of Group x Day, $F(2,18) = .87, p = .44$, were significant. However, the main effect of Day, $F(1,18) = 395.64, p <. 001$, was significant. η^2 (.96) indicates that a large proportion of the variance is explained by the Days effect, which simply means that all of the subjects weighed more on the final day than on the initial day.

For the three week treatment period (see Figure 2), the main effect of Group, $F(2,18) = 8.99, p = .002$, and Week, $F(2,36) = 336.77, p < .001$, were significant. More importantly, the interaction of Group x Week, $F(4,36) = 89.82, p < .001$, was also

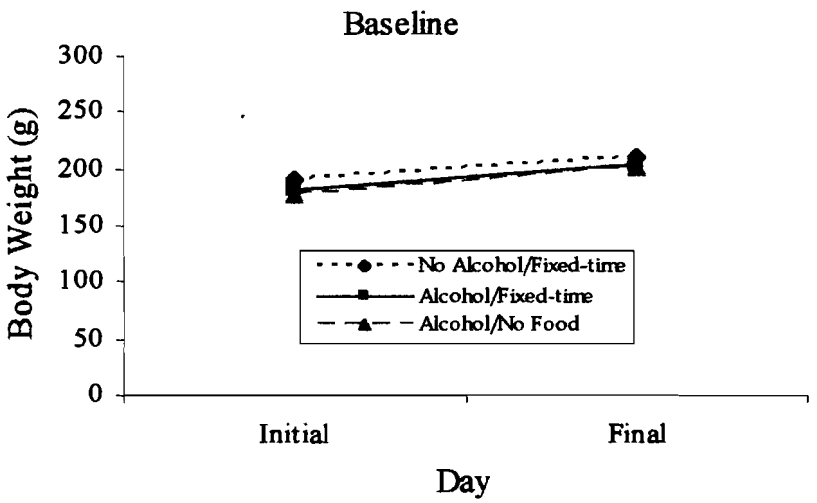


Figure 1. Group mean of body weights on the initial and final day of baseline for all three groups of rats (n = 7/group). Means and standard deviations for each group for each session are as follows: No Alcohol/ Fixed-time: Initial, M = 190.77, SD = 10.37; Final, M = 211.06, SD = 9.42; Alcohol/ Fixed-time: Initial, M = 181.91, SD = 18.04; Final, M = 203.07, SD = 21.73; Alcohol/ No food: Initial, M = 177.34, SD = 8.63; Final, M = 201.03, SD = 6.89.

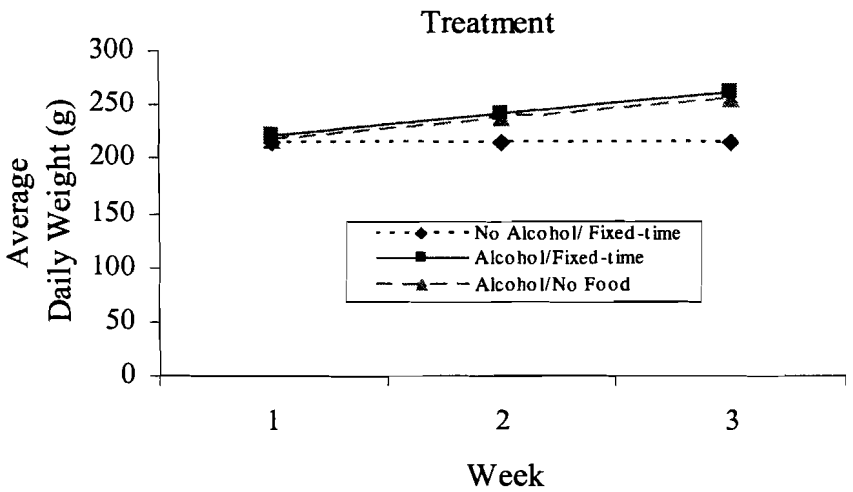


Figure 2. Group mean of average daily weights across the 3 week drinking solution treatment for all 3 groups of rats, $n = 7$ for each group. Means and standard deviations for each group for each session are as follows: No Alcohol/ Fixed-time: Week 1, $M =$ 214.63, $SD = 8.77$; Week 2, $M = 214.24$, $SD = 9.24$; Week 3, $M = 213.84$, $SD = 9.92$; Alcohol/ Fixed-time: Week 1, $M = 220.14$, $SD = 20.34$; Week 2, $M = 240.54$, $SD =$ 17.57; Week 3, $M = 260.9$, $SD = 15.41$; Alcohol/ No Food: Week 1, $M = 217.64$, $SD =$ 6.95; Week 2, $M = 236.44$, $SD = 8.53$; Week 3, $M = 255.21$, $SD = 11.03$.

significant. η^2 (.91) indicates that the proportion of the variance accounted for by the interaction was large. Alcohol treated subjects gained weight over the treatment period, whereas the control subjects did not.

For the initial day of testing (see Figure 3), a one way ANOVA revealed a significant difference in body weights between groups, $F(2,18) = 29.7, p < .001$, which means that the three groups differed from each other on body weight on the initial day of testing. The proportion of variance accounted for by the effect (η^2) was .77, which is a large effect size. Fisher LSD tests ($ps < .05$) indicated that groups treated with alcohol (alcohol/ fixed-time, alcohol/ no food) weighed more than the group of subjects that only drank water. It is likely that this is because of the increased caloric intake resulting from drinking the alcohol solution.

Fluid consumption. The means and standard deviations were calculated for each of the three groups of subjects for fluid consumption (see Figure 4). For the three week treatment period, the Group, $F(2,18) = 52.5, p < .001$ and Week, $F(2,36) = 308.61, p < .001$ were significant. More importantly, the interaction between Group x Week, $F(4,36) = 56.13, p < .001$, was also significant. η^2 (.86) indicated a large proportion of the variance was explained by the interaction. This indicates that the subjects in the two alcohol groups developed a tolerance to the alcohol solution and had to drink more to achieve the desired effect.

For the initial day of testing (see Figure 5), a one way ANOVA indicated significance for the main effect of Groups, $F(2,18) = 46.17, p <. 001$. The proportion of the variance accounted for by the effect (η^2) was .84, which is a large effect size.

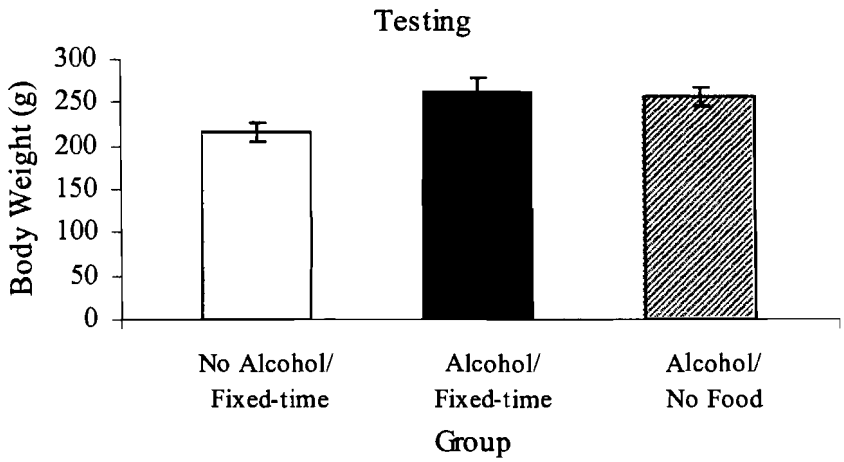


Figure 3. Body weights in grams for No Alcohol/Fixed-time: $M = 215.56$, $SD = 10.15$; Alcohol/Fixed-time: $M = 262.21$, $SD = 15.36$; Alcohol/No Food: $M = 256.23$, $SD = 10.95$ on the initial day of testing. Error bars depict standard deviations ($n = 7$/group).

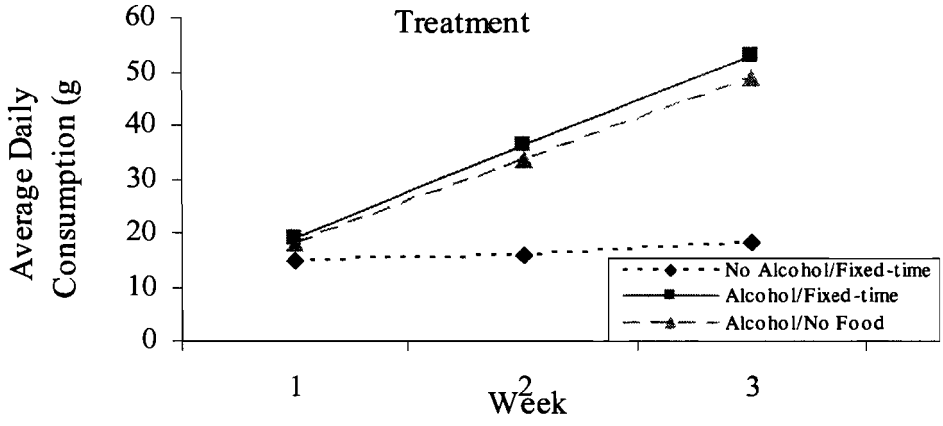


Figure 4. Group mean of average daily fluid consumption across the three week drinking solution treatment for all three groups of rats (n = 7/ group). Means and standard deviations for each group and for each week are as follows: No Alcohol/ Fixed-time: Week 1, M = 14.74, SD = 1.94; Week 2, M = 15.7, S SD = .99; Week 3, M = 18.9, SD = 2.81; Alcohol/ Fixed-time: Week 1, M = 19.15, SD = 3.08; Week 2, M = 36.41, SD = 4.87; Week 3, M = 53.17, SD = 6.71; Alcohol/ No Food: Week 1, M = 18.7, SD = 2.81; Week 2, M = 33.47, SD = 4.99; Week 3, M = 48.86, SD = 8.38.

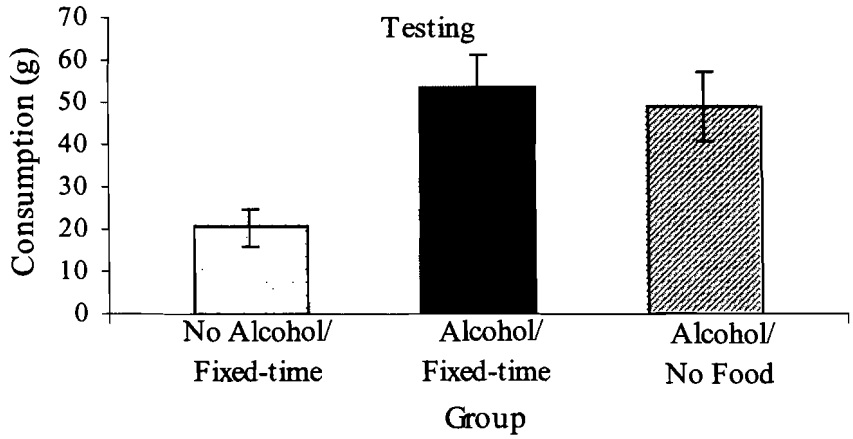


Figure 5. Fluid consumption for groups No Alcohol/Fixed-time: $M = 20.41$, $SD =$; Alcohol/Fixed-time: $M = 53.67$, $SD =$; Alcohol/No Food: $M = 48.77$, $SD =$;on the initial day of testing. Error bars depict the standard deviations ($n = 7$/group).

Fisher LSD tests ($ps < .05$) indicate that the groups exposed to alcohol consumed more fluid than the group that drank water. This latter finding was probably because of increased tolerance.

Total behavior. The means and standard deviations were determined for initial and final day of baseline total behavior scores (see Figure 6). The main effect of Group, $F(1,18) = 1.69, p = .21$, and the interaction of Group x Day, $F(2,18) = .9, p = .42$, were not significant, but the main effect of Day, $F(1,18) = 13.48, p = .002$, was significant. The proportion of the variance accounted for by the score was large as indicated by η^2 of .43. Unfortunately, the collapsed mean total behavior score was 86.81 ($SD = 27.38$) on the initial day, and was 67.86 ($SD = 21.71$) on the final day of baseline. This clearly indicates that superstitious behavior did not develop across the baseline sessions.

The means and standard deviations were calculated for baseline versus testing total behavior scores (see Figure 7). The main effect of Group, $F(2,18) = 2.25, p = .13$, was not significant, while the main effect of Session, $F(1,18) = 5.81, p = .03$ was significant. More importantly, the interaction between Group x Session, $F(2,18) = 2.8, p = .09$, was marginally significant and supercedes the main effects. The proportion of the variance accounted for by the interaction was large as indicated by η^2 of .23. It appears there was an increase in fixed-time schedule induced behavior, albeit not superstitious behavior, and only a non-significant trend.

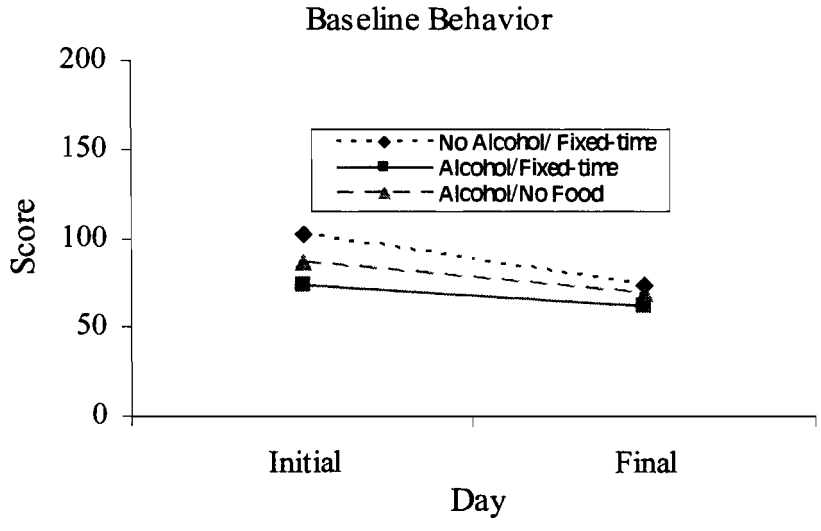


Figure 6. Group mean of average total behavior scores on the initial and final day of baseline for all three groups of rats, (n = 7/group). Means and standard deviations for each group for each session are as follows: No Alcohol/ Fixed-time: Initial, M = 101.71, SD = 18.57; Final, M = 73.57, SD = 28.62; Alcohol/ Fixed-time: Initial, M = 72.71, SD = 20.81; Final, M = 61.29, SD = 21.1; Alcohol/ No Food: Initial, M = 86, SD = 35.07; Final, M = 68.71, SD = 14.76.

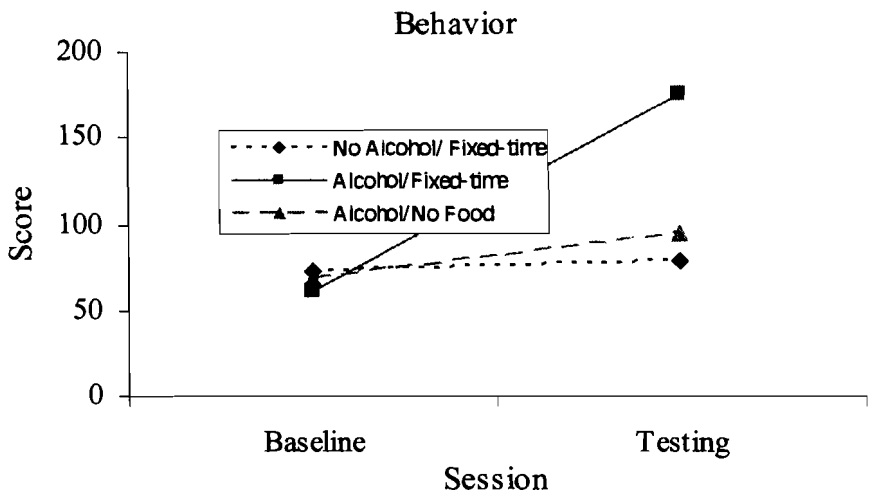


Figure 7. Group means of baseline and testing total behavior scores for all three groups of rats, (n = 7/ group). Means and standard deviations for each group are as follows: No Alcohol/ Fixed-time: Baseline, M = 73.57, SD = 28.62; Testing, M = 78.29, SD = 14.65; Alcohol/ Fixed-time: Baseline, M = 61.29, SD = 21.1; Testing, M = 175.14, SD = 115.88; Alcohol/ No Food: Baseline, M = 68.71, SD = 14.76; Testing, M = 95.29, SD = 85.59.

CHAPTER 4

DISCUSSION

The present study was designed to determine whether chronic alcohol exposure results in an increase in superstitious behavior. Though only marginally significant, the results of this study indicate that chronic alcohol exposure may at least contribute some to an increase in the development of fixed-time schedule induced behaviors in the laboratory rat. However, I cannot claim that the behavior was superstitious in the present study, because there was no development of superstitious behavior during baseline.

It is interesting to note that during the actual testing phase, the seven alcohol treated subjects that were just placed in the test chamber and observed without receiving food, did not show an increase in behavior relative to baseline. Furthermore, they did not exhibit any of the increased responding that the group that received alcohol and food on a fixed-time schedule exhibited during testing. This difference suggests that the behavior exhibited by the alcohol treated fixed-time food group was not just an increase in general, or stereotypical motor activity.

In the present study chronic alcohol exposure caused a marginally significant increase in fixed-time interval induced behavior. It appeared that the more prominent behaviors that were exhibited by the subjects were pawing, licking, or biting at the feeder cup, and leaving the feeder cup and then quickly returning to check the feeder cup. Two of the subjects continuously turned around in circles in front of the feeder cup and would then check the feeder cup after each circle. In contrast to pawing, licking, or biting the feeder cup, subjects frequently and deliberately left the vicinity of the feeder cup only to almost immediately return to the feeder cup to see if any reinforcement had been

delivered. Some animals repeatedly pressed the lever or bar on the front panel of the test chamber or sniffed or pawed around the under side of the lever. Two of the animals were so intense during this behavior that they actually rolled completely over several times while nosing the under side of the lever. This is probably just consumatory behavior, and not superstitious behavior. Appetitive behaviors occur because the animal is hungry and it is getting fed. A report (Kiefer, Badia-Elder, & Bice 1995) that high alcohol consuming rats show increased ingestive behaviors (which promotes consumption) is consistent with the interpretation in the present study that chronic alcohol exposure increased appetitive behavior.

Some of the animals performed a lot of other behaviors, but many of these other behaviors appeared to be in the form of escape responses or trying to find a way out of the test chamber. One animal did several complete back flips against the chamber lid, perhaps in an attempt to lift the lid in order to get out of the chamber. The majority of the animals frequently sniffed around at the top corners of the test chamber or pushed the lid of the chamber up a few centimeters with their nose possibly to see if they could escape the chamber. The animals also exhibited this behavior during the baseline testing.

One could argue that the behavioral differences observed in the present study might have been due to group differences in body weight. Both alcohol treated groups continued to gain weight more so than control rats. This is because while all three groups were maintained on food deprivation, the alcohol treated rats received additional calories from the alcohol. There have been studies that indicate weight loss in individuals that chronically drink alcohol such as reported by Colditz (as cited in The Secretary, 1993). Colditz suggests that the dramatic weight loss exhibited by alcoholics indicates that more

energy is expended by these individuals than is supplied by the alcohol. The present study probably did not expose subjects to alcohol long enough for weight loss to occur.

Additionally, there was a difference between the groups in the amount of fluid they drank. The subjects that were exposed to alcohol drank more than the subjects that were only given water. Carroll and Meisch (as cited in The Secretary, 1993) suggest that alcohol is a food that provides energy or calories just like sugar does, and that when an animal's food intake is reduced and low body weight is maintained, the animal shows large increases in alcohol consumption. Though they are not suggesting that the animal drinks more alcohol because they are trying to obtain calories, weight reduction apparently increases the reinforcing value of almost any kind of drug regardless of the caloric value of the drug. Likewise, Meisch and Thompson (1974) have reported that food deprived rats have greater alcohol intake.

Kiefer and Lawrence (as cited in The Secretary, 1993) indicate that alcohol is like food in that it has a flavor that they say the rat perceives as tasting bittersweet. Kiefer and Dopp (as cited in The Secretary, 1993) explain that rats with a history of self-administration of alcohol perform more ingestive responses during taste tests which indicates that the development of a preference for the flavor of alcohol results from experiencing the pharmacological effects of the alcohol.

Research Questions and Hypotheses

Even though there was marginal significance for an increasing effect of alcohol on fixed-time schedule induced behavior, caution should be used in interpreting these results. Is what occurred really superstitious behavior and do the results really support the hypothesis that chronic alcohol exposure would cause an increase in superstitious

behavior? Timberlake and Lucas (1985) suggested that these types of behaviors may be nothing more than species specific appetitive behaviors. Consistent with this suggestion, the increase in fixed-time schedule induced behavior in the present study only occurred when food was delivered and the subject happened to be one that was exposed to the alcohol treatment. It could be that the animal simply behaves this way when it experiences a gap in the continuance of the delivery of the food, during which time it is going to do behaviors that will promote consumption when food is again available. For example, licking, chewing and producing saliva, which are essential in digestion of the food.

The research question was therefore not answered and the hypothesis was not supported. However, an interesting finding did occur. Alcohol did increase the amount of fixed-time schedule induced behavior between pre-alcohol and post-alcohol situations, and this was not just an increase in nonspecific motor activity. How this will generalize to human populations is a very interesting question. On the one hand, there is new knowledge to consider in relation to the person who consumes alcohol. On the other hand, not everyone who consumes alcohol does so chronically. The effects that were obtained in the present study are marginal. Future research might aim at studying acute or moderate exposure to alcohol. Also, the group size in the present experiment was somewhat small, and future researchers might find more significant results by using larger groups of animals. It would be difficult to study the effects of chronic alcohol exposure on human beings.

If different procedures for inducing superstitious behavior were employed experimentally it would not be surprising to find that alcohol does increase certain

superstitions. Of course, there are many ways that the phenomenon of superstitious behavior might be tested. One could for instance look at attempts to escape from an aversive stimuli as a method to test for superstitious behavior. Matute (1994) did just that, and used human subjects. The main implication concerning the effects of alcohol is that there must somehow be something to compare to, such as a preliminary test without alcohol, and/ or a control group that did not receive alcohol.

It is not certain whether or not humans make adequate research participants in a study like this, because their learning is presumably more efficient. Also, there would more than likely be a lack of control over other variables such as food intake or the amount of alcohol they consume. It is also unethical to force them to drink alcohol at a chronic level. Subjects could have been scored on several different tests of superstitious behavior such as the removal of an aversive stimuli, a fixed-time reinforcement such as the present experiment, and a variable interval reinforcement schedule all combined. The more different tests that an experimenter could run, the more certain we could be that the alcohol was actually causing the increases in superstitious behavior. Regarding the task, it does not necessarily matter what the subject is doing as long as the subject is under the impression that the particular behavior is causing the reinforcement.

In conclusion, it is clear that alcohol has deleterious effects. Whether or not it has an effect on superstitious behavior has yet to be discovered.

REFERENCES

Adams, M. L. & Cicero, T. J. (1998). Alcohol intoxication and withdrawal: The role of nitric oxide. *Alcohol, 16*, 153-158.

Darbra, S., Prat, G., Pallares, M., & Ferre, N. (2002). Tolerance and sensitization to the hypnotic effects of alcohol induced by chronic voluntary alcohol intake in rats. *Journal of Psychopharmacology, 16*, 79-83.

Davis, H., & Hubbard, J. (1972). An analysis of superstitious behavior in the rat. *Behaviour, 43*, 1-12.

Devenport, L. (1979). Superstitious bar pressing in hippocampal and septal rats. *Science, 205,* 721-723.

Ehlers, C., & Slawecki, C. (2000). Effects of chronic ethanol exposure on sleep in rats. *Alcohol, 20,* 173-179.

Eldridge, G., Pear, J., Torgrud, L., & Evers, B. (1988). Effects of prior response-contingent reinforcement on superstitious behavior. *Animal Learning & Behavior, 16*, 277-284.

Gianoulakis, C. (2001). Influence of the endogenous opioid system on high alcohol consumption and genetic predisposition to alcoholism. *Journal of Psychiatry and Neuroscience 26*, 304-318.

Grover, C. A., Frye, G. D., & Griffith, W. H. (1994). Acute tolerance to ethanol inhibition of NMDA-mediated EPSPs in the CA1 region of the rat hippocampus. *Brain Research, 642*, 70-76.

Hendry, D. P., & Van-Toller, C. (1964). Performance on a fixed-ratio schedule with correlated amount of reward. *Journal of the Experimental Analysis of Behavior 7*, 207-209.

Hergenhahn, B. R., & Olson, M. H. (2001). *An introduction to theories of learning* (6th ed.). Upper Saddle River, New Jersey: Prentice Hall.

Justice, T., & Looney, T. (1990). Another look at "superstitions" in pigeons. *Bulletin of the Psychonomic Society, 28*, 64-66.

Keinan, G. (2002). The effects of stress and desire for control on superstitious behavior. *Personality and Social Psychology Bulletin, 28*, 102-108.

Kekki, M., Pentikainen, P., & Mustala, O. (1974). Effect of acute and prolonged ethanol administration on serotonin metabolism and excretion in urine and bile of rats. *Quarterly Journal of Studies on Alcohol, 35,* 1195-1204.

Kiefer, S. W., Badia-Elder, N., & Bice, P. J. (1995). Taste reactivity in high alcohol drinking and low alcohol drinking rats. *Alcoholism: Clinical and Experimental Research, 19*, 279-284.

Lattal, K., & Shahan, T. (1997). Differing views of contingencies: How contiguous? *The Behavior Analyst, 20*, 149-154.

Liddell, A., & Morgan, G. (1978). Superstitious compulsions. *British Journal of Medicine and Psychology, 51*, 369-374.

Lobmeyer, D., & Wasserman, E.A. (1986). Preliminaries to free throw shooting: Superstitious behavior? *Journal of Sport Behavior, 9*, 70-78.

Madeira, M. D., Andrade, J. P., Lieberman, A. R., Sousa, N., Almeida, O. F. X., & Paula-Barbosa, M. M. (1997). Chronic alcohol consumption and withdrawal do not induce cell death in the suprachiasmatic nucleus, but lead to irreversible depression of peptide immunoreactivity and mRNA levels. *The Journal of Neuroscience, 17*, 1302-1319.

Matute, H., (1994). Learned helplessness and superstitious behavior as opposite effects of uncontrollable reinforcement in humans. *Learning and Motivation, 25,* 216-232.

Matute, H., (1995). Human reactions to uncontrollable outcomes: Further evidence for superstitions rather than helplessness. *The Quarterly Journal of Experimental Psychology, 48*, 142-157.

Meisch, R. A., Thompson, T. (1974). Ethanol intake as a function of concentration during food deprivation and satiation. *Pharmacology, Biochemistry, and Behavior, 2*, 589-596.

Obernier, J. A., White, A. M., Swartzwelder, H. S., & Crews, F. T. (2002). Cognitive deficits and CNS damage after a 4-day binge ethanol exposure in rats. *Pharmacology, Biochemistry, and Behavior, 72,* 521-532.

Ono, K. (1987). Superstitious behavior in humans. *Journal of the Experimental Analysis of Behavior, 47*, 261-271.

Pear, J. J. (1985). Spatiotemporal patterns of behavior produced by variable-interval schedules of reinforcement. *Journal of the Experimental Analysis of Behavior, 44,* 217-231.

Pisacreta, R. (1998). Superstitious behavior and response stereotypy prevent the emergence of efficient rule-governed behavior in humans. *Psychological Record, 48*, 251-274.

Reberg, D., Mann, B., & Innis, N. K. (1977). Superstitious behavior for food and water in the rat. *Physiology and Behavior, 19*, 803-806.

Reeve, J. (2001). *Understanding motivation and emotion.* Orlando: Harcourt College Publishers.

Rintala, J., Jaatinen, P., Lu, W., Sarviharju, M., Eriksson, C.J.P., Laippala, P. et al. (1997). Effects of lifelong ethanol consumption on cerebellar layer volumes in AA and ANA rats. *Alcoholism: Clinical and Experimental Research, 21*, 311-317.

Roberto, M., Nelson, T.E., Ur, C.L., & Gruol, D.L. (2002). Long-term potentiation in the rat hippocampus is reversibly depressed by chronic intermittent ethanol exposure. *Journal of Neurophysiology, 87*, 2385-2397.

Roy, A., & Pandey, S. C. (2002). The decreased cellular expression of neuropeptide Y protein in rat brain structures during ethanol withdrawal after chronic ethanol exposure. *Alcoholism: Clinical and Experimental Research, 26*, 796-803.

The Secretary of Health and Human Services. (1993). *Eighth special report to the U.S. Congress on alcohol and health,* Alexandria, VA: Author.

Strongman, K. T. (1984). Replacement, a theory of stereotypy: a review. *Current Psychological Research & Reviews,* 72-83.

Timberlake, W., & Lucas, G. (1985). The basis of superstitious behavior: Chance contingency, stimulus substitution, or appetitive behavior? *Journal of the Experimental Analysis of Behavior, 44,* 279-299.

White, A. P., & Liu, Z. (1995). Superstitious learning and induction. *Artificial Intelligence Review, 9,* 3-18.

APPENDIX

EMPORIA STATE UNIVERSITY

1200 Commercial
Emporia, KS
66801-5087

316-341-1200
www.emporia.edu

April 24, 2003

Mr. Robert Buford
Department of Psychology & Special Education
Emporia State University

Mr. Buford,

Please be informed that the ESU Animal Care and Use Committee has received and reviewed your Application for Approval to Use Vertebrate Animals in Research and Training entitled *An Examination of the Effects of Chronic Alcohol Exposure on Superstitious Behavior in the Rat* (ESU-PROTOCOL-03-004).

The committee has determined that this protocol is in compliance with currently applicable standards for such studies as specified by Federal Regulations and ESU policy and therefore is approved. We request that you submit a notice of completion shortly after you have concluded the project (i.e., a letter stating that the project has been completed, how many animals were actually used, and the manner in which they were disposed of). Please be aware that if any changes are to be made to the study the ACUC should be notified before-hand, and if the project needs to be extended beyond the proposed ending date you will need to submit a letter to the ESU ACUC requesting an extension. Your approval number for this project is ESU-ACUC-03-004. Also, as a reminder, please label all cages housing animals used solely for this project with this protocol number.

Best wishes with your research, and if you have any questions please contact me at your convenience.

Sincerely,

Mark Runge, Vice Chair
Animal Care & Use Committee

Permission to Copy Page

I, Robert L. Buford III, hereby submit this thesis to Emporia State University as partial fulfillment of the requirements for an advanced degree. I agree that the Library of the University may make it available for use in accordance with its regulations governing materials of this type. I further agree that quoting, photocopying, or other reproduction of this document is allowed for private study, scholarship (including teaching) and research purposes of a nonprofit nature. No copying which involves potential financial gain will be allowed without written permission of the author.

Robert L. Buford III

Signature of Author

An Examination of the Effects of Chronic Alcohol Exposure on Superstitious Behavior in the Rat

Title of Thesis

Signature of Graduate Office Staff Member

12/12/2003

Date