

THE ACUTE EFFECTS OF ALCOHOL ON ATTENTION USING THE ATTENTION NETWORK TEST

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By

Shayma M. Khalil

Director: Dr. David McCord
Head, Department of Psychology

Committee Members: Dr. Hal Herzog, Psychology
Dr. Alvin Malesky, Psychology

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ABSTRACT

THE ACUTE EFFECTS OF ALCOHOL ON ATTENTION USING THE ATTENTION NETWORK TEST

Shayma M. Khalil, MA

Western Carolina University (November 2009)

Director: Dr. David McCord

The body of research on alcohol and its effects on the brain and human body is extensive. Recent advances in neuroimaging have led to a better understanding of the underlying physiology of attention coupled with new innovations in methods to measure the construct, have made the pursuit of research on Attention even more accessible. Using the Attention Network Test (ANT), this study investigated the acute effects of alcohol on the three neuronal networks within the brain associated with attention: Alerting, Orienting, and Executive Control. Little is known about whether similar observations as those reported in the literature on the acute effects of alcohol on attention can be made using the ANT. College students (n=11) from the Western Carolina University Community were administered the ANT during three test sessions. The first session was a baseline assessment, followed by two sessions in which the participant received either a placebo or an active dose of alcohol (.6g/kg). Results indicate that a moderate dose of alcohol had no effect on Alerting or Orienting. However, this dose did impair individuals' Executive Control. These results illustrate the relatively specific effect of a moderate dose of alcohol on frontally mediated Executive Control. These findings are especially important when we consider the many functions of the frontal cortex and the importance of Executive Control in complex human cognition and behavior.

INTRODUCTION

Alcohol is the mostly widely abused drug in the US and is the second most used drug next to caffeine (Meyer & Quenzer, 2005). In 2001, the Center for Disease Control (CDC) estimated Alcohol Attributed Deaths (AAD's) at 75,766 for the year, 54 percent of which were attributed to acute conditions (Center for Disease Control [CDC], 2004). In 2007, the National Highway Traffic Safety Administration (NHTSA) reported that 37% of all fatal traffic accidents were alcohol related (National Highway Traffic Safety Administration, 2007). Hingson, Heeren, Zakocs, Kopstein, and Wechsler (2002) found that nearly 1,400 alcohol-related fatalities occur among college students per year with approximately 1100 of those being traffic-related.

Research suggests that consumption of alcohol is initially accompanied by a decrease in attention (Mukherjee, Das, Vaidyanathan, Vasudevan, 2008). Even when Blood Alcohol Concentration (BAC) is decreasing towards baseline levels following alcohol consumption, it was demonstrated that decision-making ability does not recover as does response speed (Schweizer, Jolicoeur, Vogel-Sprott, and Dixon, 2004). This finding is especially disturbing when a person who has been drinking considers driving when they feel sober and have regained full motor functioning. The consumption of alcohol not only impairs a person's ability to identify performance error, it also negatively impacted their ability to adjust or correct for those errors (Ridderinkhof, Vlugt, Bramlage, Spaan, Elton, Snel, and Band, 2002). If one uses the driving example again, it is easy to see how this detail could have very grave implications.

Alcohol rapidly passes through the blood-brain barrier and affects human behavior by altering the release of various neurotransmitters such as glutamate,

dopamine, and Gama-aminobutyric-acid (GABA) and by altering the sensitivity of their respective receptors as in the glutamate receptor N-methyl-D-aspartate (NMDA) and GABA receptor (Meyer & Quenzer, 2005). However, relatively little is known about the alterations in cognition that result from the pharmacological effects of alcohol and how these in turn affect behavioral control (Vogel-Sprott, Easdon, Filmore, Finn, & Justus, 2001).

Alcohol has adverse effects on attention. More specifically, alcohol has been shown to have a different effect on the various subtypes of attention (Goodwin, Othmer, Halikas, & Freemon, 1970; Koelega, 1995; Minocha, Barth, Roberson, Herold, & Spyker, 1985; Nichols & Martin, 1996; Weissenborn & Duka, 2003). Recent advancements in neuroimaging have allowed researchers to identify three neuronal networks in the brain associated with three aspects of attention. The Attention Network Test (ANT) has provided researchers with a fast and relatively simple means of studying these three aspects of attention simultaneously: Alerting, Orienting, and Executive Control. This task, however, has never been used to study the acute effects of alcohol. This study examined the effect of alcohol on the three aspects of attention mentioned above, which are known to be mediated by different neural substrates.

LITERATURE REVIEW

Attention

Attention is the capacity of an organism to respond selectively to certain stimuli among other stimuli (Concise Dictionary of Psychology, 1998). Attention has been a popular subject of study since the late 1800's but had a resurgence of popularity after the behaviorist movement in the 1950's. However, it has been defined in multiple ways depending upon the context in which it is being used. To add to the confusion, other names such as concentration, consciousness, perception, awareness, set, vigilance and various other terms have been used to either describe attention, forms of attention, or as a supplement to what is essentially a selectivity of processing. Ambiguity in terminology complicated this area of study further, not only in the differences of the use of such terms but also in each of their meaning (Gregory, 2004; Milton, 1994).

The most widely accepted definition comes from William James (1890) who stated "Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of several simultaneously possible objects or trains of thought. Focalization, concentration of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others" (James, 1890, pp. 403-404). Milton (1994) points out that this definition brings to light an important problem within the area of attention, its relationship to consciousness. He blames the behaviorist movement of intentionally ignoring the unconscious, consequently obscuring the study of attention, which acts largely in the preconscious and unconscious domain (Milton, 1994; Raz, 2004).

Attention has been applied to the concept of state of arousal, also referred to as

implicit interest (Berlyne, 1960). Eysenck and Keane stated that “there is an obvious danger that a concept that is used to explain everything will turn out to explain nothing” (Eysenck & Keane, 2002, p. 363). For the sake of clarity, I investigated the acute effect of alcohol on three independent attention processes (Alerting, Orienting, and Executive Control) known to be mediated by independent networks in the brain, rather than a single attention construct.

Early Research on Attention

Much research has been devoted to studying how attention relates to behavior. James, Titchener, Wundt, and W.B. Pillsbury studied attention in terms of its relationship to consciousness in the early 1900’s. Titchener studied prior entry, binocular rivalry, and fluctuations of attention. He viewed attention as an attribute of sensation and found that if attention is diverted to one out of two stimuli produced at the same time, the one that attention was paid seemed to occur first (Titchener, 1908). Pillsbury studied what is now known as divided attention and found that the capacity for which stimuli can be attended varied in every respect including within and among individuals, from trial to trial, the stimuli itself, and its location in the visual field (Pillsbury, 1908). His 1908 book, *Attention*, included many observations that were later confirmed by more contemporary research and is still frequently referenced to this day.

The ability to do two things simultaneously was demonstrated as early as 1887 by a French scholar who demonstrated that a person could recite a poem while simultaneously writing a different poem (Gregory, 2004). In 1935, Stroop demonstrated that when presented with written names of colors that are different in color than the text presented, a person could typically recite the written word despite its color discrepancy.

Conversely, when asked to recite the color of the word while ignoring the written text, participants found it nearly impossible (Stroop, 1935). The difference in reaction time (RT) between these two tasks was thought to be representative of the amount of interference in attention.

The behaviorist movement caused a sharp decline in the study of attention because it was deemed a mental phenomenon that could not be overtly studied. Because attention is an unobservable cognitive process, there was a decrease in research on the vague features of attention such as those that dealt with the details of consciousness and sensation that required the use of introspection, a process deemed unreliable for scientific inquiry (Paschal, 1941). Meanwhile there was an increase in research on the “observable” aspects of attention that require experimentation. Research on overt behavior was thought to be more credible because it could be observed and thus, easier to measure. Research on the observable aspects of attention such as the activity of attending worked to strengthen ideas about the importance of the internal mechanisms involved in responding (Gregory, 2004).

In the late 1950's, research on the selectivity of processing integrated psychology with information processing concepts of computer science (Itti, Rees, & Tsotsos, 2005). Broadbent's (1958) filter theory examined information overload and proposed that human cognitions were limited-capacity information processing systems. One technique that provided evidence for a limited-capacity information processing system, the split span, found that when a listener was required to recall digits read three at a time at the rate of two words per second simultaneously in each ear, they remembered the digits that were read in each ear as a group rather than by pairs of digits. He tried to measure what he

believed to be a switching of auditory attention by examining the rates at which the pairs of numbers could be recalled (Broadbent, 1958).

Broadbent's filter theory dominated this area of research. A succession of studies tried to modify his work to explain why this filter did not successfully block all the information and that selection could be extended beyond only the sensory system. Attention began to be thought of as acting in various different ways, at different levels, and on a variety of operations in the nervous system (Gregory, 2004).

A Shift to Psychophysiological Perspectives

Consistent with the trends towards studying measurable behavior is the study of the physiology of attention as it relates to the brain. Only recently with the technological advances in science, have we been able to increase our understanding of the neural mechanisms of attention. Researchers have also examined the neuroanatomy, neurophysiology, and neurochemistry of attention (Itti et al., 2005). In the 1970's, the P-300 component of the brain-evoked potential was used to gain a better understanding of the physiological aspect of attention. Donchin and colleagues demonstrated the role of brain-evoked potentials in decision-making and attention (Gregory, 2004). Neuroimaging has allowed us to view the anatomical areas involved in attention, while the Human Genome Project has enabled us to isolate the genes involved in these networks (Raz, 2004).

Some researchers now define attention in terms of the underlying organ system involved. This physiological definition is an attempt to make the complexities of this cognitive structure and its functions more tangible (Posner & Fan, 2004; Raz, 2004). According to Raz (2004) "as with other biological systems, attention has a distinct

anatomy that carries out basic psychological functions” (Barnea, Rassis, Raz, Othmer, & Zaidel, 2004, p. 21). Specifically, it has been found that three independent neuronal networks in the brain relate to three unique aspects of attention: Alerting, Orienting, and Executive Control (Fossella, Posner, Fan, Swanson, & Pfaff, 2002).

Cognitive Types

Posner (1995) described the multiple facets of attention as serving three basic functions: maintaining an alert state, sensory attention comprised of orienting and stimulus evaluation, and executive attention or cognitive control. Fan et al. (2002) defined Alerting as achieving and maintaining an alert state. This is similar to the idea of vigilance. Orienting is the ability to select information from sensory output and as the ability to shift attention from one stimulus to another. It is in this process of disengagement, then shifting, and re-engagement that researchers have been able to locate neuronal networks acting together for one operation (Posner & Fan, 2004). Executive Control is the ability to resolve disagreement among responses and most resembles the idea of selective attention (Posner & Fan, 2004).

Selective attention has been the topic of much research. In an unpublished manuscript, J.J. Gibson (1974) explained that studies in this area focus on the idea that an individual will make a choice that is followed by an action that is relevant to what has been asked of them and their subsequent choice. He wrote on attention, “It is a circular process of input and output combined, where the input is modified by the output as well as the output being dependent on the input, not a linear process where each stage of the input follows a previous stage” (Gibson, 1974).

The idea that these three facets of attention work as networks within the brain is

highlighted by Posner and Fan (2004). They noted that brain lesion studies from the 1980's showed that extinction was the result of the action of a neuronal network because separate damage could produce the same local outcome. Thus, in order to execute a task, the brain has to coordinate with various networks within the brain even though a mental process may function locally (Fan, McCandliss, Fossella, Flombaum, & Posner, 2005; Fan & Posner 2005; Posner, 1988; Posner & Fan, 2004). Neuroimaging has been shown that cognitive functioning even in a single task is distributed among various sensory processing regions of the brain and in most cortical areas (Itti et al., 2005; Posner & Raichle, 1994).

The neuroanatomy of Alerting. The Alerting network produces the vigilant state that allows for processing of an event (Fan et al., 2002). The frontal and superior parietal lobe of the right hemisphere have been linked to the Alerting system (Fan et al., 2002). Alertness, sometimes referred to as arousal, is studied by using warning signals prior to the presentation of a target stimulus. This warning signal produces an effect on the level of alertness that has been linked to the norepinephrine system in a pathway from the locus coeruleus (Fan et al., 2005; Fan & Posner, 2004; Fossella et al., 2002).

The neuroanatomy of Orienting. The Orienting network allows for selectively allocating attention to a specific stimulus while shifting focus of attention from one location to another (Fan et al., 2002). The superior parietal lobe has also been linked to the Orienting system, as has the temporal parietal junction and the frontal eye fields (Fan et al., 2005; Fan et al., 2002; Fan & Posner, 2004; Fossella et al., 2002). Orienting is studied by presenting a cue that indicates or directs the persons gaze to the cued location (Fossella et al., 2002). Lesion studies of the parietal lobe and superior temporal lobe

produce difficulties in Orienting (Posner & Fan, 2004). Subtle deficits in Orienting tasks were also linked to an area on the thalamus, specifically, the pulvinar. This area may be indicated in information processing as it relates to shifting attention in the combined functions of voluntary eye movement and processing (Posner & Fan, 2004). This system also involves cholinergic pathways (Posner, 1995).

The neuroanatomy of Executive Control. The Executive Control network selectively attends to a stimulus while resolving conflict among competing responses to that stimulus (Fan et al., 2002). The Executive Control system is associated with the midline frontal areas of the brain such as the anterior cingulate and the later prefrontal cortex involving the dopaminergic pathways (Bush, Luu, & Posner, 2000; Fan & Posner 2004; Posner, 1995). This function of attention is typically studied by using conflict as in the Stroop test. This resolution of conflict activates these regions of the brain (Posner & Fan, 2004; Fan & Posner, 2004).

Attention Network Test

The Attention Network Test (ANT) developed by Fan and associates (2002) is a computerized task designed to simultaneously measure the three functions of attention and to evaluate their interrelationships (Fan et al., 2002). This task requires that participants determine whether the target stimulus is a left or right arrow and respond to it in a congruent manner by pressing either a left or right arrow on the keyboard. The ANT combines two previous measures of attention, Posner's 1980 cued reaction time and the flanker task (Eriksen & Eriksen, 1974). Reaction time or response time (RT) is a measure of the time it takes to respond to the target stimulus. RT is often used as a means of measuring the covert aspect of Orienting since it is not directly observable (Itti et al.,

2005). A flanker is a signal that is simultaneously presented with the target stimulus and used to distract attention from the target stimulus.

The ANT does not require the use of language and can thus be operated by monkeys, children, and adults. It only requires approximately 30 minutes for administration (Fan et al., 2002). Response time to a signal is measured for each of the three networks involved in Alerting, Orienting, and Executive Control. The effectiveness of the Alerting network is measured by subtracting RT of the cued trial from those of an uncued trial. Orienting is measured by comparing the RT of trials in which a warning signal marks the appearance of the target to those in which a target appears with no warning signal. The Executive Control network is examined by comparing RT of tasks involving conflict in response to incongruent flankers surrounding the target stimulus, to those with no conflict and involves congruent flankers (Fan et al., 2002). Flankers are used to distract the participant from the target stimulus, for example, incongruent tasks require a participant to respond to the target stimulus while the distracting flankers signal an opposite response (Fossella et al., 2002).

In an attempt to study the anatomy, circuitry, pathology, and development of the three attentional networks, Fan and Posner (2004) used functional magnetic resonance imaging (fMRI), event related potentials (ERP), genetic, and computational models. Fan et al. (2002) found that efficiency estimates of the three attention networks were uncorrelated. This suggests functional independence of the networks and that the three sub-types of attention are mediated by different neuronal networks (Fan et al., 2005; Fan & Posner, 2004). This functional independence may help distinguish the acute effects of alcohol on each subtype of attention as they relate to each neuronal network.

Although Corballis and Gratton's (2003) demonstrated that the Orienting function alone may be unitary, their findings also support the use of a non-unitary measure such as the ANT in studying the organization of information processing strategies. They found that selection strategy is not a unitary function by demonstrating that respondents adjusted their processing strategies to flanker tasks independently. This is apparent in the differences in response reaction time to target stimuli in the left visual hemifield, right visual hemifield, and lateral locations, dependent upon expectancies for compatibility and incompatibility of flankers or warning signals (Corballis & Gratton, 2003). This observation is important in light of new findings that attention works through three separate neuronal networks rather than the old methodology of viewing it as one function of the mind.

Some researchers found differences of covert Orienting between visual fields. The interaction or balance of both hemispheres by joint inhibitory processes suggests rivalry may take place between the hemispheres in regards to Orienting attention (Schulte, Müller-Oehring, Strasburger, Warzel, & Sabel, 2001). This work forms a nice foundation for Corballis and Gratton's (2003) findings that suggest a difference between hemispheric brain function and attention. The notion that if this one aspect of attention can function in this capacity further propels the idea that there are various subtypes of attention, and that within each subtypes, various processes must occur in order to carry out the functions.

Fan, Wu, Fossella, and Posner (2001) used the performance of monozygotic and dizygotic twins on attention tasks to show that different networks were inherited differently. A study of selective impairment of attentional networks in schizophrenia found that there was even specificity in the deficits observed among the networks, when

their results indicated deficits in Executive Control and the Orienting networks (Wang et al., 2005). Characteristic traits inherited with such specificity as well as selective impairments with some disorders, offer even further evidence that these attentional networks function independently. Functional magnetic resonance imaging (fMRI) studies demonstrated that tasks of attention differentially activated separate anatomical networks, and support the theory of attentional networks (Fan et al., 2005).

Translating Attention

It has been difficult to parse out the many different terms used in attention literature and research. Some of these constructs have more recently been defined in terms of their underlying physiology and anatomical structures. This is precisely why the work of Michael Posner, Jin Fan, and others is so revolutionary. Michael Posner wrote “just as DNA research changed how we thought about life but did not really clarify the meaning of life, so the study of the anatomy, cellular structure, and genetics of attention but has allowed us to view attention like any other system but not change its definition” (Posner, 2004, p. 3).

The terms Alerting, Orienting, and Executive Control as used by Michael Posner and Jin Fan are terms that don't have many or any real synonymous terms in attention literature. It seems that previous terms such as selective attention as measured by tasks such as the Stroop and cognitive control are pieces of what is referred to here as the aspect of Executive Control, which are known to be connected through the same frontally mediated structures of the brain. Terms such as arousal, vigilance, and concentrated attention have been used somewhat interchangeably with the term Alerting. Although discussed using somewhat similar terminology, Orienting has been separated in

the literature into two further categories, that of overt and covert Orienting. The ANT looks at covert Orienting, as this does not involve head and/or eye movement (Fan & Posner, 2004).

Walsh (2004) states that “attention has unrestrained explanatory range; it is freely used for any side of any argument. Like the cosmological ether of pre-20th- century physics, it is everywhere, but in the end, nowhere” (Posner, 2004, p. 24). This illustrates the difficulties in making exact inferences on previous research although seemingly tackling similar constructs.

Alcohol

Pharmacology

The term alcohol refers to ethyl alcohol or ethanol and is classified as a psychoactive drug that acts similarly to sedative-hypnotic compounds or depressants (Hobbs, Rall, & Verdoorn, 1996). The difference with alcohol is that it is used for recreation rather than medical purposes. Alcohol is one of the most widely used psychoactive drugs in the United States, second only to caffeine (Meyer & Quenzer, 2005). It is important to understand how alcohol affects our body based on this fact alone. Many factors are involved in the rate of absorption, distribution, and clearance of alcohol following consumption.

Since alcohol is absorbed through the gut and distributed via the blood stream, a convenient measure of intoxication is the amount of alcohol in a person's blood. This is known as the blood alcohol concentration (BAC). The concentration depends on body weight, the quantity and rate of alcohol ingestion, and the rates of alcohol absorption and metabolism (Meyer & Quenzer, 2005). BAC is measured and reported as weight of

ethanol, typically in grams, per 100 mL of blood. Blood alcohol concentration is characterized by the behavioral effects correlated with varying levels of intoxication (Meyer & Quenzer, 2005). A conceptual view of how BAC affects a person is provided by Kowalski (2001) who reported that: reasoning ability is negatively affected and drinkers are less inhibited with a BAC of 0.04; drinkers may become violent, anxious, or depressed with a BAC of 0.16; and with confusion and stupor setting in at BAC's of 0.25 and over, drinkers risk coma and death with a BAC above 0.35. One can easily start to see how the legal driving limit of .08 is derived.

Meyer and Quenzer (2005) provide a nice descriptive overview of alcohol metabolism. The process by which alcohol passes from the stomach and intestines into the blood is called absorption. A large portion of the alcohol that is consumed is metabolized by a variety of enzymes in the liver, a process much slower than absorption. More specifically, alcohol dehydrogenase (ADH) converts alcohol to acetaldehyde which is then converted to acetic acid by way of acetaldehyde dehydrogenase. After acetaldehyde is converted into acetic acid, it is then ultimately metabolized into carbon dioxide and water (Meyer & Quenzer, 2005). Although the rate of metabolism varies across individuals, the rate of oxidation is constant and does not increase with a greater BAC. The average rate of metabolism is approximately 12 ounces of beer, 5 ounces of wine, and 1.5 ounces of 80-proof alcohol in about 30 to 45 minutes. An individual becomes intoxicated when the rate of consumption exceeds the constant metabolic rate and alcohol accumulates in the body (Meyer & Quenzer, 2005).

Meyer and Quenzer (2005) explain that in the case of chronic alcohol use, an individual may build a tolerance to the drug, in which increased doses are necessary to

achieve previous levels of intoxication. This is due to the availability of more alcohol dehydrogenase and liver microsomal enzymes that metabolize alcohol. The more rapid metabolism of alcohol results in a decrease of blood alcohol levels, and thus, less of an intoxicating effect (Meyer & Quenzer, 2005). Tolerance is one of the criteria for alcoholism designated by the American Psychiatric Association's Diagnostic and Statistical Manual IV Text Revision (*DSM IV-TR*, 2000). For these reasons, chronic alcohol users were excluded from this study.

Acute Effects

Alcohol acts as a central nervous system depressant by interfering with communication between neurons within the brain, decreasing the activity of excitatory pathways (e.g., NMDA/glutamate) and increasing activity of inhibitory pathways (e.g., GABA) (Meyer & Quenzer, 2005). The inhibition of the excitatory pathway is responsible for what may wrongly appear to be the stimulating effects of alcohol, because people might do things they don't normally do in what has been described as a euphoric state (Mukherjee et al., 2008). The reason alcohol appears to be a stimulant is because it suppresses activity in those parts of the brain that control inhibition. When a person drinks, those areas of the brain that prevent a person from acting foolishly are essentially shut down (stimulation) and may be reinforcing to an individual. Excitatory neurotransmitters (e.g., glutamate) activate those inhibitory control centers in the brain. Research on reinforcement demonstrates the involvement of the dopamine system with regards to most drugs. Regulation of the dopamine neurons by various neurotransmitters is what indirectly connects these processes (Ramchandani, 2000).

Relevant neurotransmitters

Due to the undisputed negative consequences of alcohol intoxication, much research has been devoted to examining the effects of alcohol on the brain. More specifically, research has focused on the effects of alcohol on the neurotransmitters that are critical in cognition and control subsequent behavior and performance. Alcohol affects most neurotransmitter systems in the brain in some way (Ramchandani, 2000). Glutamate, gamma-aminobutyric acid (GABA), and dopamine are the most widely discussed neurotransmitters in the alcohol literature (Meyer & Quenzer, 2005). Sometimes, however, it is more appropriate to discuss the affects of alcohol at the receptor site where alcohol has its most deleterious effects such as in the case of N-methyl-D-aspartate (NMDA), a brain receptor of glutamate.

Glutamate. Glutamate or glutamic acid is one of the most common neurotransmitters found in the brain and has receptors on many cells (Meyer & Quenzer, 2005). Glutamate is an excitatory neurotransmitter that alcohol negatively affects by impairing its effectiveness at a specific receptor known as N-methyl-D-aspartate (NMDA) (Gonzales & Jaworski, 1997). Glutamate is thought to play a role in learning and memory (Meyer & Quenzer, 2005). An excitatory rebound of glutamate at the NMDA receptor occurs when alcohol leaves the system and glutamate is no longer suppressed. The result is excessive glutamate activity called excitotoxicity a process that also occurs after stroke and prolonged seizure activity (Meyer & Quenzer, 2005). Excitotoxicity is also associated with brain damage (Kolb & Whishaw, 2003; Meyer & Quenzer, 2005).

The glutamate receptor NMDA is known to play a large role in synaptic plasticity,

the brain's mechanism for learning and memory (Meyer & Quenzer, 2005). This is likely the reason why glutamate is associated with these functions. The excitotoxicity that occurs at the NMDA receptor site plays an important role in neuronal loss resulting in cognitive deficits (Barinaga, 2000).

Gamma-aminobutyric acid (GABA). GABA is the other major amino acid neurotransmitter in the brain. Alcohol enhances the effects of this inhibitory neurotransmitter at the modulatory sites of one of its receptor complexes (Meyer & Quenzer, 2005). This response has been linked to neuronal loss resulting in alcohol-related cognitive deficits (Barinaga, 2000). GABA is a mood modulator that aids in reducing anxiety and stress. It also regulates norepinephrine and dopamine whose roles are discussed in the next section. One study found that GABA agonists could enhance ethanol-induced sedation, which may be due to the increased sensitivity of GABA receptors (the site at which sedative hypnotics and antianxiety drugs act) following the introduction of alcohol (Frye & Breese, 1982; Kolb & Whishaw, 2003; Meyer & Quenzer, 2005).

Support for alcohol's influence on GABA receptors involved spatial cueing tasks with monkeys. Robinson and Petersen (1992) found effects of chemically induced unilateral deactivation of the pulvinar dorsomedial region (PDM). This is the same region identified as being an integral part of the Orienting network as it relates to voluntary eye movement and processing. Monkeys had difficulty shifting attention to the contralateral field when injected with a GABA agonist, similar to the enhancing effect of alcohol on GABA. Conversely when injected with a GABA antagonist, the monkey could shift attention more easily to the contralateral field (Robinson & Petersen, 1992).

Dopamine. Meyer and Quenzer (2005) describe the dopaminergic systems as part of the internal neural reward circuit in the brain. Dopamine is also one of the neurotransmitters associated with attention especially clinically with disorders of attention when dopamine levels are too high as in ADHD and schizophrenia (Fan & Posner, 2004; Wang et al., 2005; Wang & Fan, 2007). More specifically, dopamine synthesizes norepinephrine which is classified as both a neurotransmitter and a hormone that affects parts of the brain associated with attention (Meyer & Quenzer, 2005). Norepinephrine works in a neurotransmitter system that effects large areas of the brain and plays a role in the alerting function and has an impact on the reward system (Meyer & Quenzer, 2005; Fan & Posner, 2004). Fan and Posner (2004) explain that theories of attention deficit disorders imply deficits in executive functions, however, both previous and recent work have shown greater deficits in the Alerting function. The relationship between these deficits and the norepinephrine system, known to be involved with the Alerting function and dopamanergic pathways, thought to be involved with the executive function are areas for further research and consideration.

The dopaminergic pathway is often discussed in the alcohol literature in terms of drug abuse and addiction because of the positive reinforcing and often addictive affect drugs have on this reward system (Julien, 1998). Although literature on the acute effects of alcohol suggests that ethanol activates the mesolimbic dopamine system, the mechanism by which this occurs is still unclear (Schmidt, Smolka, & Rommelspacher, 2003).

Attention, Alcohol, and Performance

Alcohol impacts performance on tasks that require a participant to divide their attention between stimuli before responding (Dougherty, Marsh, Moeller, Chokshi, & Rosen, 2000; Koelega, 1995). Tasks of divided attention have been used to measure Alerting and have consistently shown sensitivity to the acute effects of alcohol. However, research has not shown consistent results for tasks that require sustained attention which is also a measure of an alert state (Koelega, 1995). Some researchers have concluded that tasks of sustained or concentrated attention are less sensitive to the effects of alcohol than more complex tasks of dividing attention.

Dougherty et al. (2000) studied the effect of alcohol on concentrated attention (Alerting), impulsivity, discriminability, and response bias in immediate and delayed memory tasks. Their focus on concentrated attention was in response to previous studies that suggested tasks that require vigilance (alert state), were not affected or only minimally affected by alcohol. They demonstrated that alcohol indeed impacted performance in all areas studied. In the alcohol groups; performance error increased, commission errors increased, and discriminability decreased. These researchers also found minimal change in the moderate dose group across the BAC curve but found significant changes in performance with the high alcohol dose group. They concluded that previous findings concerning vigilance may have been due to using measures that were less sensitive in detecting impaired responding (Dougherty et al., 2000).

The body of research on alcohol is immense. There are countless theories on how humans are affected by alcohol. Josephs and Steele (1990) cited several studies that found alcohol had very little effect on practiced tasks that may require little to no

attention. However based on their attention allocation model, Josephs and Steele (1990) conducted two studies on how alcohol effects attentional processes with the idea that attention is allocated differently according to the task. The attention allocation model is based on the premise that alcohol negatively impacts performance in regards to tasks that require a lot of attention and control processing. In addition, the reduction in attention makes it possible to attend to only the most salient cues (Josephs & Steele, 1990). Researchers have based this theory on what they have termed alcohol myopia, an impairment of perception and thought, which results in a narrow view in which one can only process minimal cues and not very well (Josephs & Steele, 1990). This finding helped strengthen our understanding of the integral role of attention and behavior and lead to further research on how alcohol restricts focus of attention.

In a related model based on cognitive control, Fillmore and Vogel-Sprott (1999) argued that alcohol impairs response inhibition rather than attention. They found that alcohol impairs a person's ability to inhibit a response and alternately found no affect on their ability to activate behavior. This means a person's ability to stop a behavior is impaired while their ability to initiate behavior is unaffected. Responding in such a manner is often characterized as impulsive, a common symptom of intoxication. This response pattern is called the alcohol disinhibition model (Fillmore & Vogel-Sprott, 1999).

One study investigated the acute effects of alcohol on cognition and performance based on both the attention-allocation model and response inhibition model. Both of these use tasks that require attentional control and require subsequent adjustment of behavior (Bartholow, Pearson, Sher, Wieman, Fabiani, & Gratton, 2003). After randomly

assigning participants to placebo, moderate doses of alcohol (0.40 g/kg), and high doses alcohol (0.80 g/kg), behavioral data on a modified flanker task supported previous findings that alcohol impairs response inhibition (Bartholow, et al., 2003). Consistent with previous research on divided attention tasks, these researchers also found that distraction is actually reduced under low doses of alcohol. It seemed that alcohol interfered with response selection rather than attentional processes. If viewed differently, however, the results could actually support the attention allocation model. Since responses of those who drank the most alcohol were more affected by manipulation of flankers, it can be argued that there was either an increase in response conflict or that flankers were more salient so more attention was paid to them.

Response error and deficits in reaction time are a common theme among research on the acute effects of alcohol. It makes intuitive sense for both of these processes to be negatively impacted by alcohol. However, recent research shows that reaction and response time may not always be affected the way that we think. Response reaction time may not actually increase. This is due to the characteristic impulsivity or response disinhibition of a person under the influence of alcohol (Dougherty et al., 2000; Schweizer et al., 2004).

Wiessenborn and Duka (2003) investigated the acute effects of alcohol on tests of planning, spatial working memory, and spatial and pattern recognition tasks. They found that alcohol decreased the participant's ability to solve problems, decreased response time (indicating impulsivity), and decreased item recognition within the spatial recognition task. These researchers concluded that larger doses of alcohol impair executive-type cognitive functioning (Wiessenborn & Duka, 2003). Ridderinkhof et al. (2002) provided

additional support with their research on the anterior cingulate cortex. They found that even moderate doses of alcohol interfered with a person's ability to detect response error followed by a decreased ability to make adjustments in response to these errors. Another study demonstrated that the acute effects of alcohol impairs risky decision making by impairing one's ability to change responses in accordance to changes in prospective rewards (George, Rogers, & Duka, 2005).

Schweizer et al. (2004) reported that speed and accuracy were effected in different ways during the ascending and descending limb of the blood alcohol content (BAC) curve. The ascending limb of the BAC curve is the phase in which a person's BAC increases resulting in intoxication after the consumption of alcohol. Cessation of alcohol consumption and subsequent decrease in BAC levels during the descending limb of the BAC curve results in sobriety. These researchers found that although reaction time decreases due to impulsivity of response it recovers early during the descending limb of the BAC curve. However, response error did not recover as quickly in the descending limb of the alcohol curve (Schweizer et al., 2004). These findings have very important implications for purposes of practicality in which a person may be able to recover in terms of their processing speed but not the accuracy of response. An important consideration is how long it takes to recover these processes and their long-term effects on cognition.

Pihl, Paylan, Gentes-Hawn, and Hoaken (2003) studied the BAC curve in terms of how executive cognitive functioning is affected differently along this continuum. They found that cognitive functioning such as planning, abstract reasoning, and governance of self-directed behavior as they relate to the prefrontal cortex and the temporal lobe were

negatively impacted by the affects of acute alcohol intoxication. Greater impairment in functioning was found during the descending limb of the BAC when compared to functioning during the ascending limb (Pihl et al., 2003).

In regards to visual Orienting of attention, King and Byars (2004) compared participants given high (0.8 g/kg) and low (0.4 g/kg) doses of alcohol and found that heavy and light drinkers showed slowing of psychomotor processing and encoding. Slowing of saccadic eye movement was also found. They attributed this to the idea that alcohol is thought to impair saccadic eye movement by increasing the time the subject disengages attention from the fixation point and focus on the target (King & Byars, 2004). This finding may have an impact on response times in visual Orienting tasks with respect to visual attention. As mentioned earlier, Robinson & Petersen (1992) found that GABA agonists that function similarly to alcohol had a negative impact on Orienting in cued tasks with monkeys. Additionally, when studying the acute effects of alcohol on covert attention, Schulte et al. (2001) found that sensory-attention mechanisms were important in their effects on visual perceptual performance after consumption of alcohol. These findings have negative implications for the effect of impaired attention with regards to these three aspects of attentional function.

STATEMENT OF THE PROBLEM

There has been much research devoted to the topic of attention and the effects of alcohol on the brain and human body. To date, research shows that Alerting is far less sensitive to the effect of alcohol than more complex forms of attention. In addition, Orienting and related cognitive processes are somewhat more sensitive to the effects of alcohol than other forms of attention. However, the effects depend on the task and alcohol level administered. Finally, alcohol has a greater effect on the more complex tasks required to measure Executive Control and related cognitive processes.

Are these cognitive processes differentially affected by a single dose of alcohol? Little is known about whether the same observations on the acute effects of alcohol can be made using the ANT due to its relatively new arrival into the field (Fossella et al., 2002). I investigated the acute effects of alcohol on the three neuronal networks within the brain associated with attention: Alerting, Orienting, and Executive Control.

HYPOTHESIS

Measures of response time and accuracy have been shown to be sensitive to the acute effects of alcohol. The three aspects of attention measured by the ANT (Alerting, Orienting, and Executive Control) and acute alcohol consumption have never before been examined in a single task. The following hypotheses were based on the compilation of significant findings of alcohol and attention.

Hypothesis 1: *Consumption of alcohol will not have a significant effect on RT of Alerting.* This is based on the idea that various tasks that measure the vigilant state of Alerting such as those measuring RT are not as sensitive and thus, may not show an affect due to learning, practice affects, and the level anxiety invoked by the task (Koelega, 1995; Shulte et al., 2001). The attention allocation model and numerous studies have found minimal effect in this regard (Josephs & Steele, 1990; Koelega, 1995; Shulte et al., 2001).

Hypothesis 2: *Consumption of a moderate dose of alcohol will have a significant increase in RT for tasks that required a participant to orient their attention.* This was based on studies that have found a slowing of psychomotor processes and saccadic eye movement that work to impair response speed (King & Byars, 2004; Robinson & Petersen, 1992; Shulte et al., 2001). King and Byars (2004) attributed this increase in RT to the idea that alcohol is thought to impair saccadic eye movement by increasing the time the subject disengages attention from the fixation point and focus on the target.

Hypothesis 3: *Consumption of alcohol will significantly decrease RT for tasks that require Executive Control.* This was based on literature that states divided attention tasks are extremely sensitive to the effects of alcohol, perhaps due to the more complex

process that requires conflict resolution among competing responses (Fan et al., 2002; Fan & Posner, 2004; Koelega, 1995; Shulte et al., 2001). Based on the findings of several research studies that found a decrease in RT's due to impulsivity, a characteristic response to alcohol intoxication that produced shorter RT's (Dougherty et al., 2000; Schweizer et al., 2004; Vogel-Sprott et al., 2001).

METHOD

Participants

Eighteen male participants from the Western Carolina University community were recruited to participate in this study. Several participants were lost to attrition either after consent and baseline ($n=2$) or after the second session ($n=2$). During data analysis, it was found that ANT data for two other participants were not saved correctly and lost and one participant was excluded based on the discrepancy between his age (41 years old) and the age of the remainder of the sample. The final sample ($n=11$) consisted of graduate ($n=5$) and undergraduate ($n=6$) Caucasian male students from the WCU community. Participant age ranged from 21-27 (Mean= 23.5, SD= 1.92). Females were excluded due to lack of resources for providing pregnancy tests, an important and necessary precaution when administering alcohol to females. Each student received research credit and/or extra credit for participation in the study in addition to \$50 dollars for completion of the baseline and two experimental sessions.

In addition to alcohol and psychiatric screening measures, volunteers were excluded on the basis of self-reported alcohol and drug dependence, any report of current medical conditions, and diagnosis of mental illness. Participants were asked to abstain from the use of illicit recreational drugs, sleeping tablets, hay fever medication, medication that may potentially interact with alcohol, and medication classified as a narcotic for at least 48 hours prior to the experiment. Age, race, sex, and other general questions were also covered in the General Information form (General Information Form is included in Appendix A).

Participants were also asked to abstain from the use of alcohol for at least 24

hours prior to the experiment. In addition, participants were asked to abstain from eating and drinking on the mornings of the experiment, as they were provided with a light breakfast and would typically be released by lunch. Participants were excluded if the above guidelines were not met.

Measures

Attention Network Test (ANT)

This description is adapted from Fan et al. (2002). The Attention Network Test was created by Fan and colleagues to assess the attention networks involved in the three aspects of attention: Alerting, Orienting and Executive Control. The ANT was presented on a computer running Windows XP, on a 15-inch monitor. The participants viewed the screen from a 26 inch distance and responses were made in regards to the direction of the target stimuli (centrally located arrow) on the two input keys (left and right arrows). The current study deviated slightly from the keyboard arrows to a hand-held game controller by corresponding thumbs.

A session consists of a 24-trial practice block in which immediate feedback is given by a presentation of the word “correct” or “incorrect” after each response. The three experimental blocks of trials that follow do not provide such feedback. Each experimental block consisted of 96 trials (4 cue conditions x 2 target locations x 2 target directions x 3 flanker conditions x 2 repetitions). Trials were presented in a random order. Participants were instructed to focus on a centrally located fixation cross throughout the task, and to respond as quickly and accurately as possible. The practice block takes approximately 2 minutes and each experimental block takes approximately 5 minutes. Overall, completion of the ANT takes approximately one half hour, although, the

experimental session was extended until Blood Alcohol Concentration (BAC) reached baseline.

The target stimulus is either a black rightward or a leftward arrow centered against a lighter background. In the Executive Control condition, the target is flanked on either side by either two arrows in the same direction (congruent condition), or in the opposite direction (incongruent condition), or by lines (neutral condition) and each participant was asked to respond congruently to only the middle most arrow by pressing either the right or left arrow on the game controller with their corresponding thumb. Performance is measured by the time it takes to make each response (Response Time (RT) in milliseconds), in the congruent condition minus performance in the incongruent condition.

To measure Orienting and Alerting, targets were preceded by several cues; no cue, center cue, double cue, and a spatial cue. Neither spatial nor alerting cues were in the no cue trial. For the center cue trials, participants were shown an asterisk at the location of the fixation for 100 msec, alerting the participant to the next target stimulus. In the double cue trials, two warning cues were present for 100 msec corresponding to the two possible target locations, up or down. For the spatial-cue trials, the cue was at the target position and the time course was still 100 msec. The spatial cues are always presented at the exact locations of the targets. Alerting is measured by the difference in performance in the cued condition and performance in the no-cue condition. Orienting is measured by performance in the spatial cue condition minus performance in the center-cue condition. Previous studies found that both Alerting and Orienting are involved in the spatial cue condition (Fan et al., 2002).

Each trial was made up of five events. First, there was a fixation period for a random variable duration (400 to 1600 msec). Then, a warning cue was presented for 100 msec. There was a short fixation period for 400 msec after the warning cue and then the target and flankers appear simultaneously. The target and flankers were presented until the participant responds, but for no longer than 1700 msec. After participants make their response, the target and flankers will disappear immediately followed by a post-target fixation period for a variable duration, which is based on the duration of the first fixation and reaction time (3500 msec minus duration of the first fixation minus reaction time). After this interval the next trial began. Each trial lasts for 4000 msec. The fixation cross appears at the center of the screen during the whole trial. Orienting was examined by a component in which 5 stimuli were presented in one of two locations outside the fixation point and above or below the fixation point. Target location is always uncertain except when the spatial cue is presented.

Alcohol Use Disorders Identification Test (AUDIT)

The Audit is a 10-item alcohol-screening questionnaire. This tool was used to make initial inclusion and exclusion determinations. Research suggests that the AUDIT is more accurate than similar screening tools in accurately identifying alcohol dependence in college age students (Clements, 1998). The AUDIT specifically assesses quantity of alcohol use, frequency of consumption, and binge styles of drinking. Other advantages of this tool is that it focuses on drinking experiences within the past year and offers a range of response options that helps with precision. A study that examined the psychometric properties of the AUDIT using college students reported that the instrument exhibited a sensitivity of .84, specificity of .71, and .48 positive predictive value when using a cutoff

score of 11 and concluded that this tool has advantages over other alcohol screening tools in this regard (Fleming, Barry, MacDonald, 1991).

Symptom Checklist-90-Revised (SCL-90-R)

The Symptom Checklist-90, by Leonard R. Derogatis (1994), is a 90 item checklist used to assess a range of psychological problem at a specific point in time. This tool was also administered to make initial inclusion and exclusion determinations. For the purposes of this study, participants were asked to answer questions in regards to their symptoms within the previous week. This instrument can be administered to individuals 13 years of age and older and requires a 6th grade reading level (Derogatis, 1994). With only a 12-15 minute completion time, this instrument is ideal for assessing individual's symptoms in a relatively short time. The format used to administer this tool was paper and pencil and scoring was done by hand. The instrument is "gender-keyed" thus, gender based norms are used to score accordingly. The SCL-90-R was normed using adult psychiatric inpatients, adult psychiatric outpatients, adult nonpatients, and adolescent nonpatients (Derogatis, 1994). There are nine primary symptom dimensions; Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. In addition to these scales, there are three global indices: Global Severity Index that measures overall psychological distress, Positive Symptom Distress Index that measures the intensity of symptoms, and Positive Symptom Total that reports the number of self-reported symptoms. The Global Severity Index can be used to conveniently summarize results of the instrument (Derogatis, 1994).

Procedure

Interested students were provided with study information and full informed consent before consenting to participation in this study (Informed Consent document is included in the Appendix B). Participants were then asked to fill out a preliminary screening questionnaire as well as the AUDIT and SCL-90. The screening consisted of some demographic and general health questions such as age, general medical condition, medical history, psychiatric history including substance abuse, current substance use, allergies, and current medications. This screening helped preliminarily exclude individuals that were not able to participate due to such things as current substance use, medical condition, and medication use. Additional exclusionary criteria included those individuals who identified that they had serious current physical disease or those who were currently undergoing treatment for a psychological disorder including substance abuse. This questionnaire was also used to determine what if any medications or herbal supplements were being taken by the participants and whether or not these substances could be discontinued for the sake of their participation in this study.

AUDIT scores revealed that participants reported normal patterns of alcohol consumption and use. All participants were screened using the accepted cutoff score of 11 to screen out individuals with problematic drinking patterns. The mean AUDIT score was 5.55 ($SD = 2.77$) with minimum scores at 1 and maximum at 10.

Scores on the SCL-90 revealed that all participants were generally healthy in their report of psychological well-being. Scaled means for each clinical symptom scores fell in the non clinical range. The mean Global Severity Index, used as an overall summary of scores, is 34.64 illustrating that all participants were well within the normal range.

All identifying information was collected and stored in a locked filing cabinet in a locked office in the psychology department at WCU. Names and contact information were stored separately from any sensitive information concerning drinking, medical, or psychiatric history. An arbitrary number was assigned to each student so that no identifying information could be linked to the participants by anyone other than the researchers.

After preliminary screening, eligible students were contacted with the exact dates, times, and location of participation. Each participant was informed that they were required to commit to two days of testing after initial baseline and screening. Each participant was told they would low dose and moderate dose of alcohol but would be blind to which condition they were receiving on each of the testing occasions. There was a level of deception used as participants were not told that they were receiving a placebo condition, rather they were told they would engage in a low dose condition. The “low dose” session consisted of the placebo condition and the moderate dose session was considered the active dose condition.

It was explained that they should not drink alcohol or caffeine at least 24 hours before participation. In addition, it was explained that they should not eat or drink on the morning of their participation to control for the absorption and metabolism of alcohol. Participants were also advised to get their normal amount of sleep the night prior to participation. A light breakfast of apple juice and buttered whole grain toast was provided for the participants on the morning of participation.

Subject Preparation and Dosing

On the day of participation each student was asked to confirm their compliance

with the eating, drinking, caffeine, alcohol, and drug restrictions. They were then provided with a light breakfast of buttered whole grain toast and apple juice. Each participant was seen on an individual basis to ensure that they were well supervised and to maintain confidentiality during this process. After breakfast each student was weighed in order to calculate a weight dependant dose of ethanol. Blood Alcohol Concentration (BAC) was taken by Breathalyzer to ensure that participants had no alcohol in their system prior to participation. Once this baseline level was established the first dose of alcohol or placebo was given.

Alcohol was given in the form of a “screwdriver” or orange juice and ethanol. Ninety-five percent alcohol was used and mixed with the orange juice to equal 350 ml of fluid and divided into three equal doses or “mixed drinks.” The alcohol dose was calculated based on subject’s weight (0.6 g/kg). For placebo doses, 1 ml of alcohol was floated on the top and rim of the each portion of orange juice to give off the smell of alcohol and simulate an active does. The placebo dose also equaled 350 mls of fluid. Each of the active and placebo doses was divided into three small cups containing equal amounts of the 350ml ethanol and orange juice mixture. Participants were given a time limit of up to 5 minutes to finish each mixed drink for a total of a 15 minute dose administration period. The smaller doses are thought to make it easier for participants to drink as well as decrease the likelihood that alcohol is detected in each dose. Participants were instructed to swallow the mixture and not allow the drink to sit in their mouth, again to help avoid detection of alcohol. While the participant was seated in the testing room, BAC was taken at baseline and every 20 minutes after the dose was fully consumed. Once the participant’s BAC stabilized and was at near peak (60 minutes after completion

of dosing), he was seated in front of a computer monitor to complete the ANT.

Participants BAC was continually measured at 20 minute intervals after completion of the attention task. Participants were required to stay in the testing session until BAC returned to trace levels (< 0.02). In the placebo session, participants stayed approximately one hour and 40 minutes to simulate a low dose alcohol administration session. Participants were permitted to bring homework or entertainment materials to engage in after testing. There was approximately one day between testing sessions, however, days between baseline and the testing sessions varied.

Design and Analysis

This study was conducted to evaluate the acute effect of alcohol on the three forms of attention assessed by the ANT. A one-way within-subjects Multivariate Analysis of Variance (MANOVA) was used to group differences across the three dependant variables in this study; Alerting, Orienting, and Executive Control, as measured by performance scores on the ANT.

RESULTS

Mean estimated blood alcohol level (eBAL) was 0.049 (SD= .009) immediately prior to the ANT and 0.049 (SD=.012) immediately following the ANT. To examine the effect of alcohol on Alerting, Orienting, and Executive Control, the scores from the baseline, placebo and active alcohol dose sessions were analyzed using a one-way within subjects Multivariate Analysis of Variance (MANOVA). Analyses revealed a significant difference between the three conditions (baseline, placebo, and alcohol), Wilks' $\lambda = 0.514$, $F(6, 36) = 2.37$, $p = 0.05$, $\eta^2 0.28$. For ease, Table 1 shows means and standard deviation for the three networks across conditions.

Table 1

ANT Scores (in milliseconds) across Condition

Network	Baseline	Placebo	Alcohol
(n=11)			
Alerting	36.27(18.20)	43.09(27.35)	51.64(13.56)
Orienting	44.00(16.37)	41.00(19.20)	45.00(27.76)
Executive Control	133.09(47.35)	96.91(15.45)	129.00(59.33)

Univariate F tests were then inspected to determine if condition was significant for each network measured (Alerting, Orienting, and Executive Control). Analyses further revealed that there were no significant differences among the Alerting scores, $F(2, 20) = 2.62$, $p = 0.098$, $\eta^2 0.21$, and no significant differences among the Orienting scores, $F(2, 20) = 0.11$, $p = 0.895$, $\eta^2 0.22$. There were, however, significant differences among

the Executive Control scores, $F(2, 20) = 5.08$, $p = 0.016$, $\eta^2 0.34$. Pair-wise follow-up comparisons were completed using paired-samples T-tests. These analyses revealed that RT in the placebo Executive Control trials was significantly slower than at baseline, $t(10) = 3.47$, $p = 0.006$, and alcohol condition scores, $t(10) = 2.19$, $p = 0.05$. However, baseline and alcohol condition scores were not significantly different, $t(10) = 0.346$, $p = 0.74$.

Analysis of global accuracy (collapsing across measures) revealed no significant difference in accuracy across condition, $F(2, 20) = 1.433$, $p = 0.26$. Therefore, the effect of alcohol on Executive Control is not due to a simple change in accuracy of the participant's scores. Table 2 includes Accuracy means and standard deviation over each condition.

Table 2

Global Accuracy across Conditions

Condition	Mean	Standard Deviation
(n=11)		
Baseline	97.91	1.3
Placebo	97.36	2.06
Alcohol	96.91	2.02

DISCUSSION

This study investigated the acute effects of alcohol on the three specific neuronal networks within the brain associated with attention: Alerting, Orienting, and Executive Control using the ANT, a task designed to specifically look at said brain functions. Western Carolina University college students (n=11) were administered the ANT during three test sessions. The first session was a baseline assessment, followed by two sessions in which the participant received either a placebo or an active dose of alcohol (.6g/kg). Little is known about the acute effects of alcohol on three aspects of attention as measured by the ANT due to its relatively new arrival into the field.

It was hypothesized that Alerting RT would not be affected by the moderate dose given to participants because the literature on this aspect of attention show that tasks used to measure it are not as sensitive at lower or moderate doses of alcohol (Josephs & Steele, 1990; Koelega, 1995; Shulte et al., 2001). Secondly, it was thought that Orienting would be significantly affected by alcohol due to the characteristic slowing of saccadic eye movements. Lastly, it was hypothesized that Executive Control would be significantly affected due to the complexity of the Executive network and the tasks used to measure it.

The results only partially support these hypotheses. Results indicate that a moderate dose of alcohol had no significant effect on the Alerting or Orienting Networks. Analyses of scores on the ANT reveal that no significant change in response time was detected for Alerting and Orienting over time. However, this dose did significantly impair individuals' Executive Control Network.

The analyses reveal that participants who drank moderate doses of alcohol performed no better on the ANT than at baseline in regards to Executive Control.

Differences in this task were not due to changes in the accuracy of their responses because mean accuracy scores revealed little change across the three conditions. Further analyses revealed that differences in ANT scores were found between baseline and placebo trials and between placebo and active dose trials, however no difference was found between baseline and active dose trials.

Although it was hypothesized that alcohol would significantly affect an individual's ability to orient, this data did not support those findings. As described earlier, it is difficult to make inferences on previous works due to variations in the use of terminology and use of the term. Previous work on Orienting may have looked at just one aspect of the construct; however, this novel task measures the entire neuronal network of covert Orienting described by Fan and colleagues (2002). As Shulte et al. (2001) explain, there is little known about covert Orienting and how it is affected by alcohol as tasks to measure it are relatively new in the field.

Additionally, although it was hypothesized that RT for tasks requiring Executive Control would decrease due to the characterized impulsivity of responses observed in other studies, termed the alcohol disinhibition model, the results of this study did not support that directional hypothesis (Dougherty et al., 2000; Schweizer et al., 2004). Analyses did support the hypothesis that a significant difference would be found for tasks requiring Executive Control. The current study revealed that alcohol significantly affected Executive Control with slower RT than at Placebo levels, indicating that although impulsivity of response was not observed in this study, Executive Control was indeed impaired. The trends in the data show that at baseline, scores are comparatively lower as this is the participants' first introduction to the unfamiliar task, placebo scores

should see improved scores due to practice effects and lack of alcohol in the system, and active alcohol session scores are hindered due to effects of alcohol on the brain.

The current findings might vary from previous studies, because as Dougherty et al. (2000) found, the moderate dose of alcohol administered might not have been sufficient enough to bring about the impulsivity reported in other studies whereas higher doses of alcohol have been shown to have significant changes in performance (Shulte et al., 2001). Additionally, although the results of the executive function do not support the alcohol disinhibition model, the results do show that Executive Control is significantly affected by even moderate amounts of alcohol. However, it is possible and worthy of consideration that participants in the moderate dose session, engaged so intently during this conflicting task that, their RT actually increased because of their extra effort to not make mistakes (Fan & Posner, 2004). This theory may find some support in the attention allocation model, wherein, a person's performance during an alcohol condition is mediated by the attention allocated to that task and the amount of anxiety or stress it invokes (Josephs & Steele, 1990)

Based in the context of previous research on the attentional networks, it can be speculated that the significant effect found in the Executive Control network indicate that the midline frontal area of the brain is most affected by this moderate dose of alcohol. Specifically, it would seem that the anterior cingulate and the lateral prefrontal cortex that involve the dopaminergic pathway, areas previously described in other research, are affected by this dose of alcohol (Bush et al., 2000; Fan et al., 2002; Fan & Posner, 2004; Posner, 1995; Posner & Fan, 2004). Involvement of these areas of the brain has important implications, especially when considering the dopamine system and its involvement with

addiction and substance abuse (Julien, 1998). Executive control is thought to be most important in planning and decision making, error detection, novel responses to stimuli, difficult or dangerous conditions, and overcoming habitual actions (Fan & Posner, 2004). This last function is important in considering why the executive function showed greater sensitivity when other studies have proposed that simple tasks of attention are easily learned and thus may not show effects in RT (Josephs & Steele, 1990; Koelega, 1995; Shulte et al., 2001).

In light of the accumulating findings that even moderate doses of alcohol affect attention, continued careful prospective studies of the acute effects of alcohol on attention appear warranted. The current study has many limitations that should be considered in future research. For instance, the group studied did not include females, ethnic representation, diverse age groups, and non-college students, thus the ability to generalize to other groups is limited. Additionally, the small sample used in the study not only reduces the generalizability of results but also significantly reduces the statistical power of the study and thus, should be replicated to look for further significant values. Future studies should also explore how Alerting, Orienting, and Executive Control, as measured by the ANT, are affected with placebo, moderate, and high doses of alcohol to look for differences between the moderate and high dose groups. Other areas for future research include the relationship between the midline frontal and prefrontal areas of the brain and alcohol especially, their role in the intrinsic rewards system (dopaminergic pathway) of the brain as it is involved in these very areas. The Executive Control network associated with these areas has shown greater sensitivity to alcohol in this and as well as previous research and warrants further exploration.

This study revealed that even moderate doses of alcohol can adversely affect aspects of attention required for us to divide our attention meanwhile responding appropriately to our environment. These findings are extremely important when considering the amount of stimuli one must attend to while driving. Especially taking into account that the mean BAC both before and after the ANT did not research the legal limit of alcohol consumption to drive a vehicle. Although this study is a small snap shot of the growing body of evidence on the effects of alcohol on the brain, it brings us one step closer towards understanding how the workings of the brain are affected by the interaction with this drug.

REFERENCES

- American Psychiatric Association. (2000). Diagnostic and statistical manual of mental disorders (4th ed., text rev.). Washington, DC: Author.
- Barinaga, M. (2000). A new clue to how alcohol damages the brain. *Science*, 287, 647-649.
- Barnea, A., Rassis, A., Raz, A., Othmer, S., & Zaidel, E. (2004). Effects of neurofeedback on hemispheric word recognition. *Brain and Cognition*.
- Bartholow, B.D, Pearson, M, Sher, K.J., Wieman, L.C, Fabiani, M, Gratton, G. (2003). Effects of alcohol consumption and alcohol susceptibility on cognition: A psychophysiological examination. *Biological Psychology*, 64, 167-190.
- Berlyne, D.E. (1974). Attention. In E. C. Catrерette & M.P. Friedman (Eds.), Handbook of perception. *Historical and philosophical roots of perception*, (pp. 123-147). New York: Academic Press, 1.
- Broadbent, D.E. (1958). Perception and communication. An important work on selective listening, a classic in the field of attention. Oxford: Pergamon Press.
- Bush, G., Luu, P., & Posner, M.I. (2000). Cognitive and emotional influences in the anterior cingulate cortex. *Trends in Cognitive Science*, 4, 215-222.
- Center for Disease Control. (2004, September 24). Alcohol-attributable deaths and years of potential life lost: United States, 2001. *Morbidity and Mortality Weekly Report*, 53(37), 866-870. Retrieved March 12, 2009 from <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5337a2.htm>
- Clements, R. (1998). A critical evaluation of several alcohol screening instruments using the CIDI-SAM as a criterion measure. *Alcoholism: Clinical and Experimental*

Research, 22(5).

Concise Dictionary of Psychology (1998). (3rd ed.) Statt, D.A. (Ed.), London: Routledge.

Corballis, P.M., Gratton, G. (2003). Independent control of processing strategies for different locations in the visual field. *Biological Psychology*, 64, 191-209.

Derogatis, L.R. (1994). SCL-90-R: Administration scoring and procedural manual. Minneapolis, Mn: National Computer System.

Dougherty, D.M., Marsh, D.M., Moeller, F.G., Chokshi, R.V., Rosen, V.C. (2000). Effects of moderate and high doses of alcohol on attention, impulsivity, discriminability, and response bias in immediate and delayed memory task performance. *Alcoholism: Clinical and experimental research*, 24(11), 1702-1711.

Eriksen, B.A., & Eriksen, C.W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics*, 16, 143-149.

Eysenck, M.W., & Keane, M.T. (2002). Attention and performance limitations. In D.J. Levitin, (Ed.), *Foundation of cognitive psychology* (p. 363). Cambridge, Ma: Bradford.

Fan, J., McCandliss, B.D., Fossella, J., Flombaum, J.I., & Posner, M.I. (2005). The activation of attentional networks. *Neuroimage*, 26(2), 471-479.

Fan, J., McCandliss, B.D., Sommer, T., Raz, A., & Posner, M.I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience*, 3, 340-347.

Fan, J., Posner, M.I. (2004). Human attentional networks. *Psychiatrische Praxis*, 31,

s210-s214.

- Fan, J., Wu, Y., Fossella, J. & Posner, M.I. (2001). Assessing the heritability of attentional networks. *BioMed Central Neuroscience*, 2:14.
- Fillmore M.T, & Vogel-Sprott M. (1999). An alcohol model of impaired inhibitory control and its treatment in humans. *Experimental Clinical Psychopharmacology*, 7, 49-55.
- Fleming, M.F., Barry, K.L., MacDonald, R. (1991). The alcohol use disorders identification test (AUDIT) in a college sample. *The International Journal of the Addictions*, 26:1173-1185.
- Fossella, J., Posner, M.I., Fan, J., Swanson, J.M. & Pfaff, D.W. (2002). Attentional phenotypes for the analysis of higher mental function. *The Scientific World*, 2, 217-223.
- Frye, G.D., Breese G.R. (1982). GABAergic modulation of ethanol induced motor impairment. *Journal of Pharmacological Experimental Therapy*, 223, 750–756.
- George, S, Rogers, R.D., Duka, T. (2005). The acute effect of alcohol on decision making in social drinkers. *Psychopharmacology*, 182, 160-169.
- Gibson, J.J. (1974). Purple Perils. Unpublished Manuscript, Cornell University.
Retrieved April 7, 2006 from <http://www.huwi.org/gibson/overt.php>
- Goodwin, D.W., Othmer, E., Halikas, J.A., & Freemon, F. (1970). Loss of short term memory as a predictor of the alcoholic “blackout”. *Nature*, 227, 201–202.
- Gonzales, R.A. & Jaworski, J.N. (1997). Alcohol and glutamate. *Alcohol Health & Research World*, 21, 120-128.
- Gregory, R.L. (Ed.) (2004). The oxford companion to the mind. *Attention Oxford*

University Press. 2nd ed., 75-77.

Hingson, R.W., Heeren, T., Zakocs, R.C., Kopstein, A., & Wechsler, H. (2002).

Magnitude of alcohol-related mortality and morbidity among U.S. college students ages 18-24. *Journal of Studies on Alcohol*, 63(2), 136-144.

Hobbs, W.R, Rall, T.W., & Verdoorn, T.A. (1996). Hypnotics and Sedatives: Ethanol In,

Goodman and Gillman's The Pharmacological Basis of Therapeutics,(Hardman, J.G, Limbird, L.E, Molinoff, P.B., Ruddon, R.W, & Gilman, A.G.,eds,) The McGraw-Hill Companies, Inc. 385-386.

Itti, L., Rees, G., & Tsotsos, J.K. (Eds.). (2005). *Neurobiology of Attention*. San Diego:

Elsevier Academic Press.

James, W. (1890). The principles of psychology. Vol. 1. New York: Henry Holt. 403-

404. Retrieved April 2, 2006 from <http://psychclassics.yorku.ca/>

James/Principles/index.htm

Josephs, R.A., & Steele, C.M. (1990). The two faces of alcohol myopia: Attentional

mediation of psychological stress. *Journal of abnormal psychology*, 99(2), 115-126.

Julien, Robert, M. (1998). A primer of drug action: a concise, nontechnical guide to the

actions, uses, and side effects of psychoactive drugs. 8th ed. W.H. Freeman and Company. New York, 64-92.

King, A.C., & Byars, J.A. (2004). Alcohol-induced performance impairment in heavy

episodic and light social drinkers. *Journal of Alcohol Studies*, 65, 27-36.

Koelega, H.S. (1995). Alcohol and vigilance performance: a review.

Psychopharmacology, 118, 233–249.

- Kolb, B. & Wishaw, I.Q. (2003). *Fundamentals of Human Neuropsychology* (5th ed.). *Attention, Mental Images, and Consciousness*, (pp. 576-608). New York, NY: Worth Publishers.
- Kowalksi, K.M. (2001). Debunking myths about alcohol. *Current Health* 2, 27, 6-12.
- Lovinger, D.M. (1997). Serotonin's role in alcohol's effects on the brain. *Alcohol Health & Research World*, 21, 114-121.
- Meyer, J.S., & Quenzer, L.F. (2005). Psychopharmacology: Drugs, the brain, and behavior. *Alcohol*, (pp. 216-243). Sunderland, Ma: Sinauer Assoc., Inc.
- Milton, N. (1994, May). Early and late selection theories of attention. Retrieved April 6, 2006, from <http://www.epistemics.co.uk/staff/nmilton/papers/attention.htm>
- Minocha, A., Barth, J.T., Roberson, D.G., Herold, D.A., Spyker, D.A. (1985). Impairment of cognitive and psychomotor function by ethanol in social drinkers. *Vet Hum Toxicol* 27:533-536
- [Mukherjee, S.](#), [Das S.K.](#), [Vaidyanathan, K.](#), [Vasudevan, D.M.](#) (2008). [Consequences of alcohol consumption on neurotransmitters -an overview](#). *Current Neurovascular research*, 5(4), 266-272.
- National Highway Traffic Safety Administration. (2007). Fatality Analysis Reporting System. Retrieved March, 12 2009 from <http://www-fars.nhtsa.dot.gov/Trends/TrendsAlcohol.aspx>
- Nichols, J.M., Martin, F. (1996). The effect of heavy social drinking on recall and event-related potentials. *Journal of Studies in Alcohol* 57:125-135.
- Paschal, F.C. (1941). The trend in theories of attention. *Psychological review*, 48, 383-403.

- Pihl, R.O., Paylan, S.S., Gentes-Hawn, A., & Hoaken, P. (2003). Alcohol affects executive cognitive functions differentially on the ascending versus descending limb of the blood alcohol concentration curve. *Alcoholism: Clinical and experimental research*, 27(5), 773-779.
- Pillsbury, W. B. (1908). Attention. London: Swan Sonnenschein.
- Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology*, 41A, 19-45.
- Posner, M.I. (1988). Structures and functions of selective attention. In T. Boll and B. Bryant (Eds.), *Master Lectures in Clinical Neuropsychology and Brain Function: Research, Measurement, and Practice*, American Psychological Association, 171-202.
- Posner, M.I. (Ed.). (2004). *Cognitive Neuroscience of Attention*. Guilford Press, New York.
- Posner, M.I. & Fan, J. (2004). Attention as an organ system. In: Pomerantz, J.R., Crai, M.C., (Editors) *Topics in integrative neuroscience: from cells to cognition*. Cambridge: Cambridge University Press.
- Posner, M.I. (1995). Forward. In M.D. Rugg and M.G.H. Coles (Eds.), *Electrophysiology of Mind*, Oxford: University Press.
- Posner, M.I., & Raichle, M.E. (1994). *Images of Mind*. Scientific American Books.
- Ramchandani, V.A. (2000). *Alcohol: Neurobiology and Pharmacology*. Retrived 2/14/06, from Alcohol medical scholars program website
<http://www.alcoholmedicalsolars.org>.

- Raz, A. (2004). Anatomy of attentional networks. *The anatomical record part b: New anatomist*, 281b(1), 21-36.
- Ridderinkhof, R.K., Vlucht, Y., Bramlage, A., Spaan, M., Elton, M., Snel, J., & Band, G. (2002). Alcohol consumption, impairs detection of performance errors in mediofrontal cortex. *Science*, 298, 2209-2211.
- Robinson, D.L., & Petersen, S.E. (1992). The pulvinar and visual salience. *Trends in Neuroscience*, 15, 127-132.
- Schmidt, L.G., Smolka, M.N., & Rommelspacher, H. (2003). Dopaminergic dysfunction—a common final pathway of addiction to alcohol, opiates and nicotine? Kuntze, M.F., Müller-Spahn, F., Ladewig, D., Bullinger, A.H. (Eds.), *Basic and Clinical Science of Opioid Addiction. Bibliotheca Psychiatrica*, Basel, Karger, 170, 39-45.
- Schulte, T., Muller-Oehring, M., Strasburger, H., Warzel, H., & Sabel, B.A. (2001). Acute effects of alcohol on divided and covert attention in men. *Psychopharmacology*, 154, 61-69.
- Schweizer, T.A., Jolicoeur, P., Vogel-Sprott, M., & Dixon, M.J. (2004). Fast, but error-prone, responses during an acute alcohol intoxication: Effects of stimulus-response mapping complexity. *Alcoholism: Clinical and experimental research*, 28(4), 643-649.
- Stroop, J.R. (1935). Studies of interference in serial verbal reactions. *Journal of experimental psychology*, 18, 643-661.
- Titchener, E.B. (1908). *Lectures on the elementary psychology of feeling and attention*. Reprinted in 1973, New York, Arno Press.

- Turner, J.C. & Shu, J. (2004). Serious health consequences associated with alcohol use among college students: Demographic and clinical characteristics of patients seen in an emergency department. *Journal of Alcohol Studies, 65*, 179-183.
- Vogel-Sprott, M., Easdon, C., Fillmore, M., Finn, P., & Justus, A. (2001). Alcohol and behavioral control: Cognitive and neural mechanisms. *Alcoholism: Clinical and Experimental Research, 25(1)*, 117-121.
- Volkow, N.D., Fowler, J.S., Wolf, A.P., Hitzemann, R., Dewey, S., Bendriem, B., Alpert, R., & Hoff, A. (1991). Changes in brain glucose metabolism in cocaine dependence and withdrawal. *American Journal of Psychiatry, 148*, 621-626.
- Volkow, N.D., Mullani, N., Gould, K.L., Adler, S., Krajewski, K. (1988). Cerebral blood flow in chronic cocaine users: A study with positron emission tomography. *British Journal of Psychiatry, 152*, 641-648.
- Wang, H., Fan, J. (2007). Human Attentional Networks: A connectionist model. *Journal of Cognitive Neuroscience, 19(10)*, 1678-1689.
- Wang, K., Fan, J., Dong, Y., Wang, C.Q., Lee, T.M.C., & Posner, M.I. (2005). Selective impairment of attentional networks of orienting and executive control in schizophrenia. *Schizophrenia Research, 78(2-3)*, 235-241.
- Weissenborn, R., & Duka, T. (2003). Acute alcohol effects on cognitive function in social drinkers: their relationship to drinking habits. *Psychopharmacology, 165*, 306-312.

APPENDICES

APPENDIX A

General Information Form

920_____

Age_____ DOB_____

Gender_____ Ethnicity_____

Height_____ Weight_____

Are you currently a student or employee of WCU or a member of the community?

Education level?

How would you rate your current health status (poor, fair, good or excellent)?

Do you currently suffer from any chronic physical illness or disability (e.g., diabetes, chronic hypertension)?

Are you currently taking any medications? If so, list them.

Are you currently taking any herbal supplements? If so, list them.

Are you a smoker or do you use any smokeless tobacco? How much do you use per day?

Have you experimented with any illicit drugs in the past 90 days? If so, when was your last use and what did you take?.

Have you ever been very concerned about your use of drugs at any time during the past two years?

Have your friends or family expressed strong concerns about your use of drugs in the last two years?

Have you ever been in treatment for an alcohol or other drug problem?

Have you ever had withdrawal symptoms from alcohol or other drugs?

On Average do you consume 2-4 alcoholic drinks in a week? Y/N

More_____ Less_____

APPENDIX B

Informed Consent

What is the purpose of this research?

We are studying the acute effects of alcohol on male participants who are 21 years of age or older. Participants will complete a computerized measure of attention called the Attention Network Test (ANT) after consuming a moderate dose of alcohol or placebo (no alcohol). Our primary goal is to provide valuable information about the effect of alcohol on attention.

What will be expected of me?

You will be asked to complete a 10-question alcohol use screening questionnaire called the Alcohol Use Disorders Identification Test (AUDIT), a 90 question screening for psychological symptoms called the Symptom Checklist-90, and you will be asked to provide some general information on your current medical condition. The forms will be provided on line through Western Carolina University prior to participating in the experiment. This screening will help exclude individuals not able to participate. Exclusionary criteria include those individuals who have identified that they have a serious current physical disorder, those who meet criteria for significant substance abuse, and those who report psychological symptoms that exceed the cut-off score. The time required to complete this screening is approximately 35 minutes. If you are eligible for participation upon completion of the screening, you will be contacted by telephone to set-up testing dates and directions to the Western Carolina University campus and laboratory room. You will be required to participate in this study on two non-consecutive days for approximately six hours per session. You will also be asked to sleep your normal number of hours the night before the each test day, not to use any recreational drug or alcohol for at least 24 hours before the testing day, and not to eat breakfast the morning of testing.

The amount of alcohol provided to you will be based on your body weight. For the sake of comparison, a 150 pound man would receive the equivalent of about 3 drinks. Those that weigh more than 150 pounds would receive more alcohol; those that weigh less would receive less alcohol. As a result, you must agree to stay in the laboratory, under the supervision of the Experimenters, until your Blood Alcohol Concentration returns to .02 or less, as measured by our Breathalyzer. In extreme cases, this may take up to six hours depending on how quickly your body processes that alcohol). Please understand that if you cannot consent to this, we will not be able to allow you to be in the experiment. If during the course of the experiment, you change your mind and decide you do not want to participate, we will stop the experimental procedure, but for safety reasons, **YOU WILL STILL BE REQUIRED TO STAY IN THE LABORATORY UNTIL YOUR BLOOD ALCOHOL CONCENTRATION IS NEAR ZERO**, as measured by our Breathalyzer. If you try to leave before then, we will be required to contact WCU Campus Police to alert them of your whereabouts. Having read this, if you do not wish to

participate in the study, please let us know now.

On each of the two days of testing you will be provided with a light breakfast after you have been weighed and your blood alcohol content has been measured by Breathalyzer. You will then be given three doses of alcohol or placebo in gelatin form. You will be instructed not to hold the gelatin in your mouth before swallowing to prevent detection of alcohol. Your blood alcohol content will be measured via Breathalyzer after each dose and then several times after dosing to track when you have again reached baseline level (sobriety). Approximately 70 minutes after you are given your last dose you will be asked to complete a computerized measure of attention called the ANT that will take approximately 25 minutes. A standard lunch, consisting of a turkey sandwich and non-caffeinated soda will be provided 250 minutes into the experiment. Except for within one hour of dosing, the subjects will be allowed water as requested.

During the test session in which you may consume alcohol, the doses given to you will produce alcohol concentrations in your blood which might exceed the legal limit for driving a motor vehicle in North Carolina. However, the dose should not be high enough to produce excessive drowsiness. On each test day you will be required to stay in the laboratory until your blood alcohol level returns to trace levels.

Will my answers be confidential?

Your individual responses will be kept strictly confidential. All identifying information will be collected and stored in a secure database. Your name and contact information will be stored separately from any sensitive information concerning drinking history. Your name and corresponding information will be assigned an arbitrary "participant number" as the only form of identification. This coding system will ensure that you will only be known by your assigned number. The researcher will be the only person aware of which participant actually corresponds to each of these numbers.

Can I withdraw from the study if I decide to?

Yes, you may discontinue your voluntary participation at any time without penalty. If at any time, you feel that you must terminate the session, please do so by saying "Stop," out loud. You can terminate an experiment without giving a reason and we won't ask for one. However, if you have received active doses of alcohol, you must stay in the laboratory until your Blood Alcohol Concentration is .02 or less. If you have any questions, please ask the Experimenter. You will be given a copy of this form to take with you.

Is there any harm that I might experience from taking part in the study?

Yes, there is some risk involved in the alcohol consumption and subsequent intoxication. Typical symptoms of feeling drunk may include euphoria (excitement), talkativeness, affective lability (change of emotion), slurred speech, body sway, and loss of coordination. The minor risks of intoxication may cause you to become drowsy, nauseated, or throw up. Such risks are minimized by ensuring that after dosing, you will

always be accompanied by a researcher whenever you stand up or walk around. Personnel will not allow you to leave the laboratory until your breathalyzer reading is 0.02 or less (sober).

Since this study requires that you have experience with alcohol, the possible side effects or risks should be minimal and may not be undesirable.

How will I benefit from taking part in the research?

You will have the opportunity to earn research participation credit in one of your psychology courses. The professor of the course will decide upon the amount of credit given.

We hope that you will learn something about the research process by your participation, and although you might not personally benefit from the experiment, your data will be combined with other data so that we can learn more about the acute effects of alcohol on attention.

Who should I contact if I have questions or concerns about the research?

This research project is being conducted by Shayma Khalil under the direction of Dr. Shawn K. Acheson, Ph.D. of the Psychology Department. If you have any questions about your participation or about the study in general, you may contact Shayma (828-506-4856 or chezma82m@aol.com), Dr. Acheson (227-3368 or acheson@wcu.edu), or Dr. Chris Cooper, the Institutional Review Board Chair for WCU (828-227-7640).

CONSENT:

I, _____, state that I am over 21 years of age and that I agree to participate in a research study being directed by Dr. Shawn Acheson of the Psychology Department and conducted by his student, Shayma Khalil. I acknowledge that the researcher has informed me of the purpose of the study; that my participation is voluntary; that I may withdraw my participation at any time without penalty; and that all data will remain strictly confidential. The researcher has agreed to answer any of my questions about the research that could influence my decision to participate. I understand that I will be completing an alcohol screening that includes information about my alcohol use and current general medical condition, consuming intoxicating amounts of alcohol, and completing a computerized measure of attention. I understand that the study involves some risk associated with alcohol intoxication and the researcher has made me aware of those risks. I agree TO STAY IN THE LABORATORY UNTIL MY BLOOD ALCOHOL CONCENTRATION IS NEAR ZERO, as measured by the Breathalyzer, and I understand that WCU Campus Police will be called if I attempt to leave before then.

In addition, I certify that I do not have a physical condition that would make it dangerous for me to consume alcohol, that I am not taking any substances that will make it dangerous for me to drink alcohol, that I have not had any alcohol, nor any other psychoactive substances today and that I have not eaten a meal or a large snack in the last

three hours.

Name _____

Date _____

Signature _____

I wish to receive a copy of the results.

Address _____

Email _____

Brief Summary

There has been much research devoted to the topic of attention. Research on alcohol and its effects on the brain and human body are vast. More recent understanding of the underlying physiology of attention and proposed tasks to measure aspects of has made research in this area more accessible. Little is known about whether the same observations on the acute effects of alcohol on the attention networks can be made using the ANT due to its relatively new arrival into the field (Fossella, Posner, Fan, Swanson, & Pfaff, 2002). We are studying the acute effects of alcohol on male participants who are 21 years of age or older. Participants will complete a computerized measure of attention called the Attention Network Test (ANT) after consuming a moderate dose of alcohol or placebo (no alcohol).

We propose a placebo-controlled study of the effects of alcohol on three aspects of attention; orienting, alerting, and executive control. We will test 20 male participants 21 years of age or older with both a placebo and active alcohol dose over two settings. This within subjects design will allow for a comparison of the participant across the session. We expect that, relative to their performance baselines, the participants will show no significant effect on the alerting aspect of attention in regards to response reaction time, an increase in RT in the orienting task, and a decrease in RT in the executive control component of attention.

Specific Aims: Our primary goal is to provide valuable information about the effect of alcohol on attention.

Testing Procedures

Subject Preparation and Dosing

The subjects will be instructed to sleep their normal number of hours the night prior to each test day, and to abstain from ethanol and other drugs during the previous 24

hours, and from breakfast on the morning of testing. They will report to the laboratory at 8:00 a.m. and will be provided a light breakfast of two slices of toast, a small amount of margarine, and orange juice. Body weight and baseline measurements Blood Alcohol Concentration (BAC) will be made prior to the experimental treatment.

One hour after breakfast, an oral dose of ethanol or placebo will be administered. Doses of ethanol or placebo will be administered in random order across the two testing sessions. Alcohol will be given in the form of a gelatin "shot." Alcohol given will be calculated based on subject's weight (0.6 g/kg). Ninety-Five percent alcohol will be used and mixed with gelatin at a ratio of 1part alcohol to 5 parts gelatin mixer. For placebo doses, alcohol will be replaced by water. Participants will be instructed to swallow the mixture and not allow the gelatin shot to sit in their mouth. BAC will be taken at baseline and every 20 minutes after the dose is fully consumed. Once the participant's BAC has stabilized, he will be seated in front of a computer monitor to complete the Attention Network Test (ANT). Approximately 70 minutes after you are given your last dose you will be asked to complete a computerized measure of attention called the ANT that will take approximately 25 minutes.

A standard lunch, consisting of a turkey sandwich and non-caffeinated soda will be provided 250 minutes into the experiment. Except for within one hour of dosing, the subjects will be allowed water as requested.

Although the testing will be completed within 30 minutes after dosing, all participants will be required remain in the laboratory until BAC's return to trace levels (below 0.02) which might take up to six-hours a session, and breath samples will be taken at 30-minute intervals after testing are completed.

Potential risks and benefits

Risks involved in the alcohol consumption and subsequent intoxication include typical symptoms of feeling drunk consisting of euphoria (excitement), talkativeness, affective lability (change of emotion), slurred speech, body sway, and loss of coordination. The minor risks of intoxication may cause you to become drowsy, nauseated, or throw up. Such risks are minimized by ensuring that after dosing, you will always be accompanied by a researcher whenever you stand up or walk around. Personnel will not allow you to leave the laboratory until your breathalyzer reading is 0.02 or less (sober). Since this study requires that you have experience with alcohol, the possible side effects or risks should be minimal and may not be undesirable.

Potential benefits include the opportunity to earn research participation credit or extra credit in one of your psychology courses. The professor of the course will decide upon the amount of extra credit given. We hope that you will learn something about the research process by your participation, and although you might not personally benefit from the experiment, your data will be combined with other data so that we can learn more about the acute effects of alcohol on attention.

