Effects of acute alcohol intoxication on visual processing and oculomotor control

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Abstract English

Although alcohol consumption is known to degrade performance in a variety of tasks, the exact character of alcohol induced impairments is currently not well understood. The present work examines to what extent acute alcohol intoxication impairs visual processing and oculomotor control on different processing levels. Understanding the impact of alcohol on the visual system is critical because the most important way humans navigate in and communicate with the environment is through the acquisition and processing of visual information. Virtually all complex cognitive tasks rely on visual input, obtained via the planning and execution of rapid eye movements. Within the theoretical framework of "active vision" (Findlay & Gilchrist, 2003) the traditional dissociation of perception from motor control is loosened and eye movements are regarded as 'part and parcel' of an integrated process of information acquisition. In order to better understand the stages at which alcohol affects oculomotor control, five paradigms were used to map alcohol effects on different hierarchically organized levels of visuomotor control and additionally two complex visual cognitive tasks were examined. On the lowest level (automatic), reflexive processes were tested using the pro saccade task. The next level (automated) incorporates implicit learning and memory processes that can influence reflexive behavior, but are still unconscious. This level was examined using the double step paradigm. The highest processing level represents voluntary modification of behavior and was studied using two versions of the anti saccade paradigm and the memory guided saccade paradigm. The two complex visual cognitive tasks were task switching and reading. Task switching requires participants to switch between two or more distinct tasks, which usually results in switch costs or benefits. Such effects are explained with the interplay of inhibition and activation and to date no study has examined effects of alcohol on performance in task switching. The sentence reading experiment offered the possibility to study visuomotor control in combination with a precisely controlled cognitive processing load in an ecologically valid everyday task. For all paradigms, participant's performance were measured in an "alcohol" and a "no alcohol" session. A total of 62 students participated and the mean breath alcohol concentration in the "alcohol session" was 70mg%. Results indicate specific effects of alcohol on different levels of visual processing and oculomotor control. Functioning on the automatic level was intact, except for a slowing in saccade latencies. Even though alcohol is known to reduce simple reaction times, the present work could show for the first time that in comparison with higher processing levels, such a "general slowing" is less pronounced on this lower processing level. Regarding the automated level deficits with in the ability to adaptively reprogram saccades on the basis of new visual information were found under alcohol. More time is necessary to achieve the same amount of reprogramming when eye movements need to be directed to new target locations. This finding is especially important, because adaptive reprogramming is a core ingredient of effective visuomotor behavior in everyday tasks such as reading or visual search. Impairments on the voluntary processing level became apparent in hypermetric (i.e., prolonged) saccade amplitudes under alcohol, whenever a reprogramming of the initial saccade target was necessary. This effect was found under conditions that required endogenous representation as well as in situations when a visual marker was present at the target location. In addition, a small effect of alcohol on visuospatial short term memory was found. Interestingly, no alcohol related effects were found regarding inhibitory functioning. In addition, performance measures in the complex visual cognitive tasks did not differ between alcohol conditions as a result of compensatory mechanisms. Apparently, longer processing time that is available under alcohol can be used in the task switching condition to activate a task set more completely. In a similar way, the trade-off between increased fixation duration and decreased number of fixations during reading suggests that the extra processing time under alcohol can be used for linguistic processing, which in itself does not seem to be impaired. This finding is supported by the fact no interaction between alcohol and word frequency was found. In conclusion, this thesis explored the effects of acute alcohol intoxication on visual processing and oculomotor control. The carefully selected paradigms have yielded interesting findings that begin to map alcohol related impairments on different levels of oculomotor control. In addition, findings and

discussions afford multiple approaches for further research that should help to achieve a deeper understanding of the effects of alcohol and its underlying mechanisms.

Abstract German

Titel: **Der Einfluss von akutem Alkoholkonsum auf visuelle Informationsverarbeitung und okulomotorische Kontrolle**

Alkoholkonsum birgt das Potential, verschiedenste Aspekte menschlicher Leistungsfähigkeit zu beeinträchtigen. Der genaue Charakter solch alkoholbedingter Veränderungen und die zugrundeliegenden Mechanismen sind jedoch bislang nicht hinreichend verstanden. In der vorliegenden Arbeit steht die Frage im Mittelpunkt, wie und im welchem Maße Alkohol visuelle Informationsverarbeitung und okulomotorische Kontrolle beeinflusst. Dieser Schwerpunkt wurde gewählt, da visuelle Informationen einen wesentlichen Beitrag in der alltäglichen Orientierung in und Kommunikation mit der Umwelt leisten. Einen wertvollen theoretischen Rahmen bietet in diesem Zusammenhang der „active vision"-Ansatz, in dem die traditionelle Trennung zwischen Wahrnehmung und motorischer Kontrolle relativiert wird. Blickbewegungen werden als wesentlicher Bestandteil eines integrierten Prozesses der Informationserfassung und so als Konsequenz und Voraussetzung visueller Informationsverarbeitung verstanden. Sieben Paradigmen wurden ausgewählt, um Alkoholeffekte auf verschiedene hierarchisch organisierte Ebenen der okulomotorischen Kontrolle sowie auf komplexere kognitive Anforderungen zu untersuchen. Die erste untersuchte Verarbeitungsstufe ist die automatische, auf der reflexive Prozesse angesiedelt sind. Auf dem nächsthöheren Niveau (automatisiert) können implizite Lern- und Gedächtnisprozesse, die noch unterhalb der Bewusstseinsschwelle ablaufen, die Verarbeitung beeinflussen. Auf der höchsten Ebene werden Prozesse subsummiert, die eine „willentliche" Modulation des Verhaltens erlauben. Zusätzlich zu fünf Paradigmen zur Untersuchung des Einflusses von Alkohol auf diese verschiedenen Verarbeitungsebenen wurden zwei weitere Aufgaben zu kognitiv komplexerem Verhalten gewählt. Beim Task Switching steht der Wechsel zwischen zwei Aufgaben im Vordergrund, wobei Kosten oder Vorteile durch den Wechsel entstehen können. Solche Wechselkosteneffekte können durch das Zusammenspiel von Inhibition und Aktivation beschrieben werden und sind bislang nicht in Bezug auf alkoholbedingte Effekte untersucht worden. Ein Satzleseexperiment bot eine ideale Gelegenheit, in einer ökologisch validen Aufgabe visuomotorische Steuerung in Kombination mit präzise kontrolliertem kognitiven Verarbeitungsaufwand zu untersuchen. In allen Paradigmen wurden Leistungen der Teilnehmer jeweils in einer „kein Alkohol" und einer „Alkohol"-Sitzung erhoben. Die mittlere Atemalkoholkonzentration in der „Alkohol"-Sitzung betrug 0.7 Promille. Insgesamt nahmen 62 Personen an der Studie teil. Die Ergebnisse zeigen spezifische Effekte des Alkohols auf den verschiedenen Ebenen der visuellen Verarbeitung und okulomotorischen Kontrolle. Auf der automatischen Ebene sind grundlegende Funktionen intakt, es kommt jedoch bereits hier zu alkoholbedingten Verzögerungen. Während eine solche Verlangsamung von einfachen Reaktionszeiten durch Alkohol bekannt ist, kann durch den Vergleich mit anderen Verarbeitungsstufen zum ersten Mal gezeigt werden, dass eine solche „generelle Verzögerung" auf diesem Niveau weniger ausgeprägt ist, als bei komplexeren Prozessen. Für die automatisierte Ebene konnten erstmalig charakteristische Defizite in Bezug auf die Neu- und Reprogrammierung von Sakkaden auf der Basis neuer visueller Information nachgewiesen werden. Unter Alkohol wird deutlich mehr Zeit benötigt, um Blickbewegungen adaptiv an neue Informationen anzupassen. Auf der willentlichen Ebene zeigte sich, dass Sakkaden unter Alkohol verlängerte Amplituden aufwiesen, wenn eine Reprogrammierungen des Sakkadenziels notwendig war. Zusätzlich wurden spezifische Effekte von Alkohol auf das visuell-räumliche Kurzzeitgedächtnis gefunden. Im Gegensatz dazu zeigten sich keine alkoholbedingten Unterschiede mit Blick auf inhibitorische Funktionen. Auch in den kognitiv komplexeren Aufgaben wurde ein relativ geringer Einfluss von Alkohol gefunden. Hier

zeigte sich, dass kompensatorische Mechanismen greifen, so dass kaum Leistungsunterschiede zwischen den Alkoholbedingungen sichtbar wurden. Die Ergebnisse werden dahingehend interpretiert, dass im Task Switching Paradigma die durch Alkohol verlängerte Reaktionszeiten entstandene zusätzliche Verarbeitungszeit genutzt wird, um ein erforderliches Task Set vollständiger zu aktivieren. Ähnlich lassen verlängerte initiale Fixationszeiten beim Lesen gepaart mit einer geringeren Anzahl von Fixationen darauf schließen, dass zusätzliche Verarbeitungszeit für die linguistische Verarbeitung genutzt wird. Zusammenfassend konnten in der vorliegenden Arbeit spezifische Effekte des Alkohols auf unterschiedliche Ebenen visueller Verarbeitung und okulomotischer Kontrolle nachgewiesen werden. Die sorgfältig ausgewählten Paradigmen lieferten ein konsistentes Muster interessanter Ergebnisse, mit dem alkoholbasierte Beeinträchtigungen auf verschiedenen Verarbeitungsebenen detailliert beschrieben werden. Zusätzlich zeigen die Befunde und Diskussionen eine Vielzahl von Ansätzen für zukünftige Forschung auf.

1 Introduction: Adverse effects of alcohol intoxication on human information processing

The interplay of alcohol and information processing in the consequences of human drinking behavior is at the heart of several key questions that are addressed in the present work: (1) what effects does acute alcohol intoxication have on basic functions related to performance of simple tasks; (2) what are the implications of these effects for more complex activities critical to everyday life; and (3) what mechanisms might underlie the impact of alcohol on information processing?

Despite the obvious importance of answers to questions like these, the extensive extant literature is somewhat fragmented and more descriptive rather than theory-driven or explanatory. Moreover, with the notable exception of driving and piloting, the dependent variables used in research on alcohol and information processing have typically been confined to traditional neuropsychological testing or simple laboratory analogue tasks. Fortunately, recent advances in the study of information processing in terms of *active vision* (the integrated process of visual information acquisition, planning and execution of eye movements, and cognitive control; Findlay & Gilchrist, 2003; see section 2.3) provide a unique and promising opportunity to improve upon the rather unsatisfying state of affairs in the alcohol literature. To this end, this thesis pursues a course that capitalizes on three developments in active vision research: (a) the evolution of measurement and analysis of eye movements to the point where they now provide highly reliable and sensitive indicators of visuomotor processing; (b) the elaboration of conceptual linkages between eye movements and distinct levels and types of information processing; and (c) the application of eye movement methodology and theory to the study of complex cognitive tasks such as task switching and reading, both representing skills critical to many aspects of human success in everyday life.

1.1 Effects of Alcohol Intoxication on Human Behavior

Research on the consequences of alcohol intoxication on information processing has a long and venerable history, represented in the related domains of effects due to acute exposure versus effects due to chronic exposure. Much in the first domain revolves around episodic, alcohol-induced impairment of the highly important practical tasks of driving and piloting of other transportation vehicles (e.g., Moskowitz, Burns, Fiorentino, Smiley, & Zador, 2000; Moskowitz & Fiorentino, 2000). There is also a more recent interest in how alcohol intoxication might influence important social behaviors like aggression (Giancola, 2000) and

emotional responses such as fear (Lang, Patrick, & Stritzke, 1999) *through* its impact on information processing. The second domain is comprised of studies designed to evaluate the sometimes persistent, pernicious effects of alcoholism on the brain (e.g., Oscar-Berman, 2000; Parsons, 1996). Relatedly, researchers are interested in how information processing might be altered by addiction in such a way that alcoholics become hypersensitive to alcohol-related stimuli or cues (Robinson & Berridge, 2002; Weirs & Stacy, 2006). The great importance of such work in both domains is clear. Unfortunately, it is beyond the scope of the present work to delve deeply into both domains. Instead, the current study was designed to examine the effects of acute alcohol intoxication, thus the presentation of related findings is restricted to this first domain.

1.1.1 Physiological Effects of Alcohol

Alcohol is a neurotoxin that can affect almost every organ in the human body, including the brain, due to its ability to pass through the blood-cerebral barrier. Notable physiological symptoms of alcohol intoxication are dizziness, increased heart rate and blood pressure, analgesia, and in more severe cases of intoxication also depressed respiration and loss of consciousness. Some of the most damaging negative consequences of alcohol consumption include hypertension, various liver diseases, and increase the risk of different cancers. Furthermore, alcohol can cause inflammation of the stomach, pancreas, and intestines, which impairs the digestion of food and absorption of nutrients into blood. Moreover, oxidation products can interfere with the activation of vitamins. Ethanol[1] itself on the other hand, does not have any minerals, vitamins, carbohydrates, fats or protein associated with it, therefore, providing only empty calories.

When consuming alcoholic beverages of any kind, ethanol absorption starts in the mucous membranes along the way to the small intestine, where the vast majority (80%) of alcohol absorption takes place. Once in the bloodstream, alcohol is transported throughout the whole body. Because ethanol is water soluble, it is distributed into body tissue in proportion to the water content. In contrast to other drugs, there are no specific receptors for ethanol in the brain, which makes it more difficult to determine the exact loci and mechanisms of alcohol induced effects on human behavior. The body metabolizes ethanol mainly through oxidation in the liver. Smaller amounts are excreted unchanged in breath, sweat and urine. The rate of alcohol metabolism is constant; in other words, only a certain amount of ethanol can be

[1] Ethanol (C_2H_5OH) is used as a synonym for alcohol in the present work. Even though there are many different forms of alcohol, ethanol is the one used in alcoholic beverages.

oxidized per hour independent from the amount of alcohol consumed. Furthermore, absorption and metabolism are influenced by gender and diet, or more specifically the content of the gastrointestinal tract when alcohol is consumed. The more time emptying the stomach requires, for example due to a high fat content diet, the longer the absorption will take. Attributable to differences in body water ratio (women have less body water) men are somewhat less affected by alcohol than similarly build women. In addition, women have a lower concentration of the ADH enzyme, which helps in metabolizing alcohol. More interesting in the context of the present work are psychological and behavioral effects of acute alcohol intoxication. A brief introduction into this field is given in the next section.

1.1.2 Psychological and Behavioral Effects of Acute Alcohol Intoxication

A very prominent concept that is associated with alcohol abuse is loss of behavioral control. Since the introduction by pioneering alcohol researchers (Jellinek, 1952; Keller, 1972) this concept has been modified to a less extreme view of impaired control (see Fillmore, 2003 for a recent review). The core assumption is that organisms that are more higly evolved can usually exert control over environmentally triggered behavior to completely inhibit, delay, or alter responses, a capability that seems reduced under alcohol intoxication. The key mechanism of behavioral control is the interplay of two processes that govern behavior: one that activates behavior and one that inhibits behavior (Fowles, 1987; Logan & Cowan, 1984). The frontal and prefrontal brain regions are thought to be the neural substrate underlying this ability. Recently, a series of studies using the stop-signal and cued go/no-go paradigms demonstrated that moderate doses of alcohol intoxication can impair the ability to inhibit a prepotent response in these relatively simple tasks (Abroms, Fillmore & Marczinski, 2003; Fillmore & Vogel-Sprott, 1999; Marczinski & Fillmore, 2003a, 2003b; Marczinski & Fillmore, 2005; Mulvihill, Skilling & Vogel-Sprott, 1997; de Wit, Crean & Richards, 2000). However, studies using the anti saccade task, in which a reflexive response has to be replaced with a voluntary action, has yielded contradictory results concerning alcohol induced impairments (see chapter 7).

Although the deleterious effects of alcohol on human behavior are recognized, the specific mechanisms underlying these impairments are still far from being understood well. Findings of recent studies suggest that the effect of alcohol intoxication is limited, if stimuli are presented without competing demands, involve automatic processing and are linked to immediate responses (Casbon, Curtin, Lang & Patrick, 2003; Fillmore, Vogel-Sprott & Gavrilescu, 1999; Holloway, 1994; Melia, Corodimas, Ryabinin, Wilson & LeDoux, 1996). These results seem to be in line with the concept of *alcohol myopia*, a term coined by Steele and Josephs (1990), reflecting the observation that alcohol intoxication causes one to focus

more intently on the most obvious, central or proximal stimuli in a particular context, to the neglect of more subtle, peripheral or distal stimuli. This hypothesis has been developed in the context of socially and emotionally relevant stimuli, and testing has been confined to these areas. In the present work, this concept will be used for the first time in the context of simple sensory-motor tasks as well as a complex cognitive task, namely reading. Further research on effects of acute alcohol intoxication is discussed at the beginning of the relevant chapters.

This thesis aims at providing the most comprehensive account to date of how acute alcohol intoxication affects visuomotor performance in basic oculomotor tasks. Performance will be studied on the automatic, automated, and voluntary levels of cognitive control (see chapters 2 and 3), as well as in complex cognitive tasks that are highly relevant in everyday life. It should also advance understanding of the role of alcohol in the disinhibition of behavioral and cognitive performance and of some of the mechanisms underlying these deficits.

The next chapter will provide general theoretical background that led to the selection of paradigms, which are suited to map the influence of moderate alcohol intoxication on different levels of oculomotor control. These paradigms are described in chapter 3. The experimental design is outlined in chapter 4, followed by the presentation of the single experiments in the remainder of the thesis.

2 Theoretical Background

The most important way humans navigate in and communicate with the environment is through acquisition and processing of visual information. Virtually all complex cognitive tasks rely on visual input, obtained via the planning and execution of rapid eye movements. Within the theoretical framework of "active vision," the traditional dissociation of perception from motor control is loosened; eye movements are regarded as 'part and parcel' of an integrated process of information acquisition (Findlay & Gilchrist, 2003). Research on eye movements and their use as indicators of perceptual and cognitive processes has become a flourishing field of study (see Hyönä, Radach, & Deubel, 2003; Van Gompel, Fischer, Murray, & Hill, 2007 for current reviews). The present thesis aims to capitalize on recent advances in the study of active vision to achieve a better understanding of the effects of alcohol intoxication. Although alcohol might well degrade performance of almost any task, the specific impairments of visual processing and visuomotor control occasioned by acute intoxication are currently not understood well. Where in the complex machinery of perception and motor control do alcohol effects come into play, and precisely which of these effects have what consequences in the context of real-world tasks? The analysis that follows could indicate some answers.

The first part of this chapter gives a brief introduction into the basics of eye movements followed by some neurophysiological background on eye movement control. Next, a functional model for the generation of saccades is explained in more detail. This modular framework, developed by Findlay and Walker (1999), specifies basic elements of visuomotor control and gives a sound theoretical foundation for the selection of experimental paradigms. The use of these theoretically based paradigms allows a systematic mapping of the effects of acute alcohol intoxication on different levels of visuomotor control (see Chapter 3). The last two sections of this chapter provide theoretical background for complex cognitive tasks, specifically task switching and reading. Complex tasks can complement findings on the effect of alcohol from the various levels of visuomotor control with regard to cognitive flexibility and inhibitory mechanisms. In addition, these tasks have a high ecological validity and are performed by a majority of people almost every day.

2.1 Eye Movements: Basics

When navigating through the environment (e.g., when reading a page of text, viewing a picture, or following a sports game), visual information enters the body through the eyes. First, light rays pass through the opening of the eye called the pupil, are then refracted by the

lens in order to be brought into focus on the ***retina***, the light sensitive tissue at the back of the eye. Visual information is then relayed to the brain. ***Visual acuity*** is distributed unevenly across the ***visual field*** (i.e., the area projected onto the retina), which is commonly divided into three regions. The area of highest visual acuity is called ***fovea*** and extends only about 1 degree of visual angle in the center of the visual field. Acuity sharply drops in the ***parafovea***, about the area composing 3 degrees of visual angle on either side of the center of the visual field. Acuity is even poorer in the ***periphery*** of the visual field, which is everything beyond the parafoveal area.

A consequence of this structure of the visual system is that eye movements are necessary to bring the object of interest into the center of the visual field, the fovea. To achieve this, the eyes jump abruptly from point to point, rather than making smooth movements. These jumps are called ***saccades*** and the average person makes more than 150,000 of these saccadic eye movements every day. However, it is only during the pauses between saccades, referred to as ***fixations***, that useful visual information is acquired. Research over the last two decades has demonstrated that various spatial and temporal parameters of eye movements represent valid and sensitive indicators of perceptual and cognitive processes (Liversedge & Findlay, 2000; Radach & Kennedy, 2004; Rayner, 1998). This 'window into the mind' can be used to 'view' and analyze alcohol-related changes in information processing. One example for a temporal parameter that is particularly important in the present thesis is ***saccadic reaction time*** or ***latency*** of saccades. Latencies are defined as the time from the onset of the event that triggers the eye movement until the start of the saccade. Latencies for saccades average at about 180-220 ms (Becker, 1989) and can be influenced by different experimental conditions (Megaw & Armstrong, 1973; Findlay, 1981). ***Saccade amplitude*** is an example for a spatial parameter and measures the length or extent of a saccade in degrees of visual angle. Other key parameters used in different experiments throughout this work will be defined when they are introduced to the reader.

2.2 Neurophysiological Background on Eye Movement Control

The oculomotor system is well researched and one of the best understood motor systems in the human body with regard to its neurophysiological background (see Leigh & Kennard, 2004 for a recent review). As mentioned before, there are no specific ethanol receptors in the brain and thus far, effects of alcohol have primarily been shown to reduce cortical activity (Davies & Alkana, 2001; Wang et al., 2000; Krull et al., 1994; Liu et al., 2000). Therefore, all subsequently described areas are potentially affected by alcohol intoxication. If paradigms that are used in this thesis lead to specific alcohol related impairments, these can be related to underlying cortical processes.

Figure 2-1 gives a schematic overview of brain structures involved in eye movement control. Visual information enters the system through multiple routes, the main route projecting from the extrastriate visual cortex to the parietal (PEF) as well as frontal (FEF) and supplementary eye fields (SEF). In addition, connections from earlier processing stages (i.e., the retina and primary visual cortex) project directly to the superficial layers of the superior colliculus (SC sup). Signals are usually place coded; in other words, size and direction of a saccade are coded by the location of the active cells (Schall, 1997, Andersen & Gnadt, 1989). The PEF as well as the FEF and SEF project heavily to the intermediate and deep layers of the superior colliculus (SCi/d). The substantia nigra pars reticularis (SNpr), which is the saccade related output nucleus of the basal ganglia also projects to the SCi/d but has an inhibitory impact. The SCi/d play a major role in the descending path of saccade related neurons. The output of the SCi/d projects to the premotor saccade generation circuitry in the mesencephalic, pontine and medullary reticular formations, where the discharge of neurons leads to innervations of the extraoculor muscles causing the desired saccade. Moreover, areas within the cerebellum that are important for the control of saccade amplitude, primarily by sending signals to end the saccade, are the oculomotor vermis and the fastigial nucleus (FN, Enderle, 2002; Scudder, Kaneko & Fuchs 2002).

The functional model that is introduced in the next section provides the theoretical framework for the current work and has links to these underlying neurophysiological structures at various points. Possible alcohol related dysfunctions of involved cortical areas will be discussed with the single experiments when appropriate.

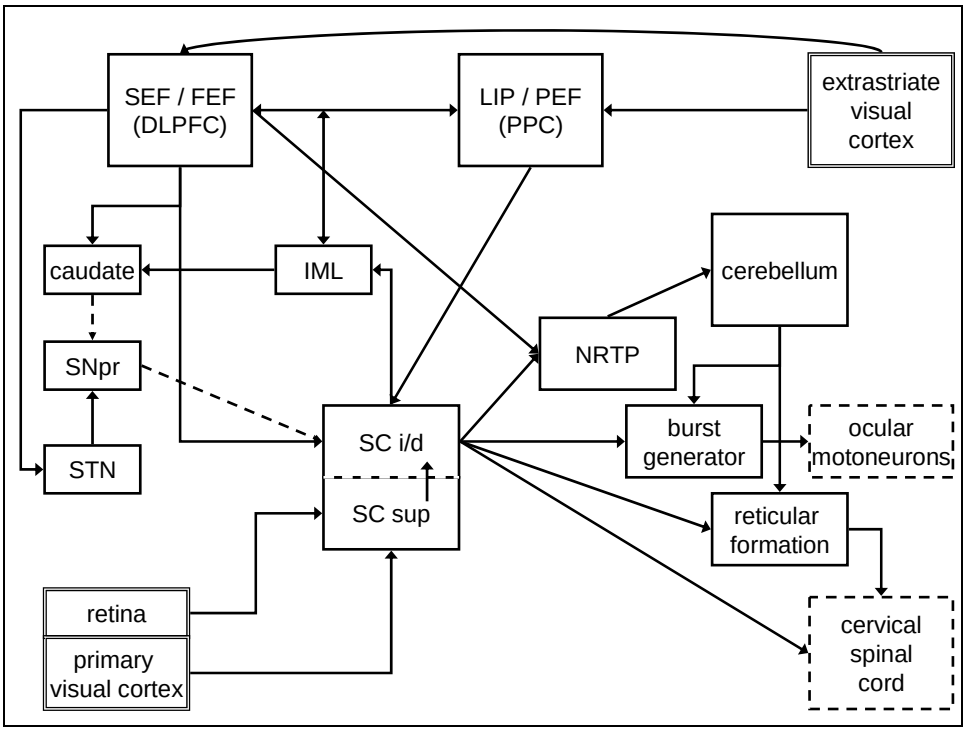

Figure 2-1. Schematic overview of brain structures involved in eye movement / saccadic control. Double lined boxes represent input, dashed boxes output units. Crossing lines do not connect. Arrows indicate direction of the connection, dashed lines indicate inhibitory connection. SEF = supplementary eye fields, FEF = frontal eye fields, DLPFC = dorsolateral prefrontal cortex, LIP = lateral intraparietal area, PEF = parietal eye fields, PPC = posterior parietal cortex, SNpr = substancia nigra, pars reticularis, IML = intramedullary lamina of thalamus, STN = subthalamic nucleus, SCi/d = intermediate and deep layers of the superior colliculus, SC sup = superficial layers of the superior colliculus, NRTP = nucleus reticularis tegmenti pontis. (Figure modified and combined from Scudder, Kaneko & Fuchs, 2002 and Leigh & Kennard, 2004).

2.3 A Theoretical Framework for the generation of saccades

Findlay and Walker (1999) offer a theoretical framework that provides an extensive integration of different experimental findings and is also considerably influenced by neurophysiological work. As can be seen in Figure 2-2, this information flow model is divided into two parallel streams of processing. One stream shows the information processing for the spatially relevant information (**where pathway**), determining the direction and extent or amplitude of the following saccade. The second stream, the **when pathway**, on the other hand shows modules that are important for the temporal aspects of the saccade, in other words when will the following saccade be triggered. Both processing streams function independently of each other; however, they are connected at several points where competitive interactions occur. In these cases reciprocal inhibitory links between two centers result in decreased activity in one center, if the connected center shows increased activity and vice-versa (see below for more details).

Furthermore, the model is structured into five levels, each representing a different processing stage. The remainder of this section will describe these processing levels of the Findlay and Walker framework, starting with lowest one.

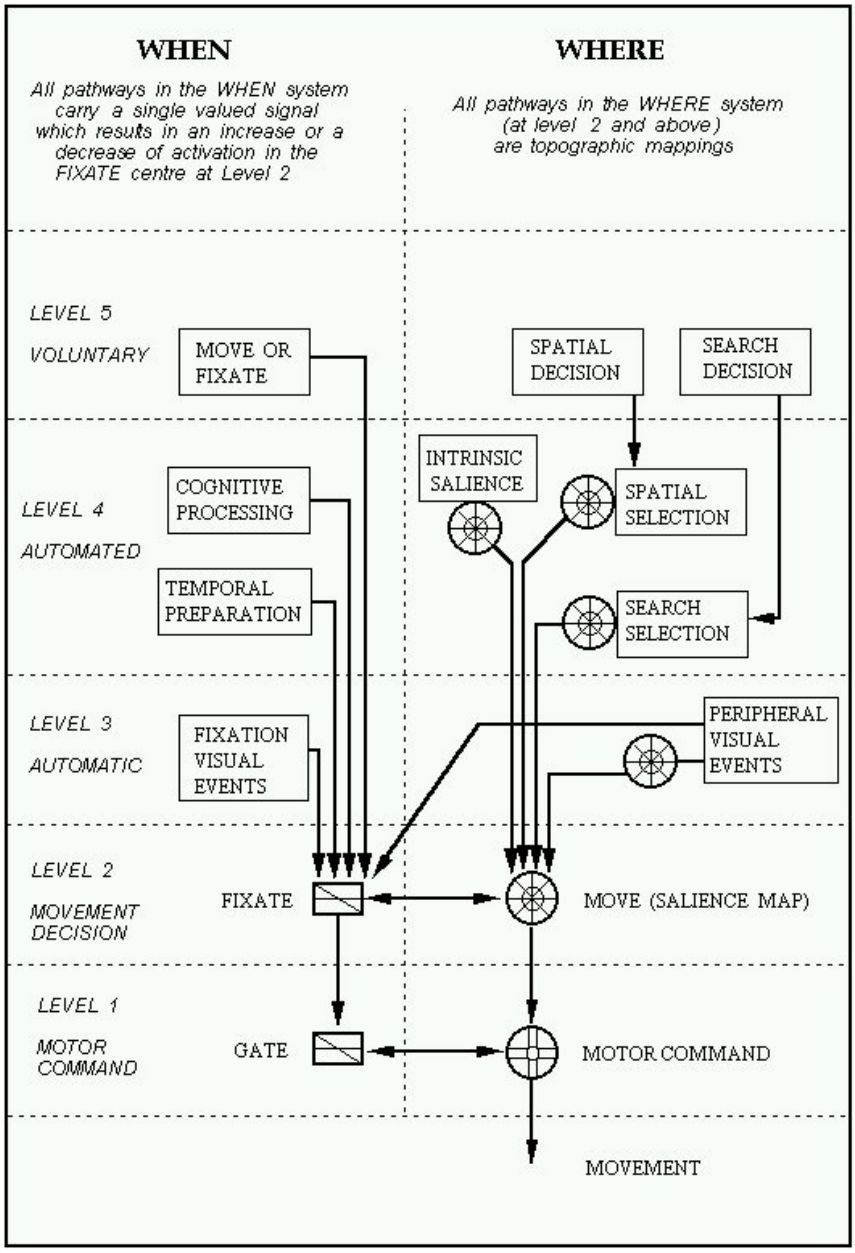

Figure 2-2. Diagram depicting the information flow routes in the temporal and spatial processing pathways, their interactions, and the different levels of control in saccade generation. From Findlay & Walker (1999).

2.3.1 LEVEL 1: Motor Command

The lowest level in the framework is labeled ***motor command*** and represents the immediate pre-motor stage. The oculomotor muscles are activated if a gate in the *when pathway* opens. Direction and amplitude of the resulting saccades are determined by the results of the processing in the *where pathway*. The neurophysiological substrate that mirrors the differentiation on this level can be found in brain stem processes. More specifically, omnipause cells fire at high rates and cease activity during saccades, whereas burst cells show the opposite behavior. The activity of the omnipause cells is independent of the spatial parameters of the saccade, but the activity of the burst cells is coded in terms of spatial metrics of the saccade (Fuchs et al, 1985; Scudder et al, 2002; Wurtz & Goldberg, 1989; Moschovakis & Highstein, 1994). Normal functioning on this level is reflected in an intact *main sequence* (Bahill et al, 1975; Ciuffreda & Tannen, 1995), which describes the relation between saccadic peak velocity and saccade amplitude. This relation should show a monotonic increase in peak velocity with longer saccades.

2.3.2 LEVEL 2: Movement Decision

The second level is composed of a *fixate center* in the when pathway and a *move center* in the where pathway. These centers are connected in a reciprocal inhibitory way. It is determined at this level, if to move and where to move, hence the label ***movement decision***. The result of this decision is directly transmitted to the gate in the motor command level (described above). Such a signal is sent, if the activation in the fixate center falls below a certain activation threshold. The activity in the fixate center is determined by the integration of various competing information signals that can come from three sources. Higher levels of the when pathway send their signals directly to the fixate center. In addition, the fixate center receives input from where pathway. The move center (connected to the fixate center through the reciprocal inhibitory link) is envisioned as a two-dimensional saliency map with spatiotopic coding. This saliency map is shaped by influences from higher levels of the where pathway. When a saccade is triggered, the location with the highest salience determines the metrics of the saccade. Furthermore, if the activation level in the move center is high, for example due to an exciting visual environment, the reciprocal inhibitory link to the fixate center reduces the activity there and makes saccades more likely. Finally, sudden onsets of visual events in the periphery result in an increased activity in the fixate center to account for findings that showed longer saccadic latencies when a visual distractor is displayed in the periphery. This increase in latency monotonically decreases with increases in eccentricity from the current fixation point and is independent of the distance between the target and the distractor. The neurophysiological substrate for the fixate center is assumed to

be the rostral pole region of the SC, which was shown to be active during fixations (Munoz & Wurtz, 1993a, 1993b). In addition, these cells in the rostral pole of the superior colliculus are also interacting in a push-pull relationship with the deep layers of the SC, which are known to code saccade metrics in a 'motor map'.

2.3.3 LEVEL 3: Automatic

The third level of processing is concerned with visual events that influence saccade programming. As effects described on this level are independent of previous experience (learning) and intentions, this level is labeled ***automatic***. The central feature of this level is the distinction between *central* and *peripheral* visual events. Events at the current fixation position directly effect the fixation centre in the when pathway. Onset of stimuli in the central region increase activity in the fixate center, whereas offset of stimuli in this are reduces activity in the fixation center. In contrast, the effect of peripheral visual stimuli on- or offset is twofold. First, the values in the *saliency map* are updated with each event in the periphery. An increase in the activity of the move center in the where pathway will render saccade triggering more likely, due to its reciprocal inhibitory connection with the fixate center. Second, peripheral visual onsets can directly influence the *fixate center*, as outlined in section 2.3.2.

2.3.4 LEVEL 4: Automated

Level four is labeled ***automated*** and represents processes that are not yet voluntary or even conscious but are influenced by implicit learning and memory. In the when pathway, two modules affect the fixate center directly. First, the *temporal preparation* module processes information that can be used to predict visual events such as warning signals (that do not need to be visual) or predictive timing sequences. This information can be used to reduce activity in the fixate center. Additionally, the *cognitive processing load* module can also directly affect the fixate center. Higher cognitive load leads to increased activity and longer fixation durations. In the where pathway three modules are located on this processing level. An *intrinsic saliency* module codes visual contours and high-contrast areas that are assumed to be intrinsically salient. This module is also thought to be responsible for carry-over effects in that previously used target stimuli retain some saliency even after a task switch. A second module on this level is called *spatial selection*. Spatial selection can either potentiate or inhibit the saliency in a particular area of the visual field. Due to constraints of distributed coding, the spatial window that can be influenced is relatively large. An example for spatial selection is a phenomenon called inhibition of return, which describes an increased difficulty to return to locations that were recently fixated manifested in longer saccadic latencies

(Klein, 1988). A last module on this level is the *search selection*. Search selection promotes saccades to particular visual features wherever in the visual field they may occur, thereby allowing selected features to have preferential access to level 2.

2.3.5 LEVEL 5: Voluntary

The highest level of the Findlay and Walker (1999) framework represents ***voluntary*** processes. Both pathways can be modified by input from the voluntary level. In the temporal pathway the influence is directly exerted on the fixate center in level 2. In the spatial processing pathway, the voluntary level exerts influence on the salience map in level 2; however this is in an indirect fashion (see Figure 2-2). A *spatial decision* module on the voluntary level is connected to the spatial selection module on level 4. As described in section 2.3.4, the spatial selection module can potentiate or inhibit saliency in a particular area of the visual field in the move center. Hence, the output of the spatial selection module is not only determined by influences from the automated level but can also be modulated itself by input from the voluntary level. Findlay and Walker assume the same indirect mechanism for the influence of a *search decision* module. Output from the *search selection* module on level 4 can be influenced by input from this *search decision* module located on the voluntary level. In a critique of this implementation of indirect influences of the voluntary level on saccade target selection, Radach (1999) suggests a direct route from the voluntary level on the saliency map on level 2. He argues that such a direct route is necessary to account for evidence from the domain of reading research, where a significant online influence on saccade target selection can be found for objects smaller than 1° of visual angle. In addition, the nature of the relevant information does not necessarily be visual. Apart from this theoretical comment, neurophysiological evidence suggests that voluntary saccades depend on functioning of the FEF (Pierrot-Deseilligny et al. 2002; Dias et al., 1995), an area that is less involved in the generation of reflexive saccades (Mort et al, 2003b).

In the next chapter, paradigms will be introduced that are well suited to examine processing on the different levels of oculomotor control and the influence of acute alcohol intoxication hereon.

3 Specific Aims and Experimental Paradigms

Alcohol consumption has the potential to affect performance of many human behaviors, however, the exact nature of this impact and the mechanisms underlying it are not well understood. Arguably, this uncertainty emanates at least in part from the extraordinary complexity of human behavior and the vast array of variables and brain structures that interact to influence it. The research plan introduced here focuses on a precise analysis of alcohol effects on visuomotor control as a promising strategy for illuminating the role of alcohol in these relationships. The legitimacy of this approach is predicated on the observation that acquisition and processing of visual information are central to the way humans navigate in and communicate with the environment. Moreover, many complex cognitive tasks rely on visual input obtained via planning and execution of rapid eye movements. Accordingly, a set of experiments is proposed, designed to evaluate the effect of alcohol on eye movements and the underlying processes they reveal.

The feasibility of using oculomotor tasks to explore alcohol effects on certain components of information processing has already been demonstrated. Some interesting findings have been reported regarding the pro and anti saccade tasks (cf. respective chapters). However, no readily interpretable pattern of results has emerged. This may reflect the so far limited attempts to develop studies or place their findings within a comprehensive theoretical framework and/or insufficient attention to the possible impact of variations in beverage manipulations. The proposed work seeks to improve upon this through more systematic applications of theory and methodology. The theoretical framework introduced above, in which the traditional separation of perception from motor control is diminished and eye movements are treated as part of an integrated process of information acquisition, serves as a foundation for the selection of experimental paradigms. This results in a set of experiments, where each experiment is designed to tap a different type or level of cognitive processing and behavioral control. Comparisons of performance across these tasks under alcohol compared with a no alcohol within subject control condition should afford to begin to map the impact of alcohol on oculomotor control in a systematic way. To the extent that particular deficits are found in different tasks, inferences can be drawn to allow for more precise identification of which levels and modules of the processing/control system are affected in what ways. Ultimately, those areas that are likely to be sensitive to alcohol intoxication could later be evaluated using appropriate measures of brain activity.

Taken together, this thesis seeks to advance understanding of alcohol effects by determining where in the complex machinery of perception and motor control effects of alcohol intoxication appear to come into play and which of them have what functional consequences.

In the following section, those experimental paradigms that were selected to examine the different processing levels established in the theoretical background (Chapter 2) are introduced, followed by a summarizing overview of the design of this thesis.

3.1 Basic oculomotor paradigms

The first and most basic level of oculomotor control that is targeted in the present work is the level of *automatic or reflexive control*. To examine functioning on this level a ***visually guided pro saccade*** task[2] was chosen. In this task a central fixation cross is presented for a certain amount of time, after which a peripheral visual target appears. Participants are instructed to look at the appearing peripheral target as quickly and accurately as possible. To examine automatic processes further, the ***gap paradigm*** (Saslow, 1967, Kingstone & Klein, 1993a; Walker et al, 1995) is implemented within the visually guided pro saccade task. In contrast to an *overlap* condition, in which a peripheral saccade target is introduced while a central fixation point is still visible, this central fixation point is removed prior to the onset of the peripheral saccade target in the *gap* condition. This offset of a central visual stimuli should reduce the activity in the fixate center and lead to shorter saccadic latencies. This *gap effect* has been shown to be maximal, if the offset of the central fixation point precedes the onset of the peripheral target by ~200ms (Fischer & Weber, 1997). The gap-effect is based on two components: a non-specific warning signal effect (Ross & Ross, 1980; 1981), which can also be induced using stimulation in non-visual modalities; and a specific oculomotor effect that is assumed to be the result of reduced activity in fixation related cells (Forbes & Klein, 1996; Dorris et al., 1995). This simple experimental design can therefore test functioning of oculomotor control on the reflexive level (see chapter 5).

On the *automated level* of control much of the visual processing in real world settings takes place. Typical examples are saccade patterns (scan paths) in visual search and reading situations. A paradigm reflecting critical components of performance on this level is the ***double-step task*** (Becker & Jürgens, 1979; Deubel, O'Regan & Radach, 2000). The basic setup is identical to that in the overlap condition of the pro saccade task, but in a certain percentage of trials the peripheral target is replaced with a second target after a specified interval, creating the appearance of a two-step jump (see chapter 6 for more details). This task indexes performance related to adaptive programming of sequential eye movements, a

[2] Note that the terms task and paradigm are used interchangeably throughout this thesis.

core ingredient of effective visuomotor behavior indicative of a substantially greater level of flexibility than is evident in automatic oculomotor responses.

A classic paradigm that is well suited to examine functioning on the *voluntary level* is the ***anti saccade task***. Again, using the identical visual setup as the pro saccade task, participants are instructed to look to the mirror position of the appearing peripheral target. This requires the inhibition of a reflexive movement to the target and a voluntary saccade to a location without visual stimulation has to be performed instead. In the functional model, this is possible, if information from the voluntary level overrides lower level information. An interesting variation that allows one to examine the importance of a visual marker at the saccade target location is the ***visually guided anti saccade task***. Visual setup and instruction are identical to the classic anti saccade task, but the possible target locations to the right and left of the central fixation cross are marked permanently with unfilled squares. This paradigm was selected to examine the extent to which additional helpful information can be used under alcohol intoxication. Another argument to include the visually guided anti saccade tasks is that fact that studies on the effect of drugs on visuomotor control have shown spatial impairments under the influence of cannabis (Ploner et al., 2002). If the same is true for alcohol intoxication, the additional use of this paradigm can help to narrow down the cause of possible spatial impairments.

Another paradigm that targets aspects of visuomotor control on the voluntary level is the ***memory guided saccade task***[3]**.** The visual setup is again identical to that of the overlap condition in the pro saccade task. The critical difference is that the peripheral target is presented for a short interval and participants are instructed to keep their fixation at the central fixation marker. Only after the offset of the central marker an eye movement is to be performed to the location where the peripheral target was displayed. The memory interval between offset of the peripheral and central fixation target is varied. This allows examination of visual-spatial short term memory effects and its modulation through alcohol intoxication. In addition, participants are required to inhibit a reflexive response to an appearing visual target and perform a saccade to a location without visual stimulation. In this respect, the task is similar to the demands of the anti saccade paradigm. However, in the memory guided paradigm no reprogramming of the spatial parameters is necessary, whereas a representation of the target location has to be kept in the visual-spatial short term memory.

[3] The use of three paradigms on the highest processing level compared to only one for the other levels is justified, as the most pronounced effects of alcohol intoxication are expected to be found on higher processing levels.

Even though a variety of other basic oculomotor paradigms is available, the five tasks chosen for this thesis are well suited to map possible effects of acute alcohol intoxication on the different levels of oculomotor control outlined in section 2.3. For each paradigm, relevant theoretical background is given at the beginning of the respective chapter.

3.2 Complex cognitive tasks

To complement the mapping of the effects of acute alcohol intoxication on oculomotor control, two complex cognitive tasks were selected. Both tasks are ecologically valid and are performed by almost everybody on a daily basis.

3.2.1 Task Switching

In everyday life, people frequently shift between cognitive tasks. There has been related research as early as the late 19th century (see Woodworth & Schlosberg, 1954), but the invention of the task switching paradigm is credited to Jersild in 1927. However, only in the last decade research on ***cognitive control*** functions went through a revolutionary revival. This development was clearly related to the advances in technology, especially with respect to improved neuroimaging techniques. Using this technology enabled researchers to examine mechanisms underlying cognitive functions with respect to the neurophysiological substrate (Kok, Ridderinkhof & Ullsperger, 2006).

Task switching experiments examine the effects of switching between different types of tasks on performance. Some typical phenomena (e.g., switch costs or benefits, preparation effects or residual costs) can be observed (see section 9.1 for details). Even though underlying mechanisms are still under discussion, it seems to be clear that the transfer of ***activation*** and ***inhibition*** from one trial to another trial can be seen as a key factor in the formation of switch costs.

Even though there are enough theoretical questions left regarding the exact mechanisms underlying task switching phenomena, including this paradigm is a valuable addition. Not only does task switching occur permanently in everyday life, but cognitive control and the undoubtedly associated mechanisms of activation and especially inhibition (see chapter 9) are closely related to, and in parts identical with, mechanism that are known to be influenced under acute alcohol intoxication and even in chronic alcoholics (when sober; Parsons & Nixon, 1993). In addition, inducing task switching by using the pro and anti saccade tasks, findings from these basic paradigms can be corroborated and extended.

3.2.2 Reading

In addition to basic visuomotor functions and the task switching paradigm, this thesis also uses a reading experiment to target higher level complex visual-cognitive processing. Compared to most other visual tasks and contexts in the natural environment, reading takes place in a ***simple perceptual setting*** and includes relatively few types of stimuli (e.g., features, letters, and words). It thus permits the study of basic oculomotor and perceptual processes under well-controlled, yet ***ecologically valid***, conditions. Its advantages in this regard have been amply demonstrated in studies addressing the role of visual attention during continuous reading. Such work can have profound implications for visual information processing in general (e. g., Inhoff, Eiter & Radach, 2005; Inhoff, Radach, Eiter, & Juhasz, 2003).

In addition, reading is among the most frequent and important of everyday tasks. Therefore, understanding the effects of acute moderate alcohol intoxication on reading performance is an important research endeavor in itself, and the effects can be regularly observed in college populations who are required to read frequently. Despite the obvious significance of the task, there has been only one prior attempt to examine the influence of alcohol on reading behavior to date (Watten & Lie, 1997, see section 10.1.4 for details).

Reading involves the coordination of two streams of processing, **linguistic** and **visual**. Both domains are relatively well understood and there are increasing numbers of computational models implementing the architecture and dynamics of various levels and modules of processing (e.g., Reichle, Rayner & Pollatsek, 2003; Reilly & Radach, 2006; Kliegl & Engbert, 2003). It is perhaps fair to say that reading is one of the most theoretically and methodologically advanced areas in cognitive research (Rayner, 1998), providing a solid base for application in a clinical field like alcohol research.

Building on the current level of theory and methodology in this field (see section 10.1), the analysis of the reading data will focus on two aspects. First, a variation of the mental workload associated with word processing via manipulation of word frequency (occurrence per million in written English) is used to examine whether detrimental alcohol effects operate on the visuomotor and/or the linguistic processing stream. Second, by using eye movement contingent display manipulations[4], the nature and extent of alcohol's effects on parafoveal

[4] This technique is commonly used in reading research and is explained in detail in section 10.1.3.

information acquisition can be examined, thus providing a novel perspective on issues of tunnel vision and alcohol myopia in the context of a natural task (see chapter 10).

3.3 Overview Paradigms

As outlined above, five basic oculomotor paradigms and two complex cognitive tasks were selected to map the influences of moderate alcohol intoxication on different levels of oculomotor control. Table 3-1 provides an overview on the selected paradigms with corresponding processing levels in the theoretical framework and associated primary neural correlates.

The paradigms were divided into two clusters. Cluster 1 included the five basic oculomotor tasks, whereas Cluster 2 consisted of the two complex cognitive tasks. This division was necessary, because the time available to test participants under alcohol is limited, as well as to minimize effects of fatigue.

Table 3-1. Selected paradigms, processing levels and primary neural correlates. PPC =posterior parietal cortex, PEF = parietal eye fields, DLPFC = dorsolateral prefrontal cortex, FEF = frontal eye fields, SEF = supplementary eye fields, BA = Broadman Area, PFC = prefrontal cortex.

Paradigm	Cluster	Processing Level	Primary Neural Correlates	Chapter
pro saccade	1	automatic	PPC	5
double step	1	automated	PPC, Thalamus, PEF	6
anti saccade	1	voluntary	DLPFC, FEF, SEF	7
visually guided *anti* saccade	1	voluntary	DLPFC, FEF, SEF	7
memory guided	1	voluntary	DLPFC (BA46, BA9)	8
task switching (pro and anti saccade)	2	voluntary / automated	medial & lateral PFC, parietal lobe, cerebellum	9
Reading	2	all levels	all saccade related + text processing modules	10

4 Experimental Methods

This chapter provides an overview of the experimental procedures used. As described above, two clusters with a total of seven experiments were conducted. The following sections describe participant screening, alcohol administration, and the eye movement recording technique, which were identical for both Clusters. Section 4.4 provides information about the experimental procedures and composition of the two samples. Stimulus material for the single experiments is described in the respective chapters.

4.1 Participant Screening

Participants had to be of legal U.S. drinking age (21+) and have recent experience with alcohol doses comparable to those administered in this study. To ensure eligibility, age was verified by the experimenter checking an official government issued picture identification. In addition, participants were administered a Drinking Behavior Survey, a Medical Screening Questionnaire, as well as the Short Michigan Alcoholism Screening Test (SMAST; Selzer et al., 1975). Grounds for exclusion were reports of an average of more than 5 drinks per day for men (or more than 4 for women)[5], any medical condition reported on the Medical Screening Questionnaire that might contraindicate alcohol consumption, or a score of >3 on the SMAST. Female participants had to have a negative result on a urine sample pregnancy test (Quick Vue One-Step hCG: Quidel, San Diego, CA). To avoid inclusion of 'outliers' in terms of beverage volumes associated with the target blood-alcohol level (BAL), the weight of participants had to be within two standard deviations of established standards for height and sex.

Participants were instructed to abstain from alcohol for at least 24hr and all other drugs for at least 72hr prior to each session. In addition, participants were asked to have a light lunch, but afterwards abstain from food and beverages (except water) for at least four hours, prior to arrival for their appointments, which were always scheduled to occur in the late afternoon or early evening. Further, regardless of other conditions of consent, all prospective participants had to agree to remain at the experimental site until their breath alcohol concentration (BrAC) were at 20 mg% or below, that is, in a range legally defined as safe and identified as such in the National Institute on Alcohol Abuse and Alcoholism "Guidelines on Ethyl Alcohol Administration in Human Experimentation" (June, 1989). They were advised that

[5] Note that "drink" refers to a standard drink equivalent to 12oz (~350ml) of beer, 5oz of wine (~150ml) or 1.5oz (~40ml) of hard liquor.

this might take up to four hours, but that they would be compensated for their time (see below). Finally, all participants had to make a commitment to comply with prescribed arrangements for transportation or escort. These latter requirements represent conservative safeguards designed to ensure that no one would drive home or walk home alone even with only negligible BrAC. If a participant could not or did not make the specified exit arrangements, the research team provided transportation.

Participants received credit toward a course research participation requirement, a payment of $5 per hour, or a prorated combination of the two. All procedures of the studies were approved by the Florida State University Institutional Review Board.

4.2 Alcohol Administration

The target BrAC in both studies was 70 mg%, a level that is well above the minimum shown to impair a wide range of complex psychomotor tasks (Holloway, 1994) and just below that constituting prima facie evidence of alcohol intoxication for driving purposes at the site of the experiment.

In the alcohol session, participants received a beverage containing chilled tonic water mixed with 100-proof vodka in a 5:1 ratio. The amount of alcohol administered to reach the target was calculated for each participant based on height, weight, age, gender, and the length of the drinking period (see Curtin et al., 1998, for details of the algorithm used). The beverage was equally distributed into four containers, each of which had to be consumed by the subject in consecutive 5 minute periods. Following this 20 min drinking period was a 20 min absorption period. Accurate information was given about the approximate equivalence for the total beverage content in terms of standard alcohol drinks. BrAC was measured before the drinking period (to insure a zero baseline), at the end of the absorption period, immediately prior to, and immediately following each task using an Alcosensor IV (Intoximeters Inc: St. Louis, MO).

In no-alcohol sessions, participants received the same total amount of liquid, consisting of tonic water only. They were also given accurate information about beverage content in this condition. The decision to use a simple no-alcohol control rather than a placebo control condition in these studies was a reasoned one. First, it was desirable to implement the simplest possible design that still included the critical contrast. Second, in this connection, it is widely acknowledged among alcohol researchers that, at least when using oral administrations of alcohol, it is quite difficult to achieve even nearly equivalent levels of either alcohol expectancy or subjective intoxication across alcohol and placebo conditions,

thereby rendering suspect any comparative inferences based on them. Third, there is mounting evidence (see Testa et al., 2006, for a review) that when dealing with drugs like alcohol, whose effects are very familiar to participants and which may therefore be subject to efforts to minimize them, placebos can invite misleading effects and might actually yield performances that exceed those obtained in simple no-alcohol conditions due to compensatory efforts. Obviously, such an artificially driven effect should be avoided. Of course, future research might do well to include all three conditions, but that seemed premature before simple effects were documented.

4.3 Eye Movement Recording

Eye movements were recorded using an EyeLink2 head-mounted video based pupil tracking system (SR Research Ltd., www.eyelinkinfo.com), sampling at 500 Hz. The system consists of two PCs: an operator PC to control and monitor camera setup; and a subject PC used for stimulus presentation. Both computers are connected through Ethernet cards that enable data transfer with minimal time delay. The recording system includes a high-speed video camera positioned 4-7 cm below the monitored eye and held in place by head-mounted gear. Infrared LEDs irradiate the eye with light of 940 nm wave length. The irradiation of the eye averages to 0.8mW/cm2[6]. The reflection of this light from the pupil serves as the basis for determining the eye position. The system has a relative spatial resolution in the order of a few minutes of arc and its absolute accuracy is better than 1/3 deg, depending on calibration.

Viewing was binocular, but eye movements were recorded from the right eye only. Participants were seated in a comfortable chair with a viewing distance of 82 cm in front of a nominal 22-inch CRT monitor. The monitor was run with a resolution of 1,024*768 at a refresh rate of 160Hz. Calibration trials with three horizontal targets were performed before each block of trials. Mean average position error in an accuracy validation routine was not to exceed 0.33°. The on-line saccade detector of the eye tracking system was set to detect saccades with an amplitude of 0.15° or greater, using an acceleration threshold of 8,000°/sec² and a velocity threshold of 30°/sec.

[6] The irradiation of the eye with infrared light at these levels does not pose any risk for the health of participants (or their eyes).

4.4 Procedure

After eligibility screening and consent, participants were seated in front of a monitor and the eye tracking equipment was set up for training. Following the training (see sections 4.4.1 and 4.4.2), participants were weighed to determine the amount of beverage to be administered and either alcoholic or non-alcoholic beverages were prepared. The order of no-alcohol and alcohol session was determined by assigning participants randomly to beverage conditions at the first session. Apart from the type of beverage administered, sessions one and two were identical.

During the drinking and absorption periods, participants answered a battery of individual difference questionnaires on alcohol and other drug use, as well as some pertinent to personality, emotional and behavioral attributes. These data were collected for later exploratory analysis of possible moderators and/or mediators of observed effects. At the end of the absorption period, initial BrAC was assessed. Next, participants performed in the experimental paradigms of the respective cluster and additional BrAC were measured between tasks. In the alcohol session, participants completed additional BrAC tests until two consecutive readings were below established criteria for release (<20 mg%), at which time they were driven or escorted home. Following the second session, participants were fully debriefed.

The next two sections provide information about the order of tasks within the sessions and sample characteristics for the two clusters.

4.4.1 Cluster 1: Basic Oculomotor Paradigms

The training for the basic oculomotor paradigms consisted of five units, one for each paradigm. During an initial phase within each unit, instructions were given and eight self-paced sample trials were executed. In the second phase, the calibration routine was introduced and 20 trials were practiced using the experimental presentation times for visual stimuli. This training procedure lasted about 10 to 15 minutes..

The order of the tasks was held constant between sessions as well as between participants[7]. After the absorption period the session started with the pro and anti saccade paradigms, followed by the visually guided anti saccade paradigm. The third task was the double step

[7] The order of the tasks was kept constant, to reduce variability caused by potential interference between tasks. Possible effects of practice or fatigue were accepted instead, as critical comparisons are within subject.

paradigm and the memory guided saccade paradigm concluded the session. Although, data for the pro and anti saccade paradigms were collected in the first of four experimental blocks, results will be presented separately for each paradigm, according to the different levels of oculomotor control introduced in chapter 2. The major reason for grouping the collection of pro and anti saccades paradigms within one experimental block was to assure that blood alcohol levels were identical in both paradigms, providing the most convincing contrast between effects of alcohol on reflexive vs. voluntary levels of oculomotor control.

BrAC was determined before the first task, between each task and immediately after the last task. A complete no-alcohol session lasted about 2 ¼ hours while a complete alcohol session lasted as long as five hours or more (see Table 4-1 for an overview).

Table 4-1. Overview of experimental protocol and timeline for basic oculomotor paradigms.

TIMELINE FOR THE EXPERIMENTAL PROTOCOLS	
Pre-laboratory Screening: The Drinking Behavior Survey was administered in mass screenings of psychology classes and results were used to establish an initial pool of participants who are eligible based on age and drinking experience. Appropriate individuals were telephoned and administered the Health Interview instrument to exclude those for whom alcohol is contraindicated. Remaining prospects were invited to participate in a study of "the effects of alcohol use on visual processing" and, if interested were scheduled and given further instructions.	
Laboratory Session	required time (min)
Introduction, Informed Consent, Pregnancy test (women), baseline BrAC	10
Instruction/familiarization with experimental tasks in the practice blocks	12
Beverage preparation (including weight & height measurement to determine appropriate dose/volume to achieve target BrAC of 70 mg% in alcohol condition)	10
Drinking Period	20
Absorption Period BrAC	20
Experimental Tasks	
Pro-saccade task (followed by BrAC)	6 (+2)
Anti-saccade task (followed by BrAC)	6 (+2)
Visually guided anti saccade task (followed by BrAC)	6 (+2)
Double-step task (followed by BrAC)	10 (+2)
Memory guided saccade paradigm (followed by BrAC)	15 (+2)
Alcohol metabolism (repeated BrACs at 15min intervals for Alcohol sessions to 20mg%)	up to180
Debriefing, Compensation, Release to pre-arranged driver or escort	10
Total Time *No-alcohol Session*	135
Total Time *Alcohol Session*	up to 315

4.4.2 Cluster 2: Complex Cognitive Tasks

The training phase for the complex cognitive tasks consisted of two units, one for each paradigm. For the task switching paradigm participants were given instruction and practiced eight self-paced sample trials, followed by 20 trials using experimental timing for the visual stimuli. For the reading experiment 10 practice sentences were provided, each followed by a comprehension question to familiarize participants with task. This training procedure took about 10 to 15 minutes.

Table 4-2. Overview of experimental protocol and timeline for complex cognitive paradigms.

TIMELINE FOR THE EXPERIMENTAL PROTOCOLS	
Pre-laboratory Screening: The Drinking Behavior Survey was administered in mass screenings of psychology classes and results were used to establish an initial pool of participants who are eligible based on age and drinking experience. Appropriate individuals were telephoned and administered the Health Interview instrument to exclude those for whom alcohol is contraindicated. Remaining prospects were invited to participate in a study of "the effects of alcohol use on visual processing" and, if interested were scheduled and given further instructions.	
Laboratory Session	required time (min)
Introduction, Informed Consent, Pregnancy test (women), baseline BrAC	10
Instruction/familiarization with experimental tasks in the practice blocks	10
Beverage preparation (including weight & height measurement to determine appropriate dose/volume to achieve target BrAC of 70 mg% in alcohol condition)	10
Drinking Period	20
Absorption Period BrAC	20
Experimental Tasks	
Task switching (followed by BrAC)	18 (+2)
Reading (followed by BrAC)	18 (+2)
Alcohol metabolism (repeated BrACs at 15min intervals for Alcohol sessions to 20mg%)	up to180
Debriefing, Compensation, Release to pre-arranged driver or escort	10
Total Time *No-alcohol Session*	120
Total Time *Alcohol Session*	up to 300

The order of the tasks was fixed, with the task switching paradigm always preceding the reading task. The task switching paradigm has a fixed time, whereas the duration of the reading tasks could vary between participants. Therefore, randomizing or counterbalancing the order of the tasks could have created an addition source of variability. The total duration of sessions in Cluster 2 was comparable with that in Cluster 1. Table 4-2 gives an overview of the experimental protocol and timeline.

4.5 Sample Characteristics

The following sections provide information regarding the sample characteristics for Clusters 1 and 2. In both studies, all participants had normal or corrected to normal visual acuity and intact color vision as determined by a test with a standard Snellen Chart.

4.5.1 Cluster 1: Basic Oculomotor Paradigms

Twenty-six eligible persons (13 male, 13 female) were scheduled for participation in the experiments after screening (see section 4.1). Two participants did not return for the second session and an additional four data sets had to be discarded, as participants did not reach a sufficient breath alcohol level (<30 mg%), resulting in 20 datasets (9 male, 11 female) for analysis. Mean age of participants was 24.7 years (range = 21-31 years). Self reported drinking behaviors for the past year indicated a mean of 1.9 (sd = 0.9) drinking episodes per week and an average of 3.2 (sd = 1.4) drinks per episode.

The beverage condition manipulation resulted in a mean BrAC of 69 mg% averaged across all points of measurement. To determine the actual BrAC for a given paradigm, BrAC measures taken before and after the each task were averaged. Table 4-3 shows that across all tasks BrACs were at the targeted level, with slightly lower values in the last task.

4.5.2 Study 2: Complex Cognitive Tasks

Thirty-six eligible persons (18 male, 18 female) were scheduled for participation in the experiments after screening (see section 4.1). Four participants did not return for the second session. Due to technical problems, for eight participants data were not recorded appropriately in the task switching paradigm. Therefore, 24 datasets (12 male, 12 female) were analyzed for this paradigm. Mean age of participants was 22.7 years (range = 21-31 years). Self reported drinking behaviors for the past year indicated a mean of 1.8 (sd = 1.0) drinking episodes per week and an average of 3.5 (sd = 1.7) drinks per episode. For the reading task, 32 datasets were available for analysis. For this extended sample (17 male, 15 female) participants had a mean age of 23.2 years (range = 21-31). Self reported drinking behaviors for the past year indicated a mean of 2.0 (sd = 1.2) drinking episodes per week and an average of 3.8 (sd = 1.6) drinks per episode.

The beverage condition manipulation resulted in a mean BrAC of 68 mg% for the task switching part of the study and 71 mg% for the reading task (see Table 4-3), again determined by averaging measurements from before and after each task.

Table 4-3. Overview of sample characteristics including sample size (N), mean age, self reported number of drinking episodes and drinks per episode for the last year as well as mean breath alcohol concentrations for all paradigms in both studies.

Study	N	mean age	mean drinking episodes per week	mean number of drinks per episode	Paradigm	mean BrAC
1	20	24.7 (21-31)	1.9 (0.9)	3.2 (1.4)	pro and traditional anti saccade	73mg%
					visually guided	74mg%
					double step	70mg%
					memory guided	64mg%
2	24	22.7 (21-31)	1.8 (1.0)	3.5 (1.7)	task switching	68mg%
2	32	23.2 (21-31)	2.0 (1.2)	3.8 (1.6)	reading	71mg%

5 Automatic Processing: The Pro Saccade Paradigm

To begin the mapping of influences of acute alcohol intoxication on oculomotor control, the visually guided pro saccade paradigm was used to examine functioning on the automatic (reflexive)[8] processing level. Processing on this level of oculomotor control is generally assumed to be automatic, because it is independent of prior learning and intentional influences. After providing specific theoretical background, hypotheses are derived and results are presented and discussed.

5.1 Theoretical Background

Under natural conditions, humans make several spontaneous saccades per second. The majority of these saccades are triggered by novel stimulation. Stimuli triggering reflexive saccades can be visual, auditory, somatosensory, or consist of a combination of stimuli from different sensory modalities. In the laboratory, reflexive visual saccades can be elicited when a novel stimulus is presented in the visual periphery. Related to the framework presented in section 2.3, this peripheral visual stimulation does not only affect the saliency map in the *where stream*, but also directly influences the fixate centre in the *when pathway*. In addition to peripheral events, visual stimulation at the current fixation location also plays a role on the automatic processing level.

5.1.1 Gap and overlap conditions

Stimulation at the current fixation location can be preserved during or extinguished before the presentation of a peripheral target, creating two conditions involving different neurophysiological structures. In the *gap condition*, when the target at the current fixation location disappears before the onset of the peripheral target, human subjects usually generate saccades with very short reaction times (Fischer & Weber, 1997; Fischer & Boch, 1983; Fischer & Ramsperger, 1984; Fischer & Weber, 1993). As discussed in section 3.1, this gap effect is not only a result of a general warning effect, but has a specific oculomotor component. While the posterior parietal cortex (PPC) has been shown to play an important role underlying the general warning aspect (Pierrot-Deseilligny et al., 1987,1991), the specific oculomotor component can be attributed to the release of a fixation mechanism that has its neurophysiological substrate in the rostral pole of the SC (Dorris et al., 2002; see also

[8] The terms automatic and reflexive are used interchangeably in this thesis.

section 2.2). In monkeys, short latency saccades in the gap condition can be eliminated with lesions of the SC (Schiller et al., 1987). Thus, the use of the gap condition provides a way of testing collicular function in humans.

Functioning in the *overlap condition*, in which the central fixation point remains visible during the onset of peripheral stimulation, is related to the FEF. Numerous studies in humans and monkeys demonstrated increased latencies for visually guided saccades when FEF functioning was impaired (Pierrot-Deseilligny, 2002; Rivaud et al., 1994; Braun et al., 1992; Dias et al., 1995).

5.1.2 Previous Studies on Alcohol and the Pro Saccade Paradigm

There were several studies that examined the influence of acute alcohol intoxication on performance in the pro saccade task (Baloh et al., 1979; Lehtinen et al., 1979; Jantti et al., 1983; Lockemann & Westhofen, 1996; Gale et al., 1996; Moser et al., 1998; Wegner & Fahle, 1999; Blekher et al., 2002; Vassallo & Abel, 2002). Generally, saccadic latencies were found to be reduced under alcohol, as are peak velocities. However, some studies failed to show such a slowing for latencies (Lehtinen et al., 1979) or peak velocities (Lockemann & Westhofen, 1996). Data on saccade accuracy in the pro saccade task were not always reported, but in those studies that did report those, most found that the accuracy of visually guided pro saccades was not (Moser et al., 1998; Blekher et al., 2002; Vassallo & Abel, 2002) or hardly affected by alcohol intoxication (Wegner & Fahle, 1999). However, none of this research had a firm theoretical foundation or tested specific hypotheses with regard to the effects of alcohol on oculomotor control.

One experiment that also implemented a variation of gap and overlap conditions (Wegner & Fahle, 1999) found that the gap effect was more pronounced under alcohol. In the alcohol session, latencies were about 18% shorter in the gap compared to the overlap condition, but latencies were only about 8% shorter in the sober session under gap conditions. The size of the gap effect in the sober condition was smaller than normally reported. This might have been a result of the large number of possible target locations (22 locations and eight directions) that were implemented in this study. The interaction between alcohol and gap condition could therefore result primarily from the difference in the overlap condition (265ms vs. 221ms). However, regarding the gap conditions, latencies were still significantly longer under alcohol compared to the placebo condition (218ms vs. 203ms), although less pronounced than in the overlap condition. Figure 5-1 gives an overview over these results.

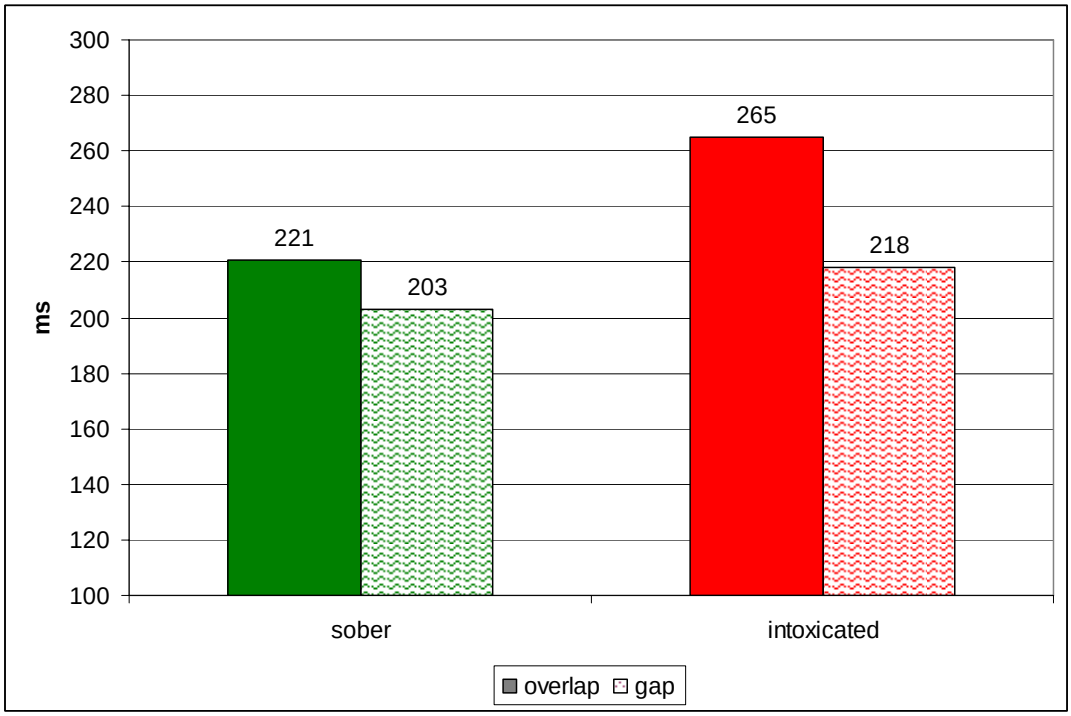

Figure 5-1. Saccade latencies in overlap and gap conditions for sober and intoxicated sessions in the study by Wegner & Fahle (1999). Note that variability measures were not extractable from the article.

Taken together, Wegner and Fahle (1999) conclude from their results that alcohol predominantly affected function of the SC, as latencies in the gap condition were longer under alcohol than in the no alcohol control condition. Other cortical regions that were assumed to mediate the gap effect (the PPC) seemed to be less affected by alcohol intoxication, due to the fact that the gap effect did emerge under alcohol intoxication.

Within the context of this thesis, the visually guided pro saccade experiment with gap and overlap conditions examines functioning on the automatic level of oculomotor control as a first step towards mapping the consequences of acute alcohol intoxication within a theoretically grounded framework.

5.1.3 Dependent variables in the pro and anti saccade task

Before stating the hypotheses, dependent variables, as used in the context of this thesis, are defined at this place. The ***error rate*** measures the proportion of saccades that leave the central fixation cross in the wrong direction. For a pro saccade trial this means moving the eye away from the visible peripheral target. The ***primary saccade latency*** measures the time from the onset of the peripheral target up to the beginning of the first following saccade. ***Primary saccade amplitudes*** measure the extent of this first response in degrees of visual

angle, and the ***peak velocity*** gives a measure of motor speed. Latency, amplitude, and velocity measures are calculated separately for *gap* and *overlap* conditions for *correct* responses.

5.2 Hypotheses

Given the theoretical considerations and the results of previously published studies outlined above, it is predicted that alcohol intoxication will somewhat slow eye movements in the visually guided pro saccade task, thus affecting processing on the automatic level of oculomotor control. This should become evident in prolonged latencies and slower saccadic peak velocities. However, error rate as well as spatial parameters of saccades are expected to be largely unaffected by alcohol.

Regarding the gap manipulation it is predicted that a gap effect will show in both alcohol conditions. However, in contrast to the results reported by Wegner & Fahle (1999), no interaction between alcohol and gap condition is expected. As outlined above, the interaction in Wegner and Fahle is largely due to the small gap effect in the sober condition, in which the current study should obtain a normal sized effect.

5.3 Materials and Method

Information regarding participants, alcohol administration, eye movement recording, and procedure were already provided in Chapter 4. The following sections will outline the experimental design in more detail and explain data analysis strategies.

5.3.1 Design

A pro saccade trial starts with the presentation of a green fixation cross with a diameter of 0.5° of visual angle, centered on an otherwise black screen. In half of the trials, a light grey circle with a diameter of 0.3° visual angle appeared after 1,000 ms at 6° either to the right or to the left of the central fixation cross, constituting the *overlap* condition. In the other half of the trials, the central fixation point disappeared after 800 ms, leaving a blank screen for 200 ms, before the peripheral target appeared (*gap* condition). The peripheral target stayed visible for 800 ms under both conditions before the next trial started. Participants were instructed to look at the peripheral target as quickly and accurately as possible as soon as it appeared.

As described in section 4.4.1, the pro saccade paradigm was implemented together with the anti saccade paradigm as the first task in a series of experiments. Eight blocks (4 pro, 4 anti) with 48 trials each were presented. The paradigm switched between pro and anti saccades every two blocks with the order of the first task counterbalanced between participants. The completion of each block took about 100 seconds, resulting in a total duration of about 15 minutes for the experiment, including calibration of the eye tracking system at the beginning of each block.

5.3.2 Data Analysis

Saccades were classified online using EyeLink software. Raw data files were converted into SPSS data matrices, using custom build software. Trials with primary saccade latencies shorter than 60 ms or longer than 800 ms or primary saccade amplitudes < 1.5° and >12 were excluded from analysis. Neither shorter nor longer latencies were regarded as conforming to instructions and were discarded, because in these cases saccades would be randomly directed towards or away from the correct response position. These restrictions resulted in 91% valid primary saccades across all trials, corresponding to a total of 6,985 primary responses. Data were analyzed using 2 x 2 x 2 repeated measures ANOVA with the within subject factors Gap Condition[9] (overlap vs. gap) and Beverage Condition (no alcohol vs. alcohol) and the between subject factor Alcohol Session. The factor alcohol session represents the fact whether alcohol was consumed in the first or the second session.

5.4 Results

Table 5-1 presents an overview of results for all cells of the experimental design, including error rates, latencies, saccade amplitudes, and peak velocities for correct responses. All analyses were based on individual subject mean values (n = 20). ANOVA tables, as well as tables with values for results that rely on data averaged across conditions were placed in the Appendix (see 0).

[9] Note that for the remainder of this thesis capital initial letters are used to indicate factors.

Table 5-1. Overview of results on key dependant variables in the pro saccade task. Means and standard error (SE) are based on 20 participants and presented for all factor combinations.

	Means (SE)							
	Alcohol Session 1				Alcohol Session 2			
	overlap		gap		overlap		gap	
	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol
proportion of errors	0.005 (0.002)	0.002 (0.002)	0.007 (0.003)	0.023 (0.006)	0.001 (0.003)	0.001 (0.002)	0.007 (0.003)	0.014 (0.007)
primary saccade latency (ms)	215 (7)	220 (7)	159 (6)	170 (6)	202 (9)	214 (9)	160 (7)	170 (7)
primary saccade amplitude (degree)	5.83 (0.08)	5.64 (0.1)	5.77 (0.1)	5.58 (0.11)	5.75 (0.1)	5.74 (0.12)	5.67 (0.12)	5.64 (0.14)
peak velocity deviation (%)	0 (4)	-21 (4)	9 (5)	-17 (5)	4 (5)	-19 (5)	15 (6)	-8 (6)

5.4.1 Error Rate

Overall, error rates (Figure 5-2) in the pro saccade task were very small (~1%) and not affected by the between subject factor Alcohol Session ($F_{(1,18)}$= 0.65, p>.10). In the gap condition, error rates were significantly higher compared with the overlap condition ($F_{(1,18)}$= 22.26, p<.001), but there are no significant differences between Beverage Conditions ($F_{(1,18)}$= 1.96, p>.10). However, a significant interaction between Gap and Beverage Condition was found ($F_{(1,18)}$= 8.76, p<.05). Figure 5-3 illustrates this interaction, showing a significant increase in error rates under alcohol for the gap condition ($F_{(1,18)}$= 6.95, p<.05) [10] and a slight, but not significant, decrease for the overlap condition under alcohol ($F_{(1,18)}$= 0.90, p>.10), resulting in a larger difference between Gap Conditions under alcohol (see A-1.1 for complete ANOVA and means tables).

[10] Single contrasts were used to determine significance for single comparisons. Critical F-values were corrected using Scheffe. Following post hoc comparisons use the same analyses unless stated otherwise.

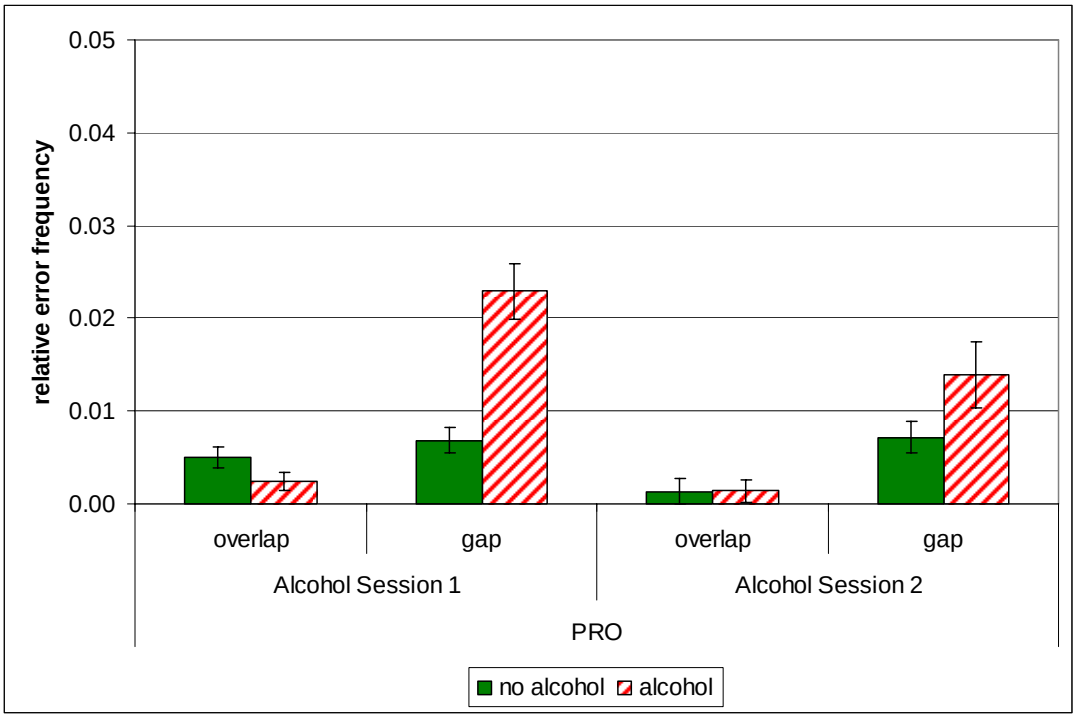

Figure 5-2. Effects of Alcohol, Gap Condition, Beverage Condition, and Alcohol Session on error rates in the pro task.

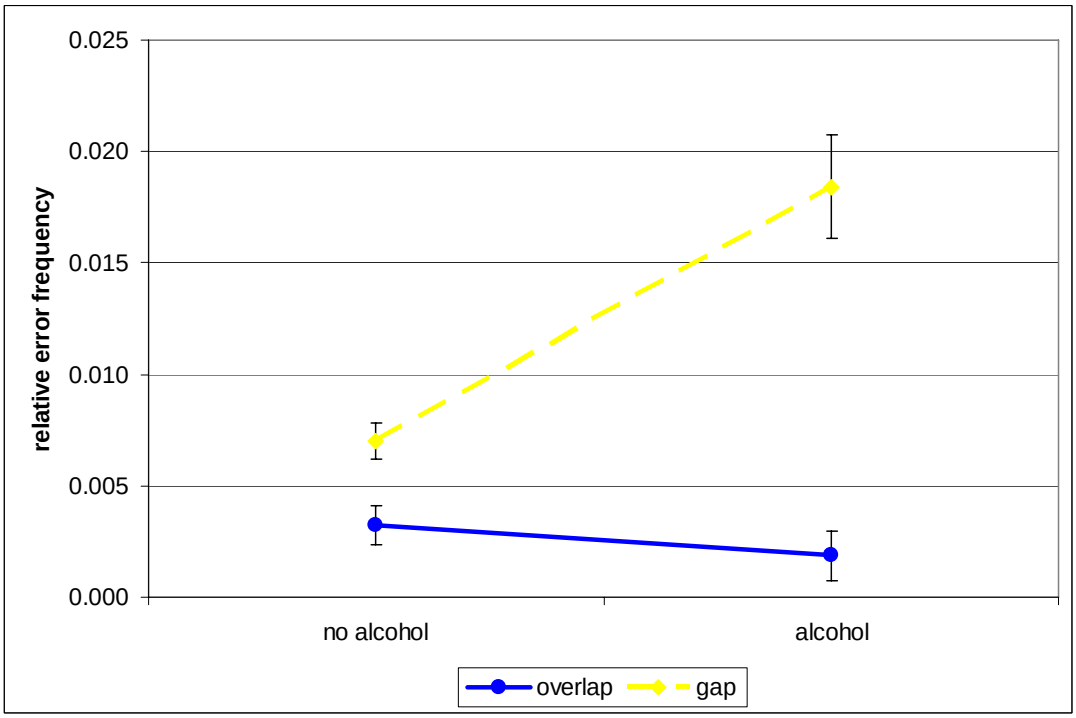

Figure 5-3. Interaction between Gap and Beverage Conditions for error rates in the pro saccade task.

For the variability of error rates, results mirror the pattern described above with significantly more variability in the gap compared to the overlap condition ($F_{(1,18)}$= 28.68, p<.001) and an interaction between Gap and Beverage Condition ($F_{(1,18)}$= 9.87, p<.05), but no alcohol related effects (see A-1.2 for complete ANOVA and means tables).

5.4.2 Saccade Latencies

Primary saccade latencies were analyzed for trials with correct responses only. As for error rates, there was no influence of the between subject factor Alcohol Session on primary saccade latencies ($F_{(1,18)}$= 0.27, p>.10). Again, there was significant effect for the factor Gap Condition ($F_{(1,18)}$= 136.42, p<.001), with latencies being about 48 ms shorter in the gap compared to the overlap condition. Latency are longer under alcohol (9 ms), a small, but statistically significant effect ($F_{(1,18)}$= 7.93, p<.05). No interactions related to saccade latencies showed significant results (all ps>.10; see 0 for complete ANOVA and means tables).

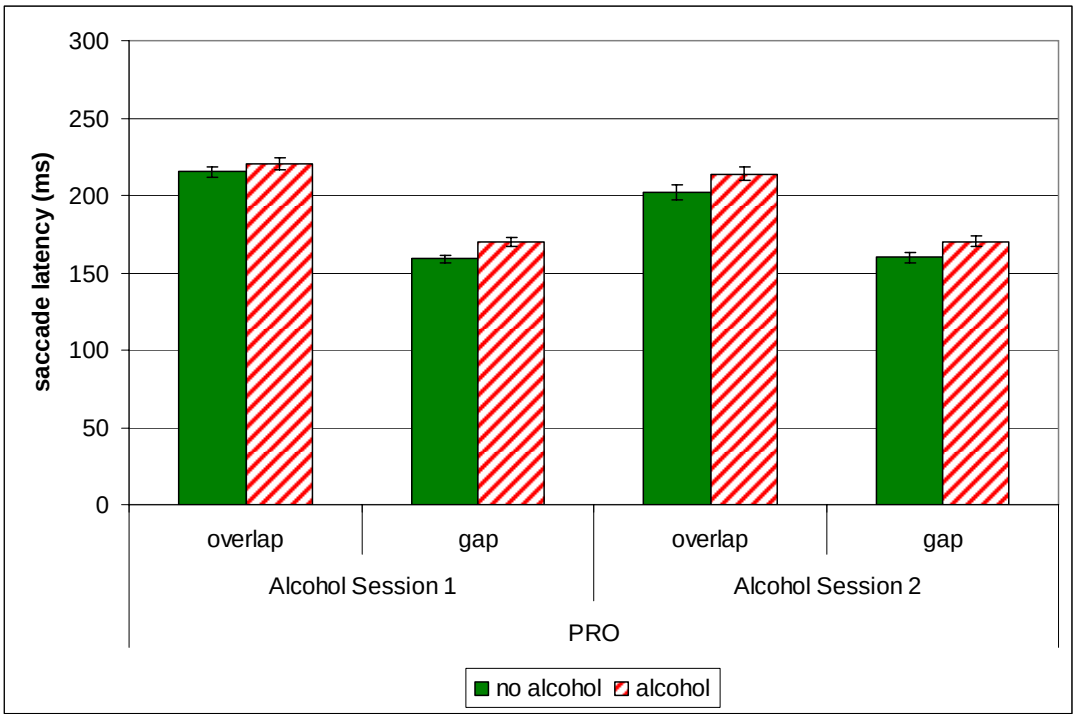

Figure 5-4. Effects of Alcohol, Gap Condition, Beverage Condition, and Alcohol Session on primary saccade latencies for trials with correct responses in the pro task.

Latencies were more variable in the gap compared to the overlap condition $F_{(1,18)}$= 15.94, p<.05) but no other significant main effects or interactions were found (all ps>.10; see A-1.4 for complete ANOVA and means tables).

5.4.3 Saccade Amplitudes

As done for latencies, only trials with correct responses were analyzed for primary saccade amplitudes. Analyses showed shorter amplitudes (5.67° vs. 5.74°) in the gap compared with the overlap condition ($F_{(1,18)}$= 6.44, p<.05). Primary amplitudes were also shorter under alcohol (5.65° vs. 5.75°) compared with the no alcohol control condition ($F_{(1,23)}$= 9.02, p<.05). Figure 5-5 depicts these results.

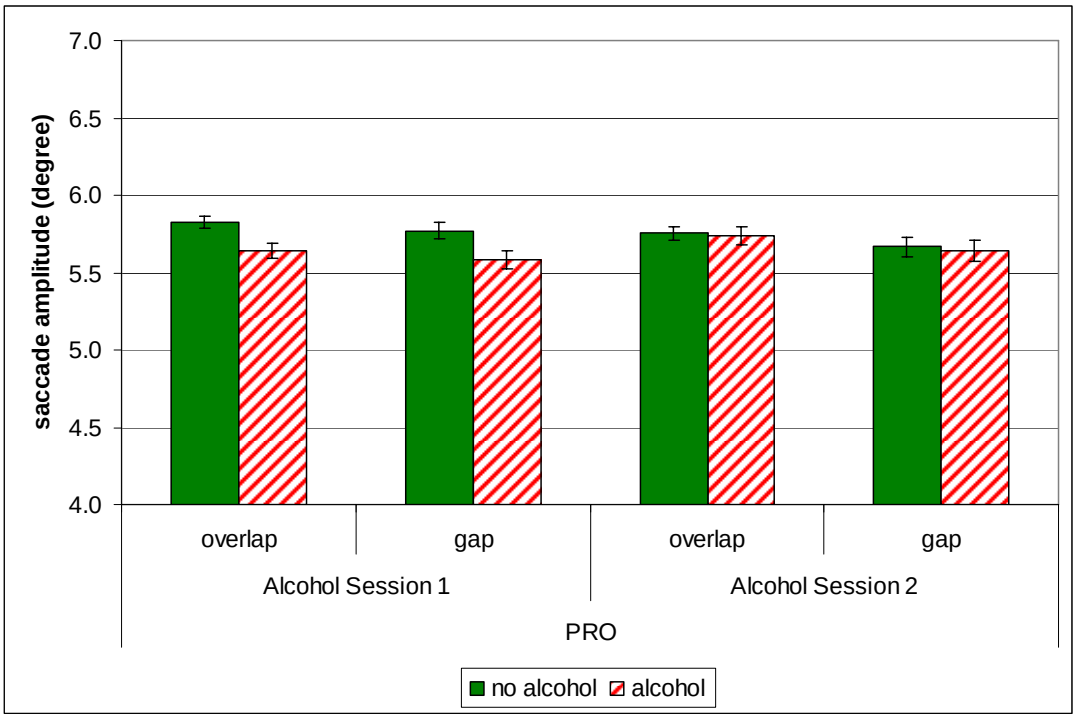

Figure 5-5. Effects of Alcohol, Gap Condition, Beverage Condition, and Alcohol Session on primary saccade amplitudes for trials with correct responses in the pro task.

In addition to these main effects, a significant interaction between Beverage Condition and Alcohol Session was found ($F_{(1,18)}$= 5.88, p<.05). As illustrated in Figure 5-6, primary saccade amplitudes differ between Beverage Conditions only, if alcohol was administered in the first session ($F_{(1,9)}$= 14.18, p<.05); in contrast, this was not true if Alcohol Session was 2 ($F_{(1,9)}$= 0.64, p>.10). No other interaction showed significant results (all ps>.10; see A-1.5 for complete ANOVA and means tables).

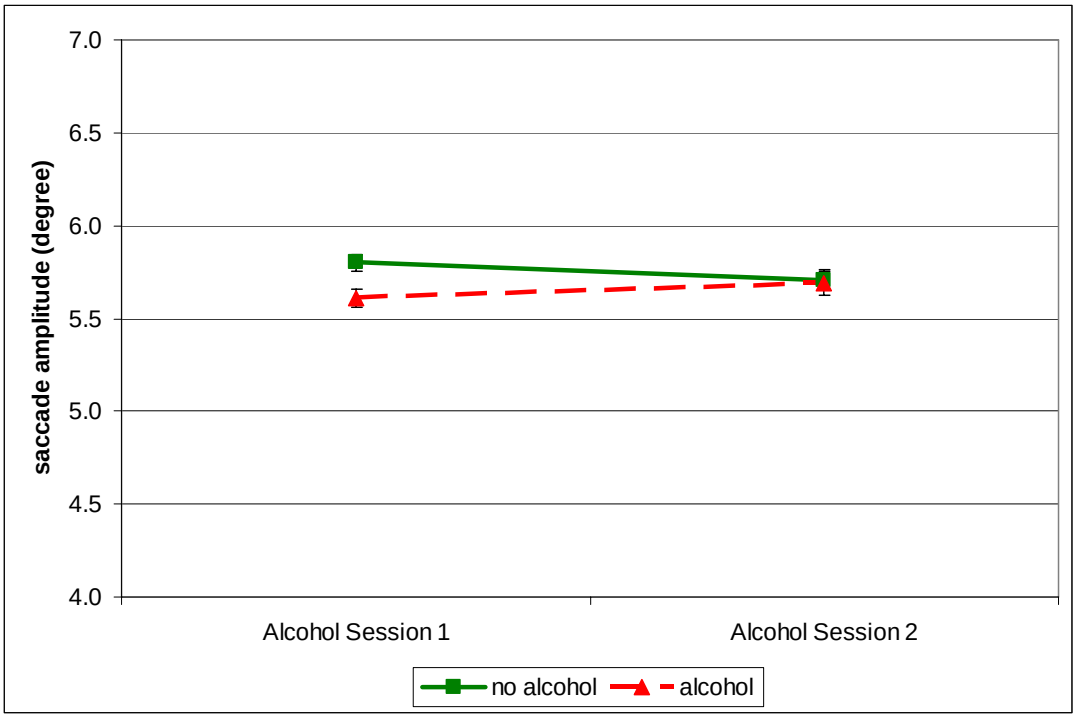

Figure 5-6. Interaction between Beverage Condition and Alcohol Session for primary saccade amplitudes in the pro saccade task.

Concerning the variability of saccade amplitudes, the only significant result was an interaction between Beverage Condition and Alcohol Session. ($F_{(1,18)}$= 4.93, p<.05). For Alcohol Session 1, amplitudes were more variable under alcohol, whereas for Alcohol Session 2, variability was higher in the no alcohol condition. This can be explained with simple training effects, as for Alcohol Session 2, the more variable no alcohol condition was conducted in the first session (see A-1.6 for complete ANOVA and means tables).

5.4.4 Peak Velocities

In trials with correct responses, peak velocities were faster in the gap compared to the overlap condition ($F_{(1,18)}$= 7.09, p<.05), as well as in the no alcohol compared to the alcohol condition ($F_{(1,18)}$= 40.64, p<.001). Figure 5-7 depicts these findings. There were no effects of Alcohol Session on peak velocities, nor reached any interactions significance (all ps>.10; see A-1.7 for complete ANOVA and means tables).Regarding the variability of peak velocities, also no significant effects were found (all ps>.10; see A-1.8 for complete ANOVA and means tables).

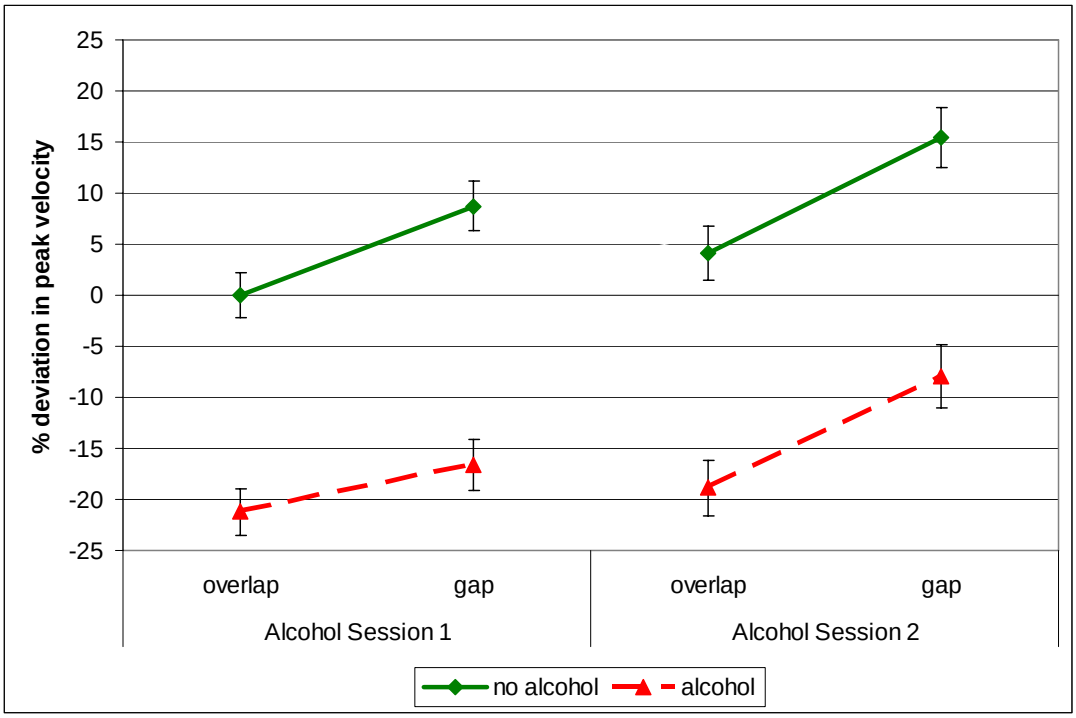

Figure 5-7. Effects of Alcohol, Gap Condition, Beverage Condition, and Alcohol Session on peak velocities for trials with correct responses in the pro task. The figure shows the percentage of deviations, with the no alcohol overlap condition in alcohol session one set to zero.

5.5 Discussion

Within the theoretical framework of this thesis, the pro saccade paradigm was used to examine effects of acute alcohol intoxication on the automatic or reflexive level of oculomotor control. Results from the pro saccade task indicated that processing on the reflexive level was altered by alcohol in terms of a general slowing of saccade preparation and execution, which was apparent in the prolonged saccade latencies and decreased peak velocities in the alcohol condition. This was a replication of findings in earlier studies that used variations of the pro saccade task to study effects of alcohol intoxication (e.g., Baloh et al., 1979; Lehtinen et al., 1979; Jantti et al., 1983; Gale et al., 1996; Moser et al., 1998; Wegner & Fahle, 1999; Blekher et al., 2002; Vassallo & Abel, 2002).

Error rates were generally very low (>1%) and overall not influenced by alcohol intoxication. However, the expected gap induced modulation was found with more errors in the gap condition. This effect was even more pronounced in the alcohol condition.

Taken together with the results on saccade latencies, which were longer under alcohol (9 ms) and in the overlap (48 ms) condition, this pattern of results suggested that functioning on the

automatic level of oculomotor control was only marginally affected by a moderate dose of alcohol. Even though saccade latencies were longer under alcohol, indicating a general reduction of cortical activity under alcohol intoxication, the gap effect was completely maintained (49 ms under no alcohol vs. 47 ms under alcohol), showing that functioning of the underlying brain structures PPC and SC remained intact under alcohol (see section 5.1.1). These findings were in line with earlier work by Wegner and Fahle (1999), who also found a preserved gap effect under alcohol. However, they reported an interaction between Beverage Condition and Gap Condition with a larger gap effect on latencies for intoxicated participants. The absence of this interaction in the present study is probably related to the slightly different experimental design. Wegner & Fahle used 22 possible target locations in 8 possible directions. The high number of targets led to a very small gap effect (~8%) for latencies in the no alcohol condition and a normal sized effect in the alcohol condition (~18%). Apparently, in the no alcohol condition, the high number of possible responses (or reduced predictability) led to a more cautious behavior, whereas under alcohol this more careful behavior was not exerted. The current study used the standard paradigm with 2 possible target locations and directions. This created a relatively high predictability (50%) and gap effects emerged in the usual range for both the no alcohol (~24%) and the alcohol condition (~22%). The interaction in the Wegner and Fahle study can therefore be interpreted as related to the predictability in the no alcohol condition.

The results on saccade accuracy in earlier studies were mixed, with some finding no effect of alcohol (Baloh et al., 1979; Lehtinen et al., 1979; Jaentti et al., 1983) and some newer work reporting decreased accuracy (e.g., Gale et al., 1996; Blekher et al., 1997). In the present experiment, saccade amplitudes under alcohol were decreased. However, this effect was caused by differences between alcohol conditions when alcohol was administered in the first session. Participants that received alcohol in the second session did not show shorter saccade amplitudes under alcohol. In combination with the results on the variability of saccade amplitudes (which were higher in the first session independently of alcohol intoxication), these findings suggest that saccade amplitudes were not generally affected by alcohol intoxication. Rather, the observed pattern could be explained in terms of training effects, with alcohol only affecting saccade amplitude, if the task is relatively new or unfamiliar.

Overall, the results of the pro saccade task demonstrated that the impact of alcohol on the automatic level of oculomotor control was relatively small. The gap effect was completely maintained and error rates were hardly affected under alcohol. A small decrease in saccade amplitudes could be attributed to training effects. Deficits were found in the temporal aspects of visually guided saccades due to alcohol intoxication, with longer latencies and slower peak velocities. Paradigms that target higher levels of oculomotor control will help to determine,

whether these temporal impairments were caused solely by an impaired motor system, or if other early stages of visual information processing were also involved.

6 Automated processing: The Double Step Paradigm

In this chapter, effects of acute alcohol intoxication on the next higher level of oculomotor control, namely the *automated level*, will be examined. Functioning at this level includes the application of learned visuomotor routines that are modified as a function of stimulus and task demands. The ability to cancel or modify saccades during the planning phase and reprogram an eye movement to a new target is a critical component of functioning at the automated level. Indeed, this is the level at which the majority of oculomotor control in natural settings takes place, with typical examples being saccade patterns (scan paths) in visual search and reading situations (cf. chapter 3.1). A basic oculomotor paradigm reflecting performance on this processing level is the double step paradigm (Becker & Jürgens, 1979). The next section gives a theoretical introduction into the double step paradigm and explains dependant variables in detail, before hypotheses and results regarding the influence of acute alcohol intoxication on the automated processing level are discussed.

6.1 Theoretical Background

In the double step paradigm, a trial starts with a fixation point in the center of a display. After a certain delay, a first visual target is presented in the periphery to the left or right of the central fixation point. In most trials, this first peripheral target is replaced with a second visual target at a different location after a variable inter stimulus interval (ISI). This visual setup gives the impression that the visual target in the periphery jumps form one position to another. Different variants can be distinguished, depending on the position of the second peripheral target. The second target can move into the same direction – in relation to the central fixation point – as the first target, creating a *stair case* condition. The second target can also be presented at a position between the first peripheral target and the initial central fixation point (*pulse undershoo*t condition), at the position of the central fixation point (*symmetrical pulse* condition) or at a position on the other side of the initial fixation target (*pulse overshoot* condition). In any case, participants are instructed to follow the peripheral target as quickly and accurately as possible.

In the double step conditions, different eye movement behaviors can occur. The most intuitive findings being that each of the two peripheral targets might receive an accurate fixation or that the first fixation might land directly on the second target. Another possible result pattern is a first fixation to an intermediate position followed by a second saccade landing accurately on the second target.

The double step paradigm is a well established task in basic oculomotor research. Experiments using the double step paradigm from the 1950s to the 1970s demonstrated that fixation durations between two saccades were shortened, if the ISI between peripheral targets was reduced (e.g. Westheimer, 1954; Levy-Schoen & Blanc-Garin, 1974). More interestingly, saccade amplitudes were longer with shorter inter-stimulus intervals, pointing to some mechanism of adaptation. However, these relations between saccade parameters and ISI did not prove that saccades can be programmed in parallel, because of the possibility that sequences of saccades could be preprogrammed (Levy-Schoen & Blanc-Garin, 1974).

Only when Becker and Jürgens (1979) introduced *reprogramming time* as a critical dependent variable, the double step paradigm could be used to examine parallel programming of saccades. The reprogramming time is defined as the time between the commencement of the first saccade and the onset of the second saccade target. This interval therefore represents a combination of the inter stimulus interval paired with the individual reaction time of a participant in a given trial. If this time interval is long, an opportunity exists to cancel or modify the imminent saccade to the initial target and replace it with a one step response to the final position. Short reprogramming times on the other hand result in a higher number of two step responses, as the new information does not have enough time to influence saccade parameters. Behavioral data confirmed these assumptions. Interestingly, in an intermediate range of reprogramming times Becker and Jürgens (1979) found that initial saccades were of intermediate amplitude. For medium reprogramming times between 80 and 150 ms a linear increase of saccade lengths was found. In contrast, shorter reprogramming times resulted in two step responses to both targets and longer reprogramming times in one step responses to the second fixation target. This relationship creates the aptly named ***amplitude transition function***, which, by plotting the average saccade amplitude for different reprogramming times, results in an ogive-shaped curve. This pattern combines previous findings on saccade durations and amplitudes. It also mirrors the temporal and spatial integration of visual information (see Becker, 1989, for a detailed discussion).

Taken together, the double step paradigm provides a tool to study performance related to adaptive programming of sequential eye movements, a core ingredient of effective visuomotor behavior (see section 3.1). Until now, there have been no studies examining the influence of acute alcohol intoxication on performance in this task. Thus the use of the double step paradigm will provide new insights on the effects of alcohol on the automated level of oculomotor control and also advance the mapping of the effects of acute alcohol intoxication.

Before stating the hypotheses in the next section, Table 6-1 gives an overview of the dependent variables (see also Figure 6-1). In addition to differentiating between single step and double step trials, it is also necessary to distinguish between single step and double step responses in double step trials.

Table 6-1. Dependent variables used in the double step paradigm.

single step trials	Amplitude	primary saccade amplitude in single step trials	
	Latency	primary saccade latency in single step trials	
		Analysis 1	Analysis 2
double step trials	% one step responses	by ISI	by reprogramming time
	Amplitude	by ISI	by reprogramming time
	Latency	by ISI	-
	Fixation Duration between Steps	by ISI	-

6.2 Hypotheses

The hypotheses for this paradigm were largely of an exploratory nature. Even though this paradigm has been well studied in the basic oculomotor research domain, there have been no prior studies examining the effects of alcohol on performance in this task.

It was expected that saccade latencies would be prolonged not only in single step trials, but also in double step trials. For double step trials, this should lead to an increase in the available reprogramming time (see Figure 6-1).

An interesting question was, whether this additional available reprogramming time under alcohol could be used within the spatial processing path. If this were the case, a higher percentage of one step responses under alcohol intoxication would be expected. If the additional time could not be utilized, the percentage of one step response should not differ between alcohol conditions. However, if alcohol intoxication impairs abilities related to spatial recoding of saccade targets, the percentage of one step responses should be lower under alcohol, largely independent of the available reprogramming time.

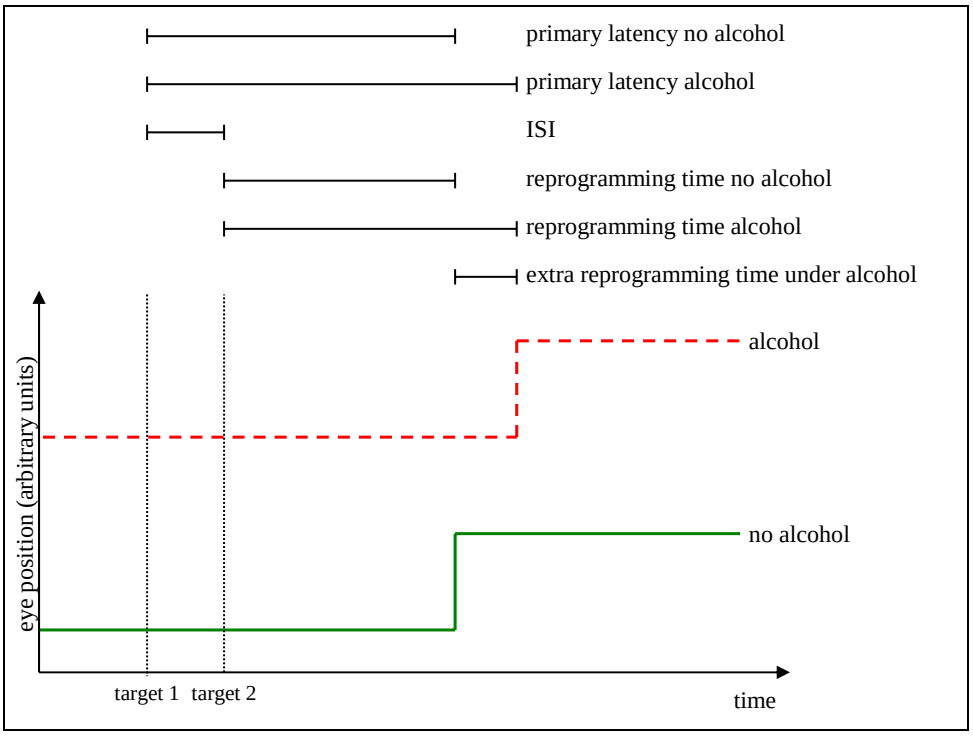

Figure 6-1. Relation between ISI, primary latencies, and reprogramming times. The figure uses arbitrary units to illustrate the expected effect of alcohol.

Analyzing the data using the available reprogramming time as independent measure effectively eliminates the additional time 'provided' via alcohol intoxication. In this analysis, the effects of alcohol for identical reprogramming intervals could be determined. It was expected to find effects of alcohol on the percentage of one step responses (i.e. fewer one step responses when reprogramming time is identical between alcohol conditions). In other words, it was predicted that acute alcohol intoxication would result in significant deficiencies in making adaptive cancellations and reprogramming of saccades on the basis of new visual information.

Performance in the one step condition was assumed to be affected by acute alcohol intoxication mostly in terms of prolonged latencies and lesser with regard to saccade amplitudes.

6.3 Materials and Method

Information regarding participants, alcohol administration, eye movement recording, and procedure were already provided in Chapter 4. The following sections will outline the experimental design in more detail and explain data analysis strategies.

6.3.1 Design

A trial starts with the presentation of a light grey fixation cross with a 0.5° diameter on an otherwise black screen. 1,000 ms after fixation cross onset, a light grey circle (0.3°) is presented as peripheral target at 3° eccentricity to the left or right of the fixation cross. In 75% of the trials, this first peripheral target is replaced with a second target at 6° eccentricity. The timing of the appearance of the second target was varied quasi-randomly, using ISIs of 40, 70 and 100 ms. These ISIs were chosen, because they had proven to produce reliable effects in similar studies (Kresser, 1996; Huestegge, 2006). The second target remained visible for 1,000 ms. After a pause of 500 ms (blank screen) the next trial started. Participants were instructed to follow the targets with their gaze as quickly and accurately as possible.

There were a total of 240 trials divided into 5 blocks. Between blocks, the number of trials was balanced for ISI for double step trials. The order of left- and rightward presentation was randomized within blocks. The completion of each block took about 120sec, resulting in a total duration of about 12min for the experiment, including calibration of the eye tracking system at the beginning of each block.

6.3.2 Data Analysis

Cut off criteria regarding latencies and amplitudes were the same as in Experiment 1 (see section 5.3.2). These restrictions resulted in 93% valid primary saccades across all trials, corresponding to a total of 8,956 primary responses. Data were analyzed separately for trials requiring a single step response and those requiring a double step response. There was no difference in the number of valid responses for trials that required one step responses (2,238 valid primary saccades, 93%) and two step responses (6,718 valid primary saccades, 93%). For all analyses, single step responses were defined as saccades with primary amplitudes of at least 1.5° and secondary saccade amplitudes of less than 0.5°. If the primary saccade was followed by a saccade larger than 0.5°, this was constituted a double step response.

For trials in which a single step response was correct, a 2 x 2 repeated measures ANOVA was used, with the within subject factor Beverage Condition (no alcohol vs. alcohol) and the between subject factor Alcohol Session.

Trials that should entail a double step response were analyzed in two different ways. A first 3 x 2 x 2 repeated measures ANOVA used the within subject factors ISI (40 vs.70 vs.100 ms) and Beverage Condition (no alcohol vs. alcohol) and the between subject factor Alcohol Session. This analysis was used to establish basic results regarding saccade parameters and

their modulation by ISIs in the two alcohol conditions. The factor Alcohol Session did not yield any significant effects and was dropped from analyses.

A second 5 x 2 repeated ANOVA with the within subject factors RepTime (reprogramming time, <80 vs. 80-120 vs. 120-160 vs. 160-200 vs. >200 ms) and Beverage Condition (no alcohol vs. alcohol) was performed in addition to test the hypotheses regarding the effects of alcohol intoxication on the ability to flexibly adjust saccade programming due to new visual information.

6.4 Results

Results for the dependent variables are presented separately for different analyses. First, results are reported for those trials that required single step responses, next double step trials are analyzed by ISI and last by Reprogramming Time (see section 6.3.2).

6.4.1 Analysis of Single Step Trials

Primary latencies in single step trials were longer in the alcohol compared to the no alcohol condition (see Figure 6-2, Table 6-2), with the main effect for Beverage Condition being significant ($F_{(1,18)}$= 28.84, p<.001; see A-2.1 for complete ANOVA and means tables). There were no differences in saccade latency variability ($F_{(1,18)}$= 2.42, p>.10). No effects regarding Alcohol Session showed significant effects (all ps>.10; see A-2.2 for complete ANOVA and means tables).

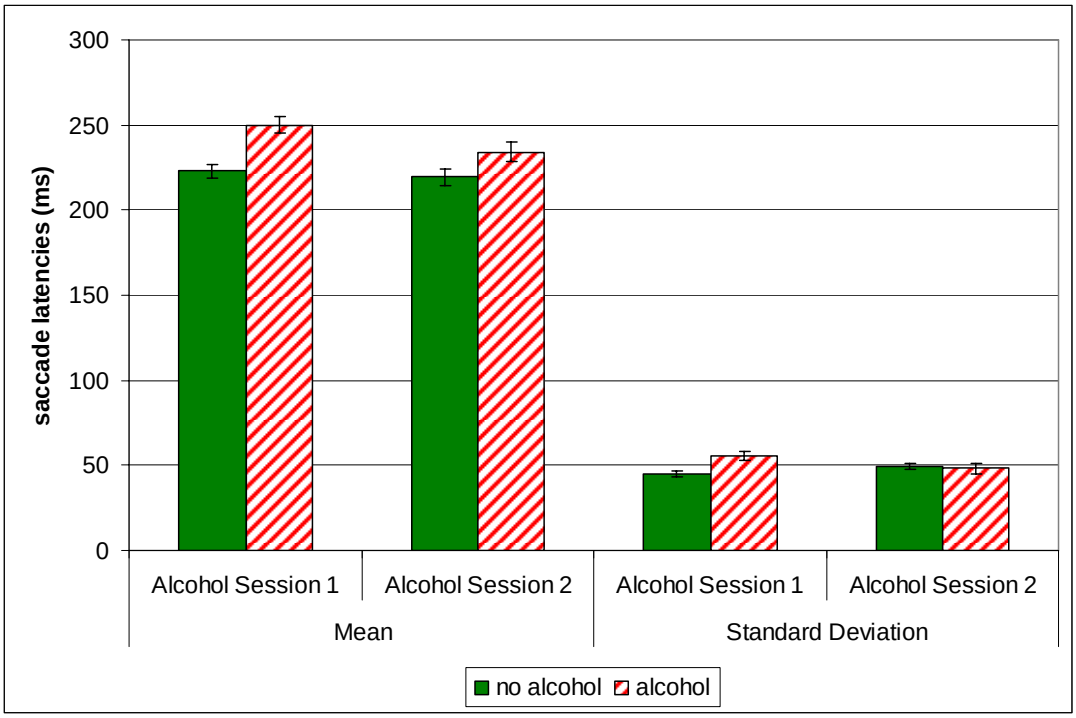

Figure 6-2. Mean primary saccade latencies and their variability for single step trials.

There were no significant differences between alcohol conditions regarding primary saccade amplitudes ($F_{(1,18)}$= 2.10, p>.10, see Table 6-2). Saccade amplitudes in the no alcohol condition as well as the alcohol condition were very accurate (2.96° vs. 2.91°; see A-2.3 for complete ANOVA and means tables). In addition, the variability of saccade amplitudes did not differ between Beverage Conditions ($F_{(1,18)}$= 1.04, p>.10) and there was no interaction with Alcohol Session ($F_{(1,18)}$= 2.68, p>.10; see A-2.4 for complete ANOVA and means tables).

Table 6-2. Means and standard error (SE) primary saccade latencies and amplitudes in single step trials. Values were based on 20 participants.

	Means (SE)			
	Alcohol Session 1		Alcohol Session 2	
	no alcohol	alcohol	no alcohol	alcohol
primary saccade latency (ms)	223 (8)	250 (9)	219 (10)	234 (11)
primary saccade amplitude (degree)	3.00 (0.07)	2.90 (0.08)	2.92 (0.09)	2.92 (0.09)

6.4.2 Analysis of Double Step Trials by ISI

The following table presents an overview of the key results for the analyses of double step trials by ISI.

Table 6-3. Summary of results for key dependent variables in the double step task, analyzed by ISI. Values were based on 20 participants.

	Means (SE)					
	ISI 40		ISI 70		ISI 100	
	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol
proportion of one step responses	0.81 (0.03)	0.79 (0.04)	0.64 (0.04)	0.67 (0.04)	0.48 (0.04)	0.51 (0.04)
primary saccade amplitude (degree)	5.40 (0.11)	5.30 (0.12)	4.93 (0.12)	5.07 (0.11)	4.49 (0.13)	4.58 (0.14)
primary saccade latency (ms)	234 (6)	251 (6)	230 (6)	255 (7)	231 (6)	259 (6)
between step saccade latency (ms)	129 (5)	151 (8)	126 (5)	146 (7)	129 (6)	144 (6)

The percentage of one step responses decreased with increasing ISI. Statistically, this was reflected in a significant main effect for ISI ($F_{(2,18)}$= 37.87, p<.001). The main effect Beverage Condition was not significant ($F_{(1,19)}$= 0.37, p>.10), and the ISI X Beverage Condition interaction showed only marginal significance ($F_{(2,18)}$= 2.85, p=.084). This effect was caused by the 40 ms inter stimulus interval, for which the percentage of one step responses was smaller under alcohol, whereas it was higher in the other ISI conditions (see Figure 6-3.; see A-3.1 for complete ANOVA and means tables). It should be noted, that the percentage of one step responses was high for all ISIs (80%, 65% and 49% for 40 ms, 70 ms and 100 ms ISI) in comparison with other studies.

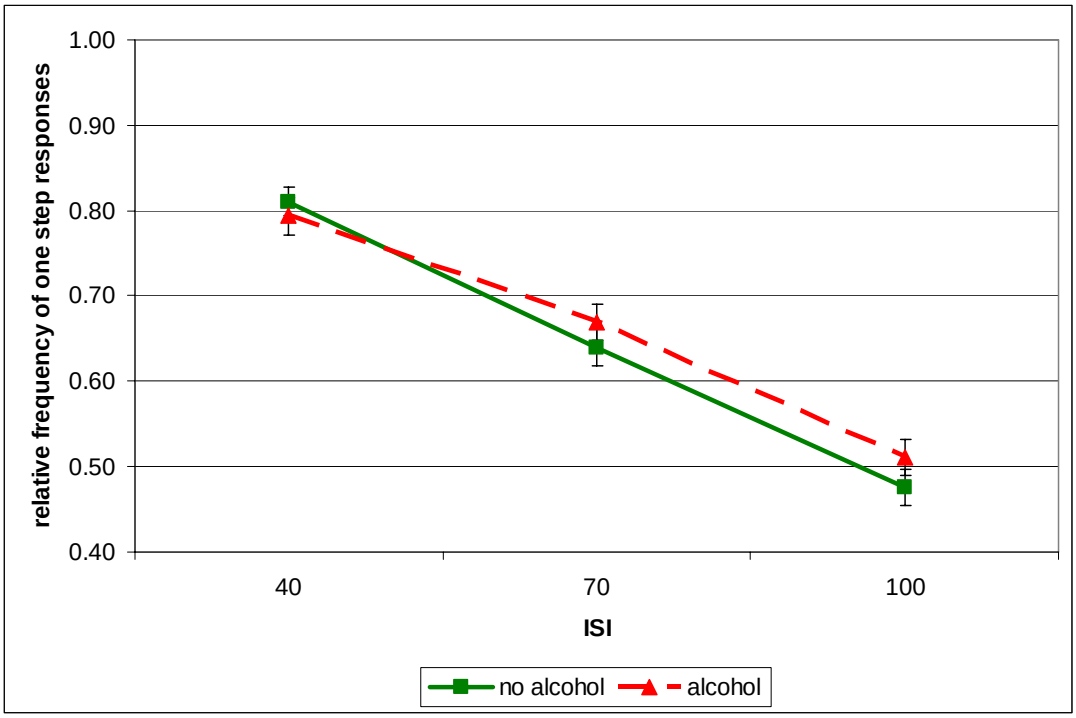

Figure 6-3. Relative frequencies of one step responses for different ISIs and Beverage Conditions.

Results for the primary saccade amplitudes exactly mirrored those from the percentage of one step responses. There was a significant decrease in saccade amplitude with increasing inter stimulus interval ($F_{(2,18)}$= 40.80, p<.001, see Figure 6-4.). The main effect for Beverage Condition was not significant ($F_{(1,19)}$= 0.35, p>.10), whereas the ISI X Beverage Condition interaction reaches significance ($F_{(2,18)}$= 6.92, p<.05). Again, this effect was caused by the 40 ms ISI, for which the difference between Beverage Conditions was significantly different from the 70 ms ($F_{(1,19)}$= 12.94, p<.05) and the 100 ms ISI ($F_{(1,19)}$= 6.39, p<.05; see A-3.2 for complete ANOVA and means tables).

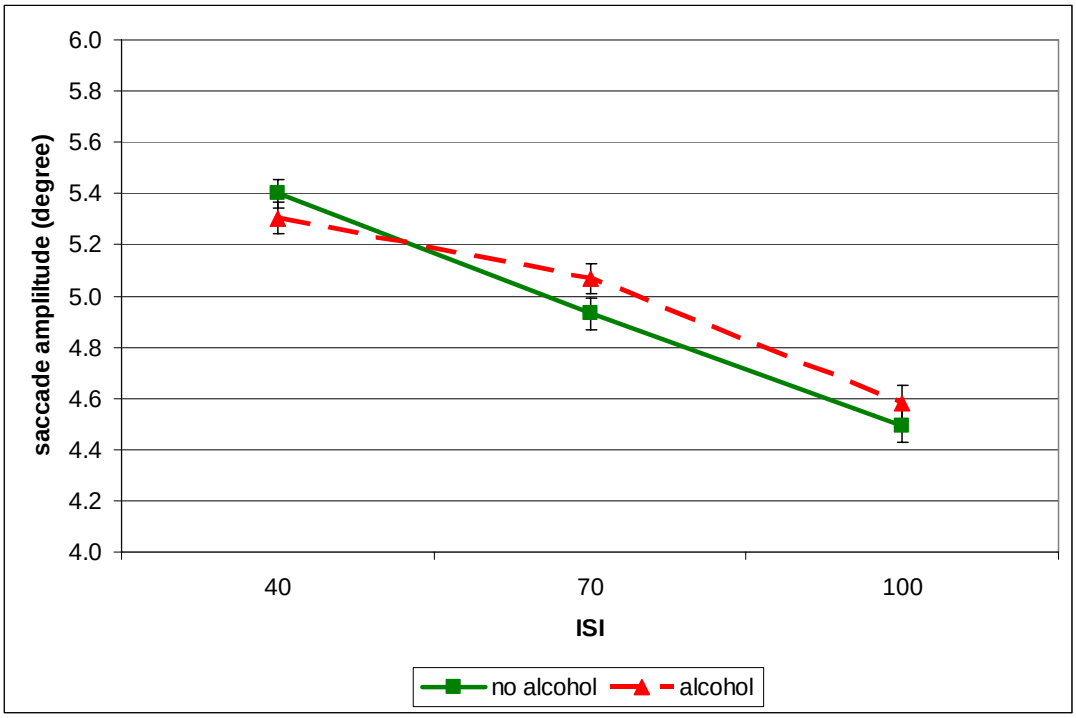

Figure 6-4. Mean primary saccade amplitudes for different ISIs and Beverage Conditions.

Primary latencies were longer under alcohol compared to the no alcohol condition (see Figure 6-5). However, the main effect of Beverage Condition ($F_{(1,19)}$= 41.62, p<.001) was qualified by a significant ISI X Beverage Condition interaction ($F_{(2,18)}$= 14.88, p<.001). This interaction was caused by the fact that the difference between Beverage Conditions was smaller in the 40 ms ISI compared to the 70 ms ($F_{(1,19)}$= 9.25, p<.05) and the 100 ms ISI ($F_{(1,19)}$= 27.06, p<.001; see A-3.3 for complete ANOVA and means tables).

Regarding the latencies of saccades for the second step, there was also a significant main effect for Beverage Condition ($F_{(1,19)}$= 14.51, p<.05), with longer latencies under alcohol (see Figure 6-5; see A-3.4 for complete ANOVA and means tables).

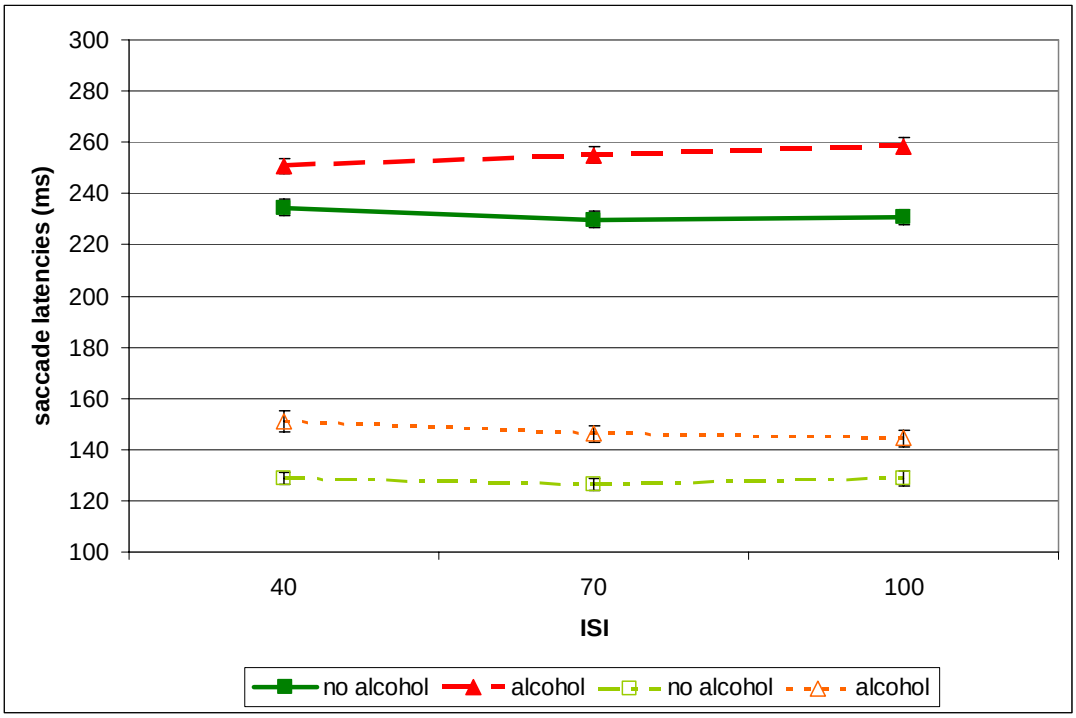

Figure 6-5. Mean primary saccade latencies (filled markers) and between step latencies (unfilled markers) for different ISIs and Beverage Conditions.

6.4.3 Analysis of Double Step Trials by Reprogramming Time

Before examining the results of alcohol intoxication on double step trials the following graphs illustrate the basic effects of reprogramming time on the two key parameters proportion of one step responses and primary amplitude. Data points were averaged across conditions and subjects and include between 63 and 2138 observations. Figure 6-6 illustrates that the absolute minimal time necessary to reprogram a saccade was around 80-90 ms, with substantial increases only starting around 100 ms of available reprogramming time. Saccade amplitudes however, were modified with as little as 60ms reprogramming time (Figure 6-7).

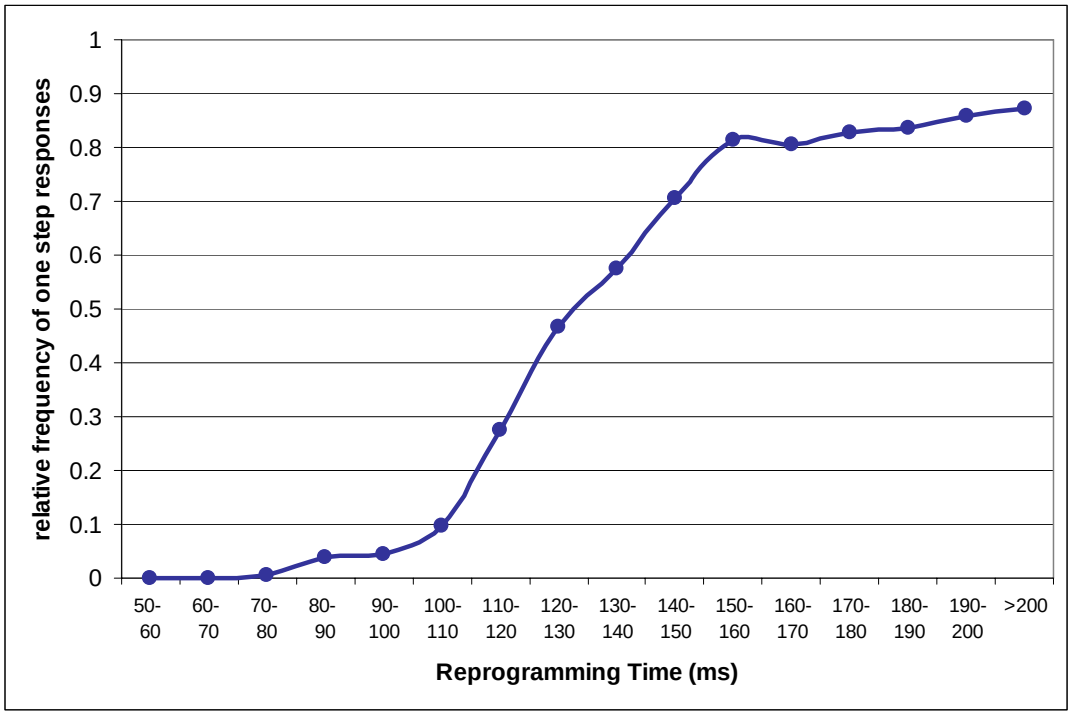

Figure 6-6. Proportion of one step responses in relation to reprogramming time. Data are averaged across Beverage Condition (see text for details).

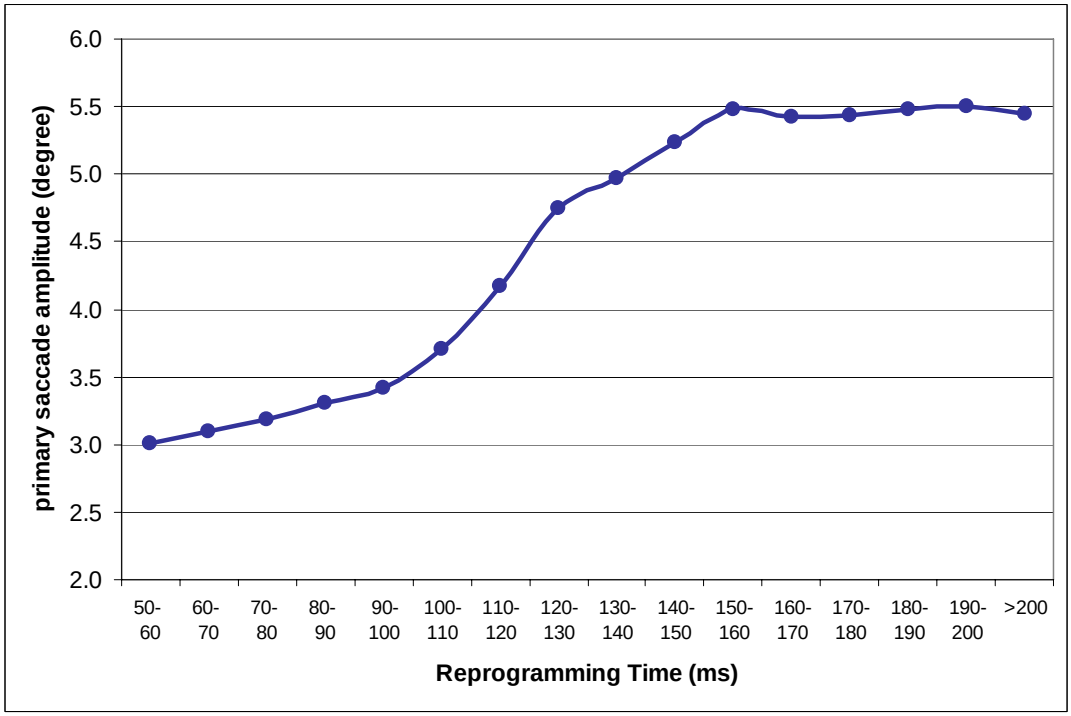

Figure 6-7. Primary saccade amplitudes in relation to reprogramming time. Data are averaged across Beverage Condition (see text for details).

For the analyses regarding alcohol effects on reprogramming time, the data for five reprocessing time intervals were averaged per subject. The mean values for each subject were then entered in the ANOVA (see 6.3.2). Two datasets had to be discarded, because they did not provide cases for every reprogramming interval.

The percentage of one step responses (see Figure 6-8) revealed significant main effects for the factors RepTime ($F_{(4,14)}$= 143.76, p<.001) and Beverage Condition ($F_{(1,17)}$= 20441, p<.001), as well as a significant interaction between the two factors ($F_{(4,14)}$= 5.84, p<.05). The percentage of one step responses was higher with longer available reprogramming times, but smaller in the alcohol compared with the no alcohol condition. This interaction was caused by the shortest reprogramming time interval, which was the only interval with no difference between Beverage Conditions ($F_{(1,17)}$= 0.41, p>.10; see A-4.1 for complete ANOVA and means tables).

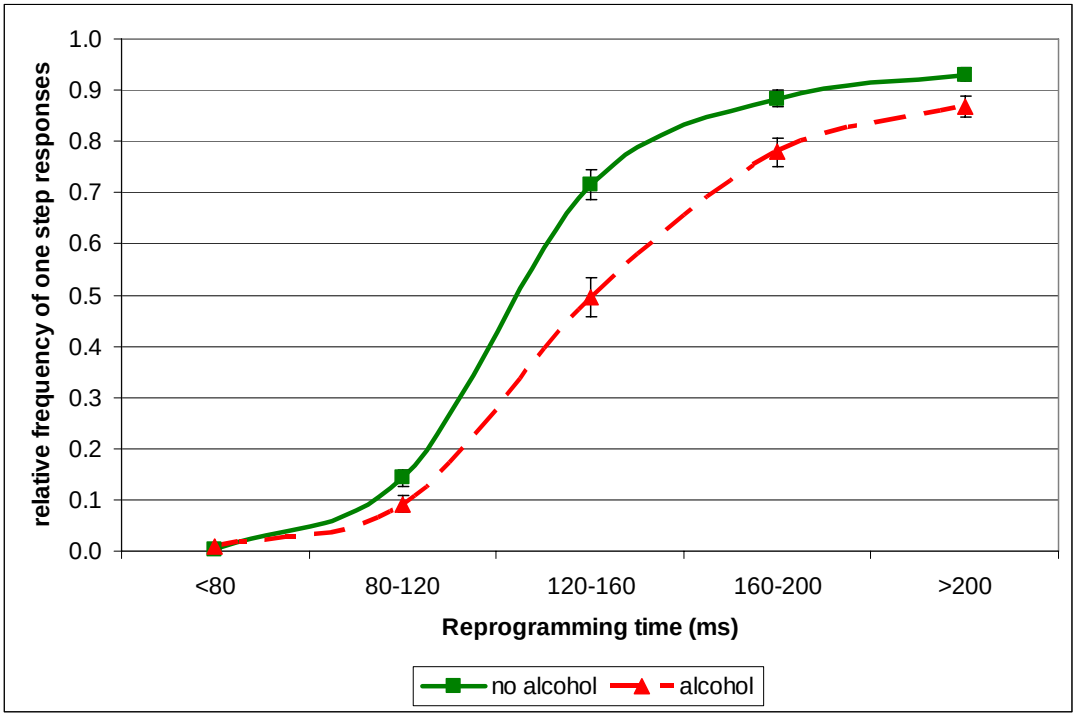

Figure 6-8. Relative frequencies of one step responses in double step trials for different reprogramming time intervals and Beverage Conditions.

Looking at the amplitude transition function (see Figure 6-9) the expected relation between reprogramming time and saccade amplitude was found. Amplitudes were significantly longer with more reprogramming time ($F_{(4,14)}$= 292.92, p<.001). In addition, saccades in the alcohol condition were significantly shorter than those in the no alcohol condition ($F_{(1,17)}$= 11.37, p<.05). Furthermore, the RepTime X Beverage Condition interaction was also significant

($F_{(4,14)}$= 5.60, p<.05). This interaction was created by the fact that differences between Beverage Conditions were only significantly different for the two medium reprogramming time intervals (80-120: $F_{(1,17)}$= 14.21, p<.05; 120-160: $F_{(1,17)}$= 16.97, p<.05), whereas for shorter and longer reprogramming times no differences between alcohol and no alcohol conditions developed (all ps>.10; see A-4.2 for complete ANOVA and means tables).

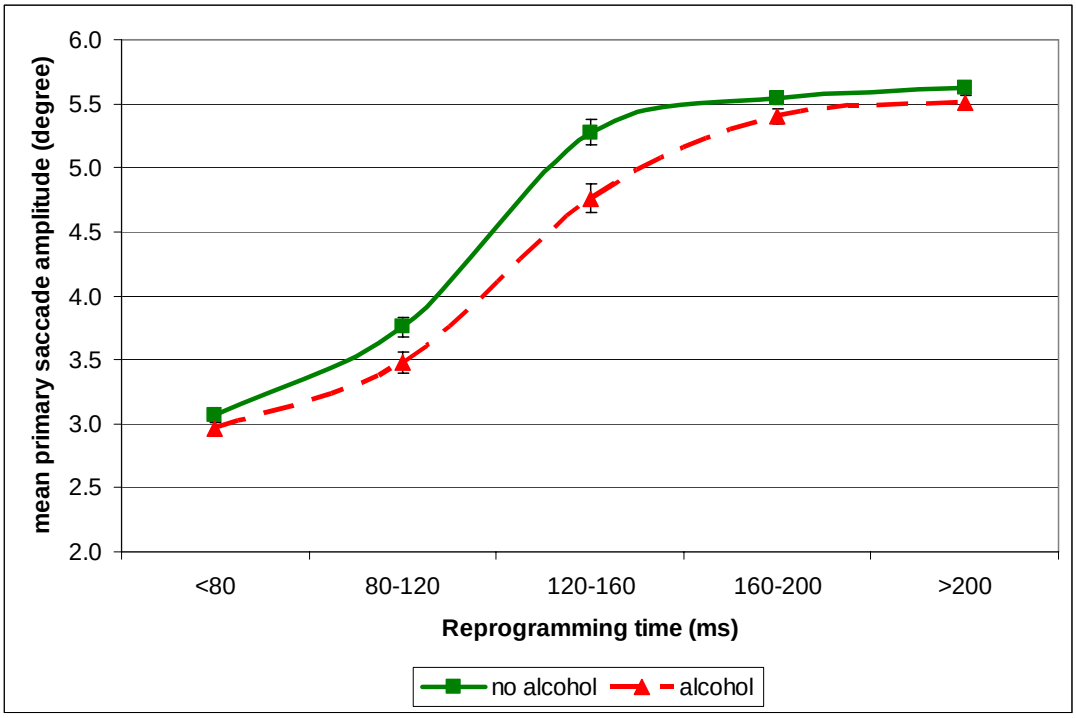

Figure 6-9. Saccade amplitudes for primary responses in double step trials for different reprogramming time intervals and Beverage Conditions.

6.5 Discussion

The double step paradigm was introduced to study performance on the automated level of oculomotor control. However, trials that required single step responses are also reflecting functioning on the automatic level and are comparable to the visually guided pro saccade paradigm. Results for these single step trials showed longer latencies under alcohol thereby replicating findings from the pro saccade paradigm. Saccade amplitudes did not differ significantly between alcohol conditions in single step trials, but showed shorter amplitudes under alcohol, again mirroring the finding from experiment 1. The pattern held even for the effect of Alcohol Session with saccade amplitudes showing a tendency to be shorter under alcohol only, if alcohol was administered in the first session. Overall, the data from the single step trials corroborate findings from the pro saccade task.

Turning to the results for double step trials, classical findings were replicated in the present study. The proportions of single step responses as well as the amplitudes of primary saccades decreased with increasing ISIs. Interestingly, even though significant interactions emerged between ISI and Beverage Conditions, at no point there were significant differences between no alcohol and alcohol condition. This was true for the proportions of single step responses and for saccade amplitudes. However, alcohol intoxication did influence saccade latencies. Not only were primary latencies longer in the alcohol condition, but between-step latencies were also prolonged under alcohol, demonstrating a general slowing effect of alcohol. Taken together, this pattern of results showed, that the additional time provided by prolonged latencies could not be utilized by the saccade programming system to decrease the number of two step responses. On the other hand, as the proportions of one step responses did not differ between alcohol conditions, underlying processes regarding the reprogramming of saccades did not seem to be impaired by alcohol intoxication.

Alcohol induced impairments became apparent however, when looking at the proportions of single step responses and saccade amplitudes for different reprogramming time intervals. Reliable differences between alcohol conditions were found for reprogramming times larger than 80ms. For both alcohol conditions the proportions of one step responses showed the typical pattern with more one step responses under longer reprogramming times, but the curve for the alcohol condition was shifted to the right. Clearly, under alcohol more reprogramming time was needed to achieve the same proportion of one step responses. The same effect was observed with respect the adjustment of saccade amplitudes, with longer reprogramming times leading to increased saccade amplitudes. Under alcohol the same effect occurred, however it was less pronounced in medium reprogramming time intervals. This is a sensible finding, because for very short reprogramming times saccades usually land on the first target and with longer reprogramming times (>160ms) the second target is reached. Only in the medium range of reprogramming times alcohol showed an effect of decreasing saccade amplitudes.

Interestingly, the necessary reprogramming time to modulate saccade amplitude and to modify the target location (i.e., program a one instead of a two step response) was different. The amplitude transition function (Figure 6-7) showed that amplitudes were modulated with reprogramming times as short as 60ms. With reprogramming times in the order of 150ms a plateau was reached. These times set a relatively tight frame regarding the timing, cancellation or reprogramming and the extraction of information during saccades and are well in line with earlier work (e.g. Deubel et al., 2000). The minimal reprogramming time necessary to perform a one instead of a two step response was approximately 100 ms (Figure

6-6), which was considerably longer than that necessary to influence saccade amplitude. This finding suggests that saccade amplitude could be modulated independent of the decision to make a one or two step response. Regarding the effects of alcohol intoxication, both processes seem to be influenced (see above). However, only for the proportions of one step responses the effect of alcohol showed even for long reprogramming times (>200ms), indicating that additional time did not help to compensate for the influence of alcohol. For the modulation of saccade amplitudes on the other hand, an impairment was only apparent for a medium range of reprogramming times (80-160ms).

Overall, the results from the double step paradigm showed that alcohol intoxication led to specific deficits in making adaptive cancellations and reprogramming of saccades on the basis of new visual information.

7 Voluntary processing: Standard and Visually Guided[11] Anti Saccades

Within the context of a longstanding acknowledgment of alcohol's impact on general cognitive processing, relevant recent research has focused more specifically on how the substance impacts voluntary control and executive functioning (see Fillmore, 2003 for a recent review). This emphasis emanates from the appreciation of the fact that a basic feature of adaptive human functioning that appears to be compromised by alcohol intoxication is the ability to exhibit flexibility in response to dynamic environmental demands. In particular, a critical component of adaptive capacity that alcohol might impair is the ability to suppress reflexive impulses and to execute voluntarily controlled actions when effective performance in a situation calls for it. One oculomotor paradigm that is well suited to examine this very process, is the anti saccade paradigm. Within the framework of this thesis, the anti saccade paradigm is related to the *voluntary processing level*. After providing the specific theoretical background for anti saccade paradigms, later sections describe the materials and methods used, followed by the presentation and discussion of results..

7.1 Theoretical Background

In this section the theoretical background for the traditional anti saccade paradigm is introduced, before previous studies, that examined the effect of alcohol intoxication on performance in this task, are reviewed. Finally, the theoretical background for a variation of the traditional[12] paradigm, the visually guided anti saccade paradigm is provided.

7.1.1 The Standard Anti Saccade Paradigm

The anti saccade paradigm involves the voluntary inhibition of a reflexive response to the sudden onset of a visual target in the periphery. In addition, not only has the reflexive eye movement to be inhibited, but at the same time the execution of a saccade to the mirror position of the displayed peripheral target has to be programmed and performed (cf. Hallet,

[11] In the present work the term "visually guided anti saccade" refers to the fact that a visual marker is present at the target location. Other work has sometimes used the same term to refer to anti saccades that are being made away from a visual target, in contrast to away from a memorized target (e.g. Van Gelder, Lebedev & Tsui, 1997).

[12] The terms "standard" and "traditional" are used interchangeably in this thesis to refer to the classic anti saccade paradigm with no stimulation at the correct saccade target location.

1978; Everling & Fischer, 1998; Massen, 2004). Since the introduction of this task, considerable knowledge has been accumulated regarding the physiological underpinnings of saccade tasks (see Munoz & Everling, 2004, for a review), thereby offering an opportunity to link alcohol's influences on performance to underlying brain systems.

Peripheral targets in the pro saccade task trigger saccades automatically, whereas in the anti saccade task this reflex has to be inhibited by intentional input from a higher cortical level to cancel the reflexive movement (when pathway). In addition, a spatial transformation of the visual target information has to take place to enable a cognitive representation and parameter specification for redirection to a new saccade target. Recent analyses of anti saccade performance suggest a 'race' between two parallel saccade programs (Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004; Walker & McSorley, 2006). Within the Findlay and Walker model this can be thought of in terms of two conflicts. The first of these is located in the when pathway, determining whether the current fixation on the central fixation cross is to be maintained or not. The second is between two simultaneously activated saccade targets, one exogenously triggered by the onset appearance of a peripheral target and the other endogenously generated in voluntary processing modules to direct the saccade to the desired location.

Generally, moderate alcohol intoxication is assumed to impair deliberate and voluntary functioning to a larger extent than automatic behaviors. Unfortunately, ethanol, unlike most other psychoactive substances, is not linked to any particular receptors in specific brain areas, thus making it more difficult to determine which brain mechanisms are most likely to be vulnerable to alcohol intoxication. Indeed, studies in humans and rodents have mainly shown a general reduction in cortical activity due to ethanol (Davies & Alkana, 2001; Wang et al., 2000; Krull et al., 1994; Liu et al., 2000). However, the underlying physiological structures that are relevant to our study can be linked to different modules of the theoretical framework introduced above. For instance, the activity of the fixation related neurons in the SC is increased prior to anti-saccade trials (Everling et al., 1999) and there is evidence that this modulation in activity is mediated by frontal cortical projections from the frontal eye fields (FEF), supplementary eye fields (SEF) and the dorsolateral prefrontal cortex (DLPFC) (Guitton et al., 1985; Munoz & Everling, 2004; Schlag-Rey et al., 1997). In relation to the Findlay and Walker (1999) framework, the SC can be thought of as part of the fixate center and the frontal areas act as the substrate for top-down modulation from the voluntary level. Therefore, the anti saccade task is well suited to examine the influence of alcohol intoxication on this level of oculomotor control and to establish links to related brain areas.

7.1.2 Previous Studies on Alcohol using the Anti Saccade Paradigms

The impact of alcohol in the reflexive, pro saccade paradigm has been studied fairly extensively (see chapter 5), but studies using the anti saccade task are still relatively rare. Moreover, the results of research on pro saccade task performance have quite consistently demonstrated prolonged latencies under alcohol intoxication, without effects on saccade accuracy. In contrast, the results from the few alcohol experiments that have included an anti saccade task have been considerably more equivocal. Of the four existing studies, two found that error rates were decreased under alcohol (Khan et al., 2003; Vassallo & Abel, 2002), one found no alcohol effect (Blekher et al., 2002), and one reported that alcohol led to increased error rates (Crevits et al., 2000). Results for saccade latencies were equally unclear. Although Khan et al. and Blekher et al. found increased latencies under alcohol, no significant differences were observed in either the Vassallo and Abel or the Crevits et al. studies. Besides these inconsistencies, measures for saccade accuracy were reported in only two of these studies, with Blekher et al. finding significant overshoots under alcohol, whereas Vassallo et al. noted no differences between alcohol conditions for this parameter. The Blekher et al. study was the only one to report saccade velocity and it appeared to be decreased by alcohol. Other potentially informative parameters, such as error correction rate and response variability have not been studied at all in previous work. In addition, the relevant pioneering work published so far has rarely included *both* reflexive *and* voluntary saccades tasks in the same study, making their direct comparison impossible. Moreover, of the two studies that did use both tasks, one involved different visual setups across tasks so that different saccade amplitudes were required for pro versus anti saccades, thereby clouding interpretation of the comparability of alcohol effects on them. This rather unsatisfying state of affairs has been compounded by the fact that most of the pertinent studies have been mainly descriptive rather than theory-driven.

This thesis seeks to improve upon the methodological precision and the conceptual framework of earlier research on performance under alcohol by integrating various tasks into the conceptualization framework for oculomotor control outlined above (section 2.3; Findlay & Walker, 1999; see also Findlay & Gilchrist, 2003) to provide a firmer theoretical grounding than has been evident in prior work. Within this effort, existing equivocation potentially can be resolved and understanding of mechanisms that underlie alcohol related impairments can be advanced. In an effort to narrow down possible mechanisms that convey alcohol induced impairments further, a variation of the traditional anti saccade task was also used within this series of experiments.

7.1.3 The Visually Guided Anti Saccade Paradigm

The visually guided anti saccade paradigm differs from the traditional in only one aspect (see section 7.1.3). During the entire experiment, the possible target locations are constantly visually marked. This additional feature results in a visual target for anti saccades, thereby eliminating the need to endogenously generate the saccade target, whereas the suppression of the reflexive response, as well as the spatial recoding are still needed to perform in this task. Some studies, examining processes related to pro and anti saccade processing, used visually guided anti saccades in their experiments (Cherkasova et al., 2002; Barton et al, 2005) without broaching the issue of possible clouding of results by potentially eliminating one of the processes underlying anti saccade performance.

The only study that systematically examined differences between traditional and visually guided anti saccades was published by Edelman et al. (2006). They introduce the visually guided anti saccade task as a "novel" task that is "used to help determine why metrics of traditional anti saccades differ from those of pro saccades" (Edelman et al., 2006, p. 1412). Their first experiment aims at determining whether the additional visual target marker would influence performance in the anti saccade task, possibly leading to a closer resemblance of anti saccade performance to pro saccade performance. Their results showed that error rates in the anti saccade task is not significantly influenced by the additional target marker. However, reported error rates in the study are very low (5.2% traditional and 4.9% visually guided), as was the number of participants. In addition, of the six participants, two were authors of the paper, who were obviously very familiar and trained in the task. As with error rate no effect was found for primary saccadic latencies with 20 9ms in the traditional and 206 ms in the visually guided version of the anti saccade task.

Regarding peak velocities, Edelman et al. demonstrated that velocities in the visually guided anti saccade task resembled those in the pro saccade more than that in the traditional anti saccade task, which were about 10% slower. The authors conclude that peak velocity is more dependent on the presence of a visual target than the task that has to be performed. Two subsequent experiments examined the question why visually guided anti saccades still showed a tendency to be slower than pro saccades. The second experiment ruled out the possibility that the suppression of the reflexive response is responsible for this difference. In the third experiment the authors provide evidence that the sudden onset in the visual periphery boost velocities in the pro saccade task rather than reducing peak velocities in the anti saccade task.

The most interesting finding for the present work is related to saccade amplitudes. The presence of the visual target marker increased saccade amplitude significantly. With the target distance being 15°, anti saccades showed the typical hypometria (13.8°) in the traditional anti saccade task compared to pro saccades (14.9°). This effect was eliminated completely in the visually guided anti saccade task, where mean amplitudes were 14.8°. Other studies using the visually guided anti saccade task did not report consequences of using a visual marker at the target location on saccade amplitudes (Cherkasova et al., 2002; Barton et al, 2005). This result is of special interest in the context of the present study, as possible differences regarding the influence of alcohol intoxication on saccade amplitudes between the traditional and visually guided anti saccade task could help to pinpoint underlying or at least related mechanisms.

7.1.4 Dependent variables in the anti saccade tasks

In addition to the variables introduced for the pro saccade paradigm in section 5.1.3, latency, amplitude and velocity measures are calculated separately for *gap* and *overlap* conditions for *correct* and *erroneous* responses. A further dependent variable is the ***error correction rate*** that gives the proportion of erroneous primary responses that were immediately corrected with the secondary saccade.

7.2 Hypotheses

Given the theoretical considerations and the results of previously published studies outlined above, it was predicted that alcohol intoxication would influence saccades in several specific ways. Higher levels of processing, especially those involving inhibition, can generally be expected to be significantly impaired by alcohol intoxication (cf. Abroms et al., 2006; Fillmore, 2003). Consequently, performance on the anti saccade task was hypothesized to be impaired by alcohol. It might be noted here that this hypothesis appears to be at odds with earlier findings by Khan et al. (2003), who reported an alcohol induced *reduction* in anti saccade error rate. Given this apparent contradiction, it was speculated that a to be expected impairment of performance under alcohol based on the voluntary level of processing might materialize, not necessarily in terms of direction errors, but rather as reduced spatial accuracy of saccades in the anti-saccade task. In other words, rather than the suppression of the reflexive movement, the spatial transformation processes necessary to perform in the anti saccade task successfully might be specifically impaired by alcohol intoxication. The design of the experiment outlined below provided an opportunity to explore these possibilities. In addition to these effects on error rate and saccade amplitudes, longer latencies and slower peak velocities were expected under alcohol.

The introduction of the visually guided anti saccade paradigm allows to further investigate involved processes, especially regarding the spatial parameters. If possible impairments were related to the need to endogenously create a saccade target, these impairments should be absent in the visually guided version of the anti saccade paradigm.

As for the traditional paradigm, longer latencies were anticipated under alcohol. Following the findings of Edelman and colleagues (2006), peak velocities were expected to be faster in the visually guided version compared to the traditional anti saccade paradigm, but still slower under alcohol.

7.3 Materials and Method

Information regarding participants, alcohol administration, eye movement recording, and procedure were already provided in Chapter 4. The following sections will outline the experimental design in more detail and explain data analysis strategies.

7.3.1 Design: Traditional Anti Saccade Paradigm

An anti saccade trial starts with the presentation of a red fixation cross with a 0.5° diameter centered on an otherwise black screen. In half of the trials, a light grey circle with a diameter of 0.3° visual angle appeared after 1,000 ms at 6° either to the right or left of the central fixation cross, constituting *overlap* trials. In the other half of the trials, the central fixation point disappeared after 800 ms, leaving a blank screen for 200 ms, before the onset of the peripheral target (*gap* condition). The peripheral target stayed visible for 800 ms under both conditions before the next trial started. Participants were instructed to look to the mirror position of the peripheral target as quickly and accurately as possible after it appeared.

As described in section 5.3.1, the pro saccade paradigm was implemented together with the anti saccade paradigm (see also section 4.4.1) as the first task in a series of experiments. Eight blocks (4 pro, 4 anti) with 48 trials each were presented. The paradigm switched between pro and anti saccades every two blocks with the order of the first task balanced between participants. The completion of each block took about 100 seconds, resulting in a total duration of about 15 minutes for the experiment, including calibration of the eye tracking system at the beginning of each block.

7.3.2 Design: Visually Guided Anti Saccade Paradigm

The visually guided anti saccade task shared the identical visual layout and timing with the traditional anti saccade task (see section 7.3.1). In addition to the presentation of the central fixation cross (0.5° diameter) with the beginning of the first trial, two unfilled rectangles (0.5° x 0.5°) with light grey colored lines marked the position of possible peripheral target presentation. These position markers stayed visible throughout the entire experiment. Participants were again instructed to look to the mirror position of the peripheral target as quickly and accurately as possible after it appeared.

There were a total of 192 trials divided into 4 blocks. Within blocks, the order of overlap and gap trials was randomized. The completion of each block took about 100 seconds, resulting in a total duration of about 7.5 minutes for the experiment, including calibration of the eye tracking system at the beginning of each block.

7.3.3 Data Analysis

For both paradigms, saccades were classified online using EyeLink software. Raw data files were converted into SPSS data matrices, using custom build software. Cut off criteria regarding latencies and amplitudes were the same as in Experiment 1 (see section 5.3.2). These restrictions resulted in 87% valid primary saccades across all trials, corresponding to a total of 6,700 primary responses for the traditional and 88% valid trials or 6,771 primary responses in the visually guided anti saccade paradigm.

For initial analyses, 2 x 2 x 2 repeated measures ANOVAs were used to examine effects of the within subject factors Gap (overlap vs. gap) and Beverage Condition (no alcohol vs. alcohol) and the between subject factor Alcohol Session in both versions of the anti saccade paradigm. In addition, a 2 x 2 x 2 x 2 repeated measures ANOVA with the within subject factors Task (traditional vs. visually guided), Gap (overlap vs. gap) and Beverage Condition (no alcohol vs. alcohol) and the between subject factor Alcohol Session was used to analyze differences between the two versions of the anti saccade paradigm.

7.4 Results

Results are presented by dependent variables. Within the variables, findings are described for the traditional and visually guided paradigm first, before outcomes are compared between tasks. All analyses were based on individual mean values (n = 20). Table 7-1 and Table 7-2 present overviews of results for all cells of the experimental design. Listed are the key

variables error rates, saccade latency, and amplitude for correct responses. Further tables can be found in the Appendix and are references in the respective passages.

Table 7-1. Overview of results for key parameters in the traditional anti saccade paradigm. Values represent means and standard error based on 20 participants (see text for details).

	Means (SE)							
	Alcohol Session 1				Alcohol Session 2			
	overlap		gap		overlap		gap	
	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol
proportion of errors	0.05 (0.03)	0.08 (0.03)	0.15 (0.04)	0.19 (0.05)	0.15 (0.04)	0.06 (0.03)	0.26 (0.05)	0.15 (0.06)
primary saccade latency (ms)	268 (9)	316 (10)	237 (9)	287 (9)	275 (11)	286 (13)	243 (11)	253 (11)
primary saccade amplitude (degree)	5.08 (0.25)	5.76 (0.25)	5.36 (0.24)	5.99 (0.25)	5.13 (0.3)	5.95 (0.31)	5.30 (0.29)	5.83 (0.3)

Table 7-2. Overview of results for key parameters in the visually guided anti saccade paradigm. Values represent means and standard error based on 20 participants (see text for details).

	Means (SE)							
	Alcohol Session 1				Alcohol Session 2			
	overlap		gap		overlap		gap	
	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol
proportion of errors	0.04 (0.02)	0.08 (0.03)	0.13 (0.04)	0.20 (0.05)	0.08 (0.03)	0.04 (0.04)	0.26 (0.05)	0.21 (0.06)
primary saccade latency (ms)	300 (16)	344 (15)	275 (14)	320 (16)	306 (19)	303 (18)	273 (17)	274 (19)
primary saccade amplitude (degree)	5.76 (0.09)	6.03 (0.11)	5.88 (0.07)	6.09 (0.1)	5.74 (0.1)	5.94 (0.13)	5.96 (0.09)	6.02 (0.12)

7.4.1 Error Rate

Error rates in the traditional anti saccade paradigm (Figure 7-1) were higher in the gap compared to the overlap condition ($F_{(1,18)}$= 49.82, p<.001). There were no differences between alcohol conditions ($F_{(1,18)}$= 1.80, p>.10), but a significant interaction between Beverage Condition and Alcohol Session ($F_{(1,18)}$= 9.66, p<.05). Looking at Figure 7-2, it becomes apparent that this interaction was caused by the fact that error rates were higher in the no alcohol condition, if Alcohol Session was 2, whereas in the alcohol condition, error rates were higher, if alcohol was administered in session 1. However, neither for the no

alcohol ($t_{(1,9)}$= -1.77, p>.10), nor for the alcohol condition ($t_{(1,18)}$= 0.63, p>.10) the differences between alcohol sessions were significant. The difference between Beverage Conditions was only significant if alcohol was administered in session 2, but not if alcohol was administered in the first session (see A-5.1 for complete ANOVA and means tables).

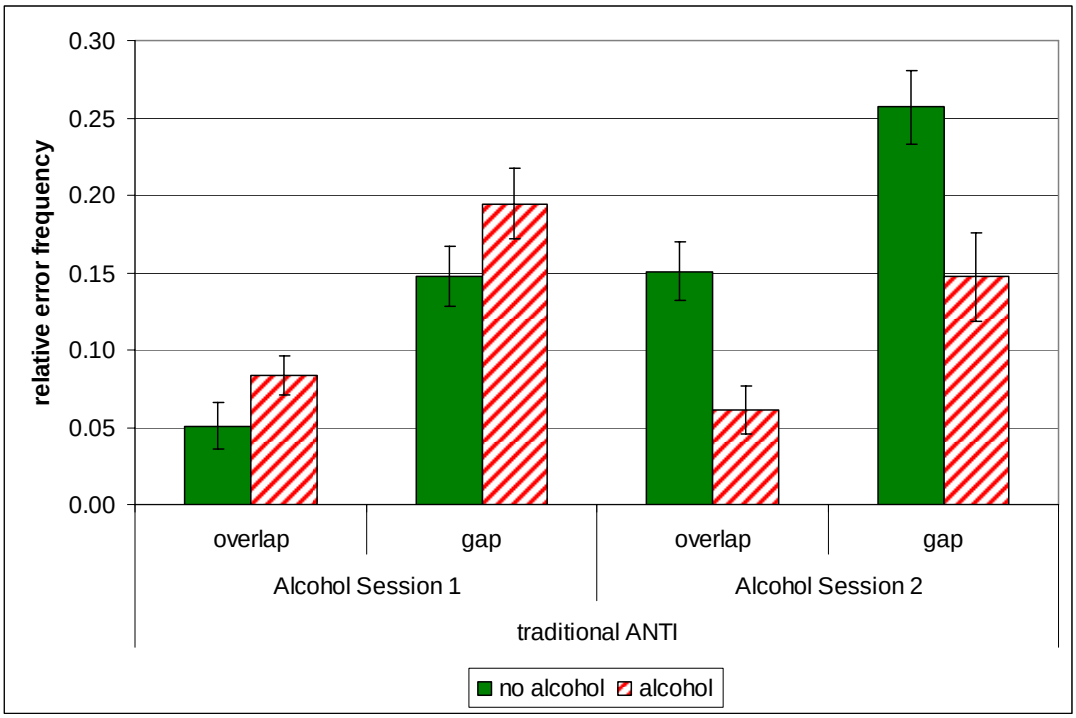

Figure 7-1. Effects of Gap Condition, Beverage Condition, and Alcohol Session on error rates in the traditional anti saccade task.

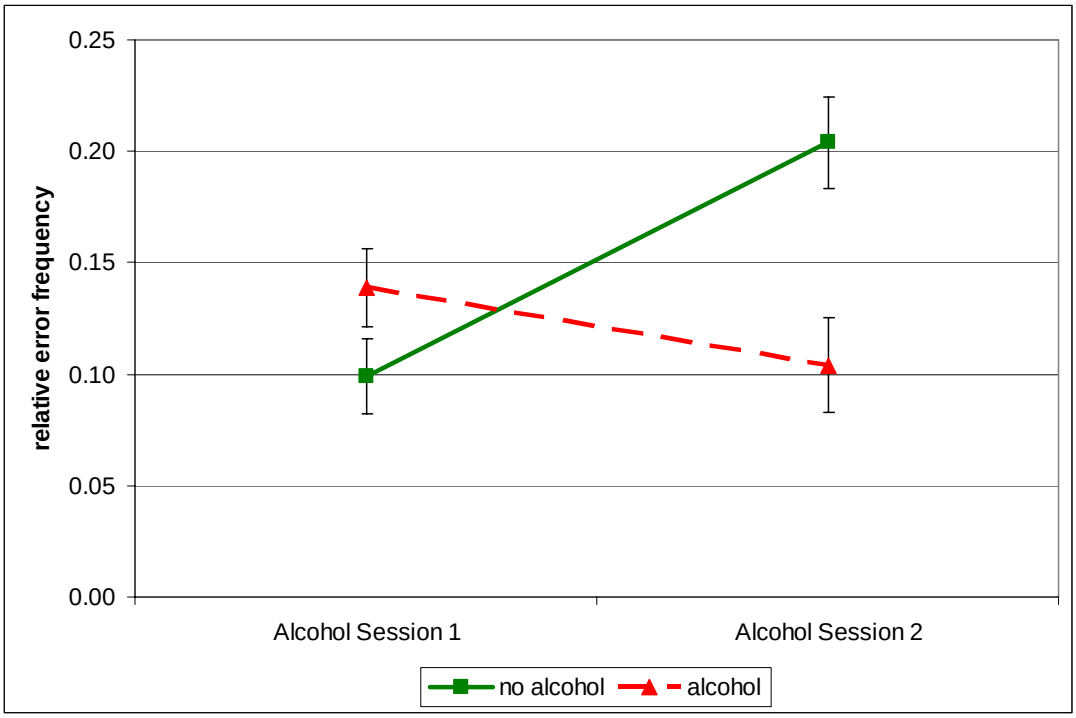

Figure 7-2. Interaction between Beverage Condition and Alcohol Session for error rates in the traditional anti saccade task.

For the visually guided anti saccade paradigm error rate results replicate findings from the traditional paradigm (Figure 7-3). Again there is a significant difference between Gap Conditions ($F_{(1,18)}$= 36.79, p<.001), but no effect of Beverage Condition ($F_{(1,18)}$= 0.01, p>.10). The Beverage Condition X Alcohol Session interaction was also significant ($F_{(1,18)}$= 7.03, p<.05), showing the same pattern as in the traditional paradigm (Figure 7-4; see A-5.2 for complete ANOVA and means tables).

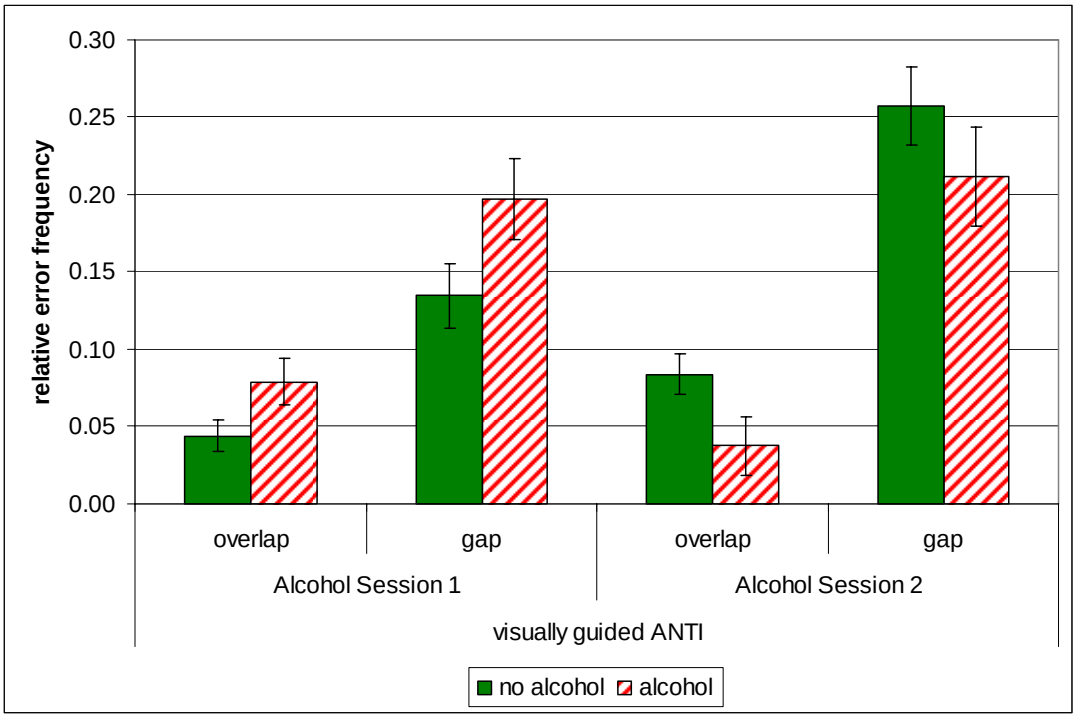

Figure 7-3. Effects of Gap Condition, Beverage Condition and Alcohol Session on error rates in the visually guided anti saccade task.

Results regarding the variability of error rates mirror those for means described above for both paradigms. There was more variability in the gap condition ($F_{(1,18)}$= 57.58, p<.001 traditional and $F_{(1,23)}$= 84.49, p<.001 visually guided), no differences between Beverage Condition (all ps>.10) and significant interactions between Beverage Condition and Alcohol Session ($F_{(1,23)}$= 6.21, p<.05 and $F_{(1,23)}$= 10.49, p<.05 respectively; see A-5.3 and A-5.4 for complete ANOVA and means tables).

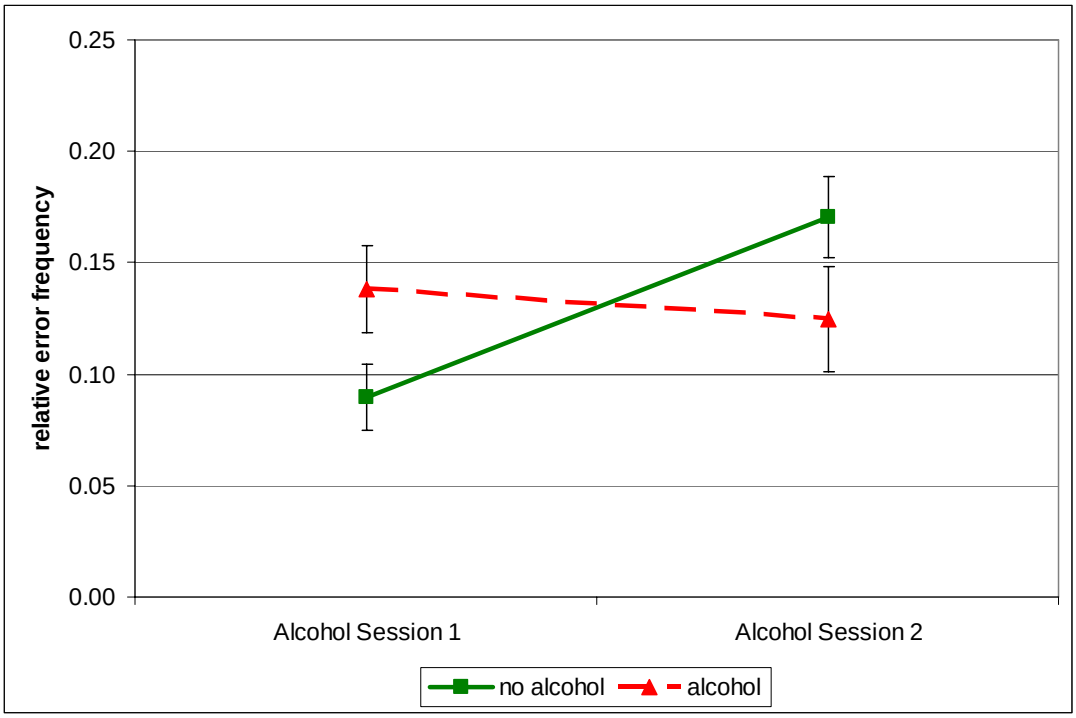

Figure 7-4. Interaction between Beverage Condition and Alcohol Session for error rates in the visually guided anti saccade task.

Comparing results on error rates from the traditional and visually guided anti saccade paradigm revealed no significant difference in error rates between the paradigms ($F_{(1,18)}$= 0.65, p>.10). In addition, even though the effect of alcohol showed a tendency to be more pronounced in the traditional compared to the visually guided anti saccade paradigm, the interaction between Task and Beverage Condition did not reach significance ($F_{(1,18)}$= 2.58, p>.10; see A-5.5 for complete ANOVA and means tables).

7.4.2 Saccade Latencies

In the traditional anti saccade paradigm, primary saccade latencies in trials with correct responses were shorter in the gap compared to the overlap condition ($F_{(1,18)}$= 93.63, p<.001). In addition, under alcohol latencies were 30 ms longer as in the no alcohol condition ($F_{(1,18)}$= 42.33, p<.001, see Figure 7-5). This main effect was qualified by a Beverage Condition X Alcohol Session interaction ($F_{(1,18)}$= 17.08, p<.05). As depicted in Figure 7-6, differences between Beverage Conditions were larger, if alcohol was administered in the first session (49 ms) compared to the second session (11 ms; see A-5.6 for complete ANOVA and means tables).

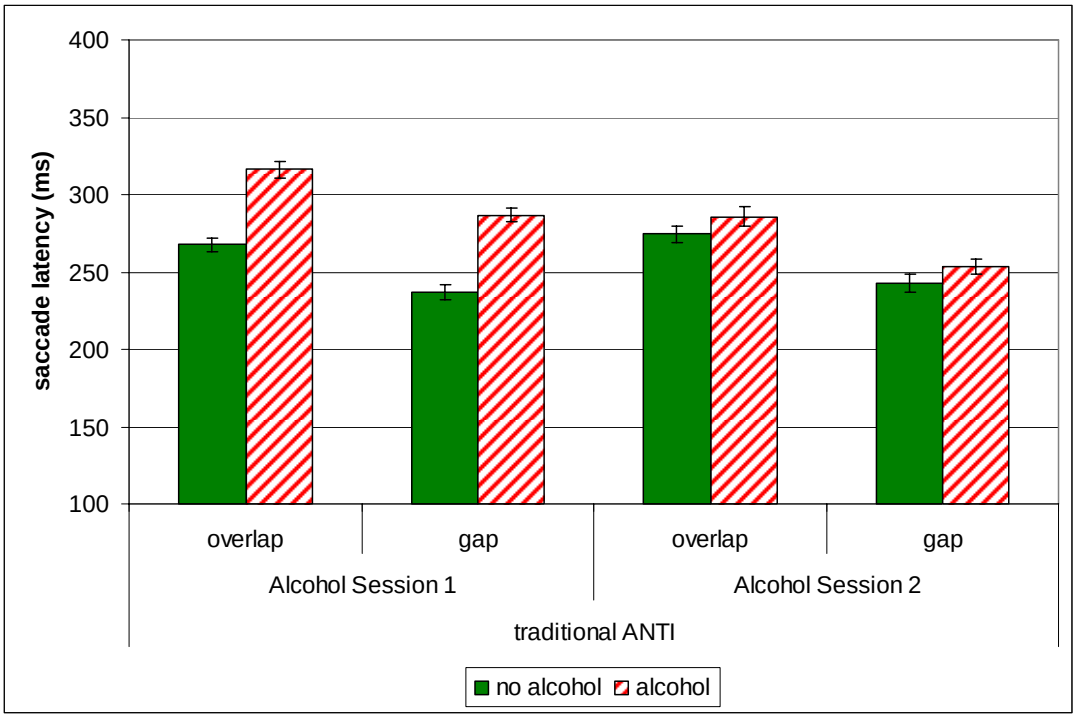

Figure 7-5. Effects of Gap Condition, Beverage Condition, and Alcohol Session on primary saccade latencies for trials with correct responses in the traditional anti saccade task.

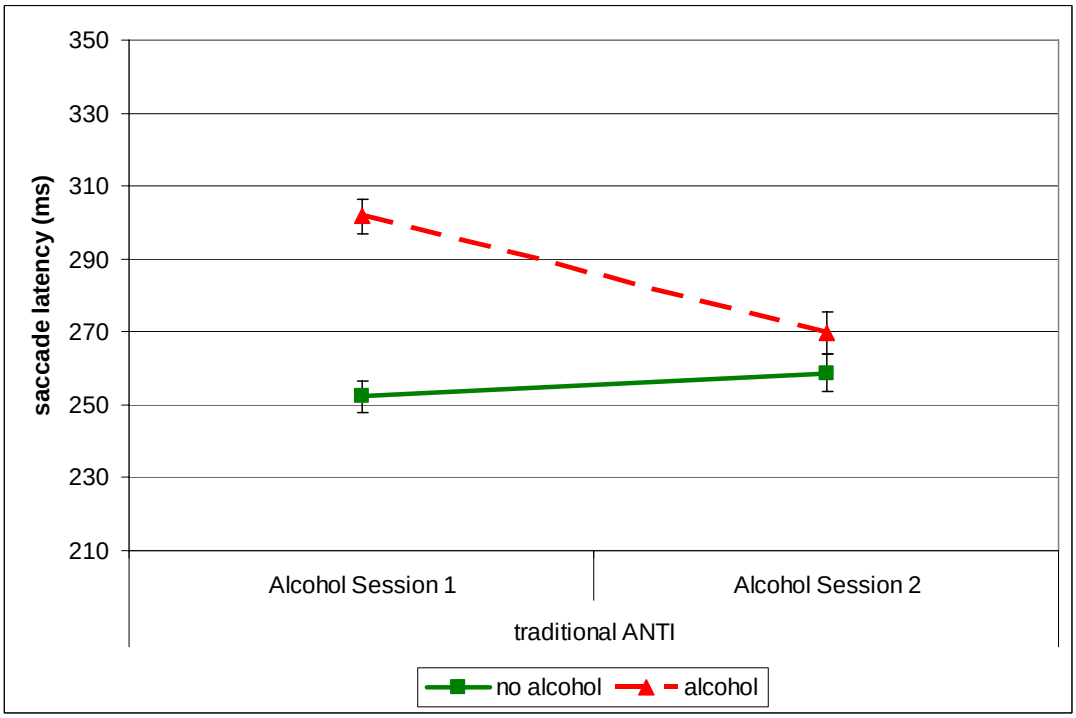

Figure 7-6. Interaction between Beverage Condition and Alcohol Session for saccade latencies in trials with correct responses in the traditional saccade task.

Again, the results of the visually guided anti saccade paradigm replicated the findings from the traditional paradigm. Saccade latencies were shorter in the gap condition ($F_{(1,18)}$= 37.52, p<.001) and longer under alcohol ($F_{(1,18)}$= 16.79, p<.05; Figure 7-7). The interaction between Beverage Condition and Alcohol Session was also significant ($F_{(1,18)}$= 19.23, p<.001), showing the same pattern for the visually guided as for the traditional anti saccade paradigm (Figure 7-8; see A-5.7 for complete ANOVA and means tables).

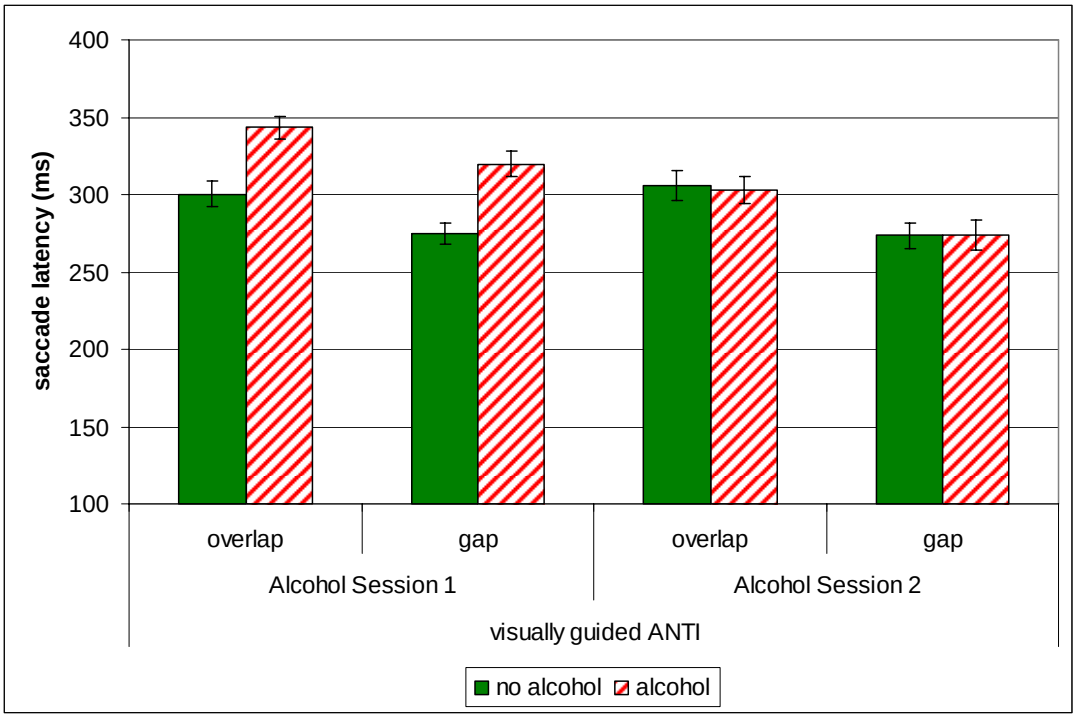

Figure 7-7. Effects of Gap Condition, Beverage Condition and Alcohol Session on primary saccade latencies for trials with correct responses in the visually guided anti saccade task.

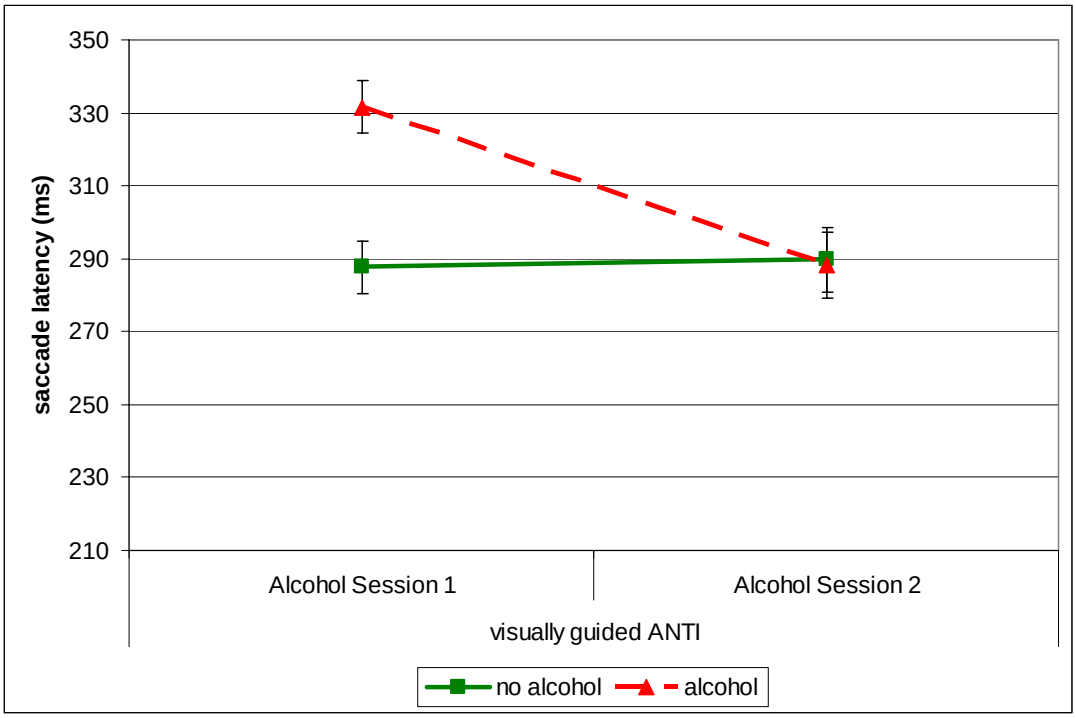

Figure 7-8. Interaction between Beverage Condition and Alcohol Session for saccade latencies in trials with correct responses in the visually guided anti saccade paradigm.

Alcohol intoxication affected the variability of saccade latencies in the traditional but not in the visually guided anti saccade paradigm ($F_{(1,18)}$= 6.91, p<.05 and $F_{(1,18)}$= 1.02, p>.10 respectively). In addition, interactions between Beverage Condition and Alcohol Session ($F_{(1,18)}$= 3.85, p=0.65 and $F_{(1,18)}$= 5.99, p<.05 respectively) showed that variability was higher for both tasks in the no alcohol conditions, if alcohol was administered in the second session, whereas variability under alcohol was higher, if Alcohol Session was 1.

A comparison of the results on primary saccade latencies confirmed that latencies in the visually guided anti saccade paradigm were longer compared to the traditional paradigm ($F_{(1,18)}$= 19.07, p<.001). However, no interactions between Task and any other variable were significant (all ps>.05; see A-5.8 for complete ANOVA and means tables).

7.4.3 Saccade Amplitudes

Saccade amplitudes in the traditional anti saccade paradigm were significantly longer under alcohol compared to the no alcohol condition (Figure 7-9; $F_{(1,18)}$= 21.54, p<.001). A Beverage Condition X Gap Condition interaction was marginally significant ($F_{(1,18)}$= 21.54, p=.084), indicating that the differences between alcohol conditions were slightly more

pronounced in the overlap condition. No other main effects or interactions are significant (all *p*s>.10; see A-5.9 for complete ANOVA and means tables).

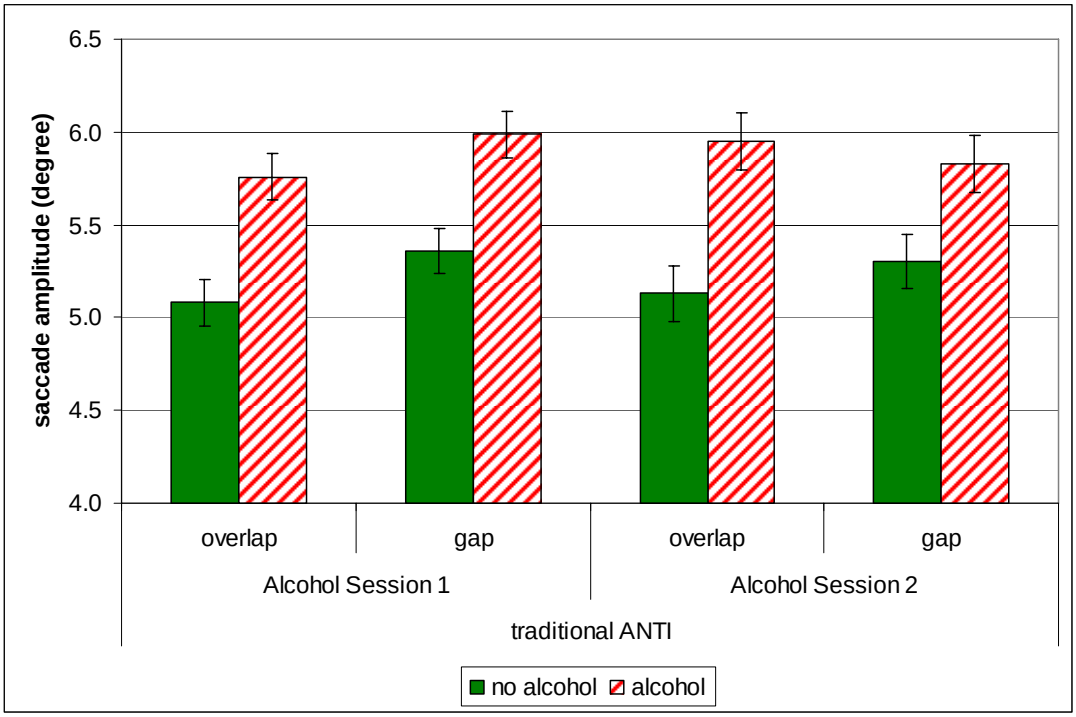

Figure 7-9. Effects of Gap Condition, Beverage Condition, and Alcohol Session on primary saccade amplitudes for trials with correct responses in the traditional anti saccade task.

In the visually guided anti saccade paradigm, saccades were also longer under alcohol ($F_{(1,18)}$=11.83, p<.05). In addition, a significant main effect for Gap Conditions indicated that saccade amplitudes were longer in the gap compared to the overlap condition ($F_{(1,18)}$= 6.81, p<.05). These main effects were qualified by a Beverage Condition X Gap Condition interaction ($F_{(1,18)}$= 9.38, p<.05; see A-5.10 for complete ANOVA and means tables).

The effect of longer amplitudes in the gap condition was more pronounced in the no alcohol condition. As a result, the difference between alcohol conditions was smaller in the gap condition compared to the overlap condition. The same pattern of results was found in the traditional task, but did not reach significance there.

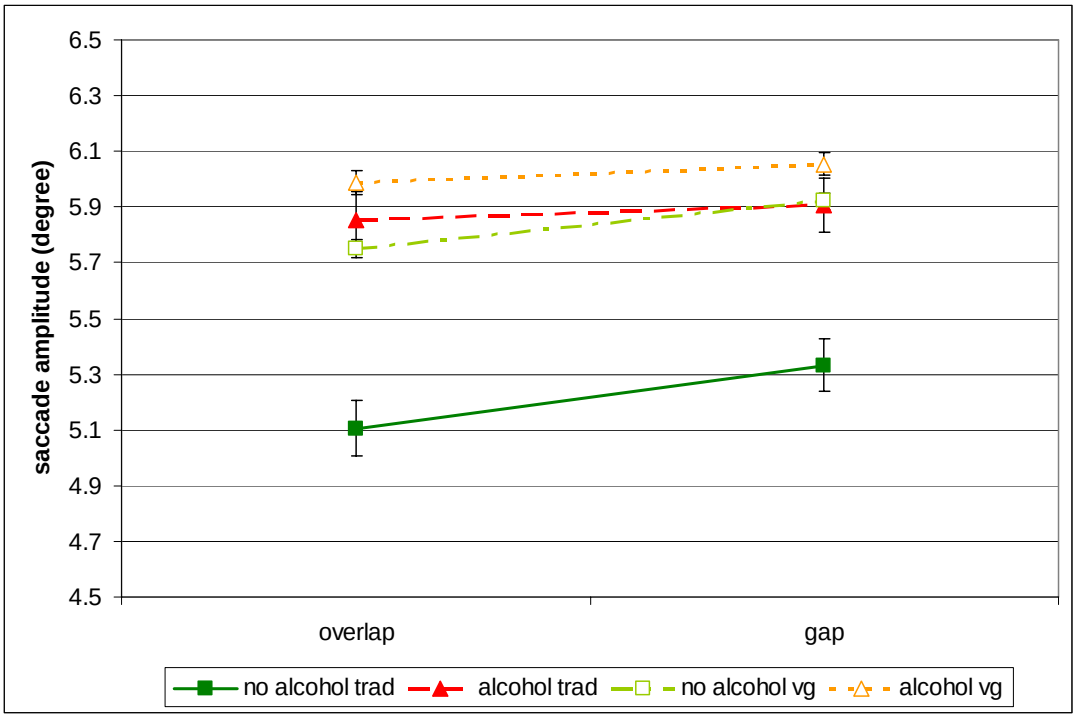

Figure 7-10. Interaction between Beverage Condition and Gap Condition for saccade amplitudes in trials with correct responses. Note that only effects for the visually guided (vg) paradigm are significant.

Regarding the variability of saccade amplitudes no significant main effects or interactions were found in either task (see A-5.11 and A-5.12 for complete ANOVA and means tables).

Analyses of the differences between the traditional and visually guided anti saccade task revealed a significant main effect for Task ($F_{(1,18)}$= 7.52, p<.05), with longer amplitudes in the visually guided paradigm. However, this effect was qualified by a Task X Beverage Condition interaction ($F_{(1,18)}$= 12.16, p<.05). Figure 7-11 illustrates that the differences between Beverage Conditions were significantly different within both the traditional ($F_{(1, 18)}$= 21.54, p<.001) and the visually guided task ($F_{(1, 18)}$= 11.86, p<.05). However, the difference between alcohol conditions was larger in the traditional paradigm. This resulted from an increase of saccade amplitude in the visually guided task under no alcohol ($F_{(1, 18)}$= 15.17, p<.05), whereas in the alcohol condition a slight increase was not significant ($F_{(1, 18)}$= 0.89, p>.10; see A-5.13 for complete ANOVA and means tables).

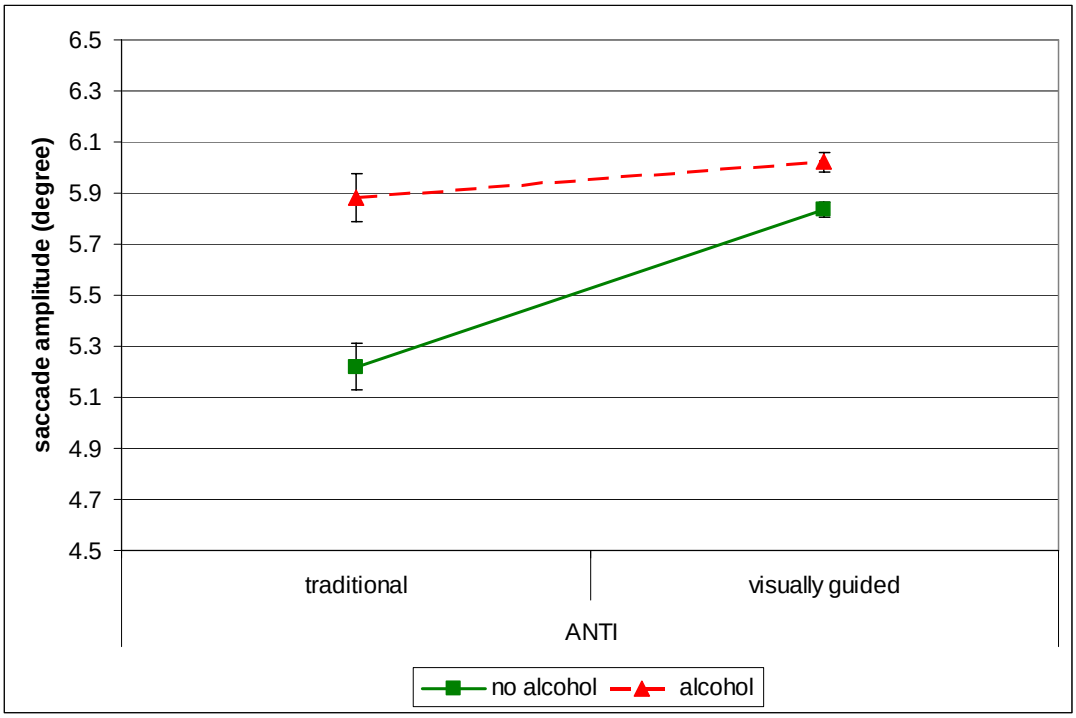

Figure 7-11. Interaction between Task and Beverage Condition for saccade amplitudes in trials with correct responses.

7.4.4 Peak Velocities

In the traditional anti saccade paradigm, peak velocities were significantly reduced under alcohol ($F_{(1,18)}$= 49.87, p<.001). The same was true for the visually guided paradigm ($F_{(1,18)}$= 26.07, p.001). No other effects were significant in both paradigm regarding (see A-5.14 and A-5.15 for complete ANOVA and means tables) and no differences were found in the variability of peak velocities (all ps>.10). When comparing the two paradigms, a significant Task X Beverage Condition interaction ($F_{(1,18)}$= 9.42, p<.05) emerged, caused by larger effects of alcohol in the traditional, compared to the visually guided anti saccade paradigm. Figure 7-12 summarizes the results on peak velocities (see A-5.16 for complete ANOVA and means tables).

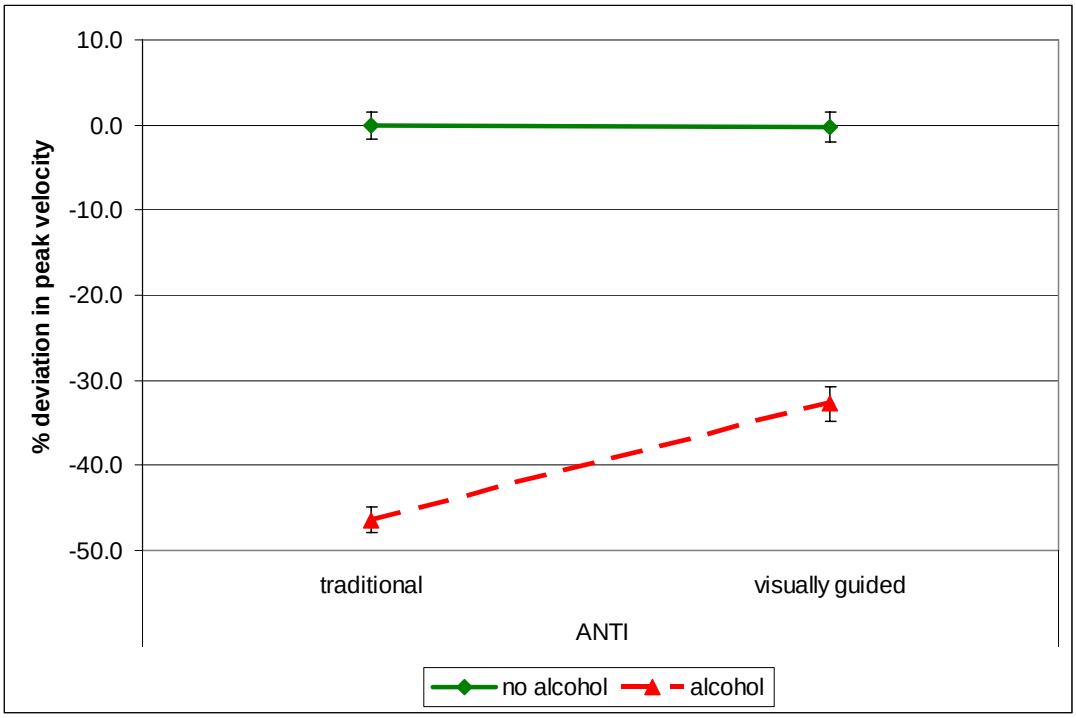

Figure 7-12. Illustration of the Beverage Condition main effects and Task X Beverage Condition interaction for peak velocities. Note that the no alcohol condition in the traditional paradigm was set to zero.

7.4.5 Erroneous Response and Error Correction

The properties of erroneous and corrective saccades were also analyzed. Only means form participants that made errors in all conditions were used for this analysis, therefore 5 datasets had to be discarded. Generally, latencies in trials with erroneous responses were shorter compared to trials with correct responses.

In the traditional anti saccade paradigm the latencies for primary saccades with erroneous responses were longer under alcohol ($F_{(1,13)}$= 4.81, p<.05) and shorter in the gap compared to the overlap condition ($F_{(1,13)}$= 29.80, p<.001; see A-5.17). Peak velocities showed a trend to be reduced under alcohol, but the effect failed to reach significance ($F_{(1,13)}$= 3.68, p=.077; see A-5.21 for complete ANOVA and means tables).

For the visually guided paradigm, the same result pattern was found with longer latencies under alcohol ($F_{(1,13)}$= 5.72, p<.05) and shorter latencies in the gap condition ($F_{(1,13)}$= 10.61, p<.05; see A-5.18), as well as reduced peak velocities under alcohol ($F_{(1,13)}$= 5.41, p<.05; see A-5.22). In addition, saccade amplitudes in trials with erroneous responses were shorter

under alcohol compared with the no alcohol condition ($F_{(1,13)}$= 8.55, p<.05; see A-5.19 for complete ANOVA and means tables).

When errors were made, they were corrected very frequently. The error correction rate was 93% in both, the traditional and the visually guided anti saccade paradigm. There were no effects of Gap Condition, Beverage Condition, or Alcohol Session on the error correction rates. The only other parameter that showed significant results was saccade amplitude of the corrective saccade. In the traditional paradigm, corrective amplitudes were longer under alcohol ($F_{(1,13)}$= 5.32, p<.05) and in the gap compared to the overlap condition ($F_{(1,13)}$= 13.54, p<.05). This gap effect was more pronounced, if alcohol was administered in session 1, leading to a significant Gap Condition X Alcohol Session interaction ($F_{(1,13)}$= 6.78, p<.05). In the visually guided paradigm, corrective saccades were also longer in the gap condition ($F_{(1,23)}$= 28.71, p<.001), but there were no effects of alcohol ($F_{(1,13)}$= 0.67, p>.10) and no other effects reached significance.

7.5 Discussion

The anti saccade paradigms were included in this thesis to examine the effects of alcohol intoxication on the voluntary level of oculomotor control. In contrast to prior work, a visually guided variation of the traditional anti saccade paradigm was also implemented and the paradigms are embedded in a firm theoretical framework, allowing the mapping of influences of alcohol intoxication on various levels of oculomotor control. Effects for the gap manipulation were generally in the expected direction, so that the discussion focuses on effects related to alcohol.

The findings of the current experiments demonstrated that error rates did not differ between the traditional and the visually guided paradigm. More importantly, in both paradigms there was no influence of alcohol intoxication. Furthermore, error correction rates were also not affected by alcohol, with very high correction rates in both paradigms (93%). Interactions between Beverage Condition and Alcohol Session for error rates (Figure 7-2 and Figure 7-4) can be attributed to training effects. Error rates were elevated in the first session, regardless whether this was the no alcohol or the alcohol session. Although finding no influence of alcohol on error rate is in line with Blekher et al.'s (2002) minority results, it appears to contradict findings from studies by Vassallo & Abel (2002) and Khan et al. (2003). Both reported a decrease in error rate under alcohol, whereas Crevits et al.'s (2000) even found increased error rates. The results of Vassallo and Abel can be attributed to a learning effect due to the unbalanced sequence of non-alcohol and alcohol sessions they used, Khan et al. accounted for this factor. The increased error rates in Crevits et al.'s (2000) can be ascribed

to blood alcohol levels ranged up to very high levels, which could have impaired additional processes not affected by moderate intoxication as used in the present study.
In contrast to error rates, primary saccade latencies are affected by alcohol. In the traditional anti saccade paradigm latencies for saccades to correct locations were 30 ms longer, in the visually guided 9 ms longer under alcohol. Latencies for trials with responses to the incorrect direction were also prolonged. Overall, this is in line with earlier work (Khan et al., 2003; Blekher et al., 2002) and can be accounted for by an attenuation of motor preparation due to alcohol intoxication (Everling et al., 1999). In addition, the comparison of the two tasks showed that latencies were longer in the visually guided paradigm, but there were no interaction between tasks and alcohol. The longer latencies can be explained by the fact that the transient onset of the target in the visual periphery is partially masked by the concurrent presentation of the rectangular frame. Indeed, participants reported that the detection of the stimulus was more difficult in the visually guided version of the task. Nevertheless, this pattern indicates that alcohol had a general slowing effect on oculomotor control. However, taking the interaction with alcohol session into account, it seems to be the case that the effect of alcohol intoxication is less pronounced, if a task is more familiar. This is a sensible finding, as with training an unfamiliar reaction pattern requires less voluntary control. In addition, the variability of saccade latencies was also lower in the second session, independent from Alcohol Session, again indicating a practice effect on latencies.

Regarding the peak velocities of saccades, alcohol intoxication led to decreased peak velocities, while leaving the main sequence intact (see Appendix A-5.23). This finding replicates results from Blekher et al. (2002), the only other study reporting velocity data for the anti-saccade task under alcohol. The comparison of the traditional with the visually guided paradigm yielded a larger effect of alcohol on peak velocities in the traditional task. This can be explained by practice effects, as the visually guided task was always performed after the pro and the traditional anti saccade paradigm. Following Edelman et al. (2006, see 7.1.3) peak velocities in the visually guided paradigm were expected to be higher. However, this effect was only found in the alcohol condition, whereas peak velocities in the no alcohol condition did not vary.

Results thus far suggest that brain stem processes and DLPFC functioning (indicated by stable error rates) are not specifically affected by moderate alcohol intoxication. However, increased latencies, in the absence of any effect on error rates, point to an alcohol-related impairment of FEF functioning (Pierrot-Deseilligny et al., 2003).

Furthermore, findings for primary saccade amplitude parameters point to specific impairments in the anti saccade tasks under alcohol. During no alcohol anti saccade trials, the

typical undershoot in saccade extent (Bell et al., 2000; Edelman et al., 2006) was found. Under alcohol, however, saccade amplitudes were significantly elongated in correct trials, thus appearing to be more "accurate." In reality, however, this pattern represents a substantial deviation from the normal hypometric saccades found in anti saccade performance under no alcohol conditions. Earlier studies did not report results on saccade amplitudes (Khan et al., 2003) or found no significant differences (Vassallo & Abel, 2002). However, despite using only a very small number of trials per subject, the authors of the latter study noted that *some* subjects showed improved accuracy when under alcohol, suggesting low power or low reliability of measurement might have masked a significant effect. In the only other study reporting saccade accuracy, Blekher et al. (2002) found a significant overshoot for saccades under alcohol for the anti-saccade task, but not the pro saccade task. As suggested above, any apparently improved "accuracy" observed in the alcohol condition might be better interpreted as a deviation from the "normal" saccadic undershoots. In this connection, the variability of saccade amplitudes was *not* significantly affected by alcohol intoxication in either task. This suggests that alcohol intoxication does not have a global effect on saccade accuracy, which, in turn, implies that cerebellar processes involved in the saccade control (Scudder at al., 2002; Enderle, 2002) did not appear to be affected by alcohol, least not at the dose used here. The question, whether the observed specific deficit in saccade programming was due to impairment of higher level processes involved in the spatial remapping process or the generation of endogenous saccade targets – both necessary in the anti saccade task – was in part addressed by using the visually guided anti saccade paradigm, in which a visual stimulus was presented at the saccade target location. The comparison of performance between the traditional and visually guided paradigm, showed that saccade amplitudes under alcohol were not affected by the additional visual marker. In the no alcohol condition, however, saccade amplitudes were significantly longer, when a visual marker was presented at the target location. The lack of a difference between paradigms for amplitudes under alcohol can be attributed to the fact that saccades already landed at the target location in the traditional paradigm. The additional target marker improved saccade accuracy in the no alcohol condition, but amplitudes under alcohol were still significantly longer. Interestingly, when making an erroneous response (i.e. performing a saccade to towards the appearing peripheral target), saccade amplitudes were shorter in the alcohol condition, thereby mirroring the results from the pro saccade paradigm, again showing that prolonged saccade amplitudes only occurred when an anti saccade was actually executed.

At this point, the presented results suggest that the impairing influence of alcohol on saccade amplitudes was not related to the fact that the saccade target needs to be represented endogenously, but is rather related to spatial remapping processes. This assumption will be

further investigated in the next chapter, using the memory guided saccade paradigm, where saccade targets need to be represented endogenously, but no reprogramming is necessary.

It is interesting to note that somewhat parallel results in connection with saccade amplitudes were recently obtained in studies on cannabis intoxication by Ploner et al. (2002). They found increased amplitudes for memory-guided saccades under THC intoxication. Unfortunately, they did not report saccade amplitudes for the anti-saccade task. More recently, Huestegge, Radach & Kunert (under review) found a very similar pattern of prolonged latencies and elongated amplitudes in chronic cannabis users tested, when sober and compared to a cannabis-naive control group.

Taken together, the results from the anti saccade paradigms indicate moderate, but specific deficits on the voluntary level of oculomotor control. In addition to an effect of general slowing, the inhibition of reflexive responses is not impaired under alcohol, but processes underlying the programming of spatial saccade parameters showed distinct effects of alcohol intoxication. These findings will be discussed in relation to affects of alcohol on other processing levels in the general discussion (see chapter 11).

8 Voluntary processing: The Memory Guided Saccade Paradigm

It is well known that alcohol can impair memory related processes, possibly culminating in severe disorders like Wernicke-Korsakoff syndrome in chronic alcoholics (e.g., Parsons & Nixon, 1993). However, there is little experimental research that examined memory related processes under acute alcohol intoxication in humans. Within the framework of this thesis, the *memory guided saccade paradigm* is used to study effects of acute alcohol intoxication on visual-spatial short term memory. In addition, this task allows examining processes related to inhibition. The anti saccade paradigms reported above required not only the inhibition of reflexive saccades, but also a reprogramming of saccade parameters to a new target location. In the memory guided saccade paradigm, no such reprogramming of the saccade target was necessary. Performance in this paradigm relies on the ability to inhibit the reflexive response and keep a representation of a target location available for a short period. Therefore the memory guided paradigm is a valuable addition to examine the influence of alcohol intoxication, targeting a new aspect on the voluntary level of oculomotor control. The next section provides the theoretical background for this paradigm.

8.1 Theoretical Background

Previous work on effects of alcohol intoxication on visual-spatial short term memory has been limited (Wegner & Fahle, 1999b; Echeverria et al., 1991; Hindmarch, 1983) and yielded mixed results. Wegner and Fahle (1999b), for example, used the Benton Visual Retention Test as a neuropsychological measure and a Vernier discrimination task to assess visual short term memory within the same sample. In the Benton test, geometric forms of increasing complexity were presented for 10sec each. Participants were instructed to draw a copy from memory, immediately after the presentation of each form. The second task used verniers, which are a pair of two vertical lines with a gap between them. The lower line can be displaced in the horizontal axis. In the vernier discrimination task, a vernier was presented for a brief interval, followed by a memory interval of either one or four seconds. Then, a second vernier was displayed and participants had to judge, whether this vernier had a smaller or larger offset than the first. Wegner and Fahle found that alcohol intoxication impaired performance on the vernier task, but not in the Benton task. These results demonstrate that simple visual stimuli are not only well suited to examine effects of alcohol on visual-spatial short term memory, but that they are also more sensitive than classic neuropsychological testing.

The memory guided saccade paradigm was selected to test the effect of alcohol on visual-spatial short term memory. In this task, saccades to remembered target positions are required. Therefore, participants are instructed to maintain fixation on a central fixation point, while a visual target is presented in the periphery for a certain amount of time. Only after the central fixation point disappears, with a certain (memory-) delay after the offset of the peripheral target, a saccade should be executed to the position where the peripheral target was displayed earlier. By varying the memory delays, temporal aspects of the visual-spatial short term memory can be studied, which was the main focus of previous studies using this paradigm. This is an interesting angle to extent the mapping of effects of acute alcohol intoxication on visuomotor functioning.

A variety of studies has used the memory guided saccade paradigm to examine voluntary processes related to saccade execution (Pierrot-Deseilligny et al, 2002; Hikosake & Wurtz, 1983; Funahashi et al, 1989; Fuster 1991). Performance of healthy participants is usually very good, with only slightly reduced accuracy compared to a traditional pro saccade task, whereas patients with lesions, especially those affecting frontal lobes and basal ganglia, show impaired performance (White et al., 1994). In order to determine neurophysiological mechanisms underlying processes of spatial integration, memorization, and saccade triggering, a number of studies have used the memory guided paradigm with rhesus monkeys and humans, using different techniques. A network of connections between the DLPFC, FEF, and PPC has been suggested to contribute to performance in the memory guided paradigm (Pierrot-Deseilligny, 1993; Sweeney et al, 1996). In humans, transcranial magnetic stimulation (TMS) over the DLPFC resulted in impaired accuracy of memory guided saccades (Brandt et al, 1998; Nyfeller et al, 2004), results corroborated by findings in lesion studies (Pierrot-Deseilligny et al, 2003). Neurons in the DLPFC of monkeys were shown to have the ability to hold memory specific to visual-spatial coordinates in a topographical memory map (Sawaguchi & Iba, 2001). Results regarding the DLPFC suggest its involvement in the control of saccade accuracy during the memorization phase in the memory guided saccade paradigm. The FEF can maintain accurate spatial representation of the environment, independent of the continuous presence of the stimulus (Umeno & Goldberg, 2001) and are thought to be mainly involved in the triggering of memory guided saccades (Pierrot-Deseilligny et al, 2002).

Taken together the memory guided saccade paradigm allows examining processes linked to *visual-spatial representation* and *inhibition*. As in the standard anti saccade paradigm, there is no visual stimulation at the target location for memory guided saccades, when saccades are executed. In both tasks, saccade targets have to be represented endogenously. However, in contrast to the anti saccade task, no reprogramming of the spatial parameters is necessary for

memory guided saccades. If alcohol intoxication has a similar impact with respect to spatial parameters in both tasks, this would indicate impairments related to the endogenous representation independent of spatial reprogramming.

Regarding the aspect of inhibition, the number of premature responses – executed during the memory interval – can be easily determined in the memory guided paradigm. This index can be used to determine the ability to inhibit a prepotent response and the possible modulation by alcohol. Abroms, Gottlob, and Fillmore (2006) have used a task similar to the memory guided saccade task, to look at alcohol effects on inhibitory control. In fact, their delayed oculomotor response task (DOR) represents a special case of the memory guided task. In the DOR task, the target of the previous saccade becomes the start point for the next saccade, instead of always using a central fixation cross as a start point for all saccades. Apart from this difference, the task is identical with the memory guided paradigm. Abroms et al. found that alcohol intoxication led to an increased proportion of premature saccades. In other words, reflexive saccades at the onset of the peripheral visual target or during the memory interval were more frequent under alcohol. This increase was dose dependent with more premature saccades under higher alcohol doses. Saccade latencies were also longer under alcohol, however, only in the higher dose (65 mg%) and not in the lower dose (45 mg%) condition. Unfortunately, no results on saccade latencies were reported and saccade accuracy was only described in terms of absolute degrees deviation from the target location. These results showed greater deviations for both alcohol conditions, compared to the placebo condition, but did not allow determining whether saccades under alcohol were hypermetric, hypometric, or generally more variable. Abroms et al. (2006) interpret their findings in terms of an impairing effect of alcohol on intentional inhibitory mechanisms.

Given these theoretical considerations and previous results from alcohol related studies, hypotheses are generated in the next section.

8.2 Hypotheses

Following the findings of Wegner & Fahle (1999b), an influence of alcohol on visual-spatial memory was predicted. In the memory guided paradigm this could become evident in a reduced percentage of responses to the correct target location, a higher variability of saccade amplitudes or a combination of both effects.

With respect to inhibitory processes, predictions were derived in parts from the finding by Abroms et al. (2006) that performance in a task demanding a delayed oculomotor response was impaired under alcohol. This suggested that alcohol reduces intentional inhibitory

control. In the memory guided saccade task this was expected to result in a substantial increase in the frequency of saccades executed prematurely with the onset of the peripheral target or during the variable memory interval. This seems to be a contradiction to the hypotheses for the anti saccade task, were no differences in error rates were expected. However, in comparison with the anti saccade task, there is no competition between an automatic and a voluntary saccade program in the memory guided paradigm. Instead, the impulse to elicit an automatic saccade needs to be delayed, creating a more pronounced measure of intentional inhibition.

In addition, latencies were hypothesized to be longer under alcohol, due to an anticipated general slowing. Regarding the spatial parameters hypotheses were exploratory. If the endogenous representation of the target location is a critical process that is targeted by alcohol intoxication, impairments of saccade amplitudes should be affected. However, if alcohol had an impact on processing regarding the remapping of spatial parameters, saccade amplitudes should not be affected in this task, because no remapping is necessary for performance in the memory guided saccade task.

8.3 Materials and Method

Information regarding participants, alcohol administration, eye movement recording, and procedure were already provided in Chapter 4. The following sections will outline the experimental design in more detail and explain data analysis strategies.

8.3.1 Design

A trial started with the presentation of a light grey fixation cross with a 0.5° diameter centered on an otherwise black screen. 1,500 ms after fixation cross onset, a light grey circle (0.3°) was presented as peripheral target at either 3° or 6° eccentricity to the left or right of the fixation cross for 1,000 ms. Following a memory delay interval that was varied quasi randomly (500 ms, 1,000 ms and 2,000 ms), the central fixation cross was extinguished, serving as the signal to execute the saccade. There was a time window of 1,000 ms for responses, before the next trial started. Participants were instructed to remain fixated on the central fixation cross until it disappeared and only then make an eye movement to the position were the last peripheral target was presented as fast and accurately as possible.

There were a total of 192 trials divided into 4 blocks. Between blocks, the number of trials was balanced for the 3 memory intervals and 2 eccentricities. The order of left- and rightward presentation was randomized within blocks. The completion of each block took about 4½

minutes, resulting in a total duration of about 20min for the experiment, including calibration of the eye tracking system at the beginning of each block.

8.3.2 Data Analysis

Primary saccades were determined by selecting the saccade in a given trial that followed the onset of the central fixation marker and had an amplitude of at least 1.5°. Responses that were shorter (790 cases) or executed to the wrong direction (322 cases) were eliminated from analyses. These criteria resulted in 6,535 valid saccades (85%). Further, saccades were classified as *primary* saccade, if a saccade was performed after the offset of the central fixation marker (62%, 4,791 cases) and as *premature*, if the saccade was elicited before the end of the memory interval (23%, 1,744 cases).

Values for valid primary responses were averaged per participant and entered into a 2 x 3 x 2 x 2 repeated measures ANOVA, with the within subject factors Eccentricity (near vs. far), Memory Interval (short, medium, long) and Beverage Condition (no alcohol vs. alcohol) and the between subject factor Alcohol Session.

As not every subject contributed cases to all possible factor level combinations for the analyses of latencies and amplitudes for premature responses, 4 datasets were discarded for these analyses.

8.4 Results

Table 8-1 and Table 8-2 provide overviews of the results in the memory guided saccade paradigm for valid and premature responses. Subsequently, results are presented in detail again for the valid responses first, followed by the analysis of the premature responses.

Table 8-1. Key results for valid responses in the memory guided saccade paradigm. Note that values presented were averaged across eccentricities, due to space restrictions. Further tables including all factors are located in the appendix (see A-5.23).

	Alcohol Session	Means (SE)					
		500		1000		2000	
		no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol
proportion of valid responses	1	0.90 (0.03)	0.84 (0.04)	0.92 (0.03)	0.84 (0.04)	0.87 (0.02)	0.84 (0.04)
	2	0.85 (0.03)	0.82 (0.05)	0.85 (0.03)	0.79 (0.04)	0.86 (0.02)	0.76 (0.04)
primary saccade latency for correct responses(ms)	1	297 (11)	331 (12)	256 (8)	288 (12)	242 (7)	268 (8)
	2	342 (13)	342 (14)	286 (9)	295 (14)	257 (8)	265 (9)
primary saccade amplitude for correct responses (degree)	1	4.21 (0.06)	4.23 (0.11)	4.23 (0.08)	4.27 (0.11)	4.39 (0.09)	4.26 (0.12)
	2	4.25 (0.08)	4.27 (0.12)	4.27 (0.09)	4.29 (0.13)	4.29 (0.11)	4.33 (0.14)

Table 8-2. Key results for premature responses in the memory guided saccade paradigm. Note that values presented were averaged across eccentricities, due to space restrictions. Further tables including all factors are located in the appendix (see A-5.23).

	Alcohol Session	Means (SE)					
		500		1000		2000	
		no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol
proportion of valid responses	1	0.25 (0.04)	0.27 (0.04)	0.25 (0.04)	0.31 (0.04)	0.32 (0.04)	0.36 (0.04)
	2	0.22 (0.04)	0.20 (0.04)	0.26 (0.04)	0.20 (0.04)	0.29 (0.04)	0.24 (0.04)
primary saccade latency for correct responses(ms)	1	1185 (121)	1197 (147)	1276 (203)	1400 (96)	1445 (162)	1558 (136)
	2	1314 (121)	1273 (147)	1411 (203)	1568 (96)	1906 (162)	1582 (136)
primary saccade amplitude for correct responses (degree)	1	3.62 (0.27)	3.78 (0.35)	3.72 (0.36)	4.21 (0.38)	3.48 (0.29)	3.75 (0.34)
	2	3.84 (0.27)	3.76 (0.35)	4.09 (0.36)	3.46 (0.38)	4.10 (0.29)	3.87 (0.34)

8.4.1 Valid Responses

First, the effect of alcohol intoxication on the proportion of responses that were directed to the correct target location is reported. In the remainder of this chapter, cases in which a valid saccade was executed to the correct target location will be referred to as *correct responses*. Under alcohol, the proportion of correct responses was significantly reduced ($F_{(1,18)}$= 10.59, p<.05). Eccentricity also produced a significant main effect ($F_{(1,18)}$= 35.96, p<.001), with more correct responses to near compared to far targets. In addition, the Beverage Condition X Eccentricity interaction was significant ($F_{(1,18)}$= 8.23, p<.05), with the difference between alcohol conditions being only different in the far ($F_{(1,18)}$= 12.11, p<.05), but not the near eccentricity ($F_{(1,18)}$= 1.53, p>.10), as depicted in Figure 8-1. There was no effect of the memory interval on the proportion of correct responses, neither showed any other interactions significance (all ps>.10; see A-6.1 for complete ANOVA and means tables).

Results for latencies and amplitudes are reported for correct responses only. One dataset with less than 1/3 of correct responses was discarded from further analysis.

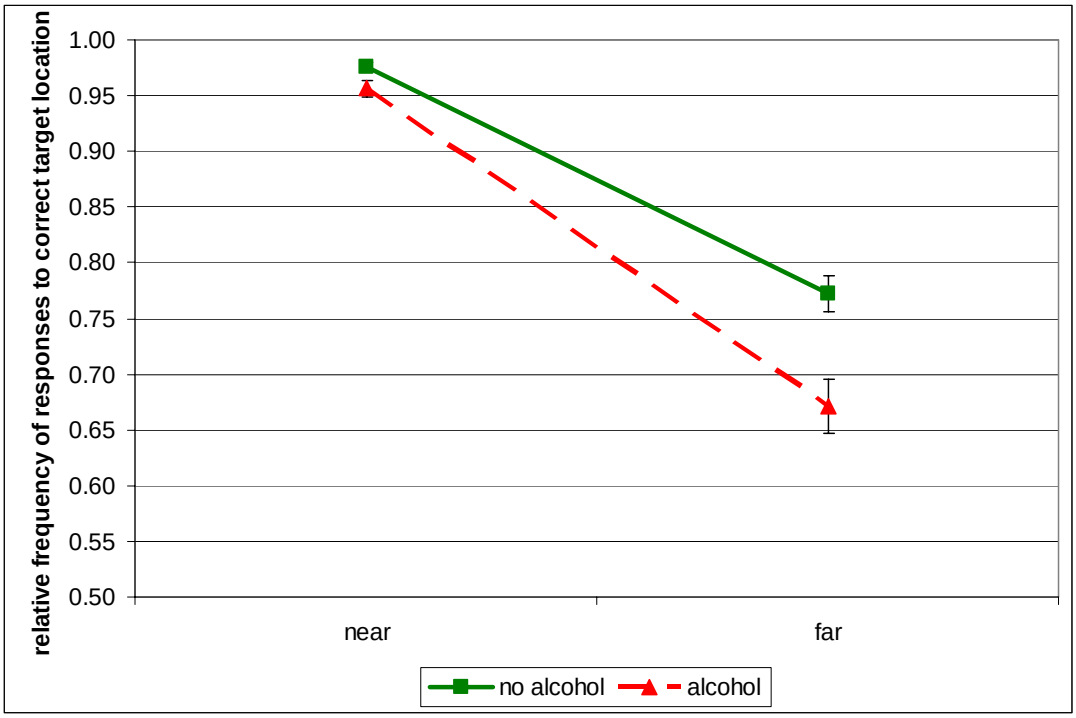

Figure 8-1. Proportion of responses to the correct target position for different alcohol conditions and eccentricities. Note that the proportion is based on the valid responses only.

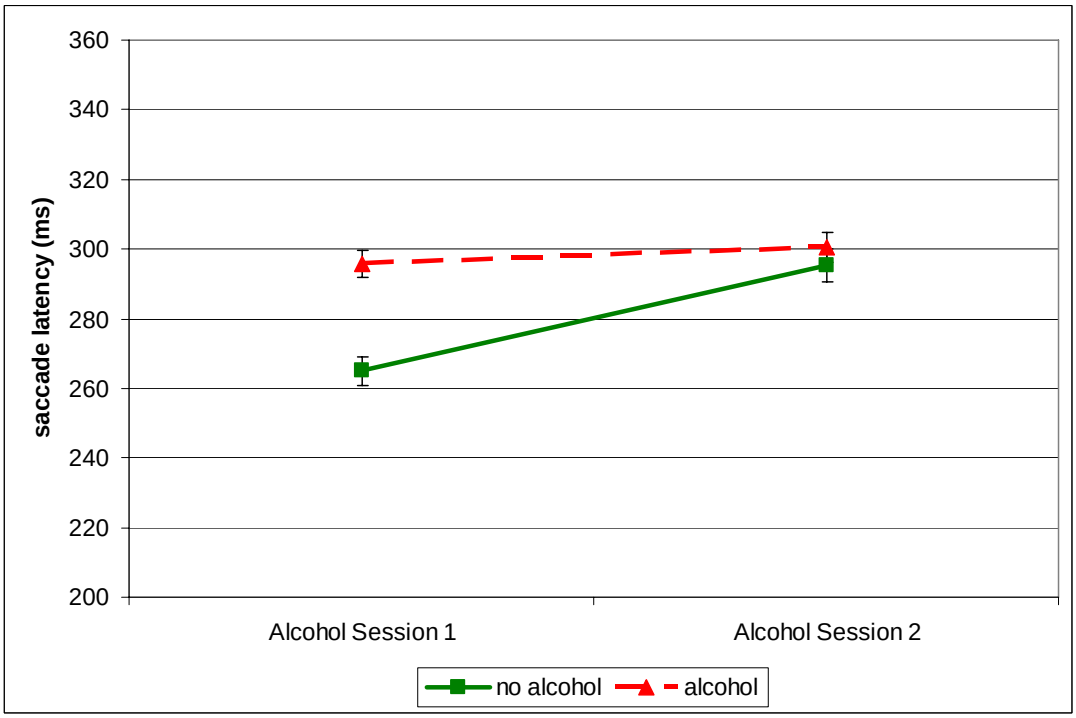

Figure 8-2. Saccade latencies for correct responses. Differences between alcohol conditions only occur, if alcohol was administered in the first session.

Saccade latencies were ~18ms longer in the alcohol condition ($F_{(1,17)}$= 18.31, p<.001). The effect of Beverage Condition was only existent, if alcohol was administered in session 1 (see Figure 8-2), resulting in a significant Beverage Condition X Alcohol Session interaction ($F_{(1,17)}$= 9.21, p<.05). Memory Interval and Eccentricity also influenced latencies, with shorter latencies for increasing memory intervals ($F_{(2,16)}$= 47.95, p<.001) and to far saccade targets ($F_{(1,17)}$= 7.49, p<.05). However, these main effects and interaction were qualified by a significant Eccentricity X Memory Interval X Beverage Condition X Alcohol Session interaction ($F_{(2,17)}$= 3.99, p<.05). Figure 8-3 illustrates this four-way interaction. The effect of Beverage Condition was more pronounced, if alcohol was administered in Session 1. Longer memory intervals led to shorter saccade latencies in the no alcohol condition independent of Alcohol Session. Latencies were longer in the no alcohol condition, if alcohol was administered in session 2. This effect was most pronounced for the near eccentricity with a short memory interval (see A-6.2 for complete ANOVA and means tables).

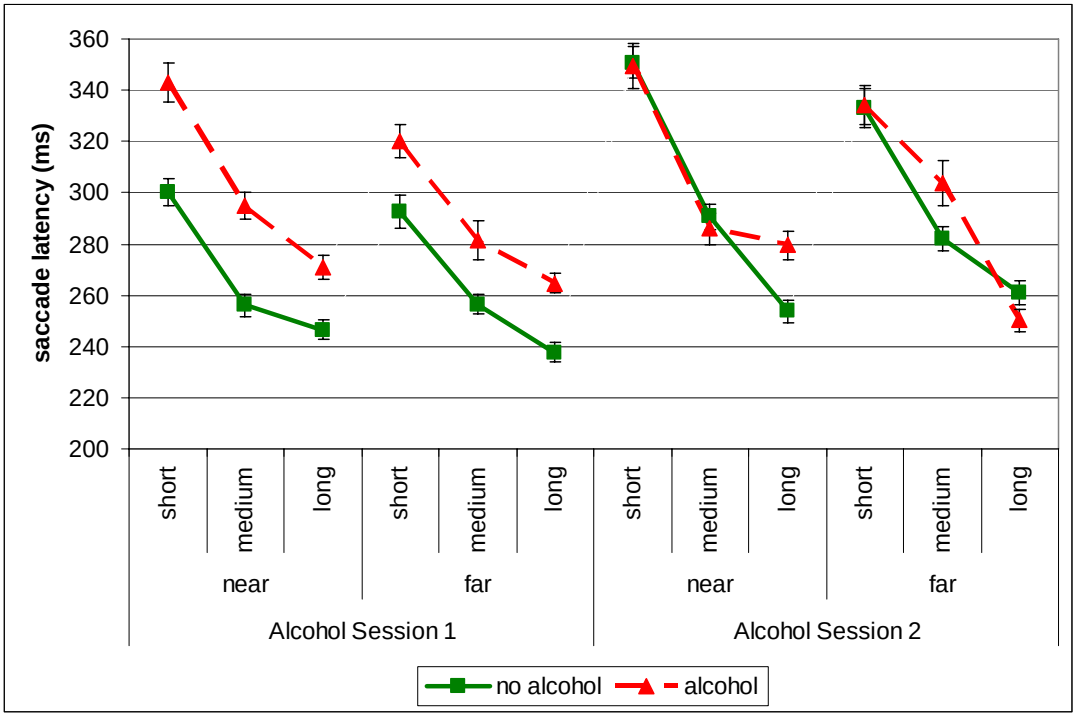

Figure 8-3. Four way interaction between Eccentricity, Memory Interval, Beverage Condition, and Alcohol Session for saccade latencies in trials with correct responses.

For saccade amplitudes, the analysis of correct responses showed, not surprisingly, a main effect for Eccentricity ($F_{(1,17)}$= 2251.64, p<.001), with longer amplitudes for far saccade target locations. There was no effect of Beverage Condition ($F_{(1,17)}$= 0.01, p>.10) or Memory Interval ($F_{(2,16)}$= 2.04, p>.10) on saccade amplitudes (see A-6.3 for complete ANOVA and

means tables). Variability of saccade amplitudes was also unaffected by alcohol ($F_{(1,17)}$= 0.10, p>.10; see A-6.4).

8.4.2 Premature Responses

For a total of 1,744 trials or 23% of all cases, responses were classified as premature (see section 8.3.2). Statistical analysis revealed that there was no significant difference between alcohol conditions for the proportions of premature saccades ($F_{(1,18)}$= 0.14, p>.10). Figure 8-4 depicts the percentage of premature reactions in relation to the duration and visual events during a trial. Apparently, the onset of the peripheral target triggers the highest percentage of premature responses, independent of the alcohol condition.

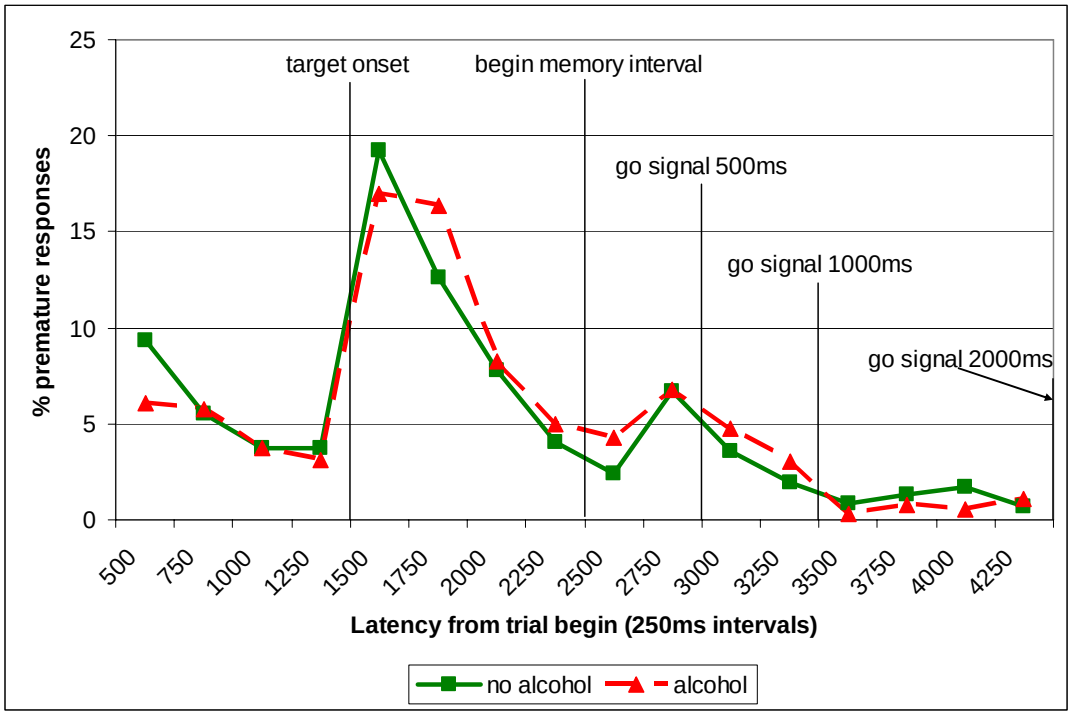

Figure 8-4. Percentage of premature responses in relation to latency intervals from trial begin. Note that percentages are based on premature responses only and not on the total number of trials.

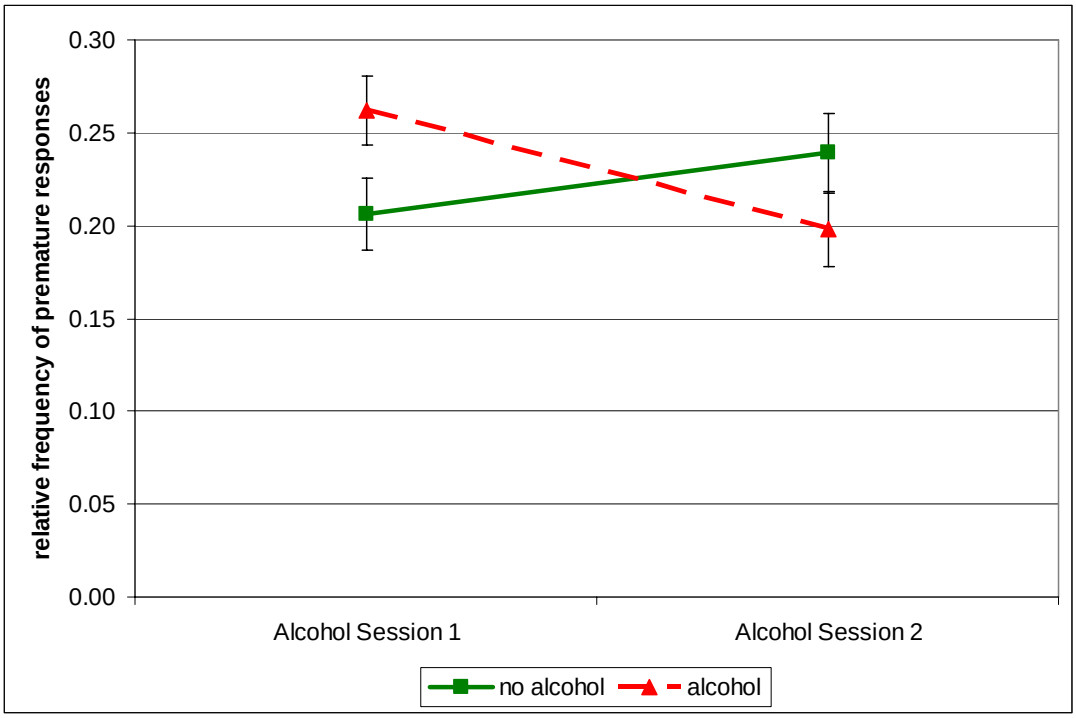

Figure 8-5. Relative frequencies of premature responses for different alcohol conditions and alcohol sessions.

In addition to these findings, a significant Beverage Condition X Alcohol Session interaction was found ($F_{(1,18)}$= 6.34, p<.05). More premature responses were made under alcohol, if administered in the first session, but the pattern reversed completely for alcohol session 2, where more premature responses were found in the no alcohol condition (Figure 8-5; see A-6.5 for complete ANOVA and means tables).

With respect to the latencies of premature responses, only a main effect of Memory Interval was found ($F_{(2,13)}$= 16.36, p<.001). Latencies were longer with increasing memory intervals (see A-6.6 for complete ANOVA and means tables).

Premature saccade amplitudes differed significantly between the two eccentricities ($F_{(1,14)}$= 16.93, p<.001), but not between alcohol conditions ($F_{(1,14)}$= 0.00, p>.10). Again the Beverage Condition X Alcohol Session interaction was significant ($F_{(1,14)}$= 8.13, p<.05), with amplitudes being shorter under alcohol, if administered in the first session and longer amplitudes under alcohol, if administered in the second session (Figure 8-6; see A-6.7 for complete ANOVA and means tables).

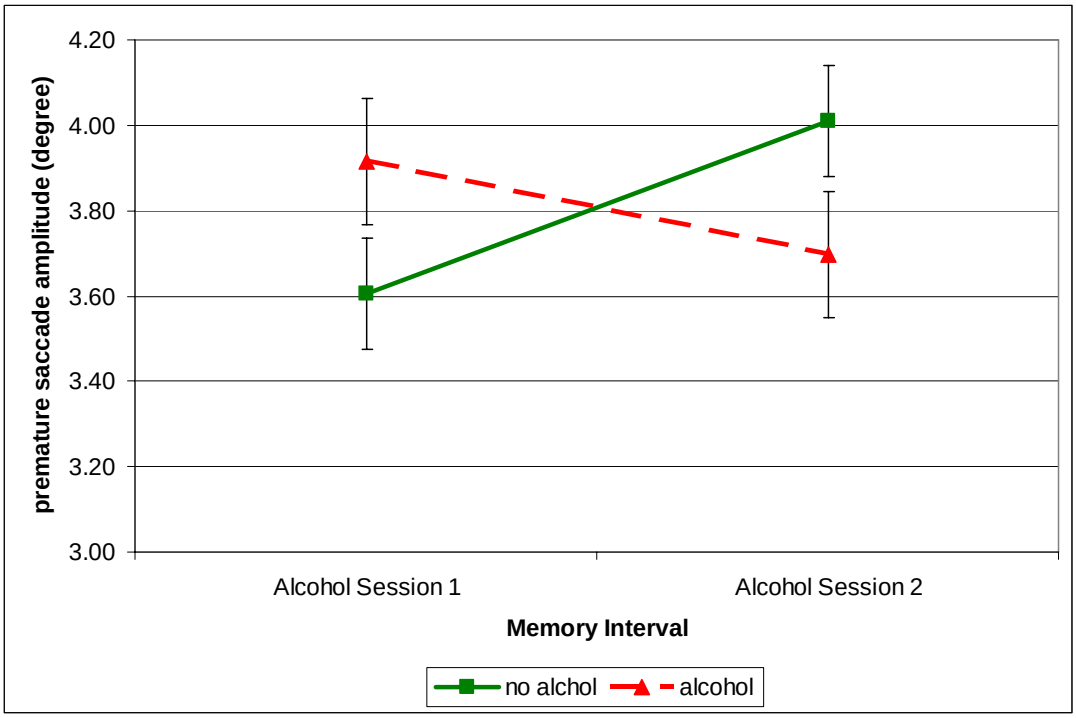

Figure 8-6. Interaction between Beverage Condition and Alcohol Session for premature saccade amplitudes.

8.5 Discussion

The memory guided saccade paradigm was selected within the framework of this thesis to examine effects of alcohol on processes related to the visual-spatial short term memory. While there is a substantial amount of literature on memory related deficits in chronic alcoholics (e.g. Parsons & Nixon, 1993), work on the influence of acute alcohol intoxication on visual-spatial short term memory has mostly focused on aspects of spatial learning mediated by the hippocampus, using the animal model (see White & Swartzwelder, 2005, for a review).

The results obtained in the current study demonstrated that moderate alcohol intoxication impaired visual-spatial memory. This was apparent in a reduced proportion of responses that were directed to the correct target location under alcohol. This finding is in line with earlier work (Wegner & Fahle, 1999b) that reported impaired performance in a different visual-spatial short term memory task under alcohol. In the vernier discrimination task Wegner and Fahle found significantly increased offset threshold under alcohol, for the detection of differences using memory intervals (1-4 seconds) comparable to the present work. However, the current results also showed that saccade amplitudes were not more variable under alcohol

and that there were no differences between alcohol conditions for correct responses. This indicates that the saccade programming system was not generally impaired by alcohol intoxication. Rather, the demonstrated effect suggests a specific deficit in the visual-spatial short term memory. The impairment was found independent of the length of the memory interval, again in line with Wegner and Fahle (1999b). It is unlikely, that this is related to insufficient differences between the memory interval conditions, which were relatively large in comparison to other studies and also yielded significant effects on other parameters. Therefore, the alcohol induced deficit is likely to be related to difficulties in the initial encoding of the visual stimulus position into memory. Interestingly, the same information on the spatial location of the stimulus can be used to reprogram the saccade target in the anti saccade task. However, this process does not rely on storing the spatial location of the visual target over a longer period.

Regarding the temporal aspects of saccade generation, alcohol led to longer latencies under alcohol, indicating a general slowing in line with results from other paradigms used in the present thesis. This effect was qualified however, by an interaction of Beverage Condition and Alcohol Session, as well as a 4 way interaction between all factors of the design. The latter interaction was predominantly caused by the results from participants that received alcohol in the second session. The effect of alcohol was not only reduced in these cases, indicating training effects, but showed also different effects of eccentricity for the medium and long memory interval. At present there is no interpretation at hand to explain this pattern

Turning to the results on premature responses, the memory guided paradigm yielded no differences between alcohol conditions for the proportion of premature responses and premature saccade amplitudes. The Beverage Condition X Alcohol Session interactions, which were found for both variables, showed a complete reversal of the alcohol effect between the Alcohol Sessions. Thus, this indicates a training effect independent of alcohol intoxication. The finding that latencies of premature responses were also unaffected by alcohol was not surprising, as the length of the interval, in which premature saccades can be elicited, amounted to up to 4,500 ms in the longest memory interval. This led to a considerable variability in premature saccade latencies, making it impossible to detect latency effects in the order of 20ms. Taken together, the intact inhibition of reflexive saccades is at odds with Abroms et al. (2006), who found an increase in premature responses under alcohol in a similar task (see 8.1). However, the timeline in their delayed oculomotor response task (DOR) was different from the current study. The presentation of the peripheral target was limited to 100 ms and the memory intervals varied between 800, 1,000 and 1,200 ms. Compared to the timing of the present experiment, the window for premature saccades was fairly limited in Abroms et al.'s work (1,500-4,500 ms vs. 900-1,700 ms). Hence, the

current implementation of the memory guided saccade paradigm should induced even more premature responses and yield larger alcohol related effects, if inhibitory processes were impaired. This was not the case and in addition, the lack of impairment in suppressing a reflexive saccade replicated results from the aforementioned anti saccade tasks. Therefore, these data indicate that moderate alcohol intoxication did not impair inhibitory processes in the context of oculomotor control. Another difference between the present study and Abroms et al. (2006) is the fact that no differences in saccade amplitudes were found in the current data, whereas Abroms et al.'s results showed a higher degree of deviation from the saccade target under alcohol. However, this is not surprisingly, as their analysis also included case with premature responses. The present data showed that eliminating this source of variability, saccade amplitudes in correct trials were not affected by alcohol intoxication.

In conclusion, the findings from the memory guided saccade task demonstrated a specific visual-spatial short term memory deficit under alcohol. In contrast, alcohol intoxication did not impair the ability to inhibit reflexive responses.

9 Complex Cognitive Processing I: Task Switching in the Pro and Anti Saccade Paradigm

In this chapter some key concepts from the task switching literature are introduced, before effects of task switching on the pro and anti saccade task are discussed. After this introduction of theoretical background, the design of the experiment is outlined and results are presented and discussed.

9.1 Task Switching: Background

Task switching experiments examine in which way switching between different types of tasks influence performance. In a typical experiment participants are first introduced to two or more different tasks. In the critical phase of the experiment, participants have to perform in both tasks, with changes between the tasks in some trials and task repetitions in other trials. There are several different methods to instruct a participant, which task to perform in a given trial. Most popular are the *alternating-runs paradigm*, in which the task alternates every N trials, the *task cueing paradigm*, where a cue immediately prior or together with the onset of a stimulus instructs the participants which task to perform or using short sequences of *pre-specified task sequences*. In either paradigm, the following phenomena can be observed (modified from Monsell, 2003):

Switch costs: Generally, performance in a switch trial is more difficult, resulting in longer response times and/or higher error rates in comparison with repetition trials. Switch costs are sometimes also referred to as task-repetition benefit.
Switch benefit: Under specific circumstances performance in a switch trial can be better than in a repetition trial. This is called switch benefit.
Preparation effect: If there is enough time to prepare the upcoming task, switch costs are usually somewhat reduced.
Residual costs: Even though switch costs are reduced with longer preparation time, substantial residual switch costs can be found even after relatively long preparation times (Sohn et al., 2000; Kimberg et al., 2000).
Mixing costs: Under conditions where the task switching occurs within an experimental block rather than between experimental blocks, a general increase in response times can be observed in comparison to pure blocks with only one task.

Even though there is an ongoing discussion of the underlying mechanisms of the above described effects, there is a consensus that the magnitude of the costs primarily depends on

the stimulus response characteristics of the pre switch task and not the task that is switched to (Wylie & Allport, 2000). Furthermore, the transfer of activation and inhibition from one trial to another trial is seen as a key factor in the formation of switch costs. Another term that can be used to describe activation and inhibition is priming. Positive priming refers to the phenomenon that performing in one task makes the following task easier, if there is overlapping stimulus-response mapping. In task repetition trials, where the stimulus response mapping is identical this is referred to as repetition priming. In these cases enduring activation from the previous trial is beneficial for the ongoing trial. In contrast negative priming occurs, when inhibition that was helpful in the previous trial (e.g., to suppress a prepotent response) is transferred to the ongoing trial and hampers performance (e.g., by slowing down execution of the now correct prepotent response).

A critical question in the context of activation and inhibition is what exactly is activated or inhibited. A task set is defined as defined as the mental representation of a given task (Roger & Monsell, 1995) and includes stimulus attributes, conceptual criteria, goal states, and action rules. Task sets for familiar tasks are stored and can be retrieved from memory, whereas task sets for novel tasks have to be specified by instruction and/or training. In a task switch condition, the active task set has to be reconfigured (Monsell, 2003). There is an ongoing debate, whether the reconfiguration of a task set depends on exogenous stimuli (Rogers & Monsell, 1995), or can also be triggered endogenously (De Jong, 2000). Koch (2003, 2001) has reported that under predictable switching conditions, a preparation interval reduces switch costs only when an external cue helps the participant to remember which task to perform. This difference leads to the suggestion that internal cues select the next task set, but external cues additionally facilitate preparatory retrieval of task specific stimulus response rules. In addition, Koch (2005) found that an observed benefit of predictability-based task preparation was not switch specific. Taken together he argues that task switching does not necessarily require a switch specific task set reconfiguration process, but that task specific control processes are needed in task switching as well as repetition trials.

Another question is in which way and to what extent task characteristics influence switch costs. A related counterintuitive finding is that switch costs are usually lower, if switching occurs form a dominant (easy) to a non-dominant (difficult) task than vice versa (Allport et al, 1994; Monsell et al., 2000). This is explained with a carryover effect of inhibition. Performing in a non-dominant task requires the active inhibition of dominant responses. In a task switch situation this active inhibition of the dominant response has to be cancelled to perform, causing the aforementioned switch costs in this case. On the other hand performing the dominant task does not require inhibitory processes regarding the non-dominant task, thus in dominant to non-dominant switch trials the costs are not as high as non-dominant to

dominant switch trials. However, these asymmetries in switch costs develop for every task pair, but only if there is both (a) a simultaneous onset of stimulus attributes, making selection of the relevant attribute difficult, and (b) an overlap in the response sets of the two tasks, making selection of the relevant response difficult (Yeung & Monsell, 2003).

This concise introduction into task switching is of course far from complete. As the aim of the present work is to examine the influence of acute alcohol intoxication on performance, the next section will focus on the effects of task switching in the paradigm used in the present thesis.

9.2 Task Switching: Using the Pro and Anti Saccade Paradigm

It has been consistently shown that performance in the anti saccade task is worse than in the pro saccade task, suggesting that processing of anti saccades is more complex. However, as discussed in chapter 7, it is not clear whether increased difficulty in anti saccade tasks reflects inhibition of an erroneous pro saccade or the more demanding programming of the desired saccade (Evdokimidis et al., 1996; Olk and Kingstone, 2003; Pratt and Trottier, 2005). The analysis of switch costs can help to gain additional insights into the components of pro and anti saccade control and the influence of alcohol intoxication hereon. The key concepts of task switching paradigms have already been introduced above. This section provides theoretical background on task switching using the pro and anti saccade paradigms.

Several studies have examined the effects of task switching on the performance in the pro and anti saccade tasks (Cherkasova et al., 2002; Reuter et al., 2006; Manoach et al., 2002; Fecteau et al., 2004; Hunt & Klein, 2002; Barton et al., 2005; Massen, 2002). Unfortunately, results in these studies regarding the effect of task switching on error rates and saccadic latencies are somewhat mixed.

The extent of switch costs in the pro and anti saccade paradigm depends on a multitude of factors. These include effects of task sequence and the length of the preparation interval, in other words, the time from knowing the next correct response until the actual go signal in a given trial. Looking at the task sequence it becomes apparent that four different combinations of task sequences are possible: pro-pro, anti-pro, pro-anti and anti-anti, with the first and last representing no switch conditions and the other two switch conditions. Given the theoretical basics provided above, performance should be impaired in the switch condition compared to the no switch condition, evident in elevated error rates and latencies. Furthermore, it could be predicted that switch costs should be more pronounced in trials that switch back to the dominant response (anti-pro) than in those that switch to a non-dominant response (pro-anti).

However, results of previous studies are equivocal. While some studies (e.g. Cherkasova et al., 2002; Hunt & Klein 2002) indeed found higher error rates in task switching compared to no task switching trials, other studies did not find switch cost, but even reported switch benefits for anti saccade error rates in switch trials (Hodgson et al., 2004). In addition, the expected greater impact of task switching when switching back to a pro saccade trial was not found by Cherkasova et al., but Hunt & Klein even report switch costs for the pro saccade task and a switch benefit for the anti saccade task regarding error rate. Looking at saccade latencies, the same confusing diversity of results can be found. Cherkasova et al. (2002) found increased latencies in a switch condition for pro saccades, but a switch benefit (decreased latencies) for anti saccades. Hunt and Klein (2002) on the other hand report switch costs in both tasks, this time even finding a greater impact when switching back to the dominant task.

As mentioned above, another factor influencing performance related to switch costs in this context is the length of the preparation interval. Hunt & Klein (2002) examined preparation intervals of 200 ms, 550 ms and 1,100 ms. They demonstrated that switch costs were only present in the short preparation interval, whereas the 550 ms interval showed only residual costs and the 1,100 ms interval even switch benefits for saccade latencies. Barton et al. (2005) used an experimental setup that allowed varying the preparation interval independent from the time interval between target stimuli, which was held constant at 3,700 ms. The reasoning behind this setup was to distinguish between effects that stem from inhibitory carry over effects of the last trial and active preparation processes (e.g., task set reconfiguration) for the following trial. The assumption being that switch costs should not be affected by the length of the preparation interval, if they result from inhibitory mechanisms of the last trial. However, if active processes needed to reconfigure the task set, longer preparation intervals should reduce switch costs. In blocks with randomized cued task switches the results for error rates showed that in the pro saccade task the effect of task switching was not modulated by the length of the preparation interval. In the anti saccade task, longer preparation led to a decrease in error rate. For the latencies, switch costs were found in the pro and anti saccade task under short preparation intervals. With long intervals, pro saccades showed moderate switch costs, whereas anti saccades showed a switch benefit. Although these results do not permit a definite answer of the question whether carry over inhibition or active task set configuration is responsible for the resulting switch cost effects, they are a replication of the results from Cherkasova et al. (2002) in that they find a switch benefit for anti saccades under long preparation intervals.

Looking at conditions with predictable task switches (a task switch endogenously triggered, in this case every two trials), results from the Barton et al. (2005) indicate that even though

the timing of the task switch is predictable, only the time during the preparation interval is used for preparing the next task. Additional time that is available during after the end of the previous trial and the onset of the preparation interval did not seem to be used. Similar results were reported by Koch (2001, 2003), who found that for predictable switching conditions, a preparation interval reduced switch costs only when an external cue helps the subject to remember which task to perform.

To complicate things further, a task switch between pro and anti saccades is confounded with the response direction. For example, a pro saccade to the right in trial N-1 can be followed either by an anti saccade with the target displayed at the right and therefore requiring a response to the left, which would constitute a task switch with a response switch. Alternatively, if the target for the anti saccade trial N is presented at the left side, the correct response would be to the right, resulting in a task switch condition without response switch. Of the 16 possible sequences in a mixed pro and anti saccade paradigm with two target locations, 50 % are task switch conditions, but in half of these cases an additional response switch is needed to perform correctly. The same is obviously true for the no task switch condition, in which in half of the cases a response switch is required in the absence of a task switch (see Table 9-1 for an overview of all 16 combinations). In addition to being confounded with the response switch condition, another issue with the switch costs related to pro and anti saccades is the fact that in anti saccade trials the reflexive response towards the visible target has to be inhibited in all anti saccade trials, independent of the switch conditions (see section 7.1). Hence, the question of the response switch condition could be more important, if the proceeding trial was in the anti saccade task, as the target position of that trial might still be inhibited in a current trial independent of the current task.

Studies examining the effect of response switch also demonstrated mixed results. Massen (2002) found higher error rates for response switches in the anti saccade task independent of task switching. Reuter et al. (2006) reported higher error rates for response switches in the anti saccade task only in task repetition trials. Response switches did not affect anti saccade performance in the task switch trials and also did not influence pro saccade error rate. In Fecteau et al. (2004) response switches also led to higher error rates, but were not modulated by the task switching condition. For saccade latencies the Reuter et al. and Fecteau et al. studies showed the same pattern with longer latencies in the no response switch condition across both tasks and task switch conditions. However, Massen (2002) reported the opposite pattern with longer latencies in the response switch condition, with the exception of the pro saccade task in task switch trials.

Table 9-1. Overview of possible combinations of task switch and response switch when using the pro and anti saccade tasks. Task switch and response switch conditions are based on trial N. N-1 refers to the trial immediately preceding this trial.

N-1		N		Task Switch	Response Switch
Task	**Target**	**Task**	**Target**		
pro	right	pro	right	***no***	***no***
pro	right	pro	left	***no***	***yes***
pro	left	pro	right	***no***	***no***
pro	left	pro	left	***no***	***yes***
anti	right	anti	right	***no***	***no***
anti	right	anti	left	***no***	***yes***
anti	left	anti	right	***no***	***no***
anti	left	anti	left	***no***	***yes***
pro	right	anti	right	***yes***	***yes***
pro	right	anti	left	***yes***	***no***
pro	left	anti	right	***yes***	***no***
pro	left	anti	left	***yes***	***yes***
anti	right	pro	right	***yes***	***yes***
anti	right	pro	left	***yes***	***no***
anti	left	pro	right	***yes***	***no***
anti	left	pro	left	***yes***	***yes***

Even though, the overall pattern of behavioral results from task switching studies using pro and saccades tasks is far from consistent, some potentially underlying mechanisms can be described. The basic principle of inhibition and activation remains identical with that introduced in section 9.1. Hunt & Klein (2002) suggest that “memory traces” are necessary to execute a given task. If memory traces from proceeding trials are still active in the ongoing trial, this can lead to facilitation effects, if the current task is identical with the previous task, or to task switching costs, if the task is different. Reuter et al. (2006) use the term “motor program” to refer to processes that are necessary to program saccades to endogenously represented targets (e.g., in the anti saccade task), whereas performance related to exogenous targets relies on the “sensorimotor transformation of stimuli” (Reuter et al, 2006, p.94).

Although there are open question regarding the factors and underlying mechanisms of task switching in the pro and anti saccade paradigms, it seems to be worthwhile to examine the influence of acute alcohol intoxication on task switching costs and benefits. Given the within subject design of the experiment (see section 9.4), the effects of alcohol on task switching can be studied in spite of a certain haziness regarding underlying mechanisms. Interestingly, an extensive literature research revealed that there have been no studies up to date that

examined the influence of alcohol on task switching. The only drug related studies on task switching examine the effect of caffeine. Tieges et al (2006, 2007) demonstrated that anticipatory control in task switching was improved by caffeine. Error rates and reaction time were significantly less influenced in switch trials in the caffeine compared to the placebo condition. The authors attribute this to a general effect of caffeine on task switching that is related to task-nonspecific anticipatory processes (e.g., actively maintaining the task set in working memory and protecting it against interference), rather than affecting task-specific processes (e.g., rule retrieval and rule-based response selection).

9.3 Hypotheses

The present experiment was not designed to resolve theoretical conflicts in the task switching literature. Rather, the task switching paradigm was used as a tool to examine the influence of alcohol on processes that are central in everyday functioning and captured in this experimental paradigm. In addition, using a second experiment with the pro and anti saccade paradigm allows replicating and corroborating findings from Cluster 1.

Given the lack of prior research on the influence of alcohol intoxication on task switching, hypotheses with regards to task switching for this experiment were of exploratory nature. However, assuming that alcohol has somewhat contrary effects on task switching performance than caffeine, results from these studies can serve as indicators of possible effects, as can the results from the basic oculomotor paradigms.

Longer latencies under alcohol can be expected (see sections 5.4.2 and 7.4.2). If alcohol reduces the ability of actively maintain a task set in working memory (contrary to the effect of caffeine), these effects might be more pronounced under task switch conditions.

Additionally, results from pro and anti saccade paradigms in Cluster 1 showed that alcohol did not increase error rate. It is possible that longer latencies – hypothesized under task switch conditions – enable the saccade programming system to generate the correct response position leading to decreased error rates under alcohol in task switch conditions. On the other hand it is also possible that alcohol decreases cognitive flexibility and error rates are higher under alcohol in the task switch condition due to perseveration.

Results for saccade amplitudes have not been reported for task switching experiments, but it was expected to replicate the findings from the anti saccade paradigm in Cluster 1 with hypermetric saccade amplitudes under alcohol. An additional effect of task switching could

not be ruled out, but there were no theoretical foundations to assume longer or shorter saccades under task switch conditions.

9.4 Materials and Method

Information regarding participants, alcohol administration, eye movement recording, and procedure were already provided in Chapter 4. The following sections will outline the experimental design in more detail and explain data analysis strategies.

9.4.1 Design

A trial started with a light grey fixation cross of 1 deg diameter presented in the center of a black screen. After 1,000 ms the color of the fixation cross changed, indicating whether a pro-saccade trial (green) or anti-saccade trial (red) was to be executed. This fixation marker remained visible during the entire duration of each trial (constituting an overlap condition; see section 5.1.1). 300 ms after the color change a light grey circle with a diameter of 0.5° visual angle appeared at 6° either to the right or to the left of the central fixation cross. For the pro-saccade trials, participants were instructed to look at the peripheral target as quickly and accurately as possible as soon as it appeared. In anti-saccade trials the task was to look to the mirror position of the appearing peripheral target as quickly and accurately as possible. The peripheral target stayed visible for 800 ms before the next trial started with a new light grey central fixation cross. If a participant moved back to the colored fixation cross while the peripheral target was still visible, an eye movement contingent display change was implemented during the return saccade to switch the display back to the neutral light grey centered fixation cross. At the same time the peripheral saccade target was erased and a new trial started 50ms later.

The decision to use a mixed block design with cued random task switching was a reasoned one. Even though performance in task-repetition trials of mixed-task blocks has been reported to be worse than in single-task blocks (Koch and Philipp, 2005; Los, 1996), these effects seem to be small for pro- and anti saccades (Cherkasova et al., 2002, Hodgson et al., 2004). In addition it seemed sensible to use a paradigm with maximal difficulty to establish effects caused by acute alcohol intoxication.

There were a total of 280 trials divided into 8 blocks. Within blocks pro- and anti-saccade trials were mixed and presented in a fixed random order. The completion of each block took about 80sec, resulting in a total duration of about 12min for the experiment, including calibration of the eye tracking system at the beginning of each block.

9.4.2 Data Analysis

Cut off criteria regarding latencies and amplitudes were the same as in Cluster 1 (see section 5.3.2). Those restrictions resulted in 91% valid primary saccades across all trials, corresponding to a total of 11,509 primary responses. Data were analyzed using a 2 x 2 x 2 x 2 x 2 repeated measures ANOVA with the within subject factors Task (pro vs. anti), TaskSwitch (no task switch vs. task switch), ResponseSwitch (no response switch vs. response switch) and Beverage Condition (no alcohol vs. alcohol) and the between subject factor Alcohol Session. The factor alcohol session represents the fact whether alcohol was consumed in the first or the second session.

9.5 Results

Findings are presented by the dependent variables. Table 9-2 presents an overview of alcohol related results for key variables. Further data tables are located in the Appendix and referenced in the respective passages.

Table 9-2. Overview of results for key findings in the pro and anti saccade task switching paradigm. Values represent means and standard error based on 20 participants (see text for more details).

	Means (SE)							
	pro saccade				anti saccade			
	no task switch		task switch		no taskswitch		taskswitch	
	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol	no alcohol	alcohol
proportion of errors	0.01 (0.01)	0.02 (0.01)	0.03 (0.01)	0.04 (0.01)	0.17 (0.03)	0.19 (0.03)	0.20 (0.03)	0.17 (0.03)
primary saccade latency (ms)	194 (6)	215 (8)	207 (7)	230 (9)	242 (7)	265 (8)	242 (8)	271 (9)
primary saccade amplitude (degree)	5.96 (0.05)	6.06 (0.08)	5.96 (0.05)	6.08 (0.08)	5.63 (0.21)	5.98 (0.23)	5.29 (0.18)	5.77 (0.23)

9.5.1 Error Rate

Results for error rate showed significant main effects for factors Task ($F_{(1,22)}$= 35.14, p<.001) and ResponseSwitch ($F_{(1,22)}$= 5.42, p<.05). Error rates were higher in the response switch condition (~11%) in comparison with the no response switch condition (~9.5%). The main effect of Task and the significant interaction between TaskSwitch and Beverage Condition ($F_{(1,22)}$= 7.60, p<.05) were further qualified by the three way interaction Task X TaskSwitch X Beverage Condition ($F_{(1,23)}$= 11.92, p<.05). Figure 9-1 depicts this interaction, showing that for the pro saccade task error rates were elevated in the task switch condition compared

to the no tasks switch condition under no alcohol ($F_{(1,22)}$= 21.16, p<.001) and alcohol ($F_{(1,22)}$= 4.71, p<.05). The difference between no alcohol and alcohol conditions did not reach significance in either TaskSwitch condition (all ps >.10) in the pro saccade task. The picture looked different for the anti saccade task. A significant increase in error rate occurred for the task switch condition only in the no alcohol beverage condition ($F_{(1,22)}$= 5.81, p<.05). Under alcohol however, error rate slightly decreased in task switch trials compared to no task switch trials, an effect just missing significance ($F_{(1,22)}$= 4.27, p=.051). Interestingly, the difference between alcohol conditions was significantly different in the task switch condition ($F_{(1,22)}$= 4.92, p<.05), but not in the no task switch condition ($F_{(1,22)}$= 2.30, p>.10; see A-7.1 for complete ANOVA and means tables).

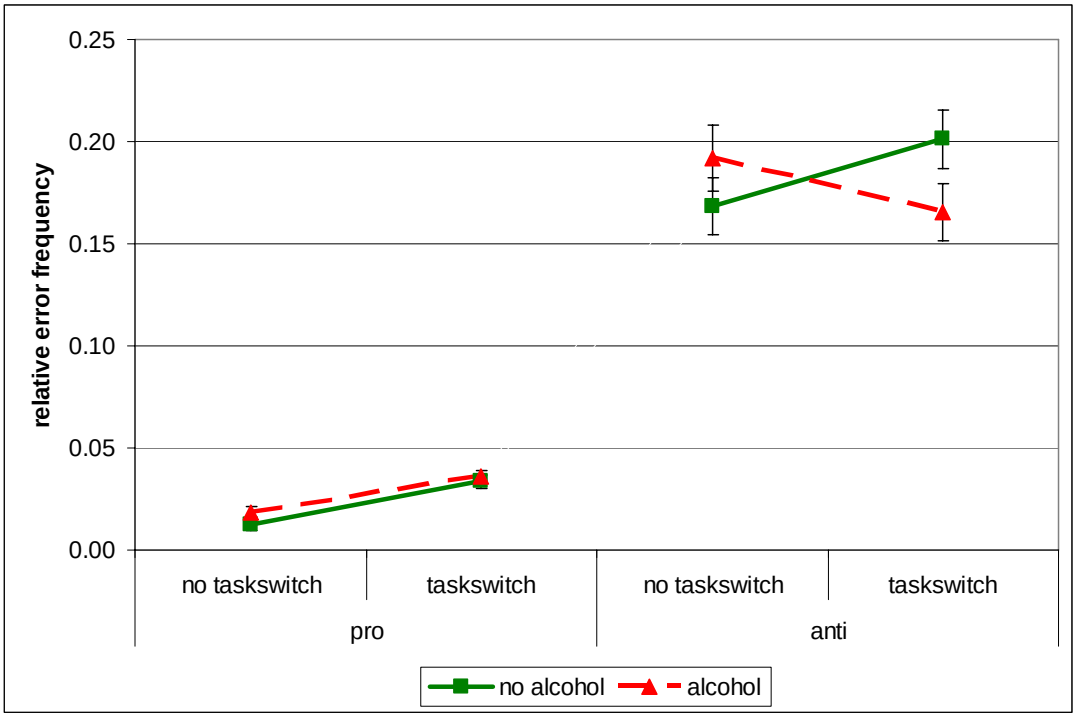

Figure 9-1. Three way interaction between Task, TaskSwitch, and Beverage Condition for error rates.

Analyzing the variability of error rates in a separate ANOVA revealed no significant main effect for Beverage Condition. However, the Task X Beverage Condition ($F_{(1,22)}$= 4.46, p<.05), as well as the Beverage Condition X Alcohol Session ($F_{(1,22)}$= 5.94, p<.05) interactions reached significance. Generally, error rates were more variable in the anti than the pro saccade task, but in the pro saccade task alcohol led to greater variability, whereas in the anti saccade task there was less variability under alcohol. The Beverage Condition X Alcohol Session interaction was again caused by the no alcohol condition, which showed higher error rate variability, if the no alcohol condition was in Session 1 (Alcohol Session 2), whereas the alcohol condition did not show any differences between groups. Tables with

means and standard errors, as well as effects on error rate variability that were not related to alcohol are presented in the appendix (see A-7.2 for complete ANOVA and means tables).

9.5.2 Saccade Latencies

Primary saccade latencies in correct trials showed significant main effects for Task ($F_{(1,22)}$= 131.42, p<.001), TaskSwitch ($F_{(1,22)}$= 28.97, p<.001) and Beverage Condition ($F_{(1,22)}$= 33.50, p<.001; see Figure 9-2).

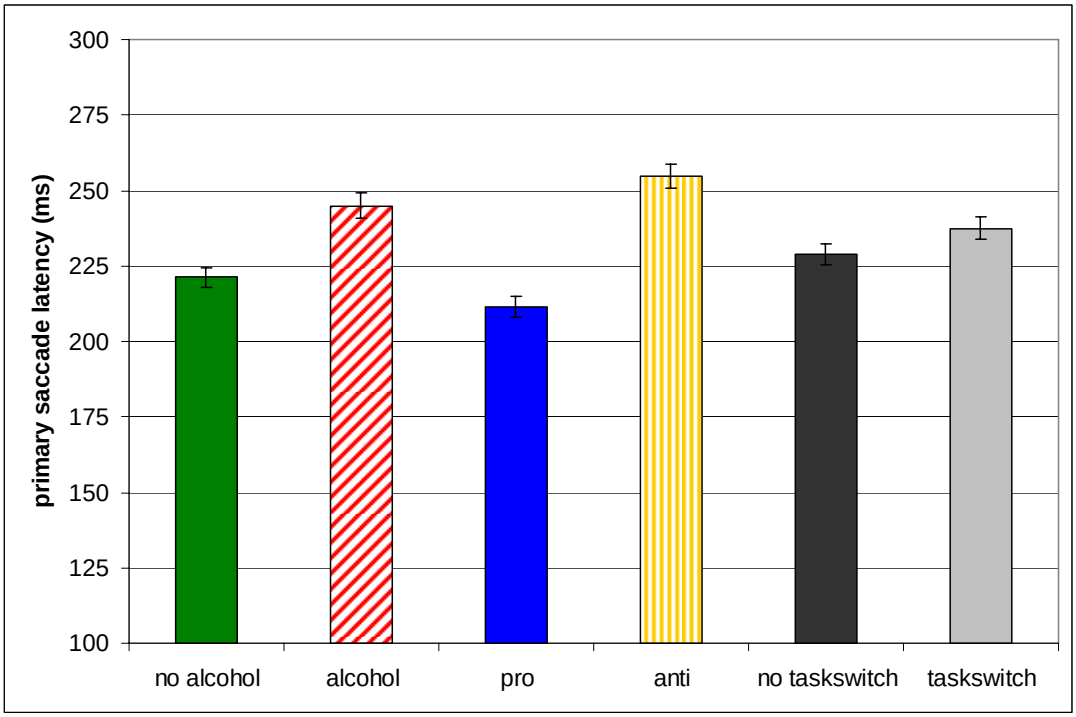

Figure 9-2. Significant main effects of Beverage Condition, Task, and TaskSwitch on primary latencies with correct responses.

These main effects were qualified by interactions, however no further alcohol related effects were found. Additional results on saccade latencies were therefore described in the appendix (see A-7.3 for complete ANOVA and means tables)

Looking at the variability of latencies, a separate ANOVA revealed a significant main effect for Beverage Condition ($F_{(1,22)}$= 5.86, p<.05), with latencies more variable under alcohol, as well as a significant Task X TaskSwitch interaction ($F_{(1,22)}$= 20.83, p<.001). The variability in latencies was not different between tasks in the task switch condition ($F_{(1,22)}$= .37, p>.10), but it was greater in the anti saccade task in the no task switch condition ($F_{(1,22)}$= 13.76, p<.001). In other words the effect of TaskSwitch was more pronounced in the pro ($F_{(1,22)}$=

18.10, $p<.001$) compared to the anti saccade task ($F_{(1,22)}= 1.67$, $p>.10$; see A-7.4 for complete ANOVA and means tables).

9.5.3 Saccade Amplitudes

Saccade amplitudes differed significantly for TaskSwitch ($F_{(1,22)}= 26.36$, $p<.001$) and Beverage Condition ($F_{(1,22)}= 6.39$, $p<.05$), with Task ($F_{(1,22)}= 4.11$, $p=.055$) almost reaching significance. The main effect for alcohol was qualified by a Task X Beverage Condition interaction ($F_{(1,22)}= 6.20$, $p<.05$), due to a significant decrease in saccade amplitude in the no alcohol condition for the anti saccade task ($F_{(1,22)}= 8.91$, $p<.05$), but not in the alcohol condition ($F_{(1,22)}= 1.02$, $p>.10$). The difference between alcohol conditions was not significant for the pro saccade task ($F_{(1,22)}= 2.15$, $p>.10$), but for the anti saccade task ($F_{(1,22)}= 7.40$, $p<.05$).

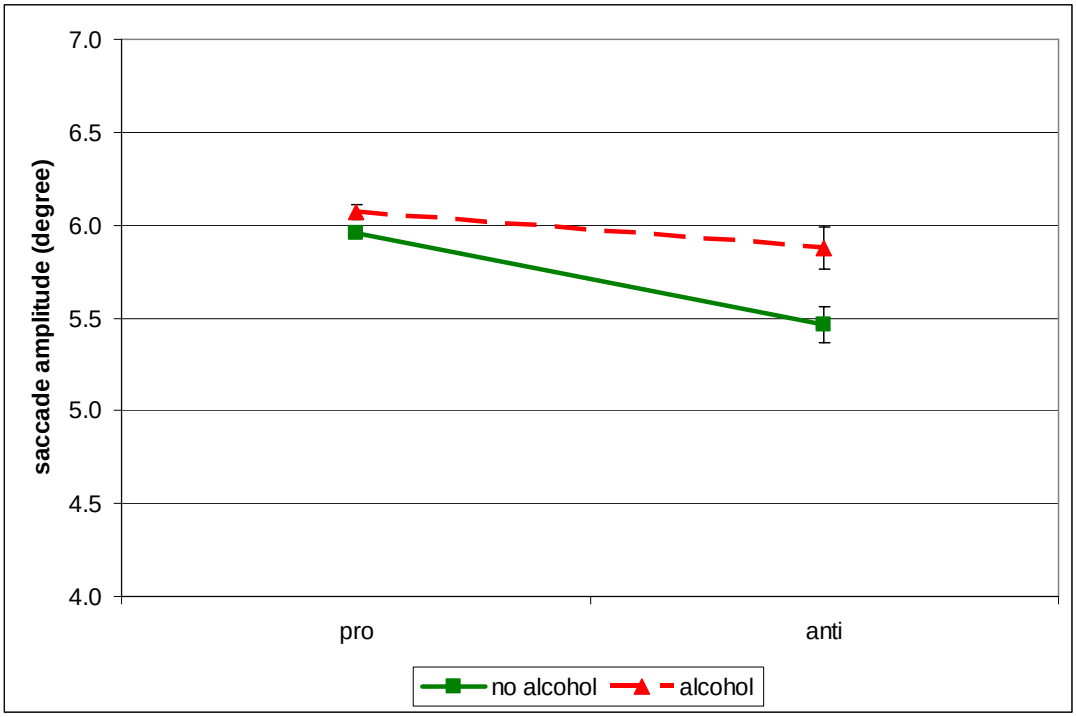

Figure 9-3. Interaction Task X Beverage Condition for primary saccade amplitudes with correct responses.

Other findings regarding saccade amplitudes and saccade amplitude variability were not alcohol related and are presented in the appendix (see A-7.5 and A-7.6 for complete ANOVA and means tables).

9.5.4 Peak Velocities

For the peak velocities the only significant effect was a difference between Beverage Conditions ($F_{(1,22)}$= 8.97, p<.05), with slower peak velocities under alcohol (see A-7.7 for complete ANOVA and means tables). Variability of peak velocities is somewhat higher in the anti saccade task ($F_{(1,22)}$= 4.24, p=.051), but did not vary between Beverage Conditions ($F_{(1,22)}$= .88, p>.10).

9.6 Discussion

The task switching experiment was included in the present thesis, representing a relatively complex cognitive task. Not only does task switching occur permanently in everyday life, but also cognitive control and associated mechanisms of activation and especially inhibition are closely related to – and in parts identical with – mechanisms that are known to be influenced under acute alcohol intoxication (e.g., Glencross, 1990). In addition, by using the pro and anti saccade tasks to induce task switching, findings from these basic paradigms can be corroborated and extended. Accordingly, the discussion is divided into two parts. Part one will focus on findings related to task switching and part two will discuss similarities and differences in results with respect to the basic pro and anti saccade tasks (chapters 5 and 7).

Regarding the effects of task switching on performance, results for the no alcohol session revealed higher error rates in the task switch condition. This finding is in line with results obtained by Cheraskova et al. (2002), Hunt & Klein (2002) as well as Reuter et al. (2006). Response switches also led to higher error rates, replicating findings from Massen (2002) and Fecteau (2004), whereas Reuter et al. (2006) found an effect of response switch only for task repetition trials in the anti saccade task. Latencies in the present experiment were also longer in switch trials, but the difference between task switch conditions was only significant in the pro saccade but not in anti saccade trials. This result adds to the equivocation of findings on effects of task switching on saccade latencies. The same is true for results related to response switches. Response switching increased error rates in the current study in both tasks, independent of the task switch condition. In addition, latencies were longer in response switch trials only, if task switching occurred at the same time. In task repetition trials, latencies were shorter in the response switch condition, compared to response repetition trials. Unfortunately, these findings add to the mixed pattern of results discussed in section 9.2.

Examining the impact of alcohol on switching effects, it became apparent that influences of alcohol were limited. The only interaction between the factors Beverage Condition and either

Task Switch or Response Switch was found for error rates. Performance in the pro saccade task was not modulated by the task switch condition. For both alcohol condition there was an increase in error rates in task switch compared to repetition trials. In the anti saccade task, there was no difference between alcohol conditions in task repetition trials as well, but in task switch trials error rates were lower under alcohol compared to the no alcohol condition. This is a counterintuitive finding and contradicted the initial hypothesis that task switching effects would be more pronounced under alcohol. While the lack of a main effect of Beverage Condition indicates that alcohol did not impair the ability to suppress a reflexive response per se, the effects on task switching performance suggest that some underlying processes are influenced by alcohol intoxication. Reduced task switching effects could be explained in two ways. It might be the case that participants cannot benefit from repetition priming under alcohol and have a problem maintaining a mental task set active in working memory. If so, there should be a significant difference between alcohol conditions in the task repetition condition as well, but this was not the case. Rather, switching from the dominant to the non dominant task was easier under alcohol, not only compared to the no alcohol condition, but also compared to the task repetition condition under alcohol. One explanation to account for this finding could be that additional processing time, available due to longer latencies under alcohol, allowed a more complete activation of a motor program or task set for the non dominant task under alcohol, while at the same time the process of maintaining the motor program or task set active was not impaired by alcohol. This would explain the result pattern found in the anti saccade condition with regard to task switching. That a similar effect was not found for the dominant task (pro saccade task) could result from the fact that the activation of the dominant task set was completed faster and therefore did not benefit from extra processing time.

Regarding the comparison to the blocked version of the pro and anti saccade tasks, findings in the current study replicated critical results. Error rates were not influenced by Beverage Condition in the pro and the anti saccade tasks. Latencies showed an increase under alcohol in both task, again mirroring previous results. For saccade amplitudes, longer saccades were found under alcohol. However, this effect was qualified by a Beverage Condition X Task interaction, which revealed that the difference between alcohol conditions was only significant for the anti saccade task. This is in line with results from Cluster 1, where anti saccades under alcohol also showed larger amplitudes.

In conclusion, the effects of alcohol intoxication on task switching were limited, at least for the intoxication level and task used in the present work. However, the effect that was found seems to be very specific and could, if replicated in further studies, not only help to understand effects of alcohol intoxication on performance in task switching, but also enhance

understanding of which processes work together in what way under task switching conditions in general. In addition, the current experiment replicated critical findings from the pro and anti saccade tasks in Cluster 1.

10 Complex Cognitive Processing II: Reading

As discussed in Chapter 3, reading was selected as a final task to examine the effects of acute alcohol intoxication in a complex cognitive task with ecological validity. Over the last three decades experimental reading research has rapidly expanded, especially due to the advances in eye tracking technology and methodology (Rayner 1998; Radach & Kennedy, 2004). Even though there is an ongoing (and fruitful) debate in the field between advocates of sequential and parallel processing models (Reichle, Rayner & Pollatsek, 2006; Richter, Engbert & Kliegl, 2006; Reilly & Radach, 2006), a large number of basic phenomena are beyond controversy. After discussing relevant theoretical background, hypotheses are generated, in turn the design and results of the present study are explained in detail.

10.1 Theoretical Background

Over the last 30 years, experimental reading research has become a flourishing field. After a period of relative stagnancy, eye movement and reading research gained new popularity, triggered by advances in eye tracking technology and the development of theories on language processing (e.g. Rayner, 1978). During this time, the field has mainly focused on two fundamental areas. The first area examines questions related to the information processing during fixations. Relevant aspects here are the size of the area from which information can be extracted, the type of information that can be extracted within a certain area and the timeline of this processing. The second area focused on eye movement control and related work studies the questions of when and where the eyes move. Within the framework of this thesis, the influence of alcohol intoxication on aspects from both areas was studied (see below). Theoretical considerations that led to the selection of well established effects within each focus area, namely the parafoveal preview effect and the frequency effect are described in subsequent sections, after a brief definition of basic parameters is given first. Finally, an earlier study on the effect of alcohol intoxication on reading is discussed.

10.1.1 Basic Eye Movement Parameters in Experimental Reading Research

Saccades and fixations can be seen as a reflection of spatial and temporal aspects of oculomotor control in reading. Consequentially, a distinction between temporal and spatial parameters is made in experimental reading research. This distinction is in accordance with the theoretical framework introduced for the basic oculomotor paradigms (see Chapter 2.3). Table 10-1 defines the most important temporal and spatial parameters (see Rayner, 1998 or Inhoff & Radach, 1998 for more details).

Table 10-1. Overview of the most important temporal and spatial eye movement paradigms used in reading research (modified after Radach & Kennedy, 2004)

Parameter	Definition
Initial / first fixation duration	Duration of the first fixation within a word, irrespective of whether more fixations follow
Gaze duration	Summed duration of all fixations before leaving the word (within the current pass)
Total reading time	Summed duration of all fixations made on the critical word
Initial landing position, *fixation position*	Position within a word (in characters) where a fixation is located, the empty space between words coded as zero
saccade amplitude *, saccade length, saccade extent*	Distance, in character positions, between the mean position of two successive fixations
launch distance *, launch site*	Distance in characters between the location of the prior fixation and the beginning (or centre) of the current word
skipping rate *, inverse measure: fixation probability*	Relative frequency with which a word is not fixated at least once
Total number of fixations	Sum of all fixations for a word
Total number of gazes	Sum of all gazes for a word
Total number of fixations per gaze, fixation frequency	Mean absolute number of fixations per word, for the current pass (defined as first, second, etc encounter with specified text)
refixation probability/frequency	Relative frequency of making at least one additional fixation before leaving a word
Regression rate/frequency	Relative frequency of making saccades against the reading direction

Spatial parameters are measured in letter units and not in visual angle, because the number of letters traversed by saccades is relatively invariant when the same text is read in different font sizes or at different distances (Morrison, 1983; Morrison & Rayner, 1981; O'Regan, 1983; O'Regan, Levy-Schoen, & Jacobs, 1983; Heller, 1982). Mean progressive saccade amplitudes reported for reading English texts are 7-9 letters and mean fixation duration range between 200 ms and 250 ms for adult readers (Rayner, 1998).

10.1.2 Eye Movement Control: Where and When to Move the Eyes

Regarding the spatial aspects of oculomotor control in reading two processes need to be distinguished (Radach & McConkie, 1998). First, a word has to be selected as a target for the next fixation. The number of targets available is fairly large, but only rarely targets are chosen that are not the currently fixated word (word N), the word proceeding the currently

fixated word (N-1) or one of the two words following the currently fixated word (N+1 and N+2). Second, the fixation position within the selected fixation target has to be determined.

When looking at factors that determine which word to chose as a fixation target, two options have been discussed in the relevant literature. Low level visual factors, such as word length or distance to the current fixation location on the hand (O'Regan, 1990) can be distinguished from high level cognitive factors, for example, word frequency or predictability on the other hand. There is a lively debate on the relative importance of low vs. high level factors on the decision to fixate a word. Kerr (1992) showed that low level factors can very well predict the fixation probability of a word, a finding corroborated by results from Vitu, O'Regan, Inhoff and Topolski (1995) who questioned the rather obvious fact that the likelihood to skip a word during reading increases with decreasing word length.

For high level factors, various studies reported higher fixation probabilities for high frequency compared to low frequency words (Henderson & Ferreira, 1993; Inhoff & Topolski, 1994; Rayner & Fischer, 1996) and lower fixation probabilities (higher skipping rates) for predictable words (Balota, Pollatsek & Rayner, 1985; Rayner & Well, 1996). A meta-analysis by Brysbaert & Vitu (1998) revealed that most of the variance in word skipping can be explained by low level factors and only a small proportion by high level factors (see Brysbaert, Drieghe & Vitu, 2005, for a recent review). However, this should not mislead to a neglect of high level factors. Radach (2003) tries to integrate this by saying that eye movement control is as automatic (low level) as possible and as cognitive (high level) as necessary.

As mentioned earlier, the second aspect that needs to be determined once a word is selected as a fixation target is the location in the word where the fixation should be made. Studies on word recognition have shown that the optimal fixation position for word recognition is the center of the word (O´Regan & Jacobs, 1992; Nazir, Heller & Sussmann, 1992; Brysbaert, Vitu & Schroyens, 1996). This position is called the ***optimal viewing position*** (O'Regan, 1981; 1990; 1992) and several studies demonstrated that the refixation probability is minimal, if a word is fixated at or near this optimal viewing position. The further away the initial fixation in a word lands, the higher the probability of an immediate refixation, resulting in a U-shaped refixation probability curve (McConkie et al., 1989; Vitu, O´Regan & Mittau, 1990; Radach & Kempe, 1993; Rayner, Sereno & Raney, 1996). Looking at initial landing positions in reading experiments, it becomes apparent that landing positions follow a normal distribution. However, the maximum of this normal distribution is not in the word center, but about half way between the beginning of the word and the word center. (McConkie, Kerr, Reddix & Zola, 1989; Rayner, Sereno & Raney, 1996; Radach & Kempe,

1993; Vitu, O'Regan, Inhoff & Topolski, 1995; Radach & McConkie, 1998). This fixation location is called ***preferred viewing position*** (Rayner, 1979, O'Regan, 1990). Main determinants of the preferred landing position are again the low level factors word length and launch distance and to a lesser extent the position on the line and the previous fixation pattern (Radach & Kempe, 1993; McConkie, Kerr & Dyre, 1994; Radach & McConkie, 1998).

In contrast to the spatial parameters, temporal aspects of eye movement control in reading are thought to be primarily influenced by high level cognitive factors. Even though the low level factor word length influences gaze durations (Just & Carpenter, 1980; Rayner et al, 1996), longer gaze durations for longer words can easily be explained by a higher number of refixations with increasing word length (Blanchard, 1985). In addition, this low level factor can be controlled by keeping the length of target words identical. One high level factor that plays an important role in determining temporal aspects of eye movements is word frequency. Word frequency refers to the printed frequency of a word in a text corpus. Several data bases are available to determine word frequencies for different languages, enabling the creation of controlled stimulus material for reading studies (e.g. CELEX, Baayen et al, 1993).

An effect of word frequency on speed and accuracy of word recognition was already established in 1935 by Preston (see Morrison & Ellis, 1995). Readers take longer to recognize low than high frequency words. This finding was replicated in the following decades in a number of studies (e.g., Forster and Chambers, 1973; Rubenstein et al., 1970) and was further corroborated by eye movement research, which revealed longer fixations on low than on high-frequency words (e.g., Rayner, 1977; Just & Carpenter, 1980). These early studies did not control for word length, therefore it could be argued that word length and word frequency are negatively correlated, thereby explaining the effects of word frequency simply by word length (Kliegl, Olson & Davidson, 1982). However, later experiments could show that the word frequency effect can also be found, if the word material is controlled for word length. (Rayner and Duffy, 1986; Inhoff and Rayner, 1986; Schilling et al., 1998; Kliegl et al., 2004; Dambacher et al., 2006).

Other variables that were thought to be confounded with the word frequency effect are age of acquisition (Brown & Watson, 1987; Morrison et al, 1992) and word familiarity (Gernsbacher, 1984). Rayner et al. (2004) demonstrated that all three factors (frequency, familiarity, and age of acquisition) have a similar influence on gaze durations in reading. Finally, word predictability (i.e. how predictable is the next word in the context, often determined using the cloze task) influences temporal parameters during reading (Rayner & Well, 1996; Rayner et al., 2004; Kliegl et al., 2004; 2006), but there is an agreement that

word frequency and predictability contribute independently to word recognition (see Dambacher, Kliegl, Hofman, & Jacobs, 2006, for a recent discussion).

Taken together, the word frequency effect is one of the most robust findings in the reading literature. Word frequency has served as one of the prime indicators of difficulty in lexical access (e.g., Hudson and Bergman, 1985; Monsell et al., 1989) and being able to simulate it is one of the benchmarks for models of word recognition (Grainger and Jacobs, 1996; Jacobs and Grainger, 1994) as well as for models of eye movement control in reading (see Radach, Inhoff & Reilly, 2007, for a detailed discussion).

The manipulation of word frequency in reading experiments can be seen as equivalent to the variations of mental workload in simple or choice reaction time experiments. In a review of the literature on alcohol and human performance Glencross (1990) summarizes that the available evidence "*suggests that the effects of alcohol are greater and performance more adversely affected as the complexity of the information processing increases*" (Glencross, 1990, p.113). The present experiment seeks to explore this possibility for the case of lexical processing in reading. As discussed above, low frequency words require longer processing times, indicating a higher information processing load. Thus, acute alcohol intoxication should interact with word frequency with more pronounced effects under alcohol.

10.1.3 Information Processing During Fixations

During reading, as during other visual tasks, information can only be extracted from a certain part of the visual field. This area is termed ***functional visual field*** or ***perceptual span***. It defines an area surrounding the visual axis, within which information of a certain quality is being processed. In languages reading from left to right the perceptual span for the detection of word boundaries extents 4 letters to the left and 14-15 letters to the right (McConkie & Rayner, 1975, 1976). Blanks between words can be used to determine word boundaries within this range[13] (McConkie & Rayner, 1975; Rayner, 1986; Pollatsek & Rayner, 1982). However, the perceptual span for letter identification extents about 4 letters to the left and only 8 to 10 letters to the right.

Due to these constraints, the eyes need to move across a line of text during reading. This is accomplished with a series of saccades and fixations, rather than a constant speed. During

[13] Saccade latencies are significantly reduced when reading texts without blank spaces (Rayner et al, 1998), but studies in languages with other alphabetic systems have shown word based reading patterns even in the absence of blank spaces between words (Reilly & Radach, 2003).

fixations information is extracted from the text and saccades serve to bring new information into the center of the visual field, making its content available for processing during the following fixation.

However, when fixating on a certain word, information is not only extracted form this word N, but also from the following word N+1[14]. The preview benefit is defined as the amount of time that readers look at a word when they have been given a full preview of it, subtracted from the amount of time that readers look at the word when they have had no preview[15]. This typically results in increased gaze duration in conditions with an illegal preview. To manipulate the amount of preview available, the boundary paradigm (McConkie & Rayner, 1975; Rayner, 1975) has traditionally been used. In the boundary paradigm an invisible boundary is located in the text, usually in front of the blank space preceding the target word. Once the gaze crosses this boundary, a display change is triggered. By manipulating the information at the location of the target word before and after the display change, the amount of parafoveal preview can be varied systematically. As the display change is taking place during the saccade, participants are usually unaware of the change. The boundary paradigm thereby allows investigating the extent to which parafoveal information is acquired and what factors (e.g. lexical, sentential) influence parafoveal processing. Virtually all reading researchers agree that low-level information can be gained from the word to the right of a fixation; the precise degree of cognitive benefit from the word to the right of fixation is still debated.

Within the recent discussion between advocates of serial attention shift (SAS) and processing gradient (PG) models, Inhoff, Eiter, and Radach (2005) demonstrated that linguistic processing of consecutive words can overlap in time. In their experiment Inhoff et al. used the boundary paradigm to allow parafoveal preview on word N+1 either for the first 140 ms of the fixation of word N and then mask word N+1 again until a saccade was made into word N+1, or word N+1 was masked for the first 140ms of the fixation on word N and unmasked thereafter. Their results showed that information of the parafoveal word is already extracted

[14] Not only information from word N and N+1 are processed during a fixation on word N. There are also spillover effects of processing from word N-1 (Rayner & Duffy, 1986; Rayner, Sereno, et al., 1989). In addition, there is an ongoing discussion, whether Information from word N+2 can also be processed while fixating word N (Rayner, Juhasz, Brown, 2007; Kliegl, Risse & Laubrock, in press; Radach & Glover, in preparation). This is a theoretically interesting debate, but clearly beyond the scope of this thesis.

[15] Note that this effect is termed parafoveal preview *benefit* in most of the literature. However, as this effect stems from taking away information that is available under normal circumstances, the neutral term *effect* is used throughout this thesis.

during the first 140 ms of the fixation of the preceding word. Overall, the result pattern led to the conclusion that linguistic processing is neither strictly serial nor strictly parallel, but that processing can overlap[16] (Inhoff, Radach & Eiter, 2006).

More important in the context of this thesis is the finding that the parafoveal preview effect is also influenced by the ongoing foveal processing. Several studies found that if the foveal processing load is high, the amount of preprocessing from the parafoveal word decreases, evident in a smaller preview effect (Henderson & Ferreira, 1990; Inhoff, 1989; Kennison, & Clifton, 1995; Rayner, 1986; Schroyens, Vitu, Brysbaert, & d'Ydewalle, 1999). This is particularly interesting for the current study, as alcohol intoxication reduces cortical activity (Krull et al., 1994; Liu et al, 2000), thereby possibly restraining cognitive processing capacities. This in turn, might lead to a relative increase in foveal processing load, reducing the parafoveal preview effect.

A related issue that is important in the context of acute alcohol intoxication is a phenomenon called ***tunnel vision***. Studies examining driving performance under alcohol have shown that intoxicated participants detected fewer critical targets in a driving simulation display, particularly when targets appeared in the periphery (e.g., Buikhuisen & Jongman, 1972). According to the tunnel vision hypothesis, alcohol degrades performance in the periphery while preserving central vision (e.g. Mills, Spruill, Kanne, Parkman & Zhang, 2001). Expressed in terms of a reading experiment, this would suggest a decreased parafoveal preview effect due to reduced information intake from areas beyond the fovea.

A somewhat conflicting account could be derived from the ***alcohol myopia*** hypothesis. Even though this hypothesis also states that alcohol restricts cognitive processing, intoxication is assumed to lead to a focus on the most salient of the available stimuli in a particular context (Steele & Josephs, 1990). Although Steele and Josephs claim that alcohol effects are "stemming from alcohol's general impairment of perception and thought" (Steele & Josephs, 1990, p.922), the alcohol myopia hypothesis has not been tested in perception experiments, but rather in social psychological studies with an emphasis on aggression and other emotional behavior. However, a letter mask presented in the parafovea in a reading experiment using the boundary paradigm could serve as a very salient stimulus. According to

[16] The discussion regarding serial attention shift models and processing gradient models for reading is ongoing. No final judgment is possible as to which class of models will prevail. However, for the present thesis this is not critical, as the effect of alcohol intoxication on the preview effect can yield interesting information independent of one's theoretical bias.

the alcohol myopia hypothesis, this unusual stimulus should attract most of the processing capacities, thereby resulting in a larger parafoveal preview effect under alcohol.

Before developing and summarizing hypotheses regarding the influence of alcohol intoxication on reading behavior and the parafoveal preview effect, the next section reviews results of an earlier study on reading under alcohol intoxication.

10.1.4 Alcohol and Reading

An extensive literature search revealed that there has been only one attempt to examine the influence of alcohol intoxication on reading. Watten and Lie (1997) studied eye movement behavior during reading under three different conditions, a 0 mg% placebo control condition and two alcohol conditions (50 mg% and 100 mg%). 18 participants were tested under all alcohol conditions with the order of alcohol conditions counterbalanced. After a 1 hour drinking period, participants read a text that was part of a short story on a computer screen with a viewing distance of 40 cm. Eye movements were recorded using a 100Hz infrared corneal reflection system.

Text lines were 13 cm long and viewing time was restricted to 20 seconds per passage. Unfortunately, there no specifications were given about the number of lines per passage, the letter size in visual angle or even the total number of passages read. The word material was not controlled for any of the factors that, as discussed above, influence reading behavior even under no alcohol conditions. In addition, the parameters used as dependent variables for analysis were fairly crude measures, compared to standard parameters used in experimental reading research (see Table 10-1). Parameters reported were *number of fixations*, representing the arithmetic sum of fixations per 100 words; *fixation duration*, measured in milliseconds; *reading speed*, not defined; *saccadic length*, which was defined as number of words covered during one saccade and finally the *number of regressions*, as the arithmetic sum of movements to the left per 100 words.

Results showed that participants made significantly more fixations per 100 words read in the alcohol conditions compared to the placebo condition. Fixation durations increased with higher blood alcohol levels, but overall reading speed did not differ significantly between conditions, even though a slight reduction was found under alcohol. Regarding saccadic length and number of regressions no significant differences were found between conditions.

It is unclear, how a significantly increased number of fixations together with a significantly higher number of fixations led to reading times that were not different from each other. The

authors address this apparent difficulty by claiming that a high variability in reading speed is responsible for this result. However, in another part of their discussion they state that all participants in the study were very skilled readers showing above average reading speed.

Taken together, the study by Watten & Lie (1999) demonstrated that there are effects of alcohol intoxication on reading behavior. However, due to severe methodological shortcomings the reported results could not provide a complete picture of possible alcohol induced impairments of reading behavior. The current experiment was designed to overcome some of these restrictions and draw a more precise picture of the effects of alcohol on reading. Furthermore, by parsing the nature and extent of alcohol's effects on parafoveal processing, this methodology also introduces a precise, on-line means for examining mechanisms that may underlie important phenomena like alcohol-induced *tunnel vision* and *alcohol myopia* (see section 10.1.3).

10.2 Hypotheses

Given the findings from the experiments in Cluster 1 a general slowing was expected to be found for temporal parameters. However, there was no reason to expect an influence of alcohol on parameters like saccade amplitude and fixation probability.

The more interesting question is, whether the expected slowing is due to impairments of the sensory-motor system or related to linguistic processing difficulties. The fact that longer latencies were found under alcohol in the basic oculomotor paradigms that require no lexical processing, suggests that at least a portion of the general slowing is related to sensory-motor impairments. However, linguistic processing was also expected to be impaired. Therefore, the effect of alcohol was assumed to be more pronounced for low frequency words, showing statistically in an interaction between Beverage Condition and word frequency.

Regarding effects of alcohol intoxication on parafoveal processing, again different scenarios were possible. Taking the theoretical considerations of section 10.1.3 into account, alcohol was hypothesized to lead to a reduction of total cognitive processing capacity. Given the fact that the focus of processing is usually on the current word, this should leave less resources for parafoveal information from adjacent words. Therefore, a decreased preview effect could be expected.

Interestingly, this pattern of results would also follow from the assumption that alcohol intoxication directly affects perception in the visual periphery, making parafoveal letters less

visible (reducing the perceptual span). Distinguishing this perceptual account from the cognitive capacity hypothesis discussed above is beyond the scope of the current study.

The hypothesis of a reduced preview effect can be contrasted, however, with an alternative prediction based on the notion of alcohol myopia (see section 10.1.3). If perceptual alcohol myopia results in focusing on the unfamiliar letter string in the parafovea as the most salient stimulus, this could lead to an increased parafoveal preview effect.

10.3 Design

The reading material consisted of 192 single line sentences. Participants read 96 sentences in each of two sessions. A 7 or 8 letter noun was used as a target word in each sentence. Half of the target words were low frequency words (<3 per million) the other half were high frequency words (>4 per million). Table 10-2 gives an overview of controlled word properties.

Table 10-2. Means (standard deviations) for controlled target word properties. Differences between frequency groups were not significant in any variable (all *p*s>.10).

	number of letters	word frequency	number of syllables	number of morphological components
low frequency	7.49 (0.5)	0.82 (0.86)	2.53 (0.5)	0.70 (0.71)
high frequency	7.48 (0.5)	44.05 (46.86)	2.42 (0.5)	0.53 (0.73)

Within the two sessions, sentences were divided into two blocks of 48 trials each, with BrAC testing between blocks. An invisible boundary was implemented in all sentences at the beginning of the blank space preceding the target word. In half of all trials the target word was masked with a visually dissimilar letter mask, preserving the first letter, before crossing the boundary. Half of the masked target words were high frequency words, the other half low frequency words.

Sentences were presented using Courier New font in size 12 pt (equivalent to 10 pixels per letter) on a 22-inch monitor, using a screen resolution of 1024*768 and a refresh rate of 160Hz. Together with a viewing distance of 80cm this resulted in a visual angle of 0.33 degrees visual angle per letter, which is a standard size in reading research. Participants were instructed to read the sentences once in their normal reading speed, so that they understand

the basic content and were able to answer questions. Comprehension questions regarding the content of the last sentences were asked at random intervals, averaging to 1 question every 6 sentences. Participants responded orally and answers were noted by the experimenter.

A block started with the calibration and validation of the camera system (with thresholds for recalibration being a mean deviation of more than 0.3 degrees or a maximal deviation of more than 0.5 degrees for one of the four calibration points). A trial started with a drift correction target, located two letter positions to the left of the sentence beginning. After a successful drift correction the sentence appeared on the screen. When finished reading, participants pressed a button on the game pad, which triggered the question or started the next trial. The camera system was recalibrated after each question, as participants tend to move during oral answers. Depending on the reading speed of the individual, a reading block took about 8-10 minutes

10.4 Data Analysis

Raw data of the eye tracking system were edited using custom build software. In a first step, output files were converted into xml format. In a second step, word based dependent variables were created. During this procedure data were also inspected visually for noticeable problems.

In a next step, data were imported into SPSS where further data reduction took place. Only the controlled target word material was kept in the analysis matrix. Fixations with durations shorter than 60 ms or longer than 80 0ms as well as cases with saccade amplitudes > 25 letters were excluded. Note that this only excludes single fixations and not the whole word from analysis. This analysis included 5,529 cases of target word fixations within the first pass reading, which corresponded to a 90% fixation rate on target words.

An initial inspection revealed that there were no differences regarding the between subject factor Alcohol Session (i.e., if alcohol was administered in session one or two). Therefore this factor was dropped in subsequent analyses. Statistical analyses were performed using 2 x 2 x 2 repeated measures ANOVAs with the factors Beverage Condition (no alcohol vs. alcohol), Preview (legal vs. illegal), and Frequency (low vs. high word frequency).

10.5 Results

Results are presented separately for spatial and temporal parameters, followed by the presentation of results for fixation frequency measures.

10.5.1 Spatial Parameters

Regarding the spatial parameters, there were no differences between alcohol conditions for initial landing position ($F_{(1,31)}$= .770, p>.10; Figure 10-1; see A-8.1), saccade amplitude ($F_{(1,31)}$= .378, p>.10; see A-8.4) or launch distance ($F_{(1,31)}$= .046, p>.10; see A-8.5). Neither were there any interactions between Beverage Condition and other factors (all ps>.10). An additional analysis of initial landing positions was carried out for different launch distances, to examine the possibility that the preview effect was only affected differentially by alcohol, if the previous fixation was close to the target word. Therefore, cases were divided into near and far launch distances per participant.

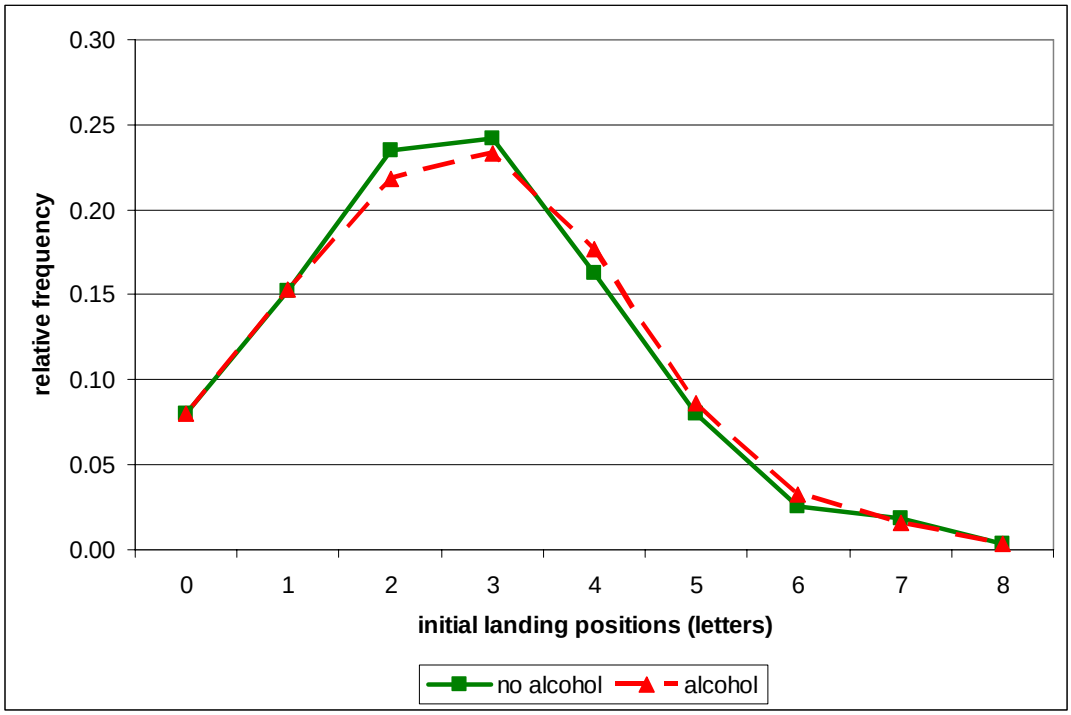

Figure 10-1. Initial landing position distribution in letters for initial target word fixations under both alcohol conditions.

Figure 10-2 illustrates the results that launch distance significantly influenced the initial landing position in target words ($F_{(1,31)}$= 279.99, p<.001), but this effect did not differ between alcohol conditions ($F_{(1,31)}$= 1.62, p>.10). More importantly, there was no interaction

between Launch Distance, Beverage Condition and Preview ($F_{(1,31)}$= .513, p>.10), showing that even for near launch distances, alcohol did not modulate the preview effect (see A-8.2 for complete ANOVA and means tables). The same pattern of results was found for an analysis of initial landing positions in relation to gaze durations on the word N-1 (see A-8.3).

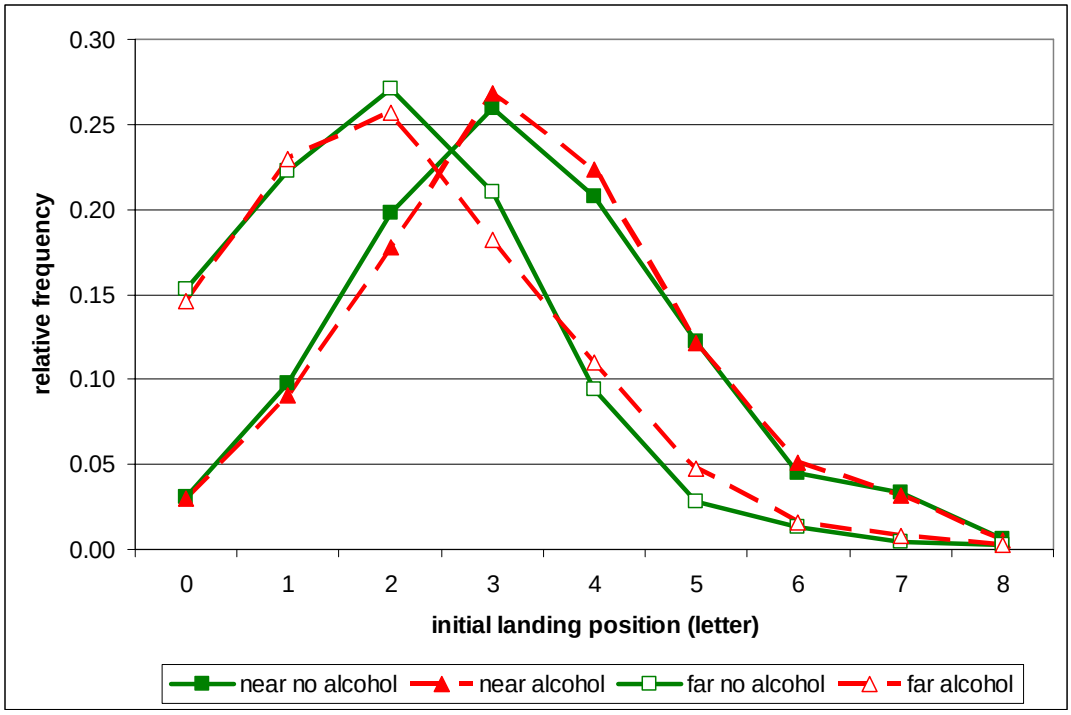

Figure 10-2. Initial landing position for near and far launch positions in both alcohol conditions.

10.5.2 Temporal Parameters

Initial fixation durations were 18 ms longer in the alcohol condition compared with the no alcohol condition, a statistically significant difference ($F_{(1,31)}$= 10.143, p<.05). Furthermore the significant main effects for Preview ($F_{(1,31)}$= 32.807, p<.001) and Frequency ($F_{(1,31)}$= 27.792, p<.05) confirm that the intended manipulations were successful. Target words with illegal preview were fixated 25 ms longer than those with a legal preview and low frequency words were fixated 16ms longer than high frequency words. Figure 10-3 summarizes the results for initial fixation durations.

Interestingly, there were no significant interaction between Beverage Condition and any other factor (all ps>.10; see A-8.6 for complete ANOVA and means tables).

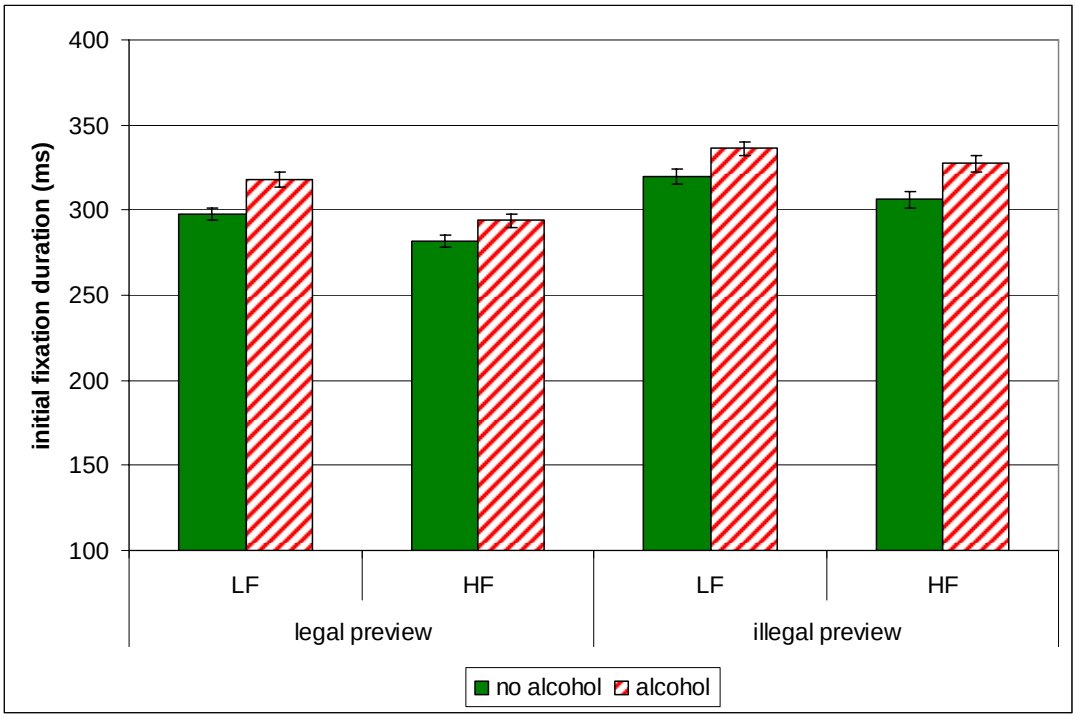

Figure 10-3. Mean initial fixation durations on target words by Beverage Condition (no alcohol vs. alcohol), Preview (legal preview vs. illegal preview) and Frequency (low frequency [LF] vs. high frequency [HF] words).

Looking at gaze durations (Figure 10-4), there was no significant difference between Beverage Conditions ($F_{(1,31)}$= 2.873, p>.10), even though averaged across all conditions, gaze durations were 15 ms longer under alcohol. As for initial fixation durations, Preview ($F_{(1,31)}$= 34.299, p<.001) and Frequency ($F_{(1,31)}$= 66.154, p<.001) showed highly significant differences in the expected direction (i.e. longer under illegal preview and for low frequency words), but again there were no interactions between Beverage Condition and any other factor (all ps>.10; see A-8.7 for complete ANOVA and means tables).

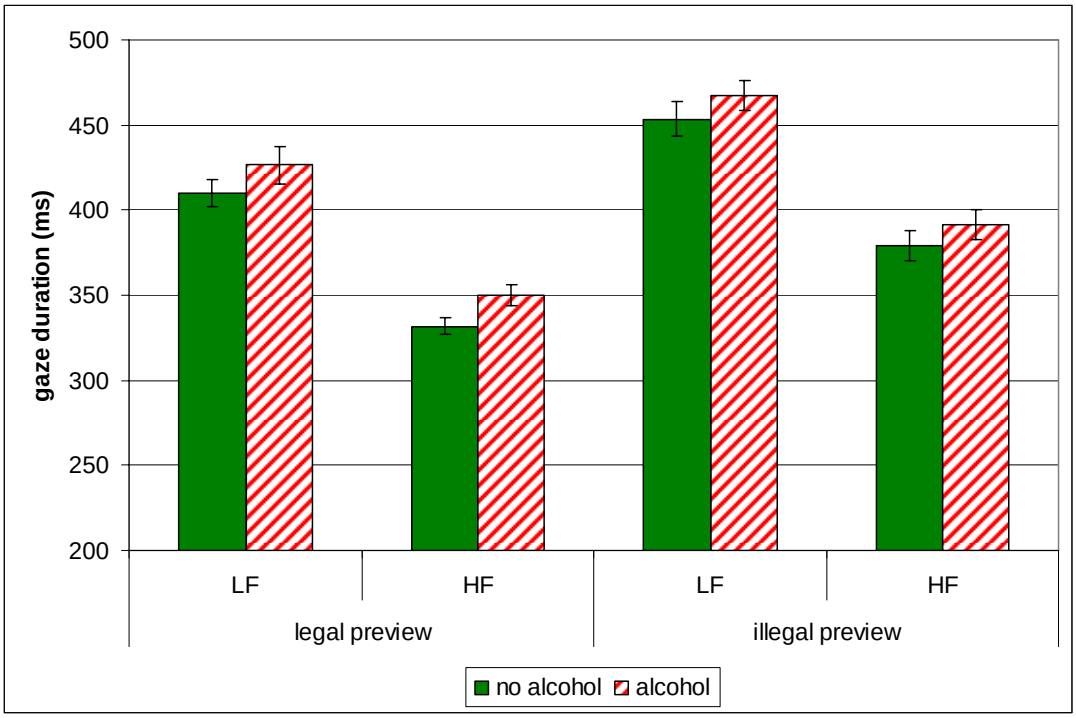

Figure 10-4. Mean gaze durations on target words by Beverage Condition (no alcohol vs. alcohol), Preview (legal preview vs. illegal preview) and Frequency (low frequency [LF] vs. high frequency [HF] words).

The pattern of results for total reading times of the target words was identical with that for gaze durations (Figure 10-5). No significant difference between Beverage Conditions ($F_{(1,31)}$= .476, p>.10), but significant main effects for Preview ($F_{(1,31)}$= 64.954, p<.001) and Frequency ($F_{(1,31)}$= 61.093, p<.001) were found. Again there were no interactions between Beverage Condition and any other factor (all ps>.10). Table 10-3 gives an overview of the results on temporal parameters (see A-8.9 for complete ANOVA and means tables).

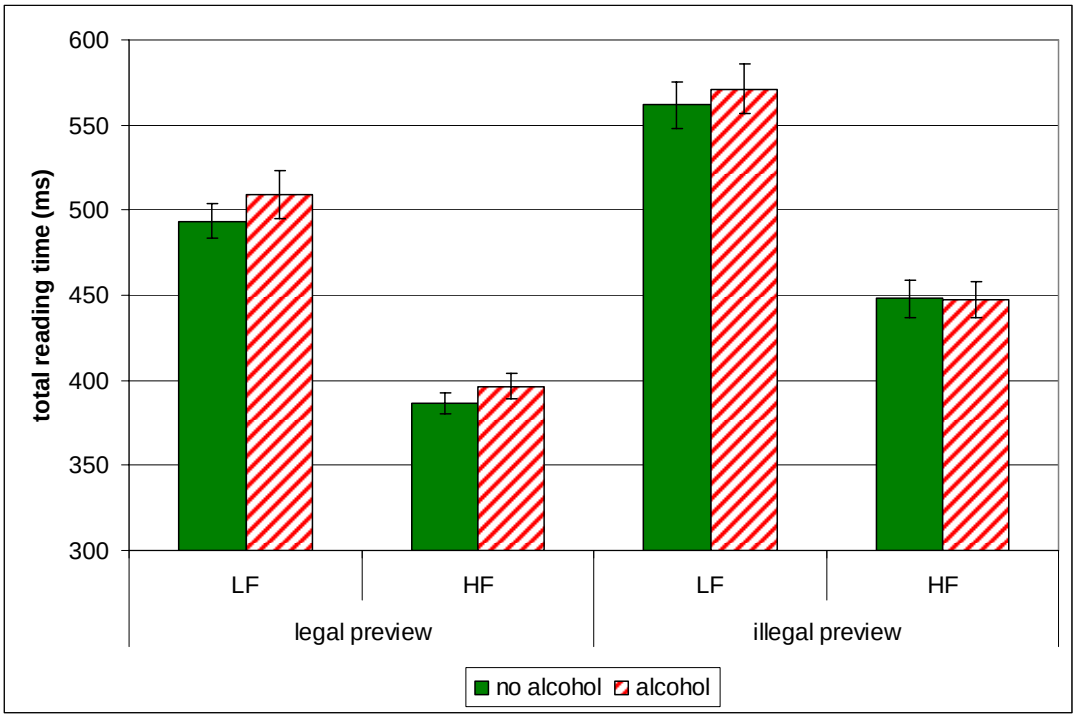

Figure 10-5. Mean total reading times for target words by Beverage Condition (no alcohol vs. alcohol), Preview (legal preview vs. illegal preview) and Frequency (low frequency [LF] vs. high frequency [HF] words).

The obtained pattern with alcohol related differences in initial fixation durations, but not gaze duration, could stem from two sources. One possibility was that refixation durations were shorter under alcohol. To examine this option, single fixation durations (SF), first of two (1 of 2) and second of two (2 of 2) fixation durations were calculated (see section 10.6 for more details), with the latter being identical with refixation durations. Looking at Figure 10-6, it becomes apparent that all fixation duration measures showed higher values in the alcohol condition. As there were not enough cases using the original ANOVA for the analysis of refixation durations, cases were collapsed across frequencies for the subsequent analyses. All differences between alcohol conditions were statistically reliable (*SF*: $F_{(1,31)}$= 6.28, p<.05; *1of2*: $F_{(1,31)}$= 7.46, p<.05; *2of2*: $F_{(1,31)}$= 4.20, p<.05). Table 10-3 presents an overview of the key temporal parameters (see A-8.10, A-8.11 and A-8.12 for complete ANOVA and means tables for SF, 1of2 and 2of2).

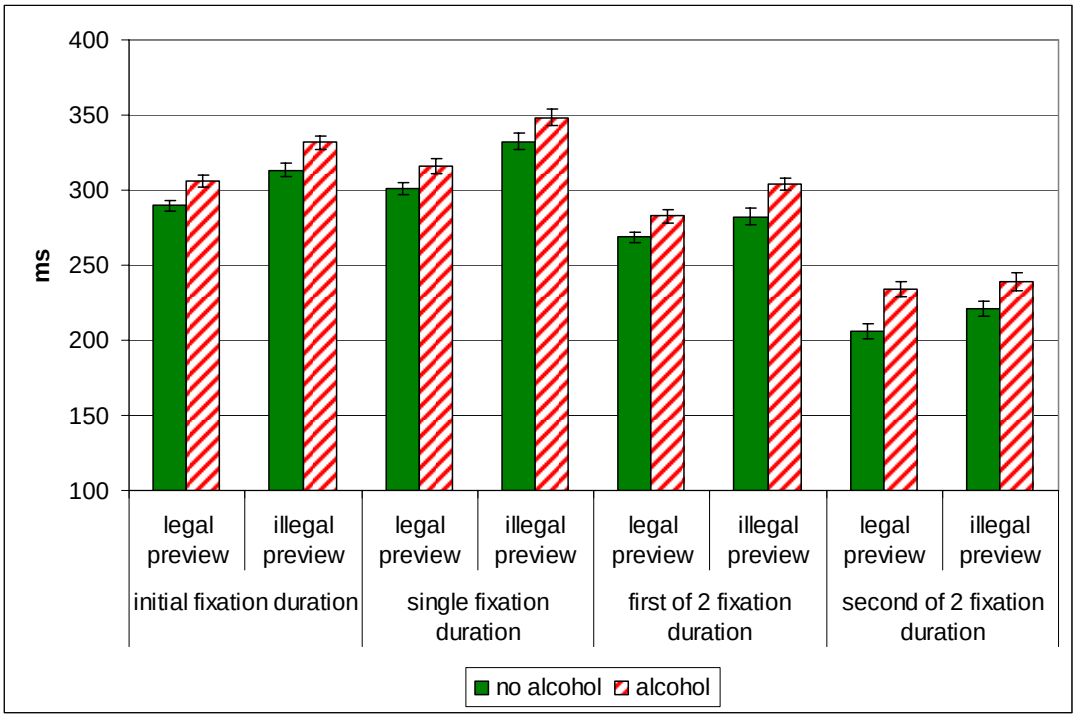

Figure 10-6. Initial, single, first of two and second of two fixation durations for no alcohol and alcohol conditions. Note that cases were collapsed across frequency conditions to obtain enough cases for the analysis of refixation durations (=second of 2 fixation durations)

Table 10-3. Overview of temporal parameters for target words under all factor combinations.

		initial fixation duration		gaze duration		total reading time	
		no alcohol	***alcohol***	***no alcohol***	***alcohol***	***no alcohol***	***alcohol***
legal preview	**HF**	282 (41)	294 (47)	332 (54)	350 (66)	387 (69)	396 (83)
	LF	298 (41)	318 (46)	410 (90)	427 (124)	494 (117)	509 (164)
illegal preview	**HF**	306 (56)	327 (58)	379 (102)	392 (99)	448 (127)	447 (117)
	LF	320 (51)	336 (48)	453 (115)	467 (101)	562 (154)	571 (165)

10.5.3 Fixation Frequency Measures

In this section, results for frequency measures including the number of fixations, refixation and skipping rates are presented.

A second possible explanation for the lack of difference between alcohol conditions in gaze durations (see above) was that fewer fixations were made under alcohol. Indeed, the number of fixations made on target words was lower under alcohol ($F_{(1,31)}$= 5.338, p<.05). There were also effects of Preview ($F_{(1,31)}$= 22.201, p<.001) and Frequency ($F_{(1,31)}$= 54.924, p<.001), with fewer fixations in the legal preview condition and for high frequency words (see A-8.13 for complete ANOVA and means tables).

The number of fixations per gaze for the first gaze, showed a trend towards fewer fixations under alcohol ($F_{(1,31)}$= 3.335, p<.077). Preview ($F_{(1,31)}$= 11.311, p<.05) and Frequency ($F_{(1,31)}$= 56.695, p<.001) again showed significant results in the expected direction with more fixations per gaze under illegal preview and for low frequency words. No interactions between Beverage Condition and any other factor reached significance (all ps>.10; see A-8.14 for complete ANOVA and means tables). Table 10-4 summarizes results on fixation and gaze frequencies.

Table 10-4. Target word fixation and gaze frequencies for all factor combinations.

		total number of fixations		**total number of fixations pre gaze**		**refixation frequency**		**skipping rate**	
		no alcohol	***alcohol***	***no alcohol***	***alcohol***	***no alcohol***	***alcohol***	***no alcohol***	***alcohol***
legal preview	**HF**	1.49 (0.27)	1.43 (0.24)	1.25 (0.17)	1.25 (0.17)	0.22 (0.15)	0.23 (0.15)	0.03 (0.04)	0.06 (0.08)
	LF	1.81 (0.41)	1.73 (0.44)	1.48 (0.25)	1.43 (0.29)	0.38 (0.16)	0.36 (0.22)	0.04 (0.04)	0.04 (0.06)
illegal preview	**HF**	1.62 (0.43)	1.49 (0.31)	1.32 (0.25)	1.26 (0.19)	0.29 (0.21)	0.25 (0.17)	0.03 (0.04)	0.05 (0.07)
	LF	1.97 (0.54)	1.86 (0.48)	1.57 (0.33)	1.50 (0.26)	0.43 (0.22)	0.39 (0.18)	0.03 (0.07)	0.06 (0.08)

Refixation frequencies (Figure 10-7) did not differ between alcohol conditions ($F_{(1,31)}$= 1.771, p>.10). Once more, significant main effects for Preview ($F_{(1,31)}$= 8.584, p<.05) and Frequency ($F_{(1,31)}$= 66.873, p<.001) were obtained. Refixations were more likely under the illegal preview condition and for low frequency words. No interactions between Beverage Condition and any other factor reached significance (all ps>.10; see A-8.15 for complete ANOVA and means tables). In addition, skipping rates for target words were not affected by Beverage Condition or any other factor (all ps>.10, see A-8.16).

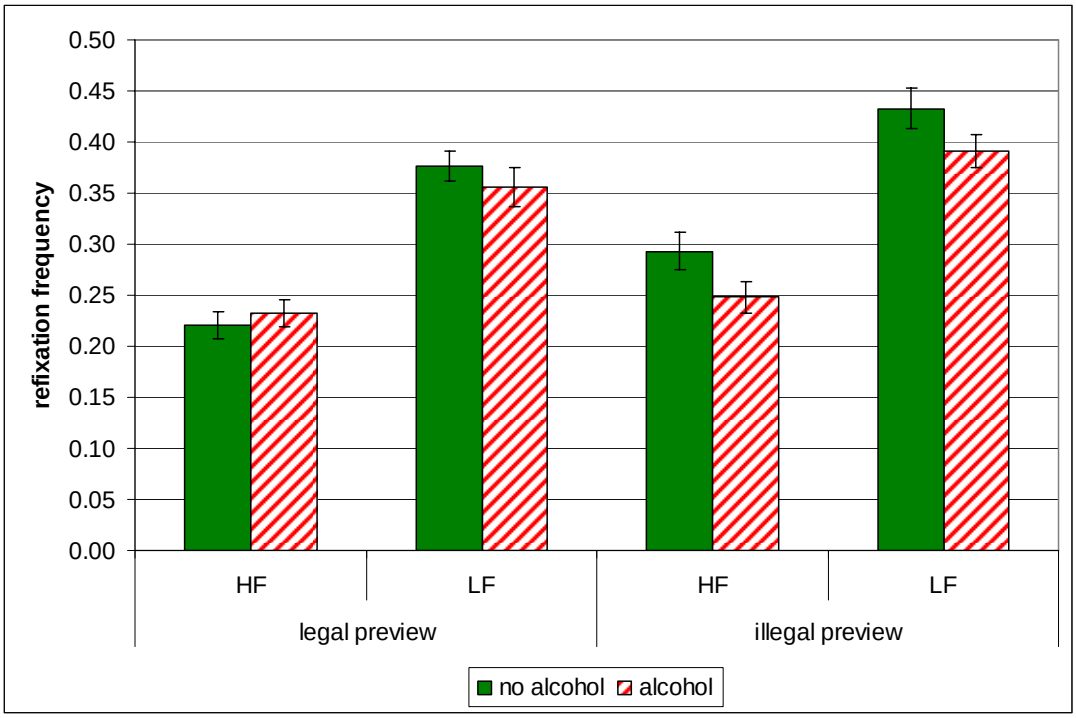

Figure 10-7. Refixation frequencies for high (HF) and low (LF) frequency target words under legal and illegal preview for different alcohol conditions.

Finally, there were fewer regressions leaving the target word in the alcohol condition ($F_{(1,31)}$= 9.67, p<.05, Figure 10-8; see A-8.17). Additional analyses showed that this pattern held for regressions that were initiated from initial landing positions at the beginning of the word (including the blank space and the first two letters) as well as for those initiated from the remaining letters of the word. Initial landing positions at the target word beginning led to higher regression rates ($F_{(1,31)}$= 11.33, p<.05, Figure 10-9). The interaction between Beverage Condition and Initial Landing Position did not reach significance ($F_{(1,31)}$= 3.34, p=.077), but there was a tendency to regress more often under alcohol, if landing at the beginning of the target word (see A-8.18 for complete ANOVA and means tables) .

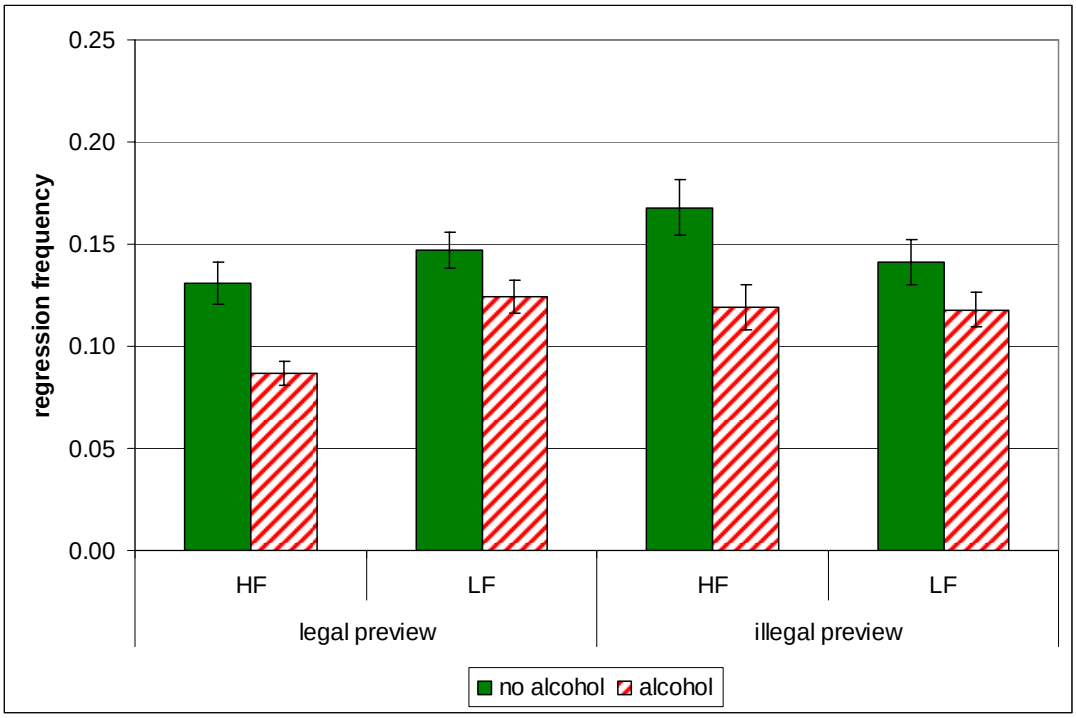

Figure 10-8. Relative frequencies of regressions leaving the target word. Note that 84% of these regressions were directed to word N-1 (the word immediately preceding the target word).

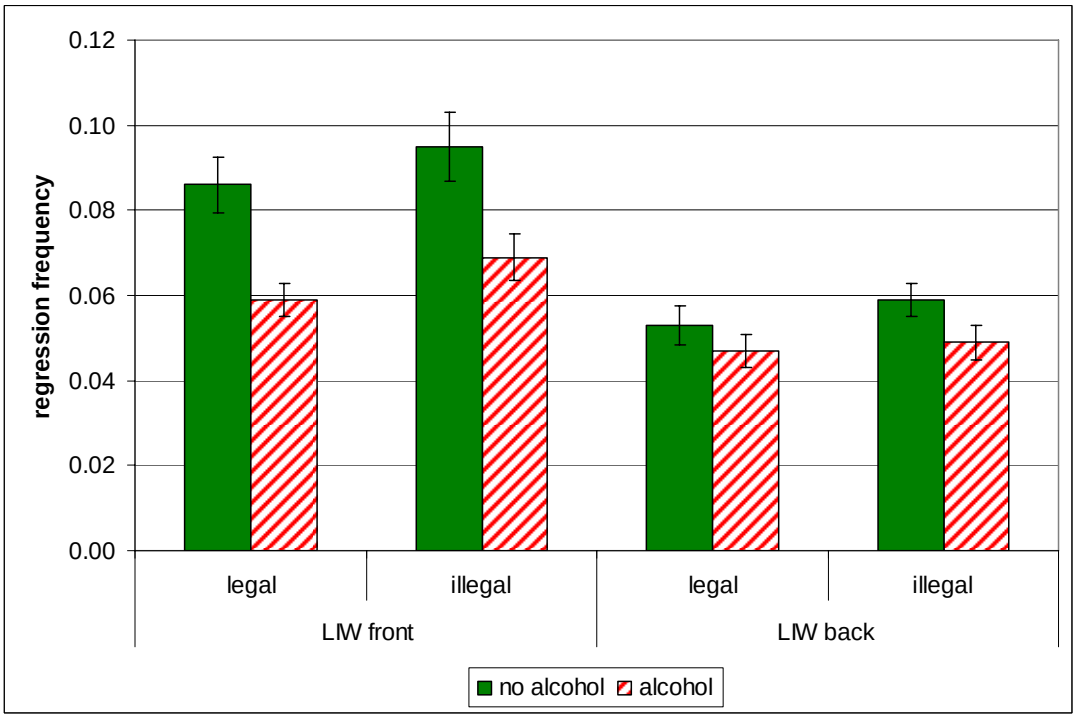

Figure 10-9. Relative frequencies of regressions leaving the target word from different landing position in the target word. Note that cases were collapsed across frequency conditions.

10.6 Discussion

Reading is an everyday task that is performed by almost everybody on a daily basis. There has been only one attempt to examine effects of alcohol on reading performance, which unfortunately suffered from severe methodological shortcomings (see section 10.1.4). Within this thesis, reading was selected not only as an ecologically valid task, but also because it allows studying complex cognitive processes in a relatively simple perceptual setting (see section 3.2.2). The results of the reading experiment showed a very specific, but moderate impact of alcohol on reading performance.

Spatial parameters were not influenced by alcohol, but showed typical effects for the preview and frequency modulations. The lack of alcohol effects on saccade amplitudes in reading is in line with findings from the double step paradigm (see section 6.4.2), when analyzes with respect to inter stimulus intervals. Processing in that paradigm, as well as in reading, is related to the automated level of oculomotor control and neither tasks showed an effect of alcohol on saccade amplitudes.

The results for the temporal parameters yielded more interesting results. Initial fixation durations were longer under alcohol. However, this effect did not carry over to gaze durations and total reading times, which did not differ between alcohol conditions. There are at least two possibilities to explain this pattern of results. First, refixation durations could be shorter under alcohol and compensate the longer fixation duration. To examine this possibility, fixation durations were also calculated for single fixations (cases in which a word received exactly one fixation), the first of two (the first fixation of cases, in which a word received exactly two fixations) and the second of two (the second fixation of cases, in which a word received exactly two fixations). Kliegl et al. (1982) found that the first and the second of two fixations are shorter than fixation durations in single fixation cases. This finding has been replicated numerous times in the literature and was also present. More importantly, refixation durations (=second of two fixation durations) were also longer under alcohol. Therefore, the refixation duration could not account for the lack of difference between alcohol conditions in gaze duration and total reading times. In addition, the consistent increase in fixation durations over all conditions once again points towards a general slowing of the oculomotor system due to alcohol intoxication.

The second possible explanation to account for the pattern of results obtained for fixation and gaze durations is that alcohol intoxication led to a smaller number of fixations. Indeed, the data showed that under alcohol fewer fixations were made. The apparent tradeoff between fixation duration and number of fixations is in harmony with an early study by Moskowitz,

Ziedman & Sharma (1976), reporting a similar pattern of effects in a visual scanning paradigm used to analyze this component of driving skill.

In reading, the decision about where to go with the next saccade must be made relatively early in each ongoing fixation. As it takes a substantial amount of time to process lexical information from the current word, the time window for the saccade decision is quite limited (Deubel, O'Regan & Radach, 2000). Alcohol intoxication led to longer fixation durations, which extended this critical time window. Hence, the chance for the cancellation of an imminent (but linguistically unnecessary) refixation saccade could have been higher, leading to the observed dissociation of fixation duration and frequency. However, overall the number of fixations per gaze was only slightly lower under alcohol, resulting in refixation rates that were not different between alcohol conditions. This lack of a difference between alcohol conditions can be attributed to the high frequency legal preview condition. For the given target word length, the refixation rate produces a floor effect in this easiest condition under no alcohol, so that there is no room for a further reduction under alcohol. The other three conditions yielded in fact lower refixation rates under alcohol. In addition to the tradeoff between fixation durations and the number of fixations, the fact that no interaction between alcohol and word frequency were found in any parameter, demonstrated that linguistic processing was unaffected at least by moderate alcohol intoxication as used in the present study. This finding is corroborated by the fact that comprehension, as measured with simple comprehension questions regarding the last read sentence, did not yield any differences between alcohol conditions. Apparently, the additional time provided by the prolonged fixation duration, can be used for linguistic processing and subsequently reduce the number of fixations.

Furthermore, an additional analysis of regressions revealed that there were fewer regressions leaving the target word under alcohol. Regressions can have different sources. One possibility is that mislocated fixations cause regressions. In this case, a saccade that was intended for word N-1 actually landed on word N. However, there are reasons that contradict this explanation for the alcohol induced difference in regression rates. First, texts were counterbalanced between alcohol conditions. Second, spatial parameters of saccades, particularly the landing position function (plotting the initial landing position against the launch distance, see Appendix A-8 Figure 2), did not show any differences between alcohol conditions. In addition, the analysis of regression rates in relation to the landing position in the target word revealed that there were fewer regressions under alcohol independently from the previous landing position (Figure 10-9). Another possible source resulting in regressions is comprehension difficulties. When encountering difficult text, regressions become more frequent (Murray & Kennedy, 1988). In the current experiment 84% of the regressions

targeted the word immediately preceding the target word. Apparently, in the no alcohol condition there was a need to move the eyes back to the previous word in a considerable proportion of cases (12-17 %). In the alcohol condition, this proportion was reduced to 9-12%. These data might indicate that a criterion to check text for consistency was more relaxed under alcohol. However, with the current available data this assumption is highly speculative and asks for more research, especially regarding high level post lexical processing. An interesting hypothesis arising from this speculation is that effects of semantic (e.g. plausibility) or syntactic (e.g. using garden path sentences) manipulations, should be less pronounced under alcohol.

Further research is also needed regarding the influence of alcohol on the preview effect. In the current study no influence of alcohol intoxication on the preview effect was found. Evidently, alcohol did not reduce the cognitive processing capacity, or perception in the visual periphery, at least not to an extent affecting the parafoveal preview effect. A perceptual alcohol myopia effect was also not found. In an effort to examine related processing further, an additional analysis was carried out (see A-8.8 for complete ANOVA and means tables). It revealed that alcohol neither affected the preview effect under conditions that were facilitating preview (near launch distances, long gaze durations on N-1) nor under conditions that impede preview (far launch distances, short gaze duration on N-1). Therefore, using the current data none of the three original hypotheses could be supported. A factor that might have contributed to this null finding is the possibility that complementary effects could have cancelled out each other. For example, even though total processing capacity might be reduced under alcohol, the preview effect might still be preserved, if the letter mask is perceived as the most salient stimulus in terms of perceptual alcohol myopia. One way to explore these interrelations further would be the use of a word mask, instead of an orthographically illegal letter string. If the cognitive capacity or the limited perception account were true, the parafoveal preview effect should be reduced in such a design, without being masked by a possible alcohol myopia effect.

Taken together, alcohol did not impair linguistic processing, as evident in the absence of interactions between alcohol and word frequency and the fact that comprehension was not comprised. Longer fixation durations indicated impairment on the sensorimotor level in terms of a general slowing. This was compensated for by a reduction of the frequency of fixations, resulting in gaze durations and total viewing times that were not different between alcohol conditions, due to the described trade-off. Finally, the preview manipulation did not result in any interactions with alcohol, hence, no evidence for either tunnel vision or increased peripheral sensitivity was found in during reading.

11 General Discussion

The present thesis aimed at providing a comprehensive account of how acute alcohol intoxication affects visual processing and oculomotor control. To achieve this, the influence of moderate alcohol intoxication in the context of seven carefully selected paradigms was studied. These paradigms were divided into two clusters, with Cluster 1 including five basic oculomotor tasks and Cluster 2 consisting of two complex cognitive tasks. Paradigms in Cluster 1 were selected to target different levels of oculomotor control, which are described in the theoretical framework of saccade generation by Findlay and Walker (1999; see section 2.3). This framework afforded a sound theoretical basis for the mapping of alcohol related effects. Paradigms in Cluster 2 afforded to extend findings from Cluster 1 with regard to effects on higher level complex visual-cognitive functioning, including inhibitory mechanisms and linguistic processing. Additionally, both tasks are ecologically valid and performed by almost everybody on a daily basis.

Experimental results have been discussed in some detail in the previous chapters. The remainder of this general discussion will focus on three aspects. First, key findings from each paradigm are briefly reviewed. Second, these findings from experiments targeting the same and different levels of oculomotor control are compared and linked to give a coherent picture. Third, an outlook on further directions to study the effects of alcohol on visual information processing is provided.

11.1 Summary of Key Findings

Key findings for Cluster 1 paradigms are summarized according to the different processing levels of oculomotor control. The summary of Cluster 2 is presented by paradigm. More detailed descriptions can be found in the respective chapters.

The impact of alcohol on the *automatic level* of oculomotor control was relatively small. This was evident in the results of the pro saccade task. In this task, where a reflexive eye movement has to be executed in response to the onset of a peripheral visual target, a further manipulation was implemented. A central fixation marker would either be present throughout the whole trial, or disappear 200 ms before the onset of the peripheral target. This offset of visual stimulation at the fixation point typically leads to shorter saccade latencies and higher error rates (see sections 3.1 and 5.1.1). Interestingly, this gap effect was completely maintained under alcohol, indicating intact basic functioning on the automatic level. In contrast, deficits were found in the temporal aspects of visually guided saccades, with longer

latencies and slower peak velocities. This finding portrayed the manifestation of a general slowing in very early stages of visuomotor processing.

For the *automated level*, results from the double step paradigm revealed that alcohol intoxication led to specific deficits with respect to adaptive cancellations and reprogramming of saccades on the basis of new visual information. In the double step paradigm an initial peripheral target was replaced with a second target after a certain interval (see sections 6.1 and 6.3.1 for more details). Critical in this paradigm are saccade amplitudes and the proportion of one step responses in relation to the time that is available for reprogramming. This reprogramming time is calculated by determining the time between the onset of the second peripheral target and the start of the saccade following this onset. Therefore, the reprogramming time represents a combination of the inter stimulus interval (ISI) paired with the individual reaction time of a participant in a given trial. Alcohol related impairments became evident in the need for longer reprogramming times to perform one step instead of two step responses as well as to adjust saccade amplitude (see also 11.2.4).

Three paradigms were used to illuminate the effects of alcohol on the *voluntary level* of oculomotor control. In the standard anti saccade task the visual layout was identical to that in the pro saccade task, but participants were instructed to look to the mirror position of the appearing peripheral target. In contrast to the pro saccade task, this required the inhibition of a reflexive movement to the target and a voluntary saccade to a location without visual stimulation instead. Therefore, not only needed the saccade to be reprogrammed, but the saccade target had to be represented endogenously. In the visually guided anti saccade task an additional marker was presented at the possible target locations, thus eliminated the need to endogenously represent the saccade target, while preserving the reprogramming element of the task. In addition, in the memory guided saccade task the endogenous representation of the saccade target was essential, but no reprogramming of the saccade target was necessary. In this paradigm, the peripheral target was presented for a short interval and participants were instructed to keep their fixation at the central fixation marker. Only after the offset of the central marker an eye movement had to be carried out to the location where the peripheral target was displayed before. Taken together, in the traditional anti saccade task the endogenous representation and the reprogramming of the saccade target was necessary, whereas in the visually guided anti saccade task and the memory guided saccade task only one of these two processes was needed. In addition to a general slowing in the anti saccade tasks, evident in longer saccade latencies, results from paradigms targeting the voluntary level indicated specific deficits. In the standard anti saccade task, saccade amplitudes were prolonged under alcohol, pointing towards impairment related to the reprogramming of spatial parameters during saccade generation. The same deficit was found in the visually

guided anti saccade task, suggesting that the endogenous representation was not the driving mechanism underlying this impairment. Regarding the ability to inhibit reflexive (prepotent) responses, however, no effect of alcohol intoxication was found. Error rates in both anti saccade tasks were not affected by alcohol. Results from the memory guided saccade paradigm corroborated and extended these findings. Again, the proportion of reflexive responses to the visual stimulus was unaffected by alcohol. In addition, the visual-spatial short term memory showed a particular deficit, manifested in a smaller proportion of responses that were directed to the correct target location.

In the first complex cognitive paradigm, pro and standard anti saccade tasks were used to induce task switching[17]. Therefore, results regarding performance on these mixed pro and anti saccade blocks could also be used to replicate key findings from the pro and anti saccade tasks in Cluster 1. Indeed, all major findings were reproduced, proving the sensitivity of the paradigms with respect to alcohol induced effects. However, the impact of alcohol on task switching was limited. Nevertheless, this first attempt to apply the task switching paradigm to examine the influence of alcohol showed that there might be very specific alcohol induced deficits. The only dependent variable influenced by alcohol was the error rate in the anti saccade task. In anti saccade trials alcohol led to *lower* error rates in task switching compared to task repetition trials, whereas in the no alcohol condition *higher* error rates were found for the task switch condition (see 9.5.1). This finding suggests that additional processing time under alcohol, caused by longer latencies, can be utilized in the task switching condition to activate the required task set more completely, compared to the no alcohol condition (see 9.6).

Interestingly, a similar effect was found in the reading task (see below). The reading task included a variation of the mental workload associated with word processing via manipulation of word frequency. This allowed determining whether detrimental alcohol effects operate on the visuomotor and/or the linguistic processing stream. Furthermore, by using eye movement contingent display manipulations (see section 10.1.3 for details), the nature and extent of alcohol's effects on parafoveal information acquisition could be examined. When reading under alcohol, initial fixation durations were longer, but fewer fixations were made. This trade off indicated that the additional processing time provided by alcohol intoxication could be utilized for linguistic processing and resulted in gaze durations

[17] As discussed in section 9.2, the use of the pro and anti saccade task in this context also allows examining the effects of response switching independently from task switching. However, no significant alcohol related results were found with respect to response switching.

and total viewing times that were not different between alcohol conditions. These findings were corroborated by the fact that there were no interactions between alcohol and word frequency, as well as intact comprehension, showing that alcohol did not compromise linguistic processing as tested in the current work. Another finding that might point towards alcohol related effects in post lexical processing stages showed that alcohol reduced the likelihood of making a regression to a word that was previously fixated. This could indicate that effects of alcohol only come into play in higher processing modules not specifically tested in the present thesis. Finally, no evidence of alcohol related effects on parafoveal processing were found in the current reading experiment.

11.2 Linking Results

As summarized above and discussed at the end of the respective chapters, the current work revealed interesting and specific effects of alcohol intoxication on the different levels of oculomotor control. However, only by combining the findings from all levels and paradigms, a comprehensive mapping of the effects of alcohol on oculomotor control can be accomplished. Therefore, in this part of the general discussion, links between the results from the different experimental paradigms are explored.

This discussion will be organized around the issues of general slowing, spatial representation, and transformation, as well as the inhibition of reflexive responses. The implications of the results from different paradigms to the emerging map of alcohol related influences on visual processing and oculomotor control will be discussed. In addition, compensatory mechanisms in the complex cognitive tasks will be set in relation to the results from Cluster 1.

As an initial point, it should be noted that throughout the various paradigms, effects of the order of alcohol sessions were found. In most cases alcohol intoxication had a greater impact, if alcohol was administered in the first session. For example, error rates in the anti saccade tasks were higher under alcohol, if administered in the first session, whereas participants who received alcohol in the second session made more errors in the first no alcohol session. However, independent of these training effects, none of the most critical results that revealed specific alcohol induced impairments were influenced by the factor Alcohol Session. This provided a strong argument that these key findings were not only alcohol specific, but also robust against training effects. Furthermore, these findings underscore the importance to counterbalance alcohol conditions between sessions in related research.

11.2.1 General Slowing

Previous research reported a general slowing effect of alcohol on oculomotor control, most likely mediated by a general reduction of cortical activity (e.g., Krull et al., 1994,; Liu et al., 2000). In the present work, longer latencies were found consistently in the alcohol condition, not only for the paradigms targeting the automatic level of oculomotor control, but also in those examining the automated and voluntary level. The extent of the slowing was smaller in the blocked version of the pro saccade task (experiment 1) compared to all other tasks (~10 ms vs. ~20 ms). This demonstrated that a 'general slowing' due to moderate alcohol intoxication was less pronounced, if task demands were absolute minimal. In this context, higher task demands did not necessarily mean that a more complex behavior was required. Pro saccades in single step trials during the double step paradigm, as well as pro saccades in the task switching paradigm, were also more affected than the pro saccades in blocked trials. In all three cases the required response was identical, namely looking at a peripheral target as quickly and accurately as possible. However, in the double step and the mixed block presentation demands were higher, because there were a greater number of possible responses. A fact that corroborates this interpretation is that Wegner and Fahle (1999) found even larger alcohol effects on saccade latencies (~ 30ms) in the pro saccade with 22 possible target locations (or possible reactions). The design of the current work allowed the detection of this modulation of the 'general slowing' by task demands for the first time.

11.2.2 Spatial Transformation and Endogenous Representation of Saccade Targets

In addition to the temporal aspects discussed above, spatial properties of eye movements were also affected by alcohol intoxication. Earlier work on the effects of alcohol on visual processing has largely ignored spatial aspects of saccade generation or reported mixed results (Khan et al., 2003; Crevits et al., 2000; Blekher et al., 2002; Moser et al., 1998). However, there are interesting results from related research domains. Ploner et al. (2002) demonstrated that acute cannabis intoxication led to decreased saccade amplitudes and impaired performance in a visual search task. Huestegge (2002) found similar results for long term cannabis users with early age of onset, when tested sober.

In the present work, results regarding the effects of alcohol intoxication on saccade amplitudes showed that performance on the automatic level was relatively unaffected. However, for the automated and the voluntary level interesting results emerged. In the double step paradigm (automated level), saccade amplitudes have to be interpreted in connection with the percentage of one step responses. Generally, shorter ISI led to longer

reprogramming times, resulting in a higher percentage of one step responses. As described in section 6.4.3, for medium reprogramming times, in a certain proportion of cases double steps were still performed, while in another proportion of cases already one step responses were executed. This led to a linear increase in saccade amplitudes, known as the amplitude transition function. Saccade amplitudes did not differ between alcohol conditions, if examined with respect to different ISI. Shorter ISI yielded longer initial saccade amplitudes independent from alcohol intoxication, as more one step responses were made with increasing ISI. However, looking at saccade amplitudes related to the available reprogramming time (which increases with shorter ISI), it became apparent that under alcohol more time was needed to adjust saccade amplitudes (see section 6.4.3). Furthermore, this effect of alcohol only occurred in the medium range of reprogramming times (80-160ms). This is a sensible finding, because for shorter reprogramming times, saccades land on the near target in either alcohol condition, as adjustments cannot be implemented that fast. For longer available reprogramming times (>160ms) the effect of alcohol can be compensated and saccades were directed to the far target under both alcohol conditions. Interestingly, for the percentage of one step responses longer reprogramming times did not help to compensate. Even with reprogramming times >200ms the proportion of one step responses was still lower under alcohol. These findings demonstrated that the reprogramming of saccades on the basis of new visual information (i.e., on the automated level) was specifically impaired by alcohol.

While the double step paradigm examined processing on the automated level, the anti saccade and the memory guided saccade tasks studied influences of alcohol on the voluntary level of oculomotor control. Results from these tasks complemented findings regarding the ability to reprogram saccades. In the anti saccade task, typical results show an undershoot, with saccades falling short of the target position. Under alcohol, saccade amplitudes were longer, resulting in saccades that appeared to be more ‘accurate’. However, as argued in section 7.5, this alcohol induced amplitude alteration led to a deviation from the normal pattern and should therefore not be interpreted as higher accuracy. To narrow down the nature of this influence further, a comparison to the visually guided anti saccade and the memory guided saccade task proved helpful (see section 8.5). The effect of longer amplitudes could result from at least two different processes. First, amplitudes need to be reprogrammed after inhibiting a reflexive response, and second, the saccade target has to be represented endogenously, as there is no visual stimulation at the target location, at the time the saccade is executed. The visually guided anti saccade task eliminates the need to represent the target location endogenously, as it is continuously visually marked. The results showed that even though anti saccades in the no alcohol condition had longer amplitudes compared to the traditional anti saccade task, amplitudes under alcohol were still longer.

Therefore, the need to represent the saccade target endogenously cannot be the underlying mechanism of this alcohol induced deficit. Furthermore, the finding of no differences for saccade amplitudes in the memory guided paradigm not only corroborates this interpretation, but also highlights that it is not the process of inhibiting a reflexive saccade itself that causes differences in saccade amplitudes under alcohol. Rather, the need to reprogram saccade parameters to a new location was critical to induce the specific alcohol related impairment.

Previous work has not able to detect such specific effects of alcohol, primarily due to the fact that most studies neglected spatial parameters or used combined gain measures, which were not suited to reveal such particular amplitude effects. In addition, most research used only one paradigm with the same sample (e.g. Khan et al, 2003). Even if more paradigms were studied, versions of tasks were substantially different regarding timing or required saccade size (e.g. Blekher et al, 2002), thereby clouding potentially revealing effect patterns.

11.2.3 Inhibition of Reflexive Responses

Inhibitory influences are generally believed to be mediated by higher level processing (e.g. Glencross, 1990), represented by the voluntary control level in the present theoretical framework. Contrary to the initial expectation that the extent of alcohol related impairments would also be greater on higher processing levels within the framework of this thesis, the results obtained drew a different picture. In the anti saccade tasks, error rates were not higher under alcohol, indicating that the suppression of the reflexive saccade was equally effective in both alcohol conditions. It could be argued that in the case of anti saccades two saccade programs were initiated in parallel and therefore no real inhibition was necessary (Massen, 2002, 2004, see also section 7.1.1). Therefore, the results from the memory guided saccade paradigm provide a more convincing argument. In this task, no reprogramming of any kind was necessary, but the execution of the saccade had to be inhibited for a certain interval. The frequency of premature saccades, however, did not differ between alcohol conditions, strongly suggesting that inhibitory mechanisms were unaffected by alcohol intoxication.

These findings seem to be at odds with a line of research using go/no-go paradigms, noting alcohol-induced impairment of inhibition (Mulvihill et al., 1997; Fillmore & Vogel-Sprott, 1999; 2000; Easdon & Vogel-Sprott, 2000). However, in the go/no-go paradigm, reactions that were already initiated had to be cancelled, whereas in the memory guided saccade task, the reaction had to be delayed. This distinction might result in different processes that were involved in the two paradigms. More recently, Abroms, Gottlob, and Fillmore suggested that alcohol reduces intentional inhibitory control on selective attention, but has no effect on automatic inhibitory influences (Abroms et al., 2006). This assertion was based on their

finding of impaired performance under alcohol in a delayed ocular response task (DOR), very similar to the memory guided saccade task (see section 8.1), but not in a saccadic interference task. The DOR task varies in three aspects from the traditional memory guided saccade task. First, in the DOR task the target was only displayed for 100ms and memory intervals varied between 800, 1,000 and 1,200 ms, compared to a 1000ms presentation followed by 500, 1,000 and 2,000 ms intervals in the memory guided task. Second, distances between targets were larger in the DOR task (4.1°-16.4° vs. 3° and 6° in the memory guided task). Finally, in the DOR tasks the last target position was the starting point of the next trial, whereas in the traditional memory guided task a trial always started in the center of the screen. These differences should make premature saccades in the memory guided saccade task more likely, because the time interval in which premature saccades could be executed was longer and overall the predictability of the target location was higher. The fact that the present study nevertheless did not find alcohol related differences, suggests that inhibitory functions were not per se impaired. However, currently there is no explanation at hand to account for these differing results. Further research using both tasks with similar timing intervals within the same sample should help to solve this contradiction (see below).

11.2.4 Compensation Mechanism in Complex Cognitive Tasks

Oculomotor control was not only expected to be impaired on higher levels in the basic oculomotor tasks, but also in the two complex cognitive tasks. However, as discussed in the respective chapters, the influence of alcohol on both tasks was relatively small. In the task switching paradigm, additional processing time was utilized to enhance performance under switch conditions in the anti saccade task apparent in reduced error rates. In the reading task, a trade-off was found between *increased* fixation durations and *decreased* fixation frequencies under alcohol. At first sight, these findings seem to be in contradiction with the claim made earlier, that adaptive cancellation and reprogramming of saccades was impaired under alcohol, an argument following the results from the double step paradigm. However, comparing the findings more closely, a critical difference becomes obvious. When examining effects of alcohol on the complex cognitive tasks, the *available reprogramming times differed* between alcohol conditions. In contrast, in the double step paradigm, the impairment to make adaptive changes in saccade programming was demonstrated for *identical* reprogramming times.

In the task switching paradigm as well as in the reading task, additional processing time was provided by a slowing in the oculomotor system (evident in longer latencies or initial fixation durations). In both cases, reprogramming time was independent from new visual information, another contrast to the double step paradigm. In the task switching paradigm, the timeframe

for the activation of the task set for performing in the anti saccade task was longer under alcohol. Therefore, no new information needed to be processed, but ongoing processing simply had more time to be completed.

The same mechanism was assumed to work during reading. No differences in comprehension and no interactions with word frequency were found, indicating that linguistic processing was not affected by alcohol intoxication in the present study. Due to longer initial fixation durations, more linguistic processing could be completed during each single fixation. This allowed a significant proportion of saccades to be cancelled, resulting in lower fixation frequencies. Again, this adaptive cancellation did not originate in new visual information, rather, ongoing processing was provided more time to be finished. In analogy to Brysbaert, Drieghe, and Vitu (2005), the relevant process that benefits from this extra time is thought to be an 'educated guess' on where to fixate next. Brysbaert et al. suggest a process underlying word skipping, where an 'educated guess' is based on information gathered during the ongoing fixation (e.g. the length of word N+1, the launch distance to N+1, the difficulty of N+1). However, this information does not need to be complete, hence the term 'educated guess'. The fact that no differences were found for skipping rates in the current study, does not limit this analogy. Target words were relatively long (7-8 characters), resulting in low skipping rates. This floor effect was intended, to maximize the number of data points available for the analysis of key temporal and spatial parameters.

Overall, it was surprising to find evidence that effects of moderate acute alcohol intoxication could be compensated in the complex cognitive tasks implemented in this thesis. However, caution needs to be exercised when interpreting these findings as a demonstration that higher level cognitive processes are not subject to alcohol related impairments. One example that points towards possible impairments of higher level processes was the decreased rate of regressions in the reading task, indicating an influence of alcohol on post lexical processing (see below). In addition, the effect of alcohol might be more pronounced, when dosages higher than the moderate one used in the current work are studied. The next section provides some critique on the present work and gives an outlook on further research related to the effects of alcohol intoxication that are thought to be a promising research avenue.

11.3 Outlook and Critique

For the current research project two principal approaches were available. In the first approach, a variety of paradigms would have been selected to achieve an understanding of how different aspects of performance are affected by alcohol. The second possible approach would have studied the effects of alcohol on a smaller selection of paradigms but in more

detail. Given the relatively sparse literature on the effects of alcohol on different levels of oculomotor control, and the lack of an integrating theoretical framework in earlier research, (see chapters 1 and 3) the first approach was chosen because it promised to be a more valuable contribution. The careful and theoretically grounded selection of paradigms was essential in revealing specific alcohol related effects on different levels of processing.

The observed results provide excellent starting points for future work, as will be outlined with some selected examples (that are not prioritized) in the following. The finding that the general slowing was more pronounced in paradigms with higher 'task difficulty', for example, is warranting more research. Which factors determine 'task difficulty' in this respect and how general is the 'general slowing'? In this context it would be desirable to conduct a series of studies that vary the task demand independently from the predictability of the response. For example the number of possible target locations (i.e. predictability) could be varied orthogonally for the pro and anti saccade task. As an example, presenting 3 blocks of pro saccades with 2, 4 and 8 possible target positions, respectively, could confirm the assumption that location predictability influences the severity of the general slowing. Using the same sample in an additional anti saccade task with identical predictability conditions, would afford to distinguish the predictability effect from 'pure' task difficulty.

Another point for subsequent studies is the comparison of the memory guided saccade paradigm with the DOR task. As already described above, including both paradigms with similar timing in a study with one sample could help to determine the cause for equivocal findings regarding the ability to inhibit a prepotent (reflexive) response. Additional variation of the memory intervals and target locations could be used to narrow down the extent of impairments further in the visual-spatial short term memory. For example, shorter memory intervals could be utilized to determine how early in the timeline of visual information processing an effect of alcohol occurs.

A further promising follow up study could examine the effects of alcohol on reading more closely. It is possible that the word frequency variations, used to manipulate the difficulty of lexical access, targeted a process that is too automatic to be influenced by alcohol intoxication. Even though lexical access is clearly a complex process, it is very automatic in the sense that one, even voluntarily, is not able to suppress this very process. Rayner and Pollatsek (1989) discuss the fact that *you cannot <u>not</u> read a word that you look at* in some detail. Higher order processes that are linked to semantic or syntactic processing might reveal alcohol related impairments that were not detected in the present work. One example to study this could include the manipulation of sentence plausibility, to target higher level post lexical processes (Clifton, Staube & Rayner, 2007; see also section 10.6). In this connection, the

manipulation of text difficulty should allow for a more valid assessment of affects of alcohol intoxication on comprehension.

Finally, the unexpected findings of very small albeit specific effects of alcohol on task switching warrant further research. As the present study was the first experiment that tried to examine the effects of alcohol on task switching, a replication of the results is necessary before drawing final conclusions. Furthermore, the use of a different paradigm that proved to induce stable task switching effect appears to be desirable, as task switching in the pro and anti saccade paradigm has provided equivocal results, even under 'simple' no alcohol conditions.

Future research suggested up to now would examine the key findings in more detail, to break down the effects of alcohol on the different processing levels further. An alternative, or additional, research avenue could use the two Clusters of paradigms from the present thesis, to study effects beyond acute intoxication. One such interesting research avenue is related to chronic impairments caused by alcohol abuse. Interesting questions in this context are, if chronic alcohol abuse leads to the same impairments or compensatory mechanisms that were found for acute intoxication and whether those effects, if found, are attenuated with prolonged abstinence.

Furthermore, the tasks used in this thesis seem to be well suited to contribute to research related to expectancy effects. Such placebo effects are an important methodological issue, because there is evidence (see Testa, et al., 2006, for a review) suggesting that under certain circumstances, particularly where cognitive control is critical, placebo participants can and often do compensate for *expected* alcohol effects. This might lead to performance that exceeds that achieved by participants who simply received no alcohol without an expectation. In an effort to single out such expectancy effects, the current paradigms could be used in different placebo designs. This would allow the study of possible effects on different levels of control, with the hypothesis that higher levels are more affected than lower processing levels. Three possible placebo designs are comparisons (1) placebo vs. alcohol, (2) placebo vs. veridical no alcohol and (3) placebo vs. veridical no alcohol vs. alcohol. The most common design is the contrast between placebo and alcohol. However, as pointed out above, these studies might exaggerate apparent alcohol effects due to compensatory mechanisms in the placebo condition. Another problem with this kind of placebo design is that counterbalancing placebo and alcohol conditions is difficult. As participants are usually very familiar with the effects of alcohol on their sensation, it is almost impossible to induce a feeling of intoxication, especially if the participant has already experienced the alcohol session within the same environment. The same argument is true for the third design, which

includes all three possible sessions. The present work demonstrated that alcohol administration should be counterbalanced to avoid misinterpretations. Therefore, the comparison between a placebo condition and a veridical no alcohol condition appears to be the best candidate to shed light on expectancy effects. Although entailing a between subject design, as differences between placebo and veridical no alcohol condition need to be compared to differences between alcohol and a veridical no alcohol condition, this design could help to determine the size and quality of expectancy effects.

In conclusion, this thesis has explored the effects of acute alcohol intoxication on visual processing and oculomotor control. The selected paradigms have yielded interesting findings that begin to map alcohol related impairments on different levels of oculomotor control. In addition, findings and discussions afford multiple approaches for further research that should help to achieve a deeper understanding of the effects of alcohol and its underlying mechanisms.

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Appendix

Chapter 5 Pro Saccade Paradigm

A-1.1 Chapter 5 Pro Saccade Paradigm: Error Rates

A-1 Table 1. Means and SE[18] for ME[19] Beverage Condition, ME Gap Condition, ME Alcohol Session and IA[20] Gap Condition X Beverage Condition

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		.005	.002	.001	.009
	alcohol		.01	.003	.004	.016
ME Gap Condition	overlap		.003	.002	-.001	.006
	gap		.013	.003	.007	.019
ME Alcohol Session	Alcohol Session 1		.009	.003	.004	.015
	Alcohol Session 2		.006	.003	-.001	.013
IA Gap * Beverage Condition	overlap	no alcohol	.003	.002	-.001	.007
		alcohol	.002	.002	-.001	.005
	gap	no alcohol	.007	.002	.002	.012
		alcohol	.018	.005	.009	.028

A-1 Table 2. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.002	1	0.002	22.255	0.000
Gap * AlcSess	0.000	1	0.000	0.209	0.653
Error(Gap)	0.002	18	0.000		
Alcohol	0.000	1	0.000	1.780	0.192
Alcohol * AlcSess	0.000	1	0.000	0.481	0.497
Error(Alcohol)	0.002	18	0.000		
Gap * Alcohol	0.001	1	0.001	8.760	0.008
Gap * Alcohol * AlcSess	0.000	1	0.000	1.966	0.178
Error(Gap*Alcohol)	0.002	18	0.000		

[18] SE = standard error

[19] ME = main effect

[20] IA = interaction

A-1 Table 3. Single Contrasts for IA Gap Condition X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	alc diff overlap	0.000	1	0.000	0.903	0.354
	alc diff gap	0.002	1	0.002	6.948	0.017
	gap diff no alc	0.000	1	0.000	4.008	0.061
	gap diff alc	0.005	1	0.005	18.255	0.000
Error	alc diff overlap	0.001	18	0.000		
	alc diff gap	0.006	18	0.000		
	gap diff no alc	0.001	18	0.000		
	gap diff alc	0.005	18	0.000		

A-1.2 Chapter 5 Pro Saccade Paradigm: Error Rate Variability

A-1 Table 4. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Gap Condition X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.039	.013	.012	.065
	alcohol			.06	.013	.033	.086
ME Gap Condition	overlap			.019	.01	-.003	.041
	gap			.08	.014	.049	.11
ME Alcohol Session	Alcohol Session 1			.055	.014	.025	.085
	Alcohol Session 2			.043	.017	.007	.08
IA Gap * Beverage Condition	overlap		no alcohol	.24	.013	-.002	.051
			alcohol	.014	.101	-.008	.035
	gap		no alcohol	.053	.016	.021	.086
			alcohol	.106	.019	.065	.147
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	.035	.016	.002	.069
			alcohol	.014	.013	-.013	.041
		gap	no alcohol	.047	.02	.006	.088
			alcohol	.124	.024	.073	.175
	Alcohol Session 2	overlap	no alcohol	.013	.02	-.028	.054
			alcohol	.013	.016	-.02	.046
		gap	no alcohol	.059	.024	.009	.11
			alcohol	.088	.03	.025	.151

A-1 Table 5. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.071	1	0.071	28.676	0.000
Gap * AlcSess	0.000	1	0.000	0.000	0.990
Error(Gap)	0.044	18	0.002		
Alcohol	0.009	1	0.009	3.082	0.096
Alcohol * AlcSess	0.001	1	0.001	0.312	0.584
Error(Alcohol)	0.050	18	0.003		
Gap * Alcohol	0.019	1	0.019	9.865	0.006
Gap * Alcohol * AlcSess	0.006	1	0.006	2.965	0.102
Error(Gap*Alcohol)	0.035	18	0.002		

A-1.3 Chapter 5 Pro Saccade Paradigm: Latencies

A-1 Table 6. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Gap Condition X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			183.99	4.68	174.16	193.81
	alcohol			193.47	4.63	183.74	203.20
ME Gap Condition	overlap			212.76	5.42	201.38	224.14
	gap			164.70	4.10	156.09	173.31
ME Alcohol Session	Alcohol Session 1			190.99	5.49	179.46	202.53
	Alcohol Session 2			186.46	6.72	172.33	200.59
IA Gap * Beverage Condition	overlap		no alcohol	208.50	5.78	196.37	220.63
			alcohol	217.02	5.75	204.94	229.09
	gap		no alcohol	159.47	4.36	150.31	168.63
			alcohol	169.92	4.48	160.51	179.33
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	215.09	7.31	199.75	230.44
			alcohol	220.23	7.27	204.96	235.50
		gap	no alcohol	158.85	5.51	147.27	170.43
			alcohol	169.81	5.67	157.90	181.71
	Alcohol Session 2	overlap	no alcohol	201.91	8.95	183.11	220.70
			alcohol	213.81	8.90	195.10	232.51
		gap	no alcohol	160.09	6.75	145.91	174.28
			alcohol	170.04	6.94	155.46	184.62

A-1 Table 7. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	44348.387	1	44348.387	136.420	0.000
Gap * AlcSess	533.452	1	533.452	1.641	0.216
Error(Gap)	5851.590	18	325.088		
Alcohol	1726.982	1	1726.982	7.934	0.011
Alcohol * AlcSess	39.759	1	39.759	0.183	0.674
Error(Alcohol)	3917.934	18	217.663		
Gap * Alcohol	17.997	1	17.997	0.517	0.482
Gap * Alcohol * AlcSess	72.482	1	72.482	2.080	0.166
Error(Gap*Alcohol)	627.200	18	34.844		

A-1.4 Chapter 5 Pro Saccade Paradigm: Latency Variability

A-1 Table 8. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Gap Condition X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			45.30	3.12	38.75	51.85
	alcohol			43.08	2.91	36.97	49.18
ME Gap Condition	overlap			51.13	4.12	42.46	59.79
	gap			37.25	2.19	32.66	41.84
ME Alcohol Session	Alcohol Session 1			44.48	3.55	37.03	51.94
	Alcohol Session 2			43.90	4.35	34.77	53.03
IA Gap * Beverage Condition	overlap		no alcohol	53.56	4.35	44.43	62.70
			alcohol	48.69	4.43	39.37	58.00
	gap		no alcohol	37.04	2.96	30.81	43.26
			alcohol	37.46	2.24	32.77	42.16
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	51.47	5.50	39.91	63.02
			alcohol	51.97	5.61	40.19	63.75
		gap	no alcohol	38.05	3.75	30.18	45.93
			alcohol	36.43	2.83	30.50	42.37
	Alcohol Session 2	overlap	no alcohol	55.66	6.73	41.51	69.81
			alcohol	45.41	6.87	30.98	59.84
		gap	no alcohol	36.02	4.59	26.38	45.67
			alcohol	38.49	3.46	31.22	45.76

A-1 Table 9. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	3696.327	1	3696.327	15.938	0.001
Gap * AlcSess	6.898	1	6.898	0.030	0.865
Error(Gap)	4174.539	18	231.919		
Alcohol	95.089	1	95.089	1.021	0.326
Alcohol * AlcSess	53.399	1	53.399	0.574	0.459
Error(Alcohol)	1675.608	18	93.089		
Gap * Alcohol	134.728	1	134.728	1.790	0.198
Gap * Alcohol * AlcSess	264.456	1	264.456	3.513	0.077
Error(Gap*Alcohol)	1355.153	18	75.286		

A-1.5 Chapter 5 Pro Saccade Paradigm: Amplitudes

A-1 Table 10. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			5.76	0.07	5.61	5.90
	alcohol			5.65	0.08	5.48	5.82
ME Gap Condition	overlap			5.74	0.07	5.60	5.88
	gap			5.67	0.08	5.49	5.84
ME Alcohol Session	Alcohol Session 1			5.70	0.09	5.51	5.90
	Alcohol Session 2			5.70	0.11	5.46	5.94
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	5.80	0.09	5.62	5.98
			alcohol	5.61	0.10	5.39	5.83
	Alcohol Session 2		no alcohol	5.71	0.11	5.49	5.93
			alcohol	5.69	0.13	5.42	5.96
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	5.83	0.08	5.66	5.99
			alcohol	5.64	0.10	5.43	5.84
		gap	no alcohol	5.77	0.10	5.56	5.98
			alcohol	5.58	0.11	5.35	5.82
	Alcohol Session 2	overlap	no alcohol	5.75	0.10	5.55	5.95
			alcohol	5.74	0.12	5.49	5.99
		gap	no alcohol	5.67	0.12	5.41	5.93
			alcohol	5.64	0.14	5.35	5.93

A-1 Table 11. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.101	1	0.101	6.437	0.021
Gap * AlcSess	0.006	1	0.006	0.363	0.554
Error(Gap)	0.281	18	0.016		
Alcohol	0.213	1	0.213	9.023	0.008
Alcohol * AlcSess	0.139	1	0.139	5.879	0.026
Error(Alcohol)	0.425	18	0.024		
Gap * Alcohol	0.000	1	0.000	0.010	0.923
Gap * Alcohol * AlcSess	0.000	1	0.000	0.017	0.899
Error(Gap*Alcohol)	0.110	18	0.006		

A-1.6 Chapter 5 Pro Saccade Paradigm: Amplitude Variability

A-1 Table 12. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Alcohol Session X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		0.60	0.05	0.50	0.69
	alcohol		0.62	0.06	0.50	0.73
ME Gap Condition	overlap		0.58	0.04	0.49	0.67
	gap		0.63	0.05	0.52	0.75
ME Alcohol Session	Alcohol Session 1		0.55	0.06	0.43	0.68
	Alcohol Session 2		0.66	0.07	0.50	0.81
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	0.50	0.06	0.37	0.62
		alcohol	0.61	0.07	0.46	0.76
	Alcohol Session 2	no alcohol	0.69	0.07	0.54	0.85
		alcohol	0.63	0.09	0.45	0.81

A-1 Table 13. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.048	1	0.048	3.972	0.062
Gap * AlcSess	0.000	1	0.000	0.000	0.984
Error(Gap)	0.219	18	0.012		
Alcohol	0.010	1	0.010	0.358	0.557
Alcohol * AlcSess	0.142	1	0.142	4.927	0.040
Error(Alcohol)	0.518	18	0.029		
Gap * Alcohol	0.000	1	0.000	0.030	0.864
Gap * Alcohol * AlcSess	0.003	1	0.003	0.370	0.550
Error(Gap*Alcohol)	0.141	18	0.008		

A-1.7 Chapter 5 Pro Saccade Paradigm: Peak Velocity

A-1 Table 14. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			-35.51	3.53	-42.93	-28.09
	alcohol			-27.80	3.71	-35.59	-20.02
ME Gap Condition	overlap			-30.18	3.40	-37.33	-23.04
	gap			-33.13	3.81	-41.13	-25.13
ME Alcohol Session	Alcohol Session 1			-30.75	4.51	-40.23	-21.27
	Alcohol Session 2			-32.57	5.53	-44.18	-20.95
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	-33.16	4.31	-42.22	-24.11
			alcohol	-26.12	4.47	-35.51	-16.73
		gap	no alcohol	-36.07	4.86	-46.27	-25.86
			alcohol	-27.64	4.94	-38.02	-17.27
	Alcohol Session 2	overlap	no alcohol	-34.54	5.28	-45.63	-23.44
			alcohol	-26.91	5.48	-38.42	-15.41
		gap	no alcohol	-38.28	5.95	-50.78	-25.79
			alcohol	-30.53	6.05	-43.24	-17.83

A-1 Table 15. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	166.746	1	166.746	7.092	0.016
Gap * AlcSess	10.388	1	10.388	0.442	0.515
Error(Gap)	423.226	18	23.513		
Alcohol	1141.116	1	1141.116	40.636	0.000
Alcohol * AlcSess	0.012	1	0.012	0.000	0.984
Error(Alcohol)	505.466	18	28.081		
Gap * Alcohol	2.724	1	2.724	0.300	0.591
Gap * Alcohol * AlcSess	1.884	1	1.884	0.208	0.654
Error(Gap*Alcohol)	163.348	18	9.075		

A-1.8 Chapter 5 Pro Saccade Paradigm: Peak Velocity Variability

A-1 Table 16. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			21.88	4.69	12.03	31.73
	alcohol			22.64	6.56	8.86	36.41
ME Gap Condition	overlap			19.34	3.24	12.54	26.14
	gap			25.18	8.39	7.55	42.81
ME Alcohol Session	Alcohol Session 1			13.67	7.04	-1.13	28.47
	Alcohol Session 2			30.85	8.63	12.72	48.97
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	13.30	4.26	4.35	22.26
			alcohol	14.26	6.45	0.71	27.81
		gap	no alcohol	14.05	11.11	-9.29	37.40
			alcohol	13.08	10.17	-8.29	34.44
	Alcohol Session 2	overlap	no alcohol	22.37	5.22	11.40	33.34
			alcohol	27.42	7.90	10.82	44.02
		gap	no alcohol	37.80	13.61	9.21	66.40
			alcohol	35.80	12.46	9.63	61.97

A-1 Table 17. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	655.808	1	655.808	0.904	0.354
Gap * AlcSess	705.193	1	705.193	0.972	0.337
Error(Gap)	13061.297	18	725.628		
Alcohol	10.957	1	10.957	0.097	0.759
Alcohol * AlcSess	11.269	1	11.269	0.100	0.756
Error(Alcohol)	2030.241	18	112.791		
Gap * Alcohol	96.935	1	96.935	0.440	0.516
Gap * Alcohol * AlcSess	31.434	1	31.434	0.143	0.710
Error(Gap*Alcohol)	3967.983	18	220.443		

A-2 Chapter 6 Double Step Paradigm: Single Step Trials

A-2.1 Chapter 6 Double Step: Saccade Latencies in Single Step Trials

A-2 Table 1. Means and SE for ME Beverage Condition, ME Alcohol Session and IA Alcohol Session X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		221.17	6.66	207.18	235.16
	alcohol		241.97	7.35	226.53	257.40
ME Alcohol Session	Alcohol Session 1		236.49	8.52	218.58	254.40
	Alcohol Session 2		226.64	10.44	204.71	248.57
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	222.99	8.42	205.29	240.68
		alcohol	250.00	9.29	230.48	269.52
	Alcohol Session 2	no alcohol	219.35	10.32	197.68	241.03
		alcohol	233.93	11.38	210.03	257.84

A-2 Table 2. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Alcohol	4152.117	1	4152.117	28.841	0.000
Alcohol * AlcSess	370.690	1	370.690	2.575	0.126
Error(Alcohol)	2591.345	18	143.964		

A-2.2 Chapter 6 Double Step: Saccade Latencies Variability in Single Step Trials

A-2 Table 3. Means and SE for ME Beverage Condition, ME Alcohol Session and IA Alcohol Session X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		47.06	2.80	41.17	52.95
	alcohol		52.07	4.00	43.66	60.48
ME Alcohol Session	Alcohol Session 1		50.18	3.87	42.06	58.31
	Alcohol Session 2		48.95	4.74	39.00	58.90
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	44.71	3.55	37.26	52.15
		alcohol	55.66	5.06	45.02	66.29
	Alcohol Session 2	no alcohol	49.42	4.34	40.30	58.54
		alcohol	48.48	6.20	35.45	61.51

A-2 Table 4. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Alcohol	240.563	1	240.563	2.416	0.138
Alcohol * AlcSess	339.264	1	339.264	2.987	0.101
Error(Alcohol)	1792.555	18	99.586		

A-2.3 Chapter 6 Double Step: Saccade Amplitudes in Single Step Trials

A-2 Table 5. Means and SE for ME Beverage Condition, ME Alcohol Session and IA Alcohol Session X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		2.96	0.06	2.84	3.08
	alcohol		2.91	0.06	2.78	3.04
ME Alcohol Session	Alcohol Session 1		2.95	0.07	2.80	3.10
	Alcohol Session 2		2.92	0.09	2.74	3.10
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	3.00	0.07	2.85	3.15
		alcohol	2.90	0.08	2.74	3.06
	Alcohol Session 2	no alcohol	2.92	0.09	2.73	3.10
		alcohol	2.92	0.10	2.73	3.12

A-2 Table 6. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Alcohol	0.022	1	0.022	2.104	0.164
Alcohol * AlcSess	0.028	1	0.028	2.682	0.119
Error(Alcohol)	0.187	18	0.010		

A-2.4 Chapter 6 Double Step: Saccade Amplitude Variability in Single Step Trial

A-2 Table 7. Means and SE for ME Beverage Condition, ME Alcohol Session and IA Alcohol Session X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		0.46	0.04	0.38	0.55
	alcohol		0.43	0.03	0.38	0.48
ME Alcohol Session	Alcohol Session 1		0.39	0.04	0.31	0.47
	Alcohol Session 2		0.51	0.05	0.41	0.60
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	0.40	0.05	0.29	0.51
		alcohol	0.38	0.03	0.32	0.45
	Alcohol Session 2	no alcohol	0.53	0.06	0.40	0.67
		alcohol	0.48	0.04	0.40	0.56

A-2 Table 8. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Alcohol	0.011	1	0.011	1.039	0.322
Alcohol * AlcSess	0.004	1	0.004	0.370	0.551
Error(Alcohol)	0.190	18	0.011		

A-3 Chapter 6 Double Step Paradigm: Double Step Trials by ISI

A-3.1 Chapter 6 Double Step Trials by ISI: Proportion one step responses

A-3 Table 1. Means and SE for ME Beverage Condition, ME ISI and IA ISI X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		0.64	0.04	0.57	0.72
	alcohol		0.66	0.04	0.58	0.74
ME ISI	40		0.80	0.04	0.73	0.88
	70		0.65	0.04	0.57	0.74
	100		0.49	0.04	0.41	0.58
IA ISI * Beverage Condition	40	no alcohol	0.81	0.03	0.74	0.88
		alcohol	0.79	0.04	0.70	0.89
	70	no alcohol	0.64	0.04	0.55	0.73
		alcohol	0.67	0.04	0.58	0.76
	100	no alcohol	0.48	0.04	0.39	0.56
		alcohol	0.51	0.04	0.42	0.60

A-3 Table 2. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ISI	1.913	2	0.957	56.476	0.000
Error(ISI)	0.644	38	0.017		
Alcohol	0.007	1	0.007	0.366	0.552
Error(Alcohol)	0.382	19	0.020		
ISI * Alcohol	0.016	2	0.008	2.488	0.096
Error(ISI*Alcohol)	0.124	38	0.003		

A-3 Table 3. Single Contrasts for IA ISI X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	no alc 40-70	0.585	1	0.585	27.424	0
	no alc 70-100	0.539	1	0.539	28.151	0
	no alc 40-100	2.247	1	2.247	75.901	0
	alc 40-70	0.312	1	0.312	24.846	0
	alc 70-100	0.502	1	0.502	41.172	0
	alc 40-100	1.605	1	1.605	60.872	0
	40 noalc-alc	0.006	1	0.006	0.373	0.549
	70 noalc-alc	0.017	1	0.017	0.732	0.403
	100 noalc-alc	0.024	1	0.024	1.679	0.211
	diffalc 40-70	0.043	1	0.043	4.453	0.048
	diffalc 70-100	0.001	1	0.001	0.044	0.835
	diffalc 40-100	0.054	1	0.054	3.773	0.067
Error	no alc 40-70	0.405	19	0.021		
	no alc 70-100	0.364	19	0.019		
	no alc 40-100	0.562	19	0.03		
	alc 40-70	0.238	19	0.013		
	alc 70-100	0.232	19	0.012		
	alc 40-100	0.501	19	0.026		
	40 noalc-alc	0.296	19	0.016		
	70 noalc-alc	0.44	19	0.023		
	100 noalc-alc	0.276	19	0.015		
	diffalc 40-70	0.182	19	0.01		
	diffalc 70-100	0.289	19	0.015		
	diffalc 40-100	0.272	19	0.014		

A-3.2 Chapter 6 Double Step Trials by ISI: Primary Amplitude

A-3 Table 4. Means and SE for ME Beverage Condition, ME ISI and IA ISI X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		4.94	0.11	4.71	5.17
	alcohol		4.99	0.12	4.73	5.24
ME ISI	40		5.35	0.11	5.12	5.58
	70		5.00	0.11	4.77	5.23
	100		4.54	0.13	4.27	4.81
IA ISI * Beverage Condition	40	no alcohol	5.40	0.11	5.17	5.63
		alcohol	5.30	0.12	5.05	5.56
	70	no alcohol	4.93	0.12	4.67	5.19
		alcohol	5.07	0.11	4.83	5.31
	100	no alcohol	4.49	0.13	4.22	4.77
		alcohol	4.58	0.14	4.28	4.88

A-3 Table 5. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ISI	13.313	2	6.657	65.164	0.000
Error(ISI)	3.882	38	0.102		
Alcohol	0.056	1	0.056	0.350	0.561
Error(Alcohol)	3.023	19	0.159		
ISI * Alcohol	0.303	2	0.152	5.855	0.006
Error(ISI*Alcohol)	0.985	38	0.026		

A-3 Table 6. Single Contrasts for IA ISI X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	no alc 40-70	4.403	1	4.403	32.941	0
	no alc 70-100	3.807	1	3.807	31.195	0
	no alc 40-100	16.397	1	16.397	83.98	0
	alc 40-70	1.108	1	1.108	17.966	0
	alc 70-100	4.726	1	4.726	58.417	0
	alc 40-100	10.41	1	10.41	59.521	0
	40 noalc-alc	0.185	1	0.185	1.645	0.215
	70 noalc-alc	0.379	1	0.379	2.498	0.13
	100 noalc-alc	0.154	1	0.154	0.978	0.335
	diffalc 40-70	1.094	1	1.094	12.938	0.002
	diffalc 70-100	0.05	1	0.05	0.412	0.529
	diffalc 40-100	0.677	1	0.677	6.394	0.02
Error	no alc 40-70	2.539	19	0.134		
	no alc 70-100	2.318	19	0.122		
	no alc 40-100	3.71	19	0.195		
	alc 40-70	1.172	19	0.062		
	alc 70-100	1.537	19	0.081		
	alc 40-100	3.323	19	0.175		
	40 noalc-alc	2.136	19	0.112		
	70 noalc-alc	2.883	19	0.152		
	100 noalc-alc	2.996	19	0.158		
	diffalc 40-70	1.606	19	0.085		
	diffalc 70-100	2.29	19	0.121		
	diffalc 40-100	2.012	19	0.106		

A-3.3 Chapter 6 Double Step Trials by ISI: Primary Latency

A-3 Table 7. Means and SE for ME Beverage Condition, ME ISI and IA ISI X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		231.66	6.18	218.73	244.58
	alcohol		254.84	6.17	241.93	267.75
ME ISI	40		242.61	5.75	230.58	254.65
	70		242.43	6.28	229.29	255.56
	100		244.70	6.04	232.07	257.34
IA ISI * Beverage Condition	40	no alcohol	234.41	6.42	220.97	247.85
		alcohol	250.81	5.74	238.80	262.83
	70	no alcohol	229.79	6.39	216.41	243.17
		alcohol	255.07	6.75	240.93	269.21
	100	no alcohol	230.77	6.26	217.66	243.88
		alcohol	258.64	6.42	245.20	272.08

A-3 Table 8. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ISI	127.932	2	63.966	0.748	0.480
Error(ISI)	3249.706	38	85.519		
Alcohol	16124.668	1	16124.668	41.615	0.000
Error(Alcohol)	7361.922	19	387.470		
ISI * Alcohol	722.308	2	361.154	8.925	0.001
Error(ISI*Alcohol)	1537.684	38	40.465		

A-3 Table 9. Single Contrasts for IA ISI X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	no alc 40-70	426.718	1	426.718	2.173	0.157
	no alc 70-100	19.363	1	19.363	0.216	0.647
	no alc 40-100	264.285	1	264.285	2.023	0.171
	alc 40-70	362.068	1	362.068	2.671	0.119
	alc 70-100	254.533	1	254.533	2.977	0.101
	alc 40-100	1223.753	1	1223.753	10.355	0.005
	40 noalc-alc	5383.605	1	5383.605	16.687	0.001
	70 noalc-alc	12782.175	1	12782.175	41.359	0
	100 noalc-alc	15528.171	1	15528.171	50.893	0
	diffalc 40-70	1574.918	1	1574.918	9.245	0.007
	diffalc 70-100	133.49	1	133.49	0.612	0.444
	diffalc 40-100	2625.438	1	2625.438	27.06	0
Error	no alc 40-70	3730.556	19	196.345		
	no alc 70-100	1703.61	19	89.664		
	no alc 40-100	2482.498	19	130.658		
	alc 40-70	2575.367	19	135.546		
	alc 70-100	1624.762	19	85.514		
	alc 40-100	2245.378	19	118.178		
	40 noalc-alc	6129.999	19	322.632		
	70 noalc-alc	5872.062	19	309.056		
	100 noalc-alc	5797.152	19	305.113		
	diffalc 40-70	3236.764	19	170.356		
	diffalc 70-100	4145.918	19	218.206		
	diffalc 40-100	1843.423	19	97.022		

A-3.4 Chapter 6 Double Step Trials by ISI: Between Step Latency

A-3 Table 10. Means and SE for ME Beverage Condition, ME ISI and IA ISI X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		128.00	4.68	118.08	137.91
	alcohol		147.13	6.75	132.81	161.45
ME ISI	40		139.86	5.55	128.10	151.62
	70		136.26	5.35	124.91	147.61
	100		136.57	5.50	124.90	148.24
IA ISI * Beverage Condition	40	no alcohol	128.67	4.54	119.05	138.30
		alcohol	151.05	8.45	133.13	168.97
	70	no alcohol	126.48	4.83	116.25	136.71
		alcohol	146.04	6.70	131.83	160.25
	100	no alcohol	128.83	5.68	116.78	140.88
		alcohol	144.30	5.97	131.65	156.96

A-3 Table 11. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ISI	270.787	2	135.394	1.074	0.354
Error(ISI)	4033.591	32	126.050		
Alcohol	9337.654	1	9337.654	14.508	0.002
Error(Alcohol)	10298.093	16	643.631		
ISI * Alcohol	204.694	2	102.347	1.109	0.342
Error(ISI*Alcohol)	2953.997	32	92.312		

A-4 Chapter 6 Double Step Trials by Reprogramming Time

A-4.1 Chapter 6 Double Step Trials by Reprogramming Time: Proportions one step responses

A-4 Table 1. Means and SE for ME Beverage Condition, ME Reprogramming Time and IA Reprogramming Time X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		0.54	0.03	0.48	0.59
	alcohol		0.45	0.03	0.38	0.52
ME Reprogramming Time	<80		0.01	0.01	0.00	0.02
	80-120		0.12	0.03	0.06	0.18
	120-160		0.61	0.07	0.47	0.74
	160-200		0.83	0.04	0.74	0.92
	>200		0.90	0.03	0.83	0.97
IA Reprogramming Time * Beverage Condition	<80	no alcohol	0.00	0.00	0.00	0.01
		alcohol	0.01	0.01	-0.01	0.03
	80-120	no alcohol	0.14	0.03	0.08	0.21
		alcohol	0.09	0.03	0.03	0.16
	120-160	no alcohol	0.72	0.06	0.59	0.84
		alcohol	0.50	0.08	0.33	0.66
	160-200	no alcohol	0.88	0.03	0.81	0.95
		alcohol	0.78	0.06	0.66	0.90
	>200	no alcohol	0.93	0.02	0.88	0.98
		alcohol	0.87	0.04	0.78	0.96

A-4 Table 2. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
RepRange	24.135	4	6.034	166.167	0.000
Error(RepRange)	2.469	68	0.036		
Alcohol	0.334	1	0.334	20.441	0.000
Error(Alcohol)	0.278	17	0.016		
RepRange * Alcohol	0.256	4	0.064	9.736	0.000
Error(RepRange*Alcohol	0.447	68	0.007		

A-4 Table 3. Single Contrasts for IA Reprogramming Time X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	1 noalc-alc	0.001	1	0.001	0.412	0.530
	2 noalc-alc	0.047	1	0.047	6.447	0.021
	3 noalc-alc	0.868	1	0.868	21.92	0.000
	4 noalc-alc	0.193	1	0.193	7.482	0.014
	5 noalc-alc	0.072	1	0.072	6.598	0.020
Error	1 noalc-alc	0.03	17	0.002		
	2 noalc-alc	0.124	17	0.007		
	3 noalc-alc	0.673	17	0.04		
	4 noalc-alc	0.438	17	0.026		
	5 noalc-alc	0.185	17	0.011		

A-4.2 Chapter 6 Double Step Trials by Reprogramming Time: Primary Saccade Amplitude

A-4 Table 4. Means and SE for ME Beverage Condition, ME Reprogramming Time and IA Reprogramming Time X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		4.66	0.12	4.40	4.91
	alcohol		4.42	0.13	4.16	4.69
ME Reprogramming Time	<80		3.01	0.09	2.82	3.21
	80-120		3.62	0.15	3.30	3.94
	120-160		5.02	0.20	4.59	5.45
	160-200		5.48	0.11	5.25	5.70
	>200		5.57	0.10	5.35	5.78
IA Reprogramming Time * Beverage Condition	<80	no alcohol	3.07	0.10	2.85	3.29
		alcohol	2.96	0.11	2.73	3.19
	80-120	no alcohol	3.76	0.15	3.44	4.08
		alcohol	3.48	0.16	3.13	3.82
	120-160	no alcohol	5.28	0.20	4.85	5.70
		alcohol	4.76	0.23	4.29	5.24
	160-200	no alcohol	5.55	0.10	5.33	5.77
		alcohol	5.40	0.12	5.14	5.67
	>200	no alcohol	5.62	0.10	5.42	5.83
		alcohol	5.51	0.13	5.24	5.77

A-4 Table 5. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
RepRange	192.100	4	48.025	204.843	0.000
Error(RepRange)	15.943	68	0.234		
Alcohol	2.448	1	2.448	11.365	0.004
Error(Alcohol)	3.663	17	0.215		
RepRange * Alcohol	1.064	4	0.266	5.032	0.001
Error(RepRange*Alcohol	3.593	68	0.053		

A-4 Table 6. Single Contrasts for IA Reprogramming Time X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	1 noalc-alc	0.222	1	0.222	1.09	0.311
	2 noalc-alc	1.435	1	1.435	14.209	0.002
	3 noalc-alc	4.758	1	4.758	16.969	0.001
	4 noalc-alc	0.367	1	0.367	3.027	0.100
	5 noalc-alc	0.242	1	0.242	1.644	0.217
Error	1 noalc-alc	3.466	17	0.204		
	2 noalc-alc	1.717	17	0.101		
	3 noalc-alc	4.767	17	0.28		
	4 noalc-alc	2.063	17	0.121		
	5 noalc-alc	2.499	17	0.147		

A-5 Chapter 7 Traditional Anti Saccade Task

A-5.1 Chapter 7 Traditional Anti Saccade Task: Error Rates

A-5 Table 1. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.152	.026	.096	.207
	alcohol			.121	.028	.063	.18
ME Gap Condition	overlap			.087	.02	.044	.129
	gap			.187	.03	.123	.25
ME Alcohol Session	Alcohol Session 1			.119	.031	.054	.185
	Alcohol Session 2			.154	.038	.074	.234
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	.099	.034	.029	.17
			alcohol	.139	.035	.065	.212
	Alcohol Session 2		no alcohol	.204	.041	.118	.29
			alcohol	.104	.043	.014	.194
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	.051	.03	-.013	.114
			alcohol	.083	.025	.031	.136
		gap	no alcohol	.148	.038	.067	.228
			alcohol	.194	.046	.098	.291
	Alcohol Session 2	overlap	no alcohol	.151	.037	.073	.229
			alcohol	.061	.031	-.003	.126
		gap	no alcohol	.257	.047	.158	.356
			alcohol	.147	.056	.029	.265

A-5 Table 2. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.192	1	0.192	49.823	0.000
Gap * AlcSess	0.000	1	0.000	0.082	0.777
Error(Gap)	0.069	18	0.004		
Alcohol	0.017	1	0.017	1.795	0.197
Alcohol * AlcSess	0.093	1	0.093	9.663	0.006
Error(Alcohol)	0.174	18	0.010		
Gap * Alcohol	0.000	1	0.000	0.025	0.875
Gap * Alcohol * AlcSess	0.001	1	0.001	0.949	0.343
Error(Gap*Alcohol)	0.027	18	0.001		

A-5 Table 3. Single Contrasts for IA Gap Condition X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	alc diff	0.016	1	0.016	2.445	0.135
	alc diff gap	0.019	1	0.019	1.199	0.288
Error	alc diff	0.116	18	0.006		
	alc diff gap	0.285	18	0.016		

A-5.2 Chapter 7 Visually Guided Anti Saccade Task: Error Rates

A-5 Table 4. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.13	.024	.081	.179
	alcohol			.131	.031	.067	.196
ME Gap Condition	overlap			.061	.018	.023	.1
	gap			.2	.035	.126	.275
ME Alcohol Session	Alcohol Session 1			.114	.033	.045	.182
	Alcohol Session 2			.148	.04	.064	.232
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	.089	.03	.027	.152
			alcohol	.138	.039	.057	.22
	Alcohol Session 2		no alcohol	.171	.036	.094	.247
			alcohol	.125	.048	.025	.225
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	.044	.021	.000	.088
			alcohol	.079	.031	.014	.144
		gap	no alcohol	.135	.041	.048	.222
			alcohol	.197	.052	.088	.307
	Alcohol Session 2	overlap	no alcohol	.084	.025	.03	.137
			alcohol	.038	.038	-.042	.117
		gap	no alcohol	.257	.051	.151	.364
			alcohol	.212	.064	.077	.346

A-5 Table 5. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	0.371	1	0.371	36.787	0.000
GapCond * AlcSess	0.023	1	0.023	2.280	0.148
Error(GapCond)	0.182	18	0.010		
Alcohol	0.000	1	0.000	0.006	0.939
Alcohol * AlcSess	0.043	1	0.043	7.034	0.016
Error(Alcohol)	0.110	18	0.006		
GapCond * Alcohol	0.001	1	0.001	0.396	0.537
GapCond * Alcohol * Alc	0.001	1	0.001	0.378	0.546
Error(GapCond*Alcohol)	0.043	18	0.002		

A-5.3 Chapter 7 Traditional Anti Saccade Task: Error Rate Variability

A-5 Table 6. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.304	.025	.252	.356
	alcohol			.258	.034	.186	.331
ME Gap Condition	overlap			.221	.03	.159	.284
	gap			.341	.028	.281	.401
ME Alcohol Session	Alcohol Session 1			.26	.035	.186	.335
	Alcohol Session 2			.302	.043	.211	.393
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	.257	.031	.191	.322
			alcohol	.264	.044	.173	.356
	Alcohol Session 2		no alcohol	.351	.038	.271	.431
			alcohol	.252	.053	.14	.364
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	.186	.036	.111	.261
			alcohol	.22	.044	.127	.313
		gap	no alcohol	.328	.029	.266	.389
			alcohol	.308	.046	.211	.405
	Alcohol Session 2	overlap	no alcohol	.302	.044	.21	.394
			alcohol	.177	.054	.063	.291
		gap	no alcohol	.4	.036	.325	.475
			alcohol	.327	.057	.209	.446

A-5 Table 7. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.275	1	0.275	57.576	0.000
Gap * AlcSess	0.000	1	0.000	0.089	0.769
Error(Gap)	0.086	18	0.005		
Alcohol	0.040	1	0.040	4.605	0.046
Alcohol * AlcSess	0.055	1	0.055	6.214	0.023
Error(Alcohol)	0.158	18	0.009		
Gap * Alcohol	0.000	1	0.000	0.001	0.978
Gap * Alcohol * AlcSess	0.013	1	0.013	2.746	0.115
Error(Gap*Alcohol)	0.020	18	0.001		

A-5.4 Chapter 7 Visually Guided Anti Saccade Task: Error Rate Variability

A-5 Table 8. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.277	.024	.227	.327
	alcohol			.259	.028	.2	.317
ME Gap Condition	overlap			.186	.027	.128	.243
	gap			.35	.025	.297	.403
ME Alcohol Session	Alcohol Session 1			.247	.031	.181	.312
	Alcohol Session 2			.289	.038	.208	.369
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	.23	.03	.167	.293
			alcohol	.263	.035	.189	.338
	Alcohol Session 2		no alcohol	.324	.037	.246	.401
			alcohol	.254	.043	.163	.345
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	.168	.033	.1	.237
			alcohol	.186	.042	.097	.275
		gap	no alcohol	.292	.035	.219	.365
			alcohol	.34	.033	.271	.41
	Alcohol Session 2	overlap	no alcohol	.243	.04	.159	.327
			alcohol	.145	.052	.036	.254
		gap	no alcohol	.405	.042	.315	.494
			alcohol	.362	.041	.277	.448

A-5 Table 9. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	0.518	1	0.518	84.485	0.000
GapCond * AlcSess	0.012	1	0.012	1.967	0.178
Error(GapCond)	0.110	18	0.006		
Alcohol	0.006	1	0.006	1.323	0.265
Alcohol * AlcSess	0.051	1	0.051	10.485	0.005
Error(Alcohol)	0.087	18	0.005		
GapCond * Alcohol	0.009	1	0.009	2.278	0.149
GapCond * Alcohol * Alc	0.001	1	0.001	0.178	0.678
Error(GapCond*Alcohol)	0.071	18	0.004		

A-5.5 Chapter 7 Task Comparison Traditional vs. Visually Guided Anti Saccade Paradigm: Error Rate

A-5 Table 10. Means and SE for ME Beverage Condition, ME Task, ME Gap Condition and IA Task X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		.141	.023	.092	.19
	alcohol		.126	.027	.07	.183
ME Task	traditional		.137	.025	.085	.188
	visually guided		.131	.026	.076	.185
ME Gap	overlap		.074	.018	.037	.111
	gap		.193	.03	.13	.257
IA Task * Beverage Condition	traditional	no alcohol	.152	.026	.096	.207
		alcohol	.121	.028	.063	.18
	visually guided	no alcohol	.13	.024	.081	.179
		alcohol	.131	.031	.067	.196

A-5 Table 11. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	0.001	1	0.001	0.101	0.754
Task * AlcSess	0.000	1	0.000	0.001	0.975
Error(Task)	0.229	18	0.013		
Gap	0.549	1	0.549	58.881	0.000
Gap * AlcSess	0.009	1	0.009	0.961	0.340
Error(Gap)	0.168	18	0.009		
Alcohol	0.008	1	0.008	0.651	0.430
Alcohol * AlcSess	0.132	1	0.132	10.875	0.004
Error(Alcohol)	0.218	18	0.012		
Task * Gap	0.015	1	0.015	3.163	0.092
Task * Gap * AlcSess	0.014	1	0.014	3.104	0.095
Error(Task*Gap)	0.083	18	0.005		
Task * Alcohol	0.009	1	0.009	2.577	0.126
Task * Alcohol * AlcSess	0.005	1	0.005	1.299	0.269
Error(Task*Alcohol)	0.066	18	0.004		
Gap * Alcohol	0.000	1	0.000	0.106	0.748
Gap * Alcohol * AlcSess	0.002	1	0.002	0.804	0.382
Error(Gap*Alcohol)	0.051	18	0.003		
Task * Gap * Alcohol	0.001	1	0.001	0.659	0.427
Task * Gap * Alcohol * AlcSess	0.000	1	0.000	0.028	0.869
Error(Task*Gap*Alcohol)	0.018	18	0.001		

A-5 Table 12. Single Contrasts for IA Task X Beverage Condition.

Source	Transformed Variable	Squares	df	Square	F	Sig.
Contrast	trad, no alcohol-alcohol	0.017	1	0.017	1.795	0.197
	vg, no alcohol-alcohol	0.000	1	0.000	0.006	0.939
	no alcohol, trad-vg	0.009	1	0.009	1.321	0.265
	alcohol, trad-vg	0.002	1	0.002	0.196	0.663
	diff alccond between trad-vg	0.019	1	0.019	2.577	0.126
Error	trad, no alcohol-alcohol	0.174	18	0.010		
	vg, no alcohol-alcohol	0.110	18	0.006		
	no alcohol, trad-vg	0.121	18	0.007		
	alcohol, trad-vg	0.174	18	0.010		
	diff alccond between trad-vg	0.133	18	0.007		

A-5.6 Chapter 7 Traditional Anti Saccade Task: Saccade Latencies

A-5 Table 13. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Alcohol Session X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		255.47	6.677	241.441	269.499
	alcohol		285.647	7.298	270.313	300.98
ME Gap Condition	overlap		286.126	7.115	271.179	301.074
	gap		254.991	6.454	241.431	268.55
ME Alcohol Session	Alcohol Session 1		276.964	8.347	259.426	294.501
	Alcohol Session 2		264.153	10.223	242.675	285.632
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	252.292	8.446	234.546	270.037
		alcohol	301.636	9.232	282.24	321.031
	Alcohol Session 2	no alcohol	258.649	10.345	236.916	280.383
		alcohol	269.658	11.307	245.903	293.412

A-5 Table 14. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	18613.098	1	18613.098	93.633	0.000
Gap * AlcSess	18.356	1	18.356	0.092	0.765
Error(Gap)	3578.171	18	198.787		
Alcohol	17483.701	1	17483.701	42.326	0.000
Alcohol * AlcSess	7054.185	1	7054.185	17.077	0.001
Error(Alcohol)	7435.337	18	413.074		
Gap * Alcohol	0.647	1	0.647	0.009	0.925
Gap * Alcohol * AlcSess	4.241	1	4.241	0.060	0.810
Error(Gap*Alcohol)	1275.654	18	70.870		

A-5.7 Chapter 7 Visually Guided Anti Saccade Task: Saccade Latencies

A-5 Table 15. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			288.773	11.467	264.681	312.865
	alcohol			309.996	11.634	285.552	334.439
ME Gap Condition	overlap			313.306	11.746	288.629	337.983
	gap			285.462	11.217	261.897	309.028
ME Alcohol Session	Alcohol Session 1			309.696	14.239	279.78	339.611
	Alcohol Session 2			289.073	17.439	252.434	325.712
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	287.727	14.505	257.252	318.201
			alcohol	331.665	14.717	300.746	362.583
	Alcohol Session 2		no alcohol	289.819	17.765	252.496	327.143
			alcohol	288.326	18.024	250.459	326.193
IA Alcohol Session * Gap Condition * Beverage Condition	Alcohol Session 1	overlap	no alcohol	300.421	15.828	267.168	333.673
			alcohol	343.536	14.584	312.897	374.175
		gap	no alcohol	300.421	15.828	267.168	333.673
			alcohol	343.536	14.584	312.897	374.175
	Alcohol Session 2	overlap	no alcohol	306.213	19.385	265.488	346.939
			alcohol	303.055	17.861	265.529	340.58
		gap	no alcohol	273.425	16.768	238.198	308.653
			alcohol	273.598	19.413	232.814	314.382

A-5 Table 16. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	14885.546	1	14885.546	37.517	0.000
GapCond * AlcSess	206.380	1	206.380	0.520	0.480
Error(GapCond)	7141.918	18	396.773		
Alcohol	8647.676	1	8647.676	16.785	0.001
Alcohol * AlcSess	9907.171	1	9907.171	19.230	0.000
Error(Alcohol)	9273.631	18	515.202		
GapCond * Alcohol	29.721	1	29.721	0.167	0.688
GapCond * Alcohol * Alc	3.409	1	3.409	0.019	0.892
Error(GapCond*Alcohol)	3207.195	18	178.177		

A-5.8 Chapter 7 Task Comparison Traditional vs. Visually Guided Anti Saccade Paradigm: Saccade Latencies

A-5 Table 17. Means and SE for ME Task and IA Task X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Task	traditional		270.558	6.599	256.694	284.423
	visually guided		299.384	11.257	275.734	323.035
IA Task * Beverage Condition	traditional	no alcohol	255.47	6.677	241.441	269.499
		alcohol	285.647	7.298	270.313	300.98
	visually guided	no alcohol	288.773	11.467	264.681	312.865
		alcohol	309.996	11.634	285.552	334.439

A-5 Table 18. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	31907.597	1	31907.597	19.067	0.000
Task * AlcSess	585.931	1	585.931	0.350	0.561
Error(Task)	30122.117	18	1673.451		
Gap	33394.625	1	33394.625	73.132	0.000
Gap * AlcSess	173.918	1	173.918	0.381	0.545
Error(Gap)	8219.405	18	456.634		
Alcohol	25361.761	1	25361.761	41.073	0.000
Alcohol * AlcSess	16840.524	1	16840.524	27.273	0.000
Error(Alcohol)	11114.565	18	617.476		
Task * Gap	104.019	1	104.019	0.749	0.398
Task * Gap * AlcSess	50.819	1	50.819	0.366	0.553
Error(Task*Gap)	2500.684	18	138.927		
Task * Alcohol	769.617	1	769.617	2.476	0.133
Task * Alcohol * AlcSess	120.833	1	120.833	0.389	0.541
Error(Task*Alcohol)	5594.403	18	310.800		
Gap * Alcohol	19.571	1	19.571	0.192	0.667
Gap * Alcohol * AlcSess	0.023	1	0.023	0.000	0.988
Error(Gap*Alcohol)	1838.389	18	102.133		
Task * Gap * Alcohol	10.798	1	10.798	0.073	0.789
Task * Gap * Alcohol * AlcSess	7.628	1	7.628	0.052	0.822
Error(Task*Gap*Alcohol)	2644.460	18	146.914		

A-5.9 Chapter 7 Traditional Anti Saccade Task: Saccade Amplitudes

A-5 Table 19. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Gap Condition X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		5.219	.184	4.833	5.605
	alcohol		5.881	.189	5.483	6.278
ME Gap Condition	overlap		5.48	.181	5.1	5.86
	gap		5.619	.177	5.247	5.991
ME Alcohol Session	Alcohol Session 1		5.546	.218	5.088	6.004
	Alcohol Session 2		5.554	.267	4.993	6.115
IA Gap Condition * Beverage Condition	overlap	no alcohol	5.106	.194	4.698	5.515
		alcohol	5.854	.198	5.439	6.27
	gap	no alcohol	5.331	.189	4.933	5.729
		alcohol	5.907	.195	5.498	6.316

A-5 Table 20. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.370	1	0.370	2.063	0.168
Gap * AlcSess	0.251	1	0.251	1.400	0.252
Error(Gap)	3.227	18	0.179		
Alcohol	8.411	1	8.411	21.538	0.000
Alcohol * AlcSess	0.002	1	0.002	0.005	0.945
Error(Alcohol)	7.029	18	0.391		
Gap * Alcohol	0.142	1	0.142	3.395	0.082
Gap * Alcohol * AlcSess	0.076	1	0.076	1.818	0.194
Error(Gap*Alcohol)	0.754	18	0.042		

A-5.10 Chapter 7 Visually Guided Anti Saccade Task: Saccade Amplitudes

A-5 Table 21. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Gao Condition X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			5.836	.058	5.715	5.958
	alcohol			6.021	.077	5.858	6.183
ME Gap Condition	overlap			5.868	.07	5.72	6.016
	gap			5.989	.063	5.856	6.122
ME Alcohol Session	Alcohol Session 1			5.941	.08	5.774	6.108
	Alcohol Session 2			5.916	.097	5.711	6.12
IA Gap Condition * Beverage Condition	overlap		no alcohol	5.75	.067	5.609	5.891
			alcohol	5.987	.084	5.81	6.163
	gap		no alcohol	5.922	.058	5.801	6.044
			alcohol	6.055	.079	5.89	6.22
IA Alcohol Session * Gap Condition * Beverage Condition	Alcohol Session 1	overlap	no alcohol	5.762	.085	5.584	5.941
			alcohol	6.031	.106	5.807	6.254
		gap	no alcohol	5.883	.073	5.729	6.037
			alcohol	6.09	.099	5.881	6.299
	Alcohol Session 2	overlap	no alcohol	5.738	.104	5.519	5.957
			alcohol	5.943	.13	5.669	6.216
		gap	no alcohol	5.962	.09	5.774	6.15
			alcohol	6.02	.122	5.765	6.276

A-5 Table 22. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	0.278	1	0.278	6.808	0.018
GapCond * AlcSess	0.018	1	0.018	0.436	0.517
Error(GapCond)	0.736	18	0.041		
Alcohol	0.655	1	0.655	11.863	0.003
Alcohol * AlcSess	0.054	1	0.054	0.974	0.337
Error(Alcohol)	0.993	18	0.055		
GapCond * Alcohol	0.052	1	0.052	9.377	0.007
GapCond * Alcohol * Alc	0.009	1	0.009	1.547	0.229
Error(GapCond*Alcohol)	0.100	18	0.006		

A-5.11 Chapter 7 Traditional Anti Saccade Task: Saccade Amplitude Variability

A-5 Table 23. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			1.387	.074	1.233	1.542
	alcohol			1.481	.072	1.329	1.633
ME Gap Condition	overlap			1.431	.065	1.295	1.566
	gap			1.437	.077	1.276	1.599
ME Alcohol Session	Alcohol Session 1			1.442	.087	1.26	1.624
	Alcohol Session 2			1.426	.106	1.203	1.649
IA Alcohol Session * Gap Condition * Beverage Condition	Alcohol Session 1	overlap	no alcohol	1.356	.094	1.158	1.553
			alcohol	1.559	.087	1.376	1.741
		gap	no alcohol	1.341	.1	1.131	1.55
			alcohol	1.515	.106	1.291	1.738
	Alcohol Session 2	overlap	no alcohol	1.412	.115	1.171	1.654
			alcohol	1.397	.107	1.173	1.62
		gap	no alcohol	1.441	.122	1.185	1.698
			alcohol	1.453	.13	1.179	1.727

A-5.12 Chapter 7 Visually Guided Anti Saccade Task: Saccade Amplitude Variability

A-5 Table 24. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.749	.069	.604	.894
	alcohol			.819	.064	.686	.953
ME Gap Condition	overlap			.785	.065	.648	.923
	gap			.783	.068	.641	.925
ME Alcohol Session	Alcohol Session 1			.765	.081	.594	.936
	Alcohol Session 2			.803	.1	.594	1.012
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	.699	.087	.516	.882
			alcohol	.831	.081	.662	1.001
	Alcohol Session 2		no alcohol	.799	.107	.575	1.023
			alcohol	.808	.099	.6	1.015
IA Alcohol Session * Gap Condition * Beverage Condition	Alcohol Session 1	overlap	no alcohol	.689	.096	.487	.891
			alcohol	.85	.079	.684	1.015
		gap	no alcohol	.709	.087	.526	.893
			alcohol	.813	.09	.624	1.001
	Alcohol Session 2	overlap	no alcohol	.805	.118	.558	1.053
			alcohol	.797	.097	.594	1.
		gap	no alcohol	.792	.107	.568	1.017
			alcohol	.818	.11	.587	1.049

A-5 Table 25. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	0.000	1	0.000	0.004	0.948
GapCond * AlcSess	0.001	1	0.001	0.034	0.856
Error(GapCond)	0.388	18	0.022		
Alcohol	0.095	1	0.095	2.446	0.135
Alcohol * AlcSess	0.073	1	0.073	3.403	0.082
Error(Alcohol)	0.385	18	0.021		
GapCond * Alcohol	0.001	1	0.001	0.048	0.829
GapCond * Alcohol * Alc	0.010	1	0.010	0.730	0.404
Error(GapCond*Alcohol)	0.243	18	0.014		

A-5.13 Chapter 7 Task Comparison Traditional vs. Visually Guided Anti Saccade Paradigm: Saccade Amplitudes

A-5 Table 26. Means and SE for ME Beverage Condition, ME Task and IA Task X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		5.527	.111	5.295	5.76
	alcohol		5.951	.124	5.691	6.211
ME Task	traditional		5.55	.172	5.188	5.912
	visually guided		5.929	.063	5.796	6.061
IA Task * Beverage Condition	traditional	no alcohol	5.219	.184	4.833	5.605
		alcohol	5.881	.189	5.483	6.278
	visually guided	no alcohol	5.836	.058	5.715	5.958
		alcohol	6.021	.077	5.858	6.183

A-5 Table 27. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	5.511	1	5.511	7.562	0.013
Task * AlcSess	0.011	1	0.011	0.015	0.904
Error(Task)	13.118	18	0.729		
Gap	0.645	1	0.645	4.881	0.040
Gap * AlcSess	0.068	1	0.068	0.511	0.484
Error(Gap)	2.378	18	0.132		
Alcohol	6.879	1	6.879	25.867	0.000
Alcohol * AlcSess	0.018	1	0.018	0.066	0.800
Error(Alcohol)	4.787	18	0.266		
Task * Gap	0.003	1	0.003	0.037	0.850
Task * Gap * AlcSess	0.201	1	0.201	2.286	0.148
Error(Task*Gap)	1.585	18	0.088		
Task * Alcohol	2.186	1	2.186	12.164	0.003
Task * Alcohol * AlcSess	0.038	1	0.038	0.211	0.651
Error(Task*Alcohol)	3.235	18	0.180		
Gap * Alcohol	0.183	1	0.183	6.696	0.019
Gap * Alcohol * AlcSess	0.068	1	0.068	2.484	0.132
Error(Gap*Alcohol)	0.492	18	0.027		
Task * Gap * Alcohol	0.011	1	0.011	0.554	0.466
Task * Gap * Alcohol * AlcSess	0.017	1	0.017	0.837	0.372
Error(Task*Gap*Alcohol)	0.362	18	0.020		

A-5 Table 28. Single Contrasts for IA Task X Beverage Condition.

Source	Transformed Variable	Squares	df	Square	F	Sig.
Contrast	trad, no alcohol-alcohol	8.411	1	8.411	21.538	0.000
	vg, no alcohol-alcohol	0.655	1	0.655	11.863	0.003
	no alcohol, trad-vg	7.320	1	7.320	15.166	0.001
	alcohol, trad-vg	0.378	1	0.378	0.886	0.359
	diff alccond between trad-vg	4.373	1	4.373	12.164	0.003
Error	trad, no alcohol-alcohol	7.029	18	0.391		
	vg, no alcohol-alcohol	0.993	18	0.055		
	no alcohol, trad-vg	8.687	18	0.483		
	alcohol, trad-vg	7.666	18	0.426		
	diff alccond between trad-vg	6.470	18	0.359		

A-5.14 Chapter 7 Traditional Anti Saccade Task: Peak Velocity Deviation

A-5 Table 29. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Gap Condition X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			-33.097	3.146	-39.707	-26.486
	alcohol			-17.743	3.156	-24.374	-11.113
ME Gap Condition	overlap			-25.436	3.118	-31.988	-18.885
	gap			-25.404	2.94	-31.58	-19.227
ME Alcohol Session	Alcohol Session 1			-28.595	3.741	-36.456	-20.735
	Alcohol Session 2			-22.245	4.582	-31.872	-12.618
IA Gap Condition * Beverage Condition	overlap		no alcohol	-33.951	3.63	-41.578	-26.325
			alcohol	-16.921	3.159	-23.558	-10.285
	gap		no alcohol	-32.242	2.827	-38.182	-26.302
			alcohol	-18.565	3.484	-25.885	-11.246
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	-37.589	4.591	-47.235	-27.943
			alcohol	-18.756	3.996	-27.15	-10.361
		gap	no alcohol	-37.183	3.576	-44.697	-29.669
			alcohol	-20.853	4.407	-30.112	-11.595
	Alcohol Session 2	overlap	no alcohol	-30.314	5.623	-42.128	-18.5
			alcohol	-15.087	4.893	-25.368	-4.806
		gap	no alcohol	-27.301	4.38	-36.503	-18.098
			alcohol	-16.278	5.397	-27.617	-4.938

A-5 Table 30. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.021	1	0.021	0.001	0.980
Gap * AlcSess	14.821	1	14.821	0.443	0.514
Error(Gap)	602.250	18	33.458		
Alcohol	4525.891	1	4525.891	49.873	0.000
Alcohol * AlcSess	95.335	1	95.335	1.051	0.319
Error(Alcohol)	1633.461	18	90.748		
Gap * Alcohol	53.980	1	53.980	1.543	0.230
Gap * Alcohol * AlcSess	3.468	1	3.468	0.099	0.757
Error(Gap*Alcohol)	629.817	18	34.990		

A-5.15 Chapter 7 Visually Guided Anti Saccade Task: Peak Velocity Deviation

A-5 Table 31. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Gap Condition X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			-33.016	3.57	-40.516	-25.516
	alcohol			-22.248	4.066	-30.791	-13.706
ME Gap Condition	overlap			-26.222	3.622	-33.832	-18.613
	gap			-29.043	3.833	-37.095	-20.99
ME Alcohol Session	Alcohol Session 1			-28.311	4.652	-38.085	-18.537
	Alcohol Session 2			-26.954	5.698	-38.924	-14.983
IA Gap Condition * Beverage Condition	overlap		no alcohol	-31.927	3.565	-39.417	-24.437
			alcohol	-20.517	4.057	-29.04	-11.994
	gap		no alcohol	-34.105	3.685	-41.848	-26.363
			alcohol	-23.98	4.216	-32.837	-15.122
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	-33.356	4.51	-42.83	-23.882
			alcohol	-21.44	5.131	-32.221	-10.66
		gap	no alcohol	-35.522	4.661	-45.316	-25.729
			alcohol	-22.924	5.333	-34.128	-11.72
	Alcohol Session 2	overlap	no alcohol	-30.498	5.523	-42.101	-18.895
			alcohol	-19.594	6.284	-32.797	-6.391
		gap	no alcohol	-32.688	5.709	-44.683	-20.694
			alcohol	-25.035	6.531	-38.757	-11.313

A-5 Table 32. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	152.737	1	152.737	5.264	0.034
GapCond * AlcSess	19.020	1	19.020	0.655	0.429
Error(GapCond)	522.324	18	29.018		
Alcohol	2226.130	1	2226.130	26.065	0.000
Alcohol * AlcSess	42.580	1	42.580	0.499	0.489
Error(Alcohol)	1537.347	18	85.408		
GapCond * Alcohol	7.919	1	7.919	0.905	0.354
GapCond * Alcohol * Alc	18.563	1	18.563	2.122	0.162
Error(GapCond*Alcohol)	157.487	18	8.749		

A-5.16 Chapter 7 Task Comparison Traditional vs. Visually Guided Anti Saccade Paradigm: Peak Velocity

A-5 Table 33. Means and SE for ME Beverage Condition, ME Task, and IA Task X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		-33.056	3.22	-39.822	-26.291
	alcohol		-19.996	3.465	-27.276	-12.716
ME Task	traditional		-25.42	2.958	-31.634	-19.206
	visually guided		-27.632	3.678	-35.359	-19.905
IA Task * Beverage Condition	traditional	no alcohol	-33.097	3.146	-39.707	-26.486
		alcohol	-17.743	3.156	-24.374	-11.113
	visually guided	no alcohol	-33.016	3.57	-40.516	-25.516
		alcohol	-22.248	4.066	-30.791	-13.706

A-5 Table 34. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	187.937	1	187.937	1.279	0.273
Task * AlcSess	239.374	1	239.374	1.629	0.218
Error(Task)	2645.546	18	146.975		
Gap	74.604	1	74.604	2.922	0.105
Gap * AlcSess	0.131	1	0.131	0.005	0.944
Error(Gap)	459.549	18	25.531		
Alcohol	6550.160	1	6550.160	42.335	0.000
Alcohol * AlcSess	132.671	1	132.671	0.857	0.367
Error(Alcohol)	2784.970	18	154.721		
Task * Gap	78.154	1	78.154	2.115	0.163
Task * Gap * AlcSess	33.711	1	33.711	0.912	0.352
Error(Task*Gap)	665.024	18	36.946		
Task * Alcohol	201.861	1	201.861	9.417	0.007
Task * Alcohol * AlcSess	5.244	1	5.244	0.245	0.627
Error(Task*Alcohol)	385.837	18	21.435		
Gap * Alcohol	51.625	1	51.625	2.667	0.120
Gap * Alcohol * AlcSess	19.040	1	19.040	0.983	0.334
Error(Gap*Alcohol)	348.476	18	19.360		
Task * Gap * Alcohol	10.274	1	10.274	0.421	0.524
Task * Gap * Alcohol * AlcSess	2.992	1	2.992	0.123	0.730
Error(Task*Gap*Alcohol)	438.828	18	24.379		

A-5.17 Chapter 7 Traditional Anti Saccade Task: Erroneous Responses Latencies

A-5 Table 35. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			178.032	9.611	157.269	198.795
	alcohol			202.318	8.308	184.369	220.267
ME Gap Condition	overlap			211.809	10.102	189.985	233.633
	gap			168.541	5.422	156.828	180.253
ME Alcohol Session	Alcohol Session 1			184.404	8.946	165.078	203.729
	Alcohol Session 2			195.946	10.956	172.277	219.615
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	186.542	18.365	146.868	226.217
			alcohol	217.071	16.586	181.24	252.902
		gap	no alcohol	152.711	7.427	136.665	168.756
			alcohol	181.291	8.164	163.654	198.927
	Alcohol Session 2	overlap	no alcohol	212.102	22.492	163.511	260.693
			alcohol	231.521	20.313	187.637	275.405
		gap	no alcohol	160.771	9.097	141.119	180.423
			alcohol	179.39	9.999	157.789	200.99

A-5 Table 36. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	26959.127	1	26959.127	29.800	0.000
Gap * AlcSess	1031.219	1	1031.219	1.140	0.305
Error(Gap)	11760.524	13	904.656		
Alcohol	8493.634	1	8493.634	4.806	0.047
Alcohol * AlcSess	399.595	1	399.595	0.226	0.642
Error(Alcohol)	22974.774	13	1767.290		
Gap * Alcohol	6.801	1	6.801	0.006	0.937
Gap * Alcohol * AlcSess	1.186	1	1.186	0.001	0.974
Error(Gap*Alcohol)	13709.287	13	1054.561		

A-5.18 Chapter 7 Visually Guided Anti Saccade Task: Erroneous Response Latencies

A-5 Table 37. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Gap Condition X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			185.909	6.67	171.229	200.59
	alcohol			225.337	13.764	195.044	255.63
ME Gap Condition	overlap			224.081	8.448	205.487	242.675
	gap			187.165	9.536	166.176	208.155
ME Alcohol Session	Alcohol Session 1			225.546	8.685	206.43	244.662
	Alcohol Session 2			185.7	10.986	161.52	209.88
IA Gap Condition * Beverage Condition	overlap		no alcohol	196.087	8.004	178.47	213.704
			alcohol	252.075	13.059	223.333	280.818
	gap		no alcohol	175.732	7.784	158.598	192.865
			alcohol	198.599	18.96	156.868	240.33
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	208.28	9.928	186.429	230.131
			alcohol	287.215	16.198	251.564	322.865
		gap	no alcohol	174.667	9.655	153.415	195.918
			alcohol	232.024	23.517	180.263	283.785
	Alcohol Session 2	overlap	no alcohol	183.894	12.558	156.254	211.534
			alcohol	216.936	20.489	171.841	262.031
		gap	no alcohol	176.797	12.213	149.916	203.678
			alcohol	165.174	29.747	99.701	230.647

A-5 Table 38. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	16772.411	1	16772.411	10.606	0.008
GapCond * AlcSess	689.797	1	689.797	0.436	0.523
Error(GapCond)	17395.517	11	1581.411		
Alcohol	19132.960	1	19132.960	5.721	0.036
Alcohol * AlcSess	10150.562	1	10150.562	3.035	0.109
Error(Alcohol)	36786.802	11	3344.255		
GapCond * Alcohol	3375.385	1	3375.385	3.200	0.090
GapCond * Alcohol * Alc	410.007	1	410.007	0.571	0.466
Error(GapCond*Alcohol)	7899.493	11	718.136		

A-5.19 Chapter 7 Traditional Anti Saccade Task: Erroneous Response Amplitude

A-5 Table 39. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			4.529	.16	4.185	4.874
	alcohol			4.445	.17	4.077	4.813
ME Gap Condition	overlap			4.363	.211	3.907	4.819
	gap			4.611	.102	4.39	4.832
ME Alcohol Session	Alcohol Session 1			4.527	.179	4.14	4.914
	Alcohol Session 2			4.447	.219	3.973	4.921
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	4.237	.26	3.674	4.799
			alcohol	4.598	.404	3.726	5.47
		gap	no alcohol	4.537	.195	4.115	4.958
			alcohol	4.737	.099	4.524	4.951
	Alcohol Session 2	overlap	no alcohol	4.665	.319	3.976	5.354
			alcohol	3.951	.494	2.884	5.019
		gap	no alcohol	4.678	.239	4.162	5.195
			alcohol	4.493	.121	4.232	4.754

A-5 Table 40. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.890	1	0.890	2.069	0.174
Gap * AlcSess	0.012	1	0.012	0.028	0.870
Error(Gap)	5.593	13	0.430		
Alcohol	0.102	1	0.102	0.248	0.627
Alcohol * AlcSess	1.921	1	1.921	4.671	0.050
Error(Alcohol)	5.347	13	0.411		
Gap * Alcohol	0.122	1	0.122	0.239	0.633
Gap * Alcohol * AlcSess	0.427	1	0.427	0.838	0.377
Error(Gap*Alcohol)	6.626	13	0.510		

A-5.20 Chapter 7 Visually Guided Anti Saccade Task: Erroneous Response Amplitude

A-5 Table 41. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			5.377	.089	5.181	5.574
	alcohol			5.026	.095	4.818	5.234
ME Gap Condition	overlap			5.213	.085	5.026	5.401
	gap			5.19	.084	5.005	5.375
ME Alcohol Session	Alcohol Session 1			5.198	.086	5.008	5.388
	Alcohol Session 2			5.206	.109	4.966	5.446
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	5.323	.194	4.896	5.751
			alcohol	5.179	.199	4.741	5.617
		gap	no alcohol	5.245	.105	5.013	5.477
			alcohol	5.043	.149	4.716	5.371
	Alcohol Session 2	overlap	no alcohol	5.405	.246	4.864	5.946
			alcohol	4.945	.252	4.391	5.499
		gap	no alcohol	5.536	.133	5.243	5.83
			alcohol	4.937	.188	4.522	5.351

A-5 Table 42. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	0.007	1	0.007	0.057	0.815
Gap * AlcSess	0.087	1	0.087	0.762	0.401
Error(Gap)	1.258	11	0.114		
Alcohol	1.519	1	1.519	8.549	0.014
Alcohol * AlcSess	0.392	1	0.392	2.208	0.165
Error(Alcohol)	1.955	11	0.178		
Gap * Alcohol	0.030	1	0.030	0.084	0.777
Gap * Alcohol * AlcSess	0.005	1	0.005	0.015	0.906
Error(Gap*Alcohol)	3.895	11	0.354		

A-5.21 Chapter 7 Traditional Anti Saccade Task: Erroneous Peak Velocity

A-5 Table 43. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			-43.121	5.155	-54.259	-31.983
	alcohol			-36.068	4.829	-46.5	-25.637
ME Gap Condition	overlap			-41.025	5.256	-52.381	-29.67
	gap			-38.164	4.254	-47.354	-28.974
ME Alcohol Session	Alcohol Session 1			-43.754	5.874	-56.443	-31.064
	Alcohol Session 2			-35.435	7.194	-50.977	-19.894
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	-49.956	7.832	-66.876	-33.036
			alcohol	-40.387	7.668	-56.953	-23.821
		gap	no alcohol	-48.859	5.58	-60.914	-36.805
			alcohol	-35.813	5.564	-47.833	-23.792
	Alcohol Session 2	overlap	no alcohol	-36.684	9.592	-57.406	-15.962
			alcohol	-37.075	9.392	-57.364	-16.785
		gap	no alcohol	-36.984	6.834	-51.748	-22.22
			alcohol	-30.998	6.815	-45.721	-16.276

A-5 Table 44. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Gap	117.934	1	117.934	1.578	0.231
Gap * AlcSess	0.010	1	0.010	0.000	0.991
Error(Gap)	971.823	13	74.756		
Alcohol	716.245	1	716.245	3.675	0.077
Alcohol * AlcSess	260.730	1	260.730	1.338	0.268
Error(Alcohol)	2533.687	13	194.899		
Gap * Alcohol	87.409	1	87.409	0.681	0.424
Gap * Alcohol * AlcSess	7.562	1	7.562	0.059	0.812
Error(Gap*Alcohol)	1669.424	13	128.417		

A-5.22 Chapter 7 Visually Guided Anti Saccade Task: Erroneous Peak Velocity

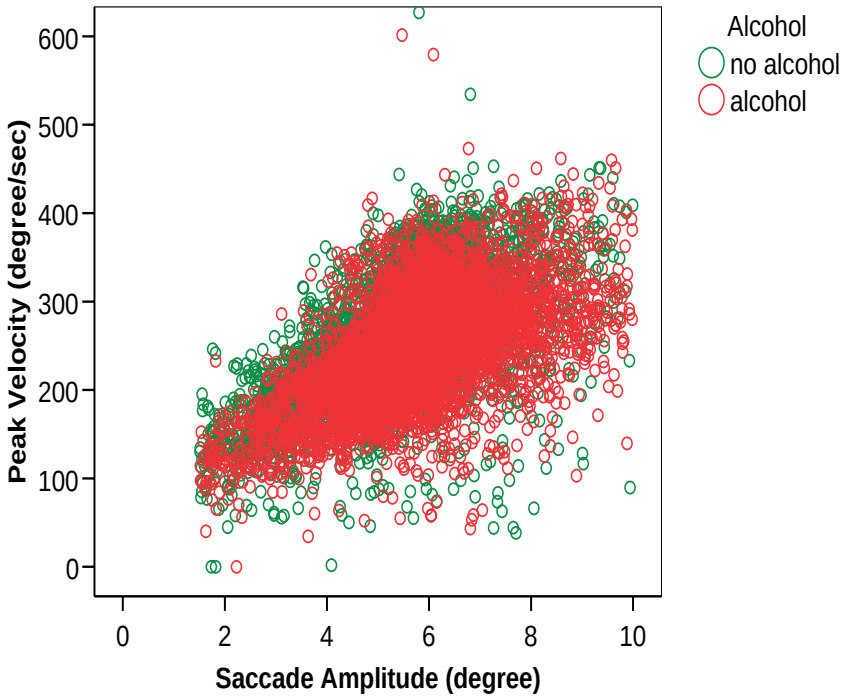
A-5 Table 45. Means and SE for ME Beverage Condition, ME Gap Condition, ME Alcohol Session, IA Alcohol Session X Beverage Condition and IA Alcohol Session X Gap Condition X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			-36.297	4.246	-45.643	-26.952
	alcohol			-30.684	5.396	-42.561	-18.808
ME Gap Condition	overlap			-31.606	3.821	-40.016	-23.196
	gap			-35.376	5.797	-48.135	-22.617
ME Alcohol Session	Alcohol Session 1			-36.234	5.806	-49.012	-23.456
	Alcohol Session 2			-30.748	7.343	-46.911	-14.585
IA Alcohol Session * Beverage Condition	Alcohol Session 1		no alcohol	-42.872	5.267	-54.464	-31.28
			alcohol	-29.596	6.693	-44.326	-14.865
	Alcohol Session 2		no alcohol	-29.723	6.662	-44.386	-15.06
			alcohol	-31.773	8.466	-50.406	-13.14
IA Alcohol Session* Gap*Beverage Condition	Alcohol Session 1	overlap	no alcohol	-41.299	4.663	-51.561	-31.037
			alcohol	-24.567	5.437	-36.535	-12.599
		gap	no alcohol	-44.444	6.283	-58.274	-30.615
			alcohol	-34.624	8.595	-53.542	-15.707
	Alcohol Session 2	overlap	no alcohol	-29.134	5.898	-42.114	-16.153
			alcohol	-31.424	6.878	-46.563	-16.286
		gap	no alcohol	-30.312	7.948	-47.806	-12.819
			alcohol	-32.122	10.872	-56.051	-8.194

A-5 Table 46. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
GapCond	174.904	1	174.904	1.618	0.230
GapCond * AlcSess	98.674	1	98.674	0.913	0.360
Error(GapCond)	1188.730	11	108.066		
Alcohol	387.752	1	387.752	4.730	0.052
Alcohol * AlcSess	722.781	1	722.781	8.818	0.013
Error(Alcohol)	901.659	11	81.969		
GapCond * Alcohol	31.822	1	31.822	0.653	0.436
GapCond * Alcohol * Alc	42.038	1	42.038	0.863	0.373
Error(GapCond*Alcohol)	535.921	11	48.720		

A-5.23 Chapter 7 Traditional Anti Saccade Task: main sequence



A-5 Figure 1. Relation between Saccade Amplitude and Peak Velocity, called main sequence. There were no differences between alcohol conditions.

A-6 Chapter 8 Memory Guided Saccade Task

A-6.1 Chapter 8 Memory Guided Saccade Task: Proportion valid responses

A-6 Table 1. Means and SE for ME Beverage Condition, ME Eccentricity, ME Memory Interval and Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	.874	.017	.838	.91
	alcohol	.814	.025	.762	.866
ME Eccentricity	near	.966	.009	.947	.985
	far	.722	.038	.641	.802
ME Memory Interval	500	.854	.024	.804	.904
	1000	.849	.02	.806	.891
	2000	.829	.02	.787	.871
ME Alcohol Session	Alcohol Session 1	.868	.026	.814	.922
	Alcohol Session 2	.82	.028	.761	.879

A-6 Table 2. Means and SE for IA Eccentricity * Beverage Condition, IA Alcohol Session * Beverage Condition and IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition.

Effect					Mean	Std.Error	95% Confidence Interval	
							Lower Bound	Upper Bound
IA Eccentricity * Beverage Condition	near			no alcohol	.976	.008	.958	.993
				alcohol	.957	.015	.926	.988
	far			no alcohol	.772	.033	.703	.841
				alcohol	.671	.048	.571	.772
IA Alcohol Session * Beverage Condition	Alcohol Session 1			no alcohol	.897	.023	.849	.945
				alcohol	.839	.033	.769	.908
	Alcohol Session 2			no alcohol	.851	.025	.798	.904
				alcohol	.789	.037	.713	.866
IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition	Alcohol Session 1	near	500	no alcohol	.982	.016	.948	1.017
				alcohol	.963	.021	.919	1.007
			1000	no alcohol	.996	.01	.975	1.018
				alcohol	.974	.017	.938	1.01
			2000	no alcohol	.981	.012	.955	1.007
				alcohol	.949	.036	.873	1.024
		far	500	no alcohol	.82	.055	.704	.936
				alcohol	.725	.074	.569	.882
			1000	no alcohol	.848	.051	.742	.955
				alcohol	.698	.069	.552	.844
			2000	no alcohol	.754	.044	.662	.847
				alcohol	.724	.071	.574	.874
	Alcohol Session 1	near	500	no alcohol	.95	.018	.911	.988
				alcohol	.944	.023	.895	.992
			1000	no alcohol	.968	.011	.944	.992
				alcohol	.966	.019	.926	1.006
			2000	no alcohol	.978	.014	.949	1.006
				alcohol	.944	.04	.861	1.027
		far	500	no alcohol	.752	.061	.624	.88
				alcohol	.695	.082	.522	.868
			1000	no alcohol	.723	.056	.606	.841
				alcohol	.615	.077	.454	.777
			2000	no alcohol	.735	.049	.632	.837
				alcohol	.571	.079	.405	.737

A-6 Table 3. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ecc	3.549	1	3.549	35.957	0.000
ecc * AlcSess	0.061	1	0.061	0.616	0.443
Error(ecc)	1.777	18	0.099		
int	0.026	2	0.013	1.153	0.327
int * AlcSess	0.006	2	0.003	0.246	0.783
Error(int)	0.409	36	0.011		
alc	0.214	1	0.214	10.594	0.004
alc * AlcSess	0.000	1	0.000	0.009	0.926
Error(alc)	0.363	18	0.020		
ecc * int	0.033	2	0.017	2.033	0.146
ecc * int * AlcSess	0.012	2	0.006	0.746	0.482
Error(ecc*int)	0.295	36	0.008		
ecc * alc	0.098	1	0.098	8.225	0.010
ecc * alc * AlcSess	0.003	1	0.003	0.257	0.618
Error(ecc*alc)	0.215	18	0.012		
int * alc	0.008	2	0.004	0.531	0.593
int * alc * AlcSess	0.030	2	0.015	2.039	0.145
Error(int*alc)	0.267	36	0.007		
ecc * int * alc	0.009	2	0.005	0.920	0.408
ecc * int * alc * AlcSess	0.020	2	0.010	1.968	0.154
Error(ecc*int*alc)	0.181	36	0.005		

A-6 Table 4. Single Contrasts for IA Eccentricity X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	alc diff near	0.131	1	0.131	8.225	0.010
	alc diff near	0.007	1	0.007	1.526	0.233
	alc diff far	0.2	1	0.2	12.106	0.003
Error	alc diff near	0.286	18	0.016		
	alc diff near	0.087	18	0.005		
	alc diff far	0.298	18	0.017		

A-6.2 Chapter 8 Memory Guided Saccade Task: Saccade Latencies for Correct Responses

A-6 Table 5. Means and SE for ME Beverage Condition, ME Eccentricity, ME Memory Interval and Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	280.098	6.206	267.004	293.192
	alcohol	298.136	5.866	285.761	310.511
ME Eccentricity	near	293.422	5.885	281.006	305.839
	far	284.811	5.86	272.447	297.176
ME Memory Interval	500	327.924	8.2	310.623	345.226
	1000	281.44	6.413	267.91	294.969
	2000	257.987	5.143	247.136	268.838
ME Alcohol Session	Alcohol Session 1	280.375	7.343	264.882	295.868
	Alcohol Session 2	297.859	8.611	279.691	316.026

A-6 Table 6. Means and SE for IA Alcohol Session * Beverage Condition and IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition.

Effect					Mean	Std.Error	95% Confidence Interval	
							Lower Bound	Upper Bound
IA Alcohol Session * Beverage Condition	Alcohol Session 1			no alcohol	264.958	8.054	247.966	281.951
				alcohol	295.792	7.612	279.732	311.853
	Alcohol Session 2			no alcohol	295.238	9.444	275.312	315.164
				alcohol	300.479	8.926	281.647	319.312
IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition	Alcohol Session 1	near	500	no alcohol	300.199	10.662	277.704	322.695
				alcohol	342.977	15.038	311.25	374.704
			1000	no alcohol	255.995	8.507	238.047	273.942
				alcohol	294.821	10.58	272.5	317.143
			2000	no alcohol	246.507	7.683	230.297	262.717
				alcohol	270.77	9.216	251.326	290.213
		far	500	no alcohol	292.869	12.89	265.673	320.066
				alcohol	319.938	13.049	292.407	347.468
			1000	no alcohol	256.546	8.184	239.28	273.813
				alcohol	281.556	14.999	249.91	313.202
			2000	no alcohol	237.634	7.759	221.263	254.004
				alcohol	264.692	7.56	248.742	280.642
	Alcohol Session 1	near	500	no alcohol	350.91	12.503	324.531	377.288
				alcohol	349.22	17.633	312.017	386.423
			1000	no alcohol	290.57	9.975	269.525	311.615
				alcohol	285.862	12.406	259.688	312.036
			2000	no alcohol	253.747	9.009	234.739	272.756
				alcohol	279.491	10.807	256.691	302.291
		far	500	no alcohol	333.195	15.115	301.305	365.086
				alcohol	334.085	15.301	301.803	366.367
			1000	no alcohol	282.175	9.597	261.928	302.422
				alcohol	303.993	17.588	266.884	341.101
			2000	no alcohol	260.83	9.099	241.633	280.026
				alcohol	250.225	8.865	231.523	268.928

A-6 Table 7. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ecc	4121.148	1	4121.148	7.493	0.014
ecc * AlcSess	62.610	1	62.610	0.114	0.740
Error(ecc)	9349.609	17	549.977		
int	187784.185	2	93892.092	65.366	0.000
int * AlcSess	4379.780	2	2189.890	1.525	0.232
Error(int)	48838.035	34	1436.413		
alc	18083.277	1	18083.277	18.307	0.001
alc * AlcSess	9100.659	1	9100.659	9.213	0.007
Error(alc)	16791.877	17	987.757		
ecc * int	2113.512	2	1056.756	2.172	0.129
ecc * int * AlcSess	588.600	2	294.300	0.605	0.552
Error(ecc*int)	16539.970	34	486.470		
ecc * alc	445.492	1	445.492	1.440	0.247
ecc * alc * AlcSess	146.565	1	146.565	0.474	0.501
Error(ecc*alc)	5259.622	17	309.390		
int * alc	138.241	2	69.121	0.078	0.925
int * alc * AlcSess	722.184	2	361.092	0.410	0.667
Error(int*alc)	29976.151	34	881.652		
ecc * int * alc	1244.671	2	622.335	1.273	0.293
ecc * int * alc * AlcSess	3899.374	2	1949.687	3.987	0.028
Error(ecc*int*alc)	16624.942	34	488.969		

A-6 Table 8. Single Contrasts for IA Eccentricity X Beverage Condition.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	alc diff near	0.131	1	0.131	8.225	0.010
	alc diff near	0.007	1	0.007	1.526	0.233
	alc diff far	0.2	1	0.2	12.106	0.003
Error	alc diff near	0.286	18	0.016		
	alc diff near	0.087	18	0.005		
	alc diff far	0.298	18	0.017		

A-6.3 Chapter 8 Memory Guided Saccade Task: Saccade Amplitudes for Correct Responses

A-6 Table 9. Means and SE for ME Beverage Condition, ME Eccentricity, ME Memory Interval and Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	4.273	.055	4.157	4.388
	alcohol	4.277	.081	4.106	4.448
ME Eccentricity	near	2.75	.064	2.614	2.885
	far	5.8	.079	5.633	5.967
ME Memory Interval	500	4.241	.062	4.111	4.371
	1000	4.266	.066	4.127	4.406
	2000	4.317	.077	4.156	4.479
ME Alcohol Session	Alcohol Session 1	4.266	.084	4.089	4.442
	Alcohol Session 2	4.284	.098	4.077	4.491

A-6 Table 10. . Means and SE for IA Eccentricity * Memory Interval and IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition.

Effect					Mean	Std.Error	95% Confidence Interval	
							Lower Bound	Upper Bound
IA Eccentricity * Memory Interval	near		500		2.77	.066	2.629	2.91
			1000		2.737	.059	2.611	2.862
			2000		2.742	.076	2.583	2.901
	far		500		5.712	.076	5.552	5.872
			1000		5.796	.088	5.609	5.982
			2000		5.893	.09	5.704	6.082
IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition	Alcohol Session 1	near	500	no alcohol	2.738	.087	2.555	2.921
				alcohol	2.805	.107	2.579	3.032
			1000	no alcohol	2.766	.084	2.588	2.944
				alcohol	2.774	.081	2.603	2.944
			2000	no alcohol	2.823	.089	2.636	3.011
				alcohol	2.731	.118	2.483	2.979
		far	500	no alcohol	5.681	.068	5.538	5.823
				alcohol	5.664	.138	5.372	5.956
			1000	no alcohol	5.691	.095	5.49	5.892
				alcohol	5.765	.158	5.432	6.098
			2000	no alcohol	5.959	.122	5.701	6.217
				alcohol	5.792	.147	5.482	6.102
	Alcohol Session 1	near	500	no alcohol	2.729	.102	2.515	2.944
				alcohol	2.806	.126	2.541	3.072
			1000	no alcohol	2.63	.099	2.421	2.838
				alcohol	2.777	.095	2.578	2.977
			2000	no alcohol	2.683	.104	2.463	2.902
				alcohol	2.732	.138	2.441	3.022
		far	500	no alcohol	5.762	.079	5.595	5.929
				alcohol	5.743	.162	5.4	6.086
			1000	no alcohol	5.914	.112	5.678	6.15
				alcohol	5.812	.185	5.422	6.203
			2000	no alcohol	5.895	.143	5.593	6.197
				alcohol	5.925	.172	5.561	6.288

A-6 Table 11. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ecc	517.273	1	517.273	2251.638	0.000
ecc * AlcSess	0.235	1	0.235	1.024	0.326
Error(ecc)	3.905	17	0.230		
int	0.225	2	0.112	1.915	0.163
int * AlcSess	0.036	2	0.018	0.310	0.735
Error(int)	1.996	34	0.059		
alc	0.001	1	0.001	0.009	0.928
alc * AlcSess	0.037	1	0.037	0.264	0.614
Error(alc)	2.378	17	0.140		
ecc * int	0.402	2	0.201	11.737	0.000
ecc * int * AlcSess	0.036	2	0.018	1.064	0.356
Error(ecc*int)	0.582	34	0.017		
ecc * alc	0.081	1	0.081	0.901	0.356
ecc * alc * AlcSess	0.028	1	0.028	0.317	0.581
Error(ecc*alc)	1.523	17	0.090		
int * alc	0.069	2	0.035	1.264	0.295
int * alc * AlcSess	0.097	2	0.049	1.774	0.185
Error(int*alc)	0.930	34	0.027		
ecc * int * alc	0.006	2	0.003	0.134	0.875
ecc * int * alc * AlcSess	0.091	2	0.045	2.044	0.145
Error(ecc*int*alc)	0.756	34	0.022		

The only other significant effect with respect to amplitudes is an interaction between Eccentricity and Memory Interval ($F_{(2,16)}$= 11.37, p<.001, see Table 7). This interaction is caused by the fact that differences between near and far eccentricities are more pronounced between the short and medium as well as the short and long memory interval, compared to the medium significant difference in saccade amplitudes to far target location between short and long memory intervals ($F_{(1,17)}$= 6.02, p<.05). All other comparisons showed no differences for saccade amplitudes (all ps>.10).

A-6 Table 12. Single Contrasts for IA Eccentricity X Memory Interval.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	near, short-medium	0.02	1	0.02	1.099	0.309
	near, short-long	0.014	1	0.014	0.75	0.398
	near, medium-long	0.001	1	0.001	0.015	0.904
	far, short-long	0.001	1	0.001	0.015	0.904
	far, short-medium	0.001	1	0.001	0.015	0.904
	far, medium-long	0.001	1	0.001	0.015	0.904
	diff near-far btw short medium	0.25	1	0.25	7.803	0.012
	diff near-far btw short long	0.8	1	0.8	30.309	0
	diff near-far btw medium long	0.156	1	0.156	3.513	0.078
Error	near, short-medium	0.312	17	0.018		
	near, short-long	0.318	17	0.019		
	near, medium-long	0.635	17	0.037		
	far, short-long	0.635	17	0.037		
	far, short-medium	0.635	17	0.037		
	far, medium-long	0.635	17	0.037		
	diff near-far btw short medium	0.545	17	0.032		
	diff near-far btw short long	0.449	17	0.026		
	diff near-far btw medium long	0.753	17	0.044		

A-6.4 Chapter 8 Memory Guided Saccade Task: Saccade Amplitude Variability for Correct Responses

A-6 Table 13. Means and SE for ME Beverage Condition, ME Eccentricity, ME Memory Interval and Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	.645	.019	.604	.685
	alcohol	.651	.026	.597	.705
ME Eccentricity	near	.58	.019	.539	.62
	far	.716	.028	.657	.775
ME Memory Interval	500	.617	.022	.57	.664
	1000	.64	.025	.588	.692
	2000	.686	.022	.639	.734
ME Alcohol Session	Alcohol Session 1	4.266	.084	4.089	4.442
	Alcohol Session 2	4.284	.098	4.077	4.491

A-6 Table 14. Means and SE for IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition.

Effect					Mean	Std.Error	95% Confidence Interval	
							Lower Bound	Upper Bound
IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition	Alcohol Session 1	near	500	no alcohol	.509	.042	.421	.597
				alcohol	.569	.038	.488	.65
			1000	no alcohol	.544	.037	.465	.622
				alcohol	.543	.036	.466	.62
			2000	no alcohol	.563	.034	.491	.635
				alcohol	.645	.049	.541	.749
		far	500	no alcohol	.657	.042	.567	.746
				alcohol	.592	.056	.474	.711
			1000	no alcohol	.655	.043	.564	.745
				alcohol	.775	.056	.657	.892
			2000	no alcohol	.775	.042	.688	.863
				alcohol	.749	.055	.633	.864
	Alcohol Session 1	near	500	no alcohol	.584	.049	.481	.687
				alcohol	.569	.045	.474	.665
			1000	no alcohol	.569	.044	.477	.661
				alcohol	.604	.043	.514	.693
			2000	no alcohol	.627	.04	.542	.711
				alcohol	.63	.058	.508	.753
		far	500	no alcohol	.726	.05	.621	.831
				alcohol	.732	.066	.592	.871
			1000	no alcohol	.748	.05	.641	.854
				alcohol	.682	.065	.544	.82
			2000	no alcohol	.782	.049	.679	.885
				alcohol	.718	.064	.583	.854

A-6 Table 15. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ecc	1.031	1	1.031	29.774	0.000
ecc * AlcSess	0.000	1	0.000	0.007	0.933
Error(ecc)	0.588	17	0.035		
int	0.183	2	0.091	7.381	0.002
int * AlcSess	0.042	2	0.021	1.705	0.197
Error(int)	0.420	34	0.012		
alc	0.002	1	0.002	0.100	0.756
alc * AlcSess	0.028	1	0.028	1.461	0.243
Error(alc)	0.331	17	0.019		
ecc * int	0.009	2	0.005	0.344	0.711
ecc * int * AlcSess	0.035	2	0.018	1.280	0.291
Error(ecc*int)	0.465	34	0.014		
ecc * alc	0.026	1	0.026	1.636	0.218
ecc * alc * AlcSess	0.000	1	0.000	0.030	0.865
Error(ecc*alc)	0.270	17	0.016		
int * alc	0.007	2	0.004	0.304	0.739
int * alc * AlcSess	0.013	2	0.007	0.556	0.578
Error(int*alc)	0.410	34	0.012		
ecc * int * alc	0.023	2	0.011	0.909	0.413
ecc * int * alc * AlcSess	0.082	2	0.041	2529.000	0.095
Error(ecc*int*alc)	0.428	34	0.013		

A-6.5 Chapter 8 Memory Guided Saccade Task: Proportion Premature Responses

A-6 Table 16. Means and SE for ME Beverage Condition, ME Eccentricity, ME Memory Interval and Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	.223	.029	.162	.283
	alcohol	.23	.028	.172	.288
ME Eccentricity	near	.237	.028	.177	.296
	far	.216	.026	.161	.271
ME Memory Interval	500	.198	.027	.142	.254
	1000	.221	.025	.167	.274
	2000	.261	.03	.198	.323
ME Alcohol Session	Alcohol Session 1	.234	.036	.159	.309
	Alcohol Session 2	.218	.039	.136	.301

A-6 Table 17. Means and SE for IA Alcohol Session * Beverage Condition and IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition.

Effect					Mean	Std.Error	95% Confidence Interval	
							Lower Bound	Upper Bound
IA Alcohol Session * Beverage Condition	Alcohol Session 1			no alcohol	.206	.039	.125	.288
				alcohol	.262	.037	.184	.339
	Alcohol Session 2			no alcohol	.206	.039	.125	.288
				alcohol	.262	.037	.184	.339
IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition	Alcohol Session 1	near	500	no alcohol	.21	.04	.126	.294
				alcohol	.216	.052	.107	.324
			1000	no alcohol	.185	.032	.117	.253
				alcohol	.259	.042	.17	.347
			2000	no alcohol	.27	.051	.162	.378
				alcohol	.324	.052	.216	.432
		far	500	no alcohol	.159	.047	.06	.258
				alcohol	.222	.031	.157	.286
			1000	no alcohol	.196	.047	.096	.296
				alcohol	.261	.04	.177	.346
			2000	no alcohol	.219	.04	.135	.303
				alcohol	.29	.036	.214	.365
	Alcohol Session 1	near	500	no alcohol	.177	.044	.084	.27
				alcohol	.198	.057	.078	.318
			1000	no alcohol	.236	.036	.161	.311
				alcohol	.188	.046	.09	.285
			2000	no alcohol	.295	.057	.176	.414
				alcohol	.281	.057	.162	.401
		far	500	no alcohol	.229	.052	.12	.338
				alcohol	.17	.034	.099	.241
			1000	no alcohol	.253	.052	.143	.364
				alcohol	.188	.044	.094	.281
			2000	no alcohol	.243	.044	.15	.336
				alcohol	.163	.04	.08	.247

A-6 Table 18. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ecc	0.025	1	0.025	2.226	0.153
ecc * AlcSess	0.000	1	0.000	0.005	0.943
Error(ecc)	0.200	18	0.011		
int	0.161	2	0.080	15.282	0.000
int * AlcSess	0.006	2	0.003	0.572	0.569
Error(int)	0.189	36	0.005		
alc	0.003	1	0.003	0.139	0.713
alc * AlcSess	0.138	1	0.138	6.339	0.021
Error(alc)	0.393	18	0.022		
ecc * int	0.058	2	0.029	7.119	0.002
ecc * int * AlcSess	0.015	2	0.007	1.837	0.174
Error(ecc*int)	0.146	36	0.004		
ecc * alc	0.004	1	0.004	0.645	0.432
ecc * alc * AlcSess	0.022	1	0.022	2.518	0.130
Error(ecc*alc)	0.110	18	0.006		
int * alc	0.000	2	0.000	0.003	0.997
int * alc * AlcSess	0.015	2	0.007	1.665	0.203
Error(int*alc)	0.159	36	0.004		
ecc * int * alc	0.000	2	0.000	0.044	0.957
ecc * int * alc * AlcSess	0.010	2	0.005	0.897	0.417
Error(ecc*int*alc)	0.205	36	0.006		

A-6.6 Chapter 8 Memory Guided Saccade Task: Saccade Latencies for Premature Responses

A-6 Table 19. Means and SE for ME Beverage Condition, ME Eccentricity, ME Memory Interval and Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	1422.904	102.89	1202.226	1643.582
	alcohol	1429.732	67.109	1285.797	1573.667
ME Eccentricity	near	1408.571	69.615	1259.262	1557.88
	far	1444.065	94.625	1241.116	1647.015
ME Memory Interval	500	1242.111	79.986	1070.558	1413.665
	1000	1413.774	85.747	1229.864	1597.684
	2000	1623.069	93.324	1422.91	1823.229
ME Alcohol Session	Alcohol Session 1	1343.55	112.791	1101.637	1585.462
	Alcohol Session 2	1509.087	112.791	1267.174	1750.999

A-6 Table 20. Means and SE for IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition.

Effect					Mean	Std.Error	95% Confidence Interval	
							Lower Bound	Upper Bound
IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition	Alcohol Session 1	near	500	no alcohol	1079.195	128.89	802.753	1355.636
				alcohol	1187.424	140.881	885.264	1489.583
			1000	no alcohol	1315.093	209.442	865.885	1764.301
				alcohol	1351.092	123.208	1086.838	1615.346
			2000	no alcohol	1347.154	174.785	972.277	1722.032
				alcohol	1596.37	143.798	1287.955	1904.785
		far	500	no alcohol	1290.557	177.905	908.989	1672.125
				alcohol	1205.838	179.27	821.343	1590.333
			1000	no alcohol	1237.887	213.201	780.617	1695.158
				alcohol	1448.516	126.934	1176.27	1720.761
			2000	no alcohol	1543.045	171.019	1176.246	1909.843
				alcohol	1520.425	151.41	1195.682	1845.167
	Alcohol Session 1	near	500	no alcohol	1245.19	128.89	968.748	1521.632
				alcohol	1290.013	140.881	987.853	1592.172
			1000	no alcohol	1416.649	209.442	967.441	1865.857
				alcohol	1694.817	123.208	1430.563	1959.071
			2000	no alcohol	1803.761	174.785	1428.884	2178.639
				alcohol	1576.092	143.798	1267.677	1884.507
		far	500	no alcohol	1381.965	177.905	1000.397	1763.532
				alcohol	1256.709	179.27	872.214	1641.205
			1000	no alcohol	1405.163	213.201	947.893	1862.434
				alcohol	1440.972	126.934	1168.726	1713.217
			2000	no alcohol	2009.187	171.019	1642.389	2375.986
				alcohol	1588.521	151.41	1263.778	1913.263

A-6 Table 21. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ecc	60473.059	1	60473.059	0.584	0.457
ecc * AlcSess	32853.591	1	32853.591	0.317	0.582
Error(ecc)	1449137.069	14	103509.791		
int	4659239.293	2	2329619.646	21.558	0.000
int * AlcSess	161530.934	2	80765.467	0.747	0.483
Error(int)	3025763.653	28	108062.988		
alc	2238.082	1	2238.082	0.010	0.922
alc * AlcSess	276960.016	1	276960.016	1.218	0.288
Error(alc)	3182958.230	14	227354.159		
ecc * int	224770.208	2	112385.104	1.621	0.216
ecc * int * AlcSess	74223.824	2	37111.912	0.535	0.591
Error(ecc*int)	1941531.792	28	69340.421		
ecc * alc	267359.438	1	267359.438	3.111	0.100
ecc * alc * AlcSess	33134.042	1	33134.042	0.386	0.545
Error(ecc*alc)	1203283.644	14	85948.832		
int * alc	493141.504	2	246570.752	1.124	0.339
int * alc * AlcSess	503886.594	2	251943.297	1.149	0.332
Error(int*alc)	6141625.768	28	219343.777		
ecc * int * alc	85085.223	2	42542.611	0.524	0.598
ecc * int * alc * AlcSess	147484.514	2	73742.257	0.908	0.415
Error(ecc*int*alc)	2274746.158	28	81240.934		

A-6.7 Chapter 8 Memory Guided Saccade Task: Saccade Amplitudes for Premature Responses

A-6 Table 22. Means and SE for ME Beverage Condition, ME Eccentricity, ME Memory Interval and Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	3.807	.185	3.411	4.204
	alcohol	3.805	.209	3.358	4.253
ME Eccentricity	near	3.5	.204	3.061	3.938
	far	4.113	.203	3.678	4.548
ME Memory Interval	500	3.75	.194	3.334	4.166
	1000	3.869	.23	3.376	4.362
	2000	3.8	.206	3.359	4.241
ME Alcohol Session	Alcohol Session 1	3.76	.268	3.185	4.334
	Alcohol Session 2	3.853	.268	3.279	4.428

A-6 Table 23. Means and SE for IA Alcohol Session * Beverage Condition and IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition.

Effect					Mean	Std.Error	95% Confidence Interval	
							Lower Bound	Upper Bound
IA Alcohol Session * Beverage Condition	Alcohol Session 1			no alcohol	3.605	.261	3.045	4.165
				alcohol	3.914	.295	3.281	4.547
	Alcohol Session 2			no alcohol	4.009	.261	3.449	4.57
				alcohol	3.697	.295	3.064	4.33
IA Alcohol Session * Eccentricity * Memory Interval * Beverage Condition	Alcohol Session 1	near	500	no alcohol	3.304	.275	2.713	3.894
				alcohol	3.26	.463	2.267	4.252
			1000	no alcohol	3.473	.42	2.571	4.375
				alcohol	3.993	.308	3.332	4.654
			2000	no alcohol	3.228	.294	2.598	3.858
				alcohol	3.474	.439	2.531	4.416
		far	500	no alcohol	3.933	.354	3.174	4.692
				alcohol	4.304	.339	3.577	5.032
			1000	no alcohol	3.965	.375	3.161	4.769
				alcohol	4.434	.561	3.23	5.638
			2000	no alcohol	3.727	.344	2.99	4.464
				alcohol	4.02	.321	3.332	4.708
	Alcohol Session 1	near	500	no alcohol	3.727	.344	2.99	4.464
				alcohol	4.02	.321	3.332	4.708
			1000	no alcohol	3.74	.42	2.839	4.642
				alcohol	3.251	.308	2.59	3.912
			2000	no alcohol	3.645	.294	3.015	4.275
				alcohol	3.691	.439	2.748	4.633
		far	500	no alcohol	4.095	.354	3.336	4.854
				alcohol	4.169	.339	3.442	4.897
			1000	no alcohol	4.434	.375	3.63	5.239
				alcohol	3.662	.561	2.458	4.866
			2000	no alcohol	4.564	.344	3.827	5.301
				alcohol	4.051	.321	3.363	4.739

A-6 Table 24. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
ecc	18.076	1	18.076	16.930	0.001
ecc * AlcSess	0.001	1	0.001	0.001	0.974
Error(ecc)	14.948	14	1.068		
int	0.456	2	0.228	0.284	0.755
int * AlcSess	2.597	2	1.298	1.615	0.217
Error(int)	22.507	28	0.804		
alc	0.000	1	0.000	0.000	0.987
alc * AlcSess	4.636	1	4.636	8.134	0.013
Error(alc)	7.978	14	0.570		
ecc * int	0.491	2	0.246	0.537	0.590
ecc * int * AlcSess	0.202	2	0.101	0.221	0.803
Error(ecc*int)	12.804	28	0.457		
ecc * alc	0.006	1	0.006	0.010	0.922
ecc * alc * AlcSess	0.305	1	0.305	0.484	0.498
Error(ecc*alc)	8.834	14	0.631		
int * alc	0.112	2	0.056	0.073	0.930
int * alc * AlcSess	1.663	2	0.831	1.084	0.352
Error(int*alc)	21.480	28	0.767		
ecc * int * alc	0.874	2	0.437	1.024	0.372
ecc * int * alc * AlcSess	0.129	2	0.064	0.151	0.861
Error(ecc*int*alc)	11.946	28	0.427		

A-7 Chapter 9 Task Switching

A-7.1 Chapter 9 Task Switching: Error Rates

A-7 Table 1. Means and SE for ME Beverage Condition, ME Task, ME TaskSwitch, ME ResponseSwitch and ME Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	.104	.015	.073	.135
	alcohol	.103	.016	.07	.136
ME Task	pro	.025	.005	.016	.035
	anti	.182	.028	.124	.24
ME TaskSwitch	no task switch	.098	.016	.065	.13
	task switch	.109	.015	.078	.14
ME ResponseSwitch	no response switch	.096	.014	.066	.125
	response switch	.111	.016	.077	.145
ME Alcohol Session	Alcohol Session 1	.079	.021	.035	.123
	Alcohol Session 2	.128	.021	.084	.172

A-7 Table 2. Means and SE for IA TaskSwitch X Beverage Condition and IA Task X TaskSwitch X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
IA TaskSwitch * Beverage Condition	no task switch		no alcohol	.09	.015	.06	.121
			alcohol	.105	.018	.068	.142
	task switch		no alcohol	.117	.016	.084	.151
			alcohol	.101	.015	.07	.132
IA Task * TaskSwitch * Beverage Condition	pro	no task switch	no alcohol	.012	.006	.	.024
			alcohol	.019	.006	.007	.031
		task switch	no alcohol	.034	.007	.02	.047
			alcohol	.036	.006	.024	.048
	anti	no task switch	no alcohol	.169	.028	.111	.226
			alcohol	.192	.033	.124	.26
		task swtich	no alcohol	.201	.029	.142	.261
			alcohol	.165	.028	.107	.224

A-7 Table 3. ANOVA Output.

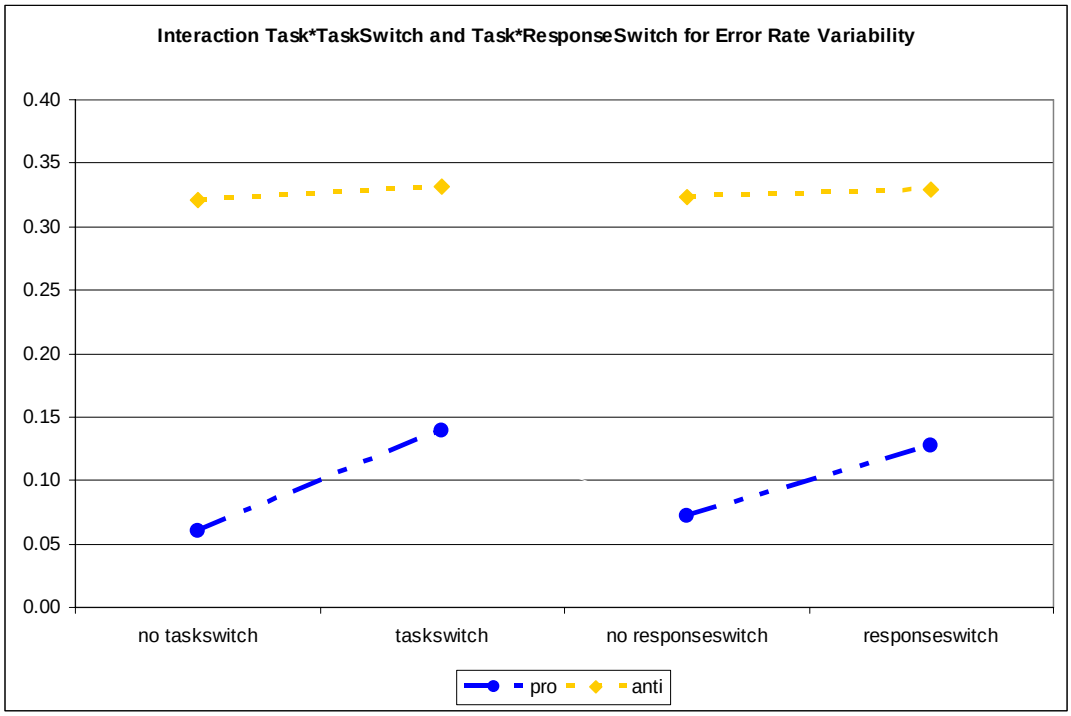
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	2.356	1	2.356	35.136	0.000
Task * AlcSess	0.216	1	0.216	3.225	0.086
Error(Task)	1.475	22	0.067		
TaskSwitch	0.012	1	0.012	3.234	0.086
TaskSwitch * AlcSess	0.001	1	0.001	0.150	0.703
Error(TaskSwitch)	0.084	22	0.004		
ResponseSwitch	0.023	1	0.023	5.418	0.030
ResponseSwitch * AlcSess	0.005	1	0.005	1.167	0.292
Error(ResponseSwitch)	0.094	22	0.004		
Alcohol	0.000	1	0.000	0.017	0.898
Alcohol * AlcSess	0.015	1	0.015	3.152	0.090
Error(Alcohol)	0.108	22	0.005		
Task * TaskSwitch	0.006	1	0.006	3.225	0.086
Task * TaskSwitch * AlcSess	0.005	1	0.005	2.798	0.109
Error(Task*TaskSwitch)	0.042	22	0.002		
Task * ResponseSwitch	0.001	1	0.001	0.254	0.619
Task * ResponseSwitch * AlcSess	0.000	1	0.000	0.003	0.956
Error(Task*ResponseSwitch)	0.077	22	0.004		
TaskSwitch * ResponseSwitch	0.001	1	0.001	0.140	0.712
TaskSwitch * ResponseSwitch * AlcSess	0.018	1	0.018	1.947	0.177
Error(TaskSwitch*ResponseSwitch)	0.200	22	0.009		
Task * TaskSwitch * ResponseSwitch	0.010	1	0.010	0.906	0.352
Task * TaskSwitch * ResponseSwitch * AlcSess	0.011	1	0.011	1.004	0.327
Error(Task*TaskSwitch*ResponseSwitch)	0.252	22	0.011		
Task * Alcohol	0.003	1	0.003	0.729	0.403
Task * Alcohol * AlcSess	3.26E-06	1	3.26E-06	0.001	0.977
Error(Task*Alcohol)	0.087	22	0.004		
TaskSwitch * Alcohol	0.024	1	0.024	7.598	0.012
TaskSwitch * Alcohol * AlcSess	7.76E-06	1	7.76E-06	0.002	0.961
Error(TaskSwitch*Alcohol)	0.069	22	0.003		
Task * TaskSwitch * Alcohol	0.018	1	0.018	11.918	0.002
Task * TaskSwitch * Alcohol * AlcSess	0.001	1	0.001	0.755	0.394
Error(Task*TaskSwitch*Alcohol)	0.034	22	0.002		
ResponseSwitch * Alcohol	0.007	1	0.007	2.078	0.164
ResponseSwitch * Alcohol * AlcSess	0	1	0	0.064	0.802
Error(ResponseSwitch*Alcohol)	0.079	22	0.004		
Task * ResponseSwitch * Alcohol	0.004	1	0.004	0.979	0.333
Task * ResponseSwitch * Alcohol * AlcSess	4.64E-05	1	4.64E-05	0.012	0.912
Error(Task*ResponseSwitch*Alcohol)	0.082	22	0.004		
TaskSwitch * ResponseSwitch * Alcohol	5.56E-05	1	5.56E-05	0.011	0.918
TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0	1	0	0.044	0.836
Error(TaskSwitch*ResponseSwitch*Alcohol)	0.113	22	0.005		
Task * TaskSwitch * ResponseSwitch * Alcohol	0.001	1	0.001	0.299	0.59
Task * TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0	1	0	0.064	0.802
Error(Task*TaskSwitch*ResponseSwitch*Alcohol)	0.099	22	0.004		

A-7 Table 4. Single Contrasts for significant IA.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	pro alc diff switch	0.007	1	0.007	4.713	0.041
	pro no alc diff switch	0.011	1	0.011	21.155	0.000
	pro no switch diff alccond	0.001	1	0.001	1.094	0.307
	pro switch diff alccond	0.000	1	0.000	0.232	0.635
	pro diff vs. anti diff noalc	0.003	1	0.003	3.223	0.086
	anti alc diff switch	0.017	1	0.017	4.271	0.051
	anti no alc diff switch	0.026	1	0.026	5.806	0.025
	anti no switch diff alccond	0.013	1	0.013	2.303	0.143
	anti switch diff alccond	0.031	1	0.031	4.923	0.037
Error	pro alc diff switch	0.034	22	0.002		
	pro no alc diff switch	0.011	22	0.001		
	pro no switch diff alccond	0.020	22	0.001		
	pro switch diff alccond	0.015	22	0.001		
	pro diff vs. anti diff noalc	0.021	22	0.001		
	anti alc diff switch	0.085	22	0.004		
	anti no alc diff switch	0.098	22	0.004		
	anti no switch diff alccond	0.123	22	0.006		
	anti switch diff alccond	0.139	22	0.006		

A-7.2 Chapter 9 Task Switching: Error Rate Variability

Error rate variability showed main effects for Task ($F_{(1,22)}$= 111.76, p<.001), TaskSwitch ($F_{(1,22)}$= 13.10, p<.05) and ResponseSwitch ($F_{(1,22)}$= 7.62, p<.05) demonstrating significant higher variability for error rates in the anti saccade task, task switch and response switch conditions. However, these main effects were qualified by significant Task X TaskSwitch ($F_{(1,22)}$= 13.25, p<.05) and Task X ResponseSwitch ($F_{(1,22)}$= 10.33, p<.05) interactions. Interestingly, in both cases the interaction effects were caused by a greater increase in variability in anti saccade compared with pro saccade performance in the no switch conditions. Differences between switch conditions were significantly different in the pro saccade task (TaskSwitch: $F_{(1,22)}$= 27.37, p<.001; ResponseSwitch: $F_{(1,22)}$= 18.49, p<.001), but not in the anti saccade task (TaskSwitch: $F_{(1,22)}$= .54, p>.10; ResponseSwitch: $F_{(1,22)}$= .16, p>.10). A-7 Figure 1 illustrates these effects.



A-7 Figure 1. Interaction Task by TaskSwitch for error rate variability.

A-7 Table 5. Means and SE for ME Beverage Condition, ME Task, ME TaskSwitch, ME ResponseSwitch and ME Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	.21	.016	.177	.244
	alcohol	.216	.015	.184	.247
ME Task	pro	.1	.012	.075	.125
	anti	.326	.023	.279	.374
ME TaskSwitch	no task switch	.191	.018	.154	.227
	task switch	.236	.014	.206	.265
ME ResponseSwitch	no response switch	.198	.015	.167	.229
	response switch	.228	.017	.194	.263
ME Alcohol Session	Alcohol Session 1	.203	.021	.16	.247
	Alcohol Session 2	.223	.021	.179	.266

A-7 Table 6. Means and SE for IA Task X Beverage Condition, IA Alcohol Session X Beverage Condition, Task X TaskSwitch and IA Task X ResponseSwitch.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
IA Task * Beverage Condition	pro	no alcohol	.089	.014	.06	.118
		alcohol	.111	.012	.086	.136
	anti	no alcohol	.332	.025	.281	.383
		alcohol	.321	.024	.272	.37
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	.188	.023	.14	.235
		alcohol	.219	.021	.175	.264
	Alcohol Session 2	no alcohol	.233	.023	.186	.281
		alcohol	.212	.021	.168	.257
IA Task * TaskSwitch	pro	no task switch	.061	.014	.031	.09
		task switch	.139	.014	.11	.169
	anti	no task switch	.32	.026	.267	.374
		task switch	.332	.022	.286	.378
IA Task * ResponseSwitch	pro	no response switch	.072	.015	.041	.103
		response switch	.127	.012	.102	.153
	anti	no response switch	.324	.022	.278	.369
		response switch	.329	.026	.276	.383

A-7 Table 7. ANOVA Output.

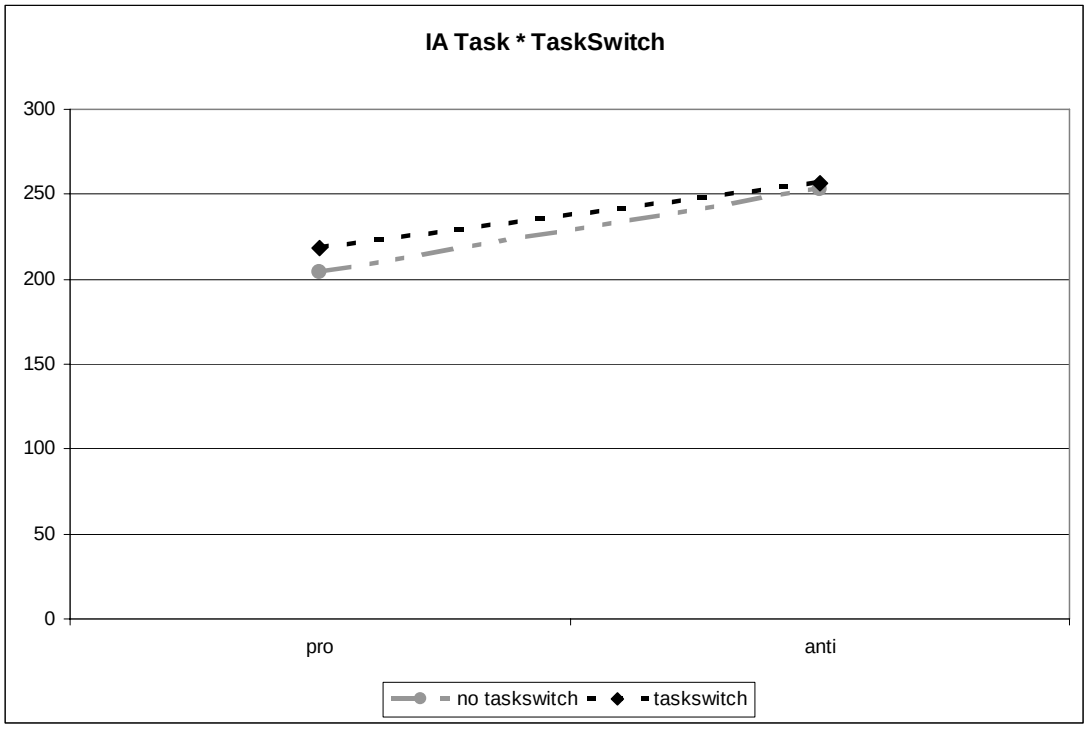
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	4.920	1	4.920	111.760	0.000
Task * AlcSess	0.016	1	0.016	0.375	0.547
Error(Task)	0.968	22	0.044		
TaskSwitch	0.196	1	0.196	13.064	0.002
TaskSwitch * AlcSess	0.006	1	0.006	0.398	0.534
Error(TaskSwitch)	0.330	22	0.015		
ResponseSwitch	0.088	1	0.088	7.623	0.011
ResponseSwitch * AlcSess	0.026	1	0.026	2.237	0.149
Error(ResponseSwitch)	0.255	22	0.012		
Alcohol	0.003	1	0.003	0.237	0.631
Alcohol * AlcSess	0.067	1	0.067	5.941	0.023
Error(Alcohol)	0.248	22	0.011		
Task * TaskSwitch	0.108	1	0.108	13.245	0.001
Task * TaskSwitch * AlcSess	0.013	1	0.013	1.546	0.227
Error(Task*TaskSwitch)	0.179	22	0.008		
Task * ResponseSwitch	0.059	1	0.059	10.330	0.004
Task * ResponseSwitch * AlcSess	0.001	1	0.001	0.151	0.701
Error(Task*ResponseSwitch)	0.126	22	0.006		
TaskSwitch * ResponseSwitch	0.022	1	0.022	1.713	0.204
TaskSwitch * ResponseSwitch * AlcSess	0.043	1	0.043	3.396	0.079
Error(TaskSwitch*ResponseSwitch)	0.280	22	0.013		
Task * TaskSwitch * ResponseSwitch	0.021	1	0.021	1.979	0.173
Task * TaskSwitch * ResponseSwitch * AlcSess	0.009	1	0.009	0.891	0.355
Error(Task*TaskSwitch*ResponseSwitch)	0.232	22	0.011		
Task * Alcohol	0.027	1	0.027	4.457	0.046
Task * Alcohol * AlcSess	7.00E-03	1	7.00E-03	1.107	0.304
Error(Task*Alcohol)	0.131	22	0.006		
TaskSwitch * Alcohol	0.018	1	0.018	1.708	0.205
TaskSwitch * Alcohol * AlcSess	1.32E-05	1	1.32E-05	0.001	0.972
Error(TaskSwitch*Alcohol)	0.232	22	0.011		
Task * TaskSwitch * Alcohol	0.007	1	0.007	1.106	0.304
Task * TaskSwitch * Alcohol * AlcSess	0.011	1	0.011	1.821	0.191
Error(Task*TaskSwitch*Alcohol)	0.13	22	0.006		
ResponseSwitch * Alcohol	0.034	1	0.034	4.113	0.055
ResponseSwitch * Alcohol * AlcSess	0.002	1	0.002	0.251	0.622
Error(ResponseSwitch*Alcohol)	0.184	22	0.008		
Task * ResponseSwitch * Alcohol	2.35E-07	1	2.35E-07	0	0.996
Task * ResponseSwitch * Alcohol * AlcSess	2.20E-02	1	2.20E-02	2.736	0.112
Error(Task*ResponseSwitch*Alcohol)	0.175	22	0.008		
TaskSwitch * ResponseSwitch * Alcohol	0.00E+00	1	0.00E+00	0.032	0.86
TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0.014	1	0.014	1.369	0.254
Error(TaskSwitch*ResponseSwitch*Alcohol)	0.217	22	0.01		
Task * TaskSwitch * ResponseSwitch * Alcohol	0.004	1	0.004	0.502	0.486
Task * TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0	1	0	0.045	0.834
Error(Task*TaskSwitch*ResponseSwitch*Alcohol)	0.155	22	0.007		

A-7 Table 8. Single Contrasts for significant IA.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	pro, no taskswitch-taskswitch	0.148	1	0.148	27.367	0.000
	anti, no taskwitch-taskswitch	0.003	1	0.003	0.535	0.472
	pro, no responsewitch-responseswitch	0.073	1	0.073	18.487	0.000
	anti, no responsewitch-responseswitch	0.001	1	0.001	0.156	0.697
	pro, no alcohol-alcohol	0.012	1	0.012	4.136	0.054
	anti, no alcohol-alcohol	0.003	1	0.003	0.531	0.474
	diff alc cond by task	0.027	1	0.027	4.457	0.046
Error	pro, no taskswitch-taskswitch	0.119	22	0.005		
	anti, no taskwitch-taskswitch	0.135	22	0.006		
	pro, no responsewitch-responseswitch	0.087	22	0.004		
	anti, no responsewitch-responseswitch	0.104	22	0.005		
	pro, no alcohol-alcohol	0.061	22	0.003		
	anti, no alcohol-alcohol	0.128	22	0.006		
	diff alc cond by task	0.131	22	0.006		

A-7.3 Chapter 9 Task Switching: Primary Saccade Latencies

TaskSwitch showed an interaction with Task ($F_{(1,22)}$= 9.52, p<.05) as well as with ResponseSwitch ($F_{(1,22)}$= 17.01, p<.001). Latencies were longer in the anti saccade compared to the pro saccade task under no task switch ($F_{(1,22)}$= 116.30, p<.001) and task switch ($F_{(1,22)}$= 102.08, p<.001) conditions, but the difference between the TaskSwitch conditions is only significant in the pro saccade task ($F_{(1,22)}$= 33.58, p<.001) and not present in the anti saccade task ($F_{(1,22)}$= 1.85, p>.10).



A-7 Figure 2. Interaction between Task and TaskSwitch for saccade latencies in trials with correct responses.

A-7 Table 9. Means and SE for ME Beverage Condition, ME Task, ME TaskSwitch, ME ResponseSwitch and ME Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	221.242	6.629	207.495	234.989
	alcohol	245.022	8.195	228.027	262.016
ME Task	pro	211.408	7.065	196.755	226.061
	anti	254.856	7.74	238.804	270.908
ME TaskSwitch	no task switch	228.91	6.897	214.606	243.215
	task switch	237.354	7.504	221.792	252.916
ME ResponseSwitch	no response switch	231.877	7.537	216.247	247.507
	response switch	234.387	6.891	220.095	248.679
ME Alcohol Session	Alcohol Session 1	236.498	10.132	215.486	257.51
	Alcohol Session 2	229.766	10.132	208.754	250.777

A-7 Table 10. Means and SE for IA Alcohol Session X Beverage Condition, Task X TaskSwitch and IA Task X ResponseSwitch.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
IA Alcohol Session * Beverage Condition	Alcohol Session 1	no alcohol	215.1	9.374	195.658	234.541
		alcohol	257.897	11.589	233.863	281.931
	Alcohol Session 2	no alcohol	227.384	9.374	207.943	246.826
		alcohol	232.147	11.589	208.113	256.181
IA Task * TaskSwitch	pro	no task switch	204.523	6.873	190.268	218.777
		task switch	218.293	7.445	202.854	233.733
	anti	no task switch	253.298	7.625	237.485	269.11
		task switch	256.414	8.019	239.784	273.045
IA Task * ResponseSwitch	pro	no response switch	232.16	7.144	217.344	246.976
		response switch	225.66	6.954	211.239	240.082
	anti	no response switch	231.594	8.138	214.717	248.471
		response switch	243.114	7.085	228.42	257.807

A-7 Table 11. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	181221.123	1	181221.123	131.417	0.000
Task * AlcSess	5752.223	1	5752.223	4.171	0.053
Error(Task)	30337.542	22	1378.979		
TaskSwitch	6844.358	1	6844.358	28.969	0.000
TaskSwitch * AlcSess	179.880	1	179.880	0.761	0.392
Error(TaskSwitch)	5197.843	22	236.266		
ResponseSwitch	604.864	1	604.864	1.918	0.180
ResponseSwitch * AlcSess	10.982	1	10.982	0.035	0.854
Error(ResponseSwitch)	6936.926	22	315.315		
Alcohol	54286.458	1	54286.458	33.500	0.000
Alcohol * AlcSess	34718.929	1	34718.929	21.425	0.000
Error(Alcohol)	35650.794	22	1620.491		
Task * TaskSwitch	2724.200	1	2724.200	9.516	0.005
Task * TaskSwitch * AlcSess	738.752	1	738.752	2.581	0.122
Error(Task*TaskSwitch)	6297.990	22	286.272		
Task * ResponseSwitch	45.929	1	45.929	0.277	0.604
Task * ResponseSwitch * AlcSess	38.665	1	38.665	0.233	0.634
Error(Task*ResponseSwitch)	3645.721	22	165.715		
TaskSwitch * ResponseSwitch	7792.529	1	7792.529	17.007	0.000
TaskSwitch * ResponseSwitch * AlcSess	4.570	1	4.570	0.010	0.921
Error(TaskSwitch*ResponseSwitch)	10080.440	22	458.202		
Task * TaskSwitch * ResponseSwitch	550.577	1	550.577	1.740	0.201
Task * TaskSwitch * ResponseSwitch * AlcSess	255.77	1	255.77	0.808	0.378
Error(Task*TaskSwitch*ResponseSwitch)	6960.439	22	316.384		
Task * Alcohol	371.979	1	371.979	0.776	0.388
Task * Alcohol * AlcSess	5.36E+01	1	5.36E+01	0.112	0.741
Error(Task*Alcohol)	10540.46	22	479.112		
TaskSwitch * Alcohol	315.469	1	315.469	2.15	0.157
TaskSwitch * Alcohol * AlcSess	2.66E+03	1	2.62E+02	1.845	0.215
Error(TaskSwitch*Alcohol)	3227.598	22	146.709		
Task * TaskSwitch * Alcohol	65.197	1	65.197	0.718	0.406
Task * TaskSwitch * Alcohol * AlcSess	0.171	1	0.171	0.002	0.966
Error(Task*TaskSwitch*Alcohol)	1998.116	22	90.823		
ResponseSwitch * Alcohol	30.933	1	30.933	0.407	0.53
ResponseSwitch * Alcohol * AlcSess	173.17	1	173.17	2.276	0.146
Error(ResponseSwitch*Alcohol)	1673.513	22	76.069		
Task * ResponseSwitch * Alcohol	7.97E+00	1	7.97E+00	0.078	0.782
Task * ResponseSwitch * Alcohol * AlcSess	2.40E+01	1	2.40E+01	0.236	0.632
Error(Task*ResponseSwitch*Alcohol)	2233.576	22	101.526		
TaskSwitch * ResponseSwitch * Alcohol	5.15E+02	1	5.15E+02	2.02	0.169
TaskSwitch * ResponseSwitch * Alcohol * AlcSess	464.493	1	464.493	1.823	0.191
Error(TaskSwitch*ResponseSwitch*Alcohol)	5604.363	22	254.744		
Task * TaskSwitch * ResponseSwitch * Alcohol	584.153	1	584.153	1.994	0.172
Task * TaskSwitch * ResponseSwitch * Alcohol * AlcSess	322.12	1	322.12	1.1	0.306
Error(Task*TaskSwitch*ResponseSwitch*Alcohol)	6444.203	22	292.918		

A-7 Table 12. Single Contrasts for significant IA.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	no switch, pro-anti	57095.814	1	57095.814	116.297	0.000
	switch, pro-anti	34876.847	1	34876.847	102.075	0.000
	pro, no switch-switch	4551.155	1	4551.155	33.583	0.000
	anti, no switch-switch	233.124	1	233.124	1.854	0.187
	diff taskswitch no rspwitch vs diff taskwitch rspswitch	7792.529	1	7792.529	17.007	0.000
	no taskswitch, no rspswitch-rspswitch	1013.828	1	1013.828	4.976	0.036
	taskswitch, no rspswitch-rspswitch	3184.868	1	3184.868	17.403	0.000
	no rspswitch, no taskswitch-taskswitch	7.686	1	7.686	0.044	0.836
	rspswitch, no taskswitch-taskswitch	7310.758	1	7310.758	42.600	0.000
Error	no switch, pro-anti	10800.871	22	490.949		
	switch, pro-anti	7516.895	22	341.677		
	pro, no switch-switch	2981.448	22	135.520		
	anti, no switch-switch	2766.469	22	125.749		
	diff taskswitch no rspwitch vs diff taskwitch rspswitch	10080.440	22	458.202		
	no taskswitch, no rspswitch-rspswitch	4482.541	22	203.752		
	taskswitch, no rspswitch-rspswitch	4026.142	22	183.006		
	no rspswitch, no taskswitch-taskswitch	3863.662	22	175.621		
	rspswitch, no taskswitch-taskswitch	3775.48	22	171.613		

A-7.4 Chapter 9 Task Switching: Saccade Latency Variability

A-7 Table 13. Means and SE for ME Beverage Condition, ME Task, ME TaskSwitch, ME ResponseSwitch, ME Alcohol Session and IA Task X TaskSwitch.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		48.377	2.571	43.045	53.708
	alcohol		52.414	2.592	47.038	57.789
ME Task	pro		48.068	2.589	42.698	53.438
	anti		52.723	2.906	46.696	58.749
ME TaskSwitch	no task switch		48.673	2.46	43.571	53.774
	task switch		52.118	2.741	46.434	57.802
ME ResponseSwitch	no response switch		51.653	2.79	45.867	57.44
	response switch		49.137	2.347	44.27	54.004
ME Alcohol Session	Alcohol Session 1		51.343	3.455	44.178	58.508
	Alcohol Session 2		49.447	3.455	42.282	56.612
IA Task * TaskSwitch	pro	no task switch	43.165	2.367	38.255	48.074
		task switch	52.971	3.235	46.262	59.679
	anti	no task switch	54.18	3.303	47.33	61.03
		task switch	51.265	2.919	45.212	57.318

A-7 Table 14. ANOVA Output.

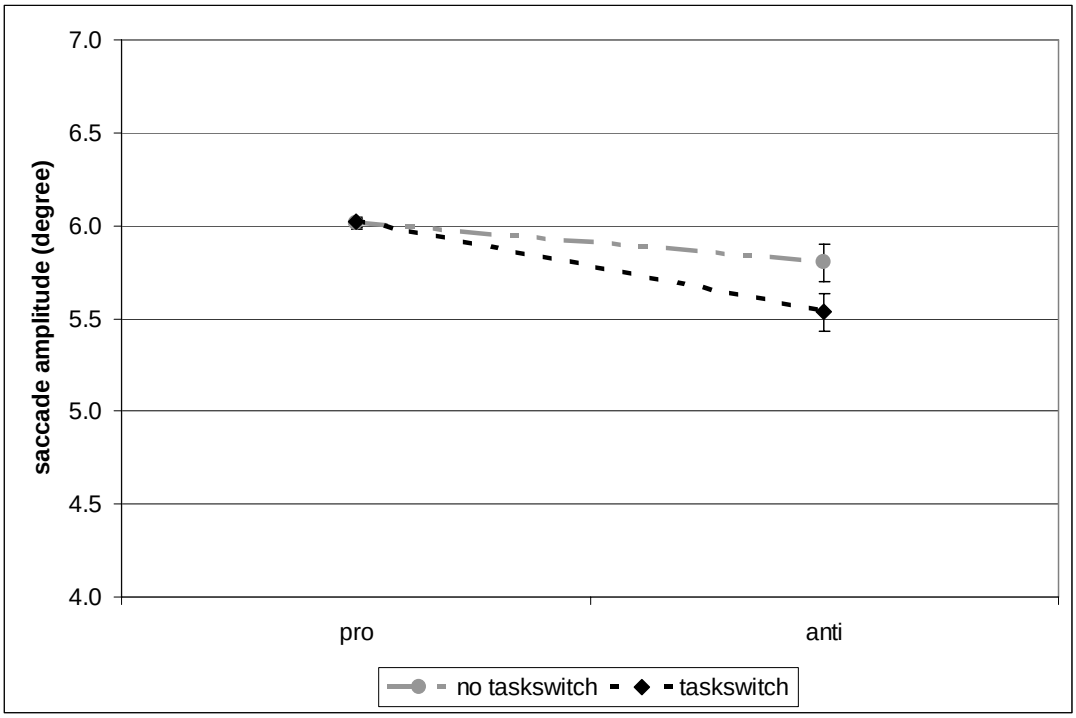
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	2080.076	1	2080.076	3.371	0.080
Task * AlcSess	11.320	1	11.320	0.018	0.893
Error(Task)	13573.350	22	616.970		
TaskSwitch	1139.467	1	1139.467	3.649	0.069
TaskSwitch * AlcSess	38.736	1	38.736	0.124	0.728
Error(TaskSwitch)	6870.662	22	312.303		
ResponseSwitch	607.903	1	607.903	2.334	0.141
ResponseSwitch * AlcSess	211.937	1	211.937	0.814	0.377
Error(ResponseSwitch)	5730.472	22	260.476		
Alcohol	1564.699	1	1564.699	5.861	0.024
Alcohol * AlcSess	1105.031	1	1105.031	4.139	0.054
Error(Alcohol)	5873.466	22	266.976		
Task * TaskSwitch	3883.572	1	3883.572	20.826	0.000
Task * TaskSwitch * AlcSess	610.989	1	610.989	3.277	0.084
Error(Task*TaskSwitch)	4102.465	22	186.476		
Task * ResponseSwitch	11.677	1	11.677	0.042	0.840
Task * ResponseSwitch * AlcSess	59.308	1	59.308	0.212	0.650
Error(Task*ResponseSwitch)	6165.679	22	280.258		
TaskSwitch * ResponseSwitch	52.565	1	52.565	0.209	0.652
TaskSwitch * ResponseSwitch * AlcSess	22.990	1	22.990	0.092	0.765
Error(TaskSwitch*ResponseSwitch)	5521.513	22	250.978		
Task * TaskSwitch * ResponseSwitch	185.875	1	185.875	0.491	0.491
Task * TaskSwitch * ResponseSwitch * AlcSess	475.864	1	475.864	1.258	0.274
Error(Task*TaskSwitch*ResponseSwitch)	8320.122	22	378.187		
Task * Alcohol	1074.039	1	1074.039	2.363	0.138
Task * Alcohol * AlcSess	5.43E+02	1	5.43E+02	1.194	0.286
Error(Task*Alcohol)	9997.576	22	454.435		
TaskSwitch * Alcohol	139.48	1	139.48	0.704	0.41
TaskSwitch * Alcohol * AlcSess	1.34E+02	1	1.34E+02	0.678	0.419
Error(TaskSwitch*Alcohol)	4355.957	22	197.998		
Task * TaskSwitch * Alcohol	0.395	1	0.395	0.004	0.948
Task * TaskSwitch * Alcohol * AlcSess	113.11	1	113.11	1.266	0.273
Error(Task*TaskSwitch*Alcohol)	1966.136	22	89.37		
ResponseSwitch * Alcohol	131.347	1	131.347	0.654	0.427
ResponseSwitch * Alcohol * AlcSess	259.493	1	259.493	1.293	0.268
Error(ResponseSwitch*Alcohol)	4416.398	22	200.745		
Task * ResponseSwitch * Alcohol	7.15E+00	1	7.15E+00	0.076	0.785
Task * ResponseSwitch * Alcohol * AlcSess	4.95E+01	1	4.95E+01	0.528	0.475
Error(Task*ResponseSwitch*Alcohol)	2063.043	22	93.775		
TaskSwitch * ResponseSwitch * Alcohol	3.43E+00	1	3.43E+00	0.016	0.902
TaskSwitch * ResponseSwitch * Alcohol * AlcSess	100.003	1	100.003	0.453	0.508
Error(TaskSwitch*ResponseSwitch*Alcohol)	4854.681	22	220.667		
Task * TaskSwitch * ResponseSwitch * Alcohol	261.71	1	261.71	0.942	0.342
Task * TaskSwitch * ResponseSwitch * Alcohol * AlcSess	178.164	1	178.164	0.641	0.432
Error(Task*TaskSwitch*ResponseSwitch*Alcohol)	6112.977	22	277.863		

A-7 Table 15. Single Contrasts for significant IA.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	pro, no taskswitch-taskswitch	2307.568	1	2307.568	18.100	0.000
	anti, no taskwitch-taskswitch	203.952	1	203.952	1.673	0.209
	no taskswitch, pro-abti	2912.014	1	2912.014	13.756	0.001
	taskswitch, pro-abti	69.810	1	69.810	0.367	0.551
Error	pro, no taskswitch-taskswitch	2804.778	22	127.490		
	anti, no taskwitch-taskswitch	2681.786	22	121.899		
	no taskswitch, pro-abti	4657.165	22	211.689		
	taskswitch, pro-abti	4180.743	22	190.034		

A-7.5 Chapter 9 Task Switching: Saccade Amplitudes

Another significant interactions was found for Task X TaskSwitch ($F_{(1,22)}$= 31.17, p<.001, see A-7 Figure 3), with shorter amplitudes in the anti saccade task compared with the pro saccade task only in the task switch condition ($F_{(1,22)}$= 8.27, p<.05), whereas the decrease in the no task switch condition did not reach significance($F_{(1,22)}$= 1.07, p>.10). The significant difference regarding TaskSwitch was therefore found only in the anti saccade task ($F_{(1,22)}$= 31.07, p<.001). A final significant interaction between Task and ResponseSwitch ($F_{(1,22)}$= 10.09, p<.05) revealed a decrease in saccade amplitudes for the anti saccade task only in the response switch condition ($F_{(1,22)}$= 6.91, p<.05), but not in the no response switch condition ($F_{(1,22)}$= 1.68, p>.10). While differences between ResponseSwitch Conditions did not differ significantly for the anti saccade task ($F_{(1,22)}$= 1.37, p>.10), pro saccades were somewhat longer under the response switch condition ($F_{(1,22)}$= 6.50, p<.05).



A-7 Figure 3. Interaction between Task and TaskSwitch for primary amplitudes in trials with correct responses.

A-7 Table 16. Means and SE for ME Beverage Condition, ME Task, ME TaskSwitch, ME ResponseSwitch and ME Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	5.71	.114	5.474	5.946
	alcohol	5.973	.14	5.681	6.264
ME Task	pro	6.016	.055	5.902	6.129
	anti	5.667	.198	5.257	6.077
ME TaskSwitch	no task switch	5.907	.119	5.661	6.153
	task switch	5.776	.116	5.534	6.017
ME ResponseSwitch	no response switch	5.827	.124	5.571	6.084
	response switch	5.855	.118	5.611	6.099
ME Alcohol Session	Alcohol Session 1	5.825	.165	5.482	6.168
	Alcohol Session 2	5.858	.165	5.515	6.2

A-7 Table 17. Means and SE for IA Task X Beverage Condition, Task X TaskSwitch and IA Task X ResponseSwitch.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
IA Task * Beverage Condition	Alcohol Session 1	no alcohol	5.96	.052	5.852	6.067
		alcohol	6.072	.079	5.908	6.235
	Alcohol Session 2	no alcohol	5.46	.193	5.06	5.861
		alcohol	5.874	.229	5.399	6.349
IA Task * TaskSwitch	pro	no task switch	6.012	.055	5.898	6.126
		task switch	6.019	.056	5.904	6.134
	anti	no task switch	5.802	.202	5.382	6.221
		task switch	5.533	.196	5.126	5.939
IA Task * ResponseSwitch	pro	no response switch	5.939	.067	5.8	6.078
		response switch	6.092	.058	5.973	6.212
	anti	no response switch	5.716	.203	5.296	6.136
		response switch	5.618	.202	5.2	6.037

A-7 Table 18. ANOVA Output.

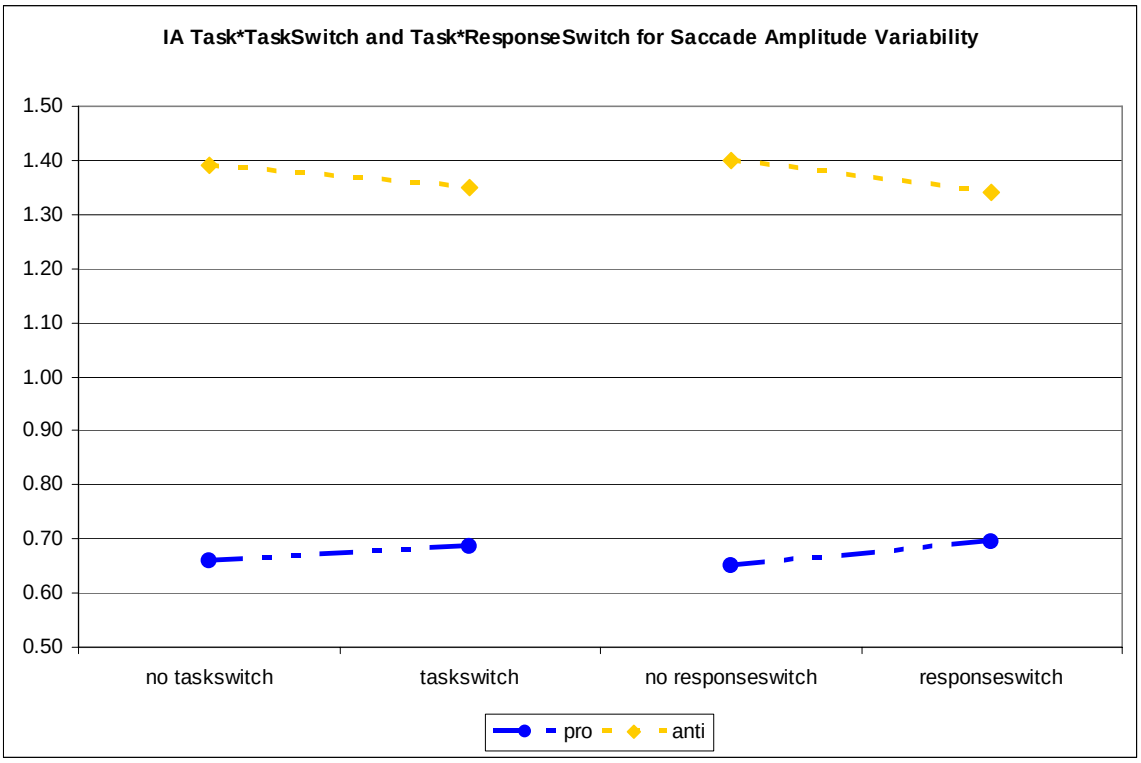
Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	11.672	1	11.672	4.113	0.055
Task * AlcSess	0.001	1	0.001	0.000	0.985
Error(Task)	62.437	22	2.838		
TaskSwitch	1.646	1	1.646	26.363	0.000
TaskSwitch * AlcSess	0.102	1	0.102	1.639	0.214
Error(TaskSwitch)	1.373	22	0.062		
ResponseSwitch	0.075	1	0.075	0.212	0.650
ResponseSwitch * AlcSess	0.000	1	0.000	0.000	0.984
Error(ResponseSwitch)	7.831	22	0.356		
Alcohol	6.628	1	6.628	6.391	0.019
Alcohol * AlcSess	0.802	1	0.802	0.773	0.389
Error(Alcohol)	22.813	22	1.037		
Task * TaskSwitch	1.830	1	1.830	31.170	0.000
Task * TaskSwitch * AlcSess	0.018	1	0.018	0.303	0.587
Error(Task*TaskSwitch)	1.292	22	0.059		
Task * ResponseSwitch	1.511	1	1.511	10.085	0.004
Task * ResponseSwitch * AlcSess	0.050	1	0.050	0.336	0.568
Error(Task*ResponseSwitch)	3.296	22	0.150		
TaskSwitch * ResponseSwitch	0.018	1	0.018	0.165	0.689
TaskSwitch * ResponseSwitch * AlcSess	0.004	1	0.004	0.035	0.854
Error(TaskSwitch*ResponseSwitch)	2.434	22	0.111		
Task * TaskSwitch * ResponseSwitch	0.064	1	0.064	0.604	0.445
Task * TaskSwitch * ResponseSwitch * AlcSess	0.028	1	0.028	0.259	0.616
Error(Task*TaskSwitch*ResponseSwitch)	2.346	22	0.107		
Task * Alcohol	2.18	1	2.18	6.203	0.021
Task * Alcohol * AlcSess	6.07E-01	1	6.07E-01	1.727	0.202
Error(Task*Alcohol)	7.733	22	0.351		
TaskSwitch * Alcohol	0.149	1	0.149	2.791	0.109
TaskSwitch * Alcohol * AlcSess	7.60E-02	1	7.60E-02	1.422	0.246
Error(TaskSwitch*Alcohol)	1.174	22	0.053		
Task * TaskSwitch * Alcohol	0.057	1	0.057	0.857	0.365
Task * TaskSwitch * Alcohol * AlcSess	1.89E-05	1	1.89E-05	0	0.987
Error(Task*TaskSwitch*Alcohol)	1.464	22	0.067		
ResponseSwitch * Alcohol	0.198	1	0.198	3.154	0.09
ResponseSwitch * Alcohol * AlcSess	0.172	1	0.172	2.739	0.112
Error(ResponseSwitch*Alcohol)	1.38	22	0.063		
Task * ResponseSwitch * Alcohol	1.00E-03	1	1.00E-03	0.037	0.85
Task * ResponseSwitch * Alcohol * AlcSess	1.10E-01	1	1.10E-01	4.862	0.038
Error(Task*ResponseSwitch*Alcohol)	0.496	22	0.023		
TaskSwitch * ResponseSwitch * Alcohol	1.00E-03	1	1.00E-03	0.023	0.882
TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0.097	1	0.097	1.591	0.22
Error(TaskSwitch*ResponseSwitch*Alcohol)	1.34	22	0.061		
Task * TaskSwitch * ResponseSwitch * Alcohol	0.08	1	0.08	2.898	0.103
Task * TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0.084	1	0.084	3.051	0.095
Error(Task*TaskSwitch*ResponseSwitch*Alcohol)	0.605	22	0.027		

A-7 Table 19. Single Contrasts for significant IA.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	no alc, pro-anti	5.985	1	5.985	8.913	0.007
	alc, pro-anti	0.941	1	0.941	1.019	0.324
	pro, no alc-alc	0.301	1	0.301	2.153	0.156
	anti, no alc-alc	4.102	1	4.102	7.402	0.012
	no switch, pro-anti	1.065	1	1.065	1.400	0.249
	switch, pro-anti	5.686	1	5.686	8.265	0.009
	pro, no switch-switch	0.001	1	0.001	0.261	0.614
	anti, no switch-switch	1.737	1	1.737	31.067	0.000
	no respsw, pro-anti	1.196	1	1.196	1.679	0.209
	respsw, pro-anti	5.396	1	5.396	6.905	0.015
	pro, no respsw-respsw	0.565	1	0.565	6.497	0.018
	anti, no respsw-respsw	0.228	1	0.228	1.374	0.254
Error	no alc, pro-anti	14.774	22	0.672		
	alc, pro-anti	20.311	22	0.923		
	pro, no alc-alc	3.079	22	0.14		
	anti, no alc-alc	12.194	22	0.554		
	no switch, pro-anti	16.729	22	0.76		
	switch, pro-anti	15.135	22	0.688		
	pro, no switch-switch	0.103	22	0.005		
	anti, no switch-switch	1.23	22	0.056		
	no respsw, pro-anti	15.675	22	0.713		
	respsw, pro-anti	17.191	22	0.781		
	pro, no respsw-respsw	1.914	22	0.087		
	anti, no respsw-respsw	3.649	22	0.166		

A-7.6 Chapter 9 Task Switching: Saccade Amplitude Variability

Regarding the variability of saccade amplitude a significant main effect of Task ($F_{(1,22)}$= 134.90, p<.001) is shown in a separate ANOVA, indicating greater variability in the anti saccade task. This main effect is qualified by significant Task X TaskSwitch ($F_{(1,22)}$= 7.16, p<.05) and Task X ResponseSwitch ($F_{(1,22)}$= 4.41, p<.05) interactions (see A-7 Figure 4). Both interactions are caused by the fact that the differences between tasks are smaller in the switch condition compared to the no switch condition, even though neither the increases in variability from no switch to switch conditions in the pro saccade task (TaskSwitch: $F_{(1,22)}$= .02, p>.10; ResponseSwitch: $F_{(1,22)}$= 4.23, p>.05), nor the decreases in variability from no switch to switch conditions in the anti saccade task (TaskSwitch: $F_{(1,22)}$= 3.42, p>.05; ResponseSwitch: $F_{(1,22)}$= 1.72, p>.10) reach significance.



A-7 Figure 4. Left side: Interaction between Task and TaskSwitch Condition for saccade amplitude variability. Right side: Interaction between Task and ResponseSwitch Condition for saccade amplitude variability.

A-7 Table 20. Means and SE for ME Beverage Condition, ME Task, ME TaskSwitch, ME ResponseSwitch and ME Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	1.007	.049	.906	1.108
	alcohol	1.036	.039	.955	1.117
ME Task	pro	.672	.03	.609	.736
	anti	1.37	.065	1.235	1.506
ME TaskSwitch	no task switch	1.025	.04	.942	1.109
	task switch	1.017	.043	.927	1.108
ME ResponseSwitch	no response switch	1.025	.04	.942	1.108
	response switch	1.018	.045	.924	1.112
ME Alcohol Session	Alcohol Session 1	1.006	.058	.886	1.127
	Alcohol Session 2	1.037	.058	.916	1.157

A-7 Table 21. Means and SE for IA Task X TaskSwitch and IA Task X ResponseSwitch.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
IA Task * TaskSwitch	pro	no task switch	.659	.03	.595	.722
		task switch	.686	.033	.617	.755
	anti	no task switch	1.392	.064	1.259	1.526
		task switch	1.349	.068	1.207	1.49
IA Task * ResponseSwitch	pro	no response switch	.651	.035	.579	.723
		response switch	.694	.029	.633	.755
	anti	no response switch	1.399	.061	1.273	1.524
		response switch	1.342	.076	1.185	1.499

A-7 Table 22. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	46.759	1	46.759	134.896	0.000
Task * AlcSess	0.065	1	0.065	0.189	0.668
Error(Task)	7.626	22	0.347		
TaskSwitch	0.006	1	0.006	0.223	0.641
TaskSwitch * AlcSess	0.013	1	0.013	0.480	0.496
Error(TaskSwitch)	0.600	22	0.027		
ResponseSwitch	0.004	1	0.004	0.079	0.782
ResponseSwitch * AlcSess	0.023	1	0.023	0.405	0.531
Error(ResponseSwitch)	1.233	22	0.056		
Alcohol	0.077	1	0.077	0.767	0.391
Alcohol * AlcSess	0.135	1	0.135	1.337	0.260
Error(Alcohol)	2.220	22	0.101		
Task * TaskSwitch	0.121	1	0.121	7.158	0.014
Task * TaskSwitch * AlcSess	0.001	1	0.001	0.064	0.803
Error(Task*TaskSwitch)	0.373	22	0.017		
Task * ResponseSwitch	0.236	1	0.236	4.411	0.047
Task * ResponseSwitch * AlcSess	0.003	1	0.003	0.058	0.812
Error(Task*ResponseSwitch)	1.179	22	0.054		
TaskSwitch * ResponseSwitch	0.035	1	0.035	0.961	0.338
TaskSwitch * ResponseSwitch * AlcSess	0.004	1	0.004	0.122	0.730
Error(TaskSwitch*ResponseSwitch)	0.803	22	0.036		
Task * TaskSwitch * ResponseSwitch	0.066	1	0.066	2.811	0.108
Task * TaskSwitch * ResponseSwitch * AlcSess	0.003	1	0.003	0.141	0.711
Error(Task*TaskSwitch*ResponseSwitch)	0.518	22	0.024		
Task * Alcohol	0.002	1	0.002	0.032	0.859
Task * Alcohol * AlcSess	1.60E-01	1	1.60E-01	3.033	0.096
Error(Task*Alcohol)	1.161	22	0.053		
TaskSwitch * Alcohol	0.049	1	0.049	0.65	0.429
TaskSwitch * Alcohol * AlcSess	0.00E+00	1	0.00E+00	0.004	0.951
Error(TaskSwitch*Alcohol)	1.642	22	0.075		
Task * TaskSwitch * Alcohol	0.002	1	0.002	0.023	0.881
Task * TaskSwitch * Alcohol * AlcSess	0.062	1	0.062	0.693	0.414
Error(Task*TaskSwitch*Alcohol)	1.973	22	0.09		
ResponseSwitch * Alcohol	0.027	1	0.027	1.963	0.175
ResponseSwitch * Alcohol * AlcSess	0.022	1	0.022	1.571	0.223
Error(ResponseSwitch*Alcohol)	0.304	22	0.014		
Task * ResponseSwitch * Alcohol	9.00E-03	1	9.00E-03	0.247	0.624
Task * ResponseSwitch * Alcohol * AlcSess	7.80E-02	1	7.80E-02	2.155	0.156
Error(Task*ResponseSwitch*Alcohol)	0.8	22	0.036		
TaskSwitch * ResponseSwitch * Alcohol	5.00E-03	1	5.00E-03	0.118	0.734
TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0.073	1	0.073	1.785	0.195
Error(TaskSwitch*ResponseSwitch*Alcohol)	0.896	22	0.041		
Task * TaskSwitch * ResponseSwitch * Alcohol	0	1	0	0.01	0.921
Task * TaskSwitch * ResponseSwitch * Alcohol * AlcSess	0.004	1	0.004	0.164	0.689
Error(Task*TaskSwitch*ResponseSwitch*Alcohol)	0.562	22	0.026		

A-7 Table 23. Single Contrasts for significant IA.

Source	Transformed Variable	Sum of Squares	df	Mean Square	F	Sig.
Contrast	pro, no taskswitch-taskswitch	0.018	1	0.018	2.076	0.164
	anti, no taskwitch-taskswitch	0.045	1	0.045	3.415	0.078
	pro, no responsewitch-responseswitch	0.044	1	0.044	4.231	0.052
	anti, no responsewitch-responseswitch	0.076	1	0.076	1.719	0.203
	(pnotasksw-anotasksw)-(ptasksw-atasksw)	0.121	1	0.121	7.158	0.014
	(pnorspsw-anorspsw)-(prspsw-arspsw)	0.236	1	0.236	4.411	0.047
Error	pro, no taskswitch-taskswitch	0.194	22	0.009		
	anti, no taskwitch-taskswitch	0.293	22	0.013		
	pro, no responsewitch-responseswitch	0.229	22	0.010		
	anti, no responsewitch-responseswitch	0.977	22	0.044		
	(pnotasksw-anotasksw)-(ptasksw-atasksw)	0.373	22	0.017		
	(pnorspsw-anorspsw)-(prspsw-arspsw)	1.179	22	0.054		

A-7.7 Chapter 9 Task Switching: Peak Velocity Deviation

A-7 Table 24. Means and SE for ME Beverage Condition, ME Task, ME TaskSwitch, ME ResponseSwitch and ME Alcohol Session.

Effect		Mean	Std.Error	95% Confidence Interval	
				Lower Bound	Upper Bound
ME Beverage Condition	no alcohol	-36.285	3.315	-43.16	-29.411
	alcohol	-28.886	3.713	-36.587	-21.186
ME Task	pro	-33.058	2.978	-39.233	-26.882
	anti	-32.114	3.889	-40.178	-24.049
ME TaskSwitch	no task switch	-32.157	3.405	-39.219	-25.095
	task switch	-33.014	3.241	-39.737	-26.292
ME ResponseSwitch	no response switch	-32.681	3.419	-39.772	-25.589
	response switch	-32.491	3.222	-39.174	-25.808
ME Alcohol Session	Alcohol Session 1	-26.266	4.661	-35.932	-16.6
	Alcohol Session 2	-38.906	4.661	-48.572	-29.24

A-7 Table 25. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Task	85.531	1	85.531	0.197	0.662
Task * AlcSess	141.535	1	141.535	0.325	0.574
Error(Task)	9572.847	22	435.129		
TaskSwitch	70.540	1	70.540	0.971	0.335
TaskSwitch * AlcSess	24.408	1	24.408	0.336	0.568
Error(TaskSwitch)	1598.941	22	72.679		
ResponseSwitch	3.457	1	3.457	0.051	0.823
ResponseSwitch * AlcSess	66.741	1	66.741	0.986	0.332
Error(ResponseSwitch)	1489.253	22	67.693		
Alcohol	5255.713	1	5255.713	8.973	0.007
Alcohol * AlcSess	2.702	1	2.702	0.005	0.946
Error(Alcohol)	12885.964	22	585.726		
Task * TaskSwitch	0.059	1	0.059	0.001	0.978
Task * TaskSwitch * AlcSess	25.617	1	25.617	0.349	0.561
Error(Task*TaskSwitch)	1616.802	22	73.491		
Task * ResponseSwitch	32.102	1	32.102	1.265	0.273
Task * ResponseSwitch * AlcSess	117.892	1	117.892	4.645	0.042
Error(Task*ResponseSwitch)	558.406	22	25.382		
TaskSwitch * ResponseSwitch	3.036	1	3.036	0.041	0.842
TaskSwitch * ResponseSwitch * AlcSess	203.554	1	203.554	2.722	0.113
Error(TaskSwitch*ResponseSwitch)	1644.920	22	74.769		
Task * TaskSwitch * ResponseSwitch	9.718	1	9.718	0.146	0.706
Task * TaskSwitch * ResponseSwitch * AlcSess	108.643	1	108.643	1.634	0.215
Error(Task*TaskSwitch*ResponseSwitch)	1463.044	22	66.502		
Task * Alcohol	430.312	1	430.312	3.203	0.087
Task * Alcohol * AlcSess	1.15E+00	1	1.15E+00	0.009	0.927
Error(Task*Alcohol)	2955.679	22	134.349		
TaskSwitch * Alcohol	9.282	1	9.282	0.114	0.739
TaskSwitch * Alcohol * AlcSess	5.79E+00	1	5.79E+00	0.071	0.792
Error(TaskSwitch*Alcohol)	1790.583	22	81.39		
Task * TaskSwitch * Alcohol	61.908	1	61.908	0.696	0.413
Task * TaskSwitch * Alcohol * AlcSess	39.467	1	39.467	0.444	0.512
Error(Task*TaskSwitch*Alcohol)	1956.851	22	88.948		
ResponseSwitch * Alcohol	14.653	1	14.653	0.19	0.667
ResponseSwitch * Alcohol * AlcSess	90.488	1	90.488	1.175	0.29
Error(ResponseSwitch*Alcohol)	1694.068	22	77.003		
Task * ResponseSwitch * Alcohol	2.07E+01	1	2.07E+01	0.506	0.484
Task * ResponseSwitch * Alcohol * AlcSess	5.80E+00	1	5.80E+00	0.142	0.71
Error(Task*ResponseSwitch*Alcohol)	900.03	22	40.91		
TaskSwitch * ResponseSwitch * Alcohol	2.64E+01	1	2.64E+01	1.16	0.293
TaskSwitch * ResponseSwitch * Alcohol * AlcSess	18.386	1	18.386	0.808	0.378
Error(TaskSwitch*ResponseSwitch*Alcohol)	500.443	22	22.747		
Task * TaskSwitch * ResponseSwitch * Alcohol	4.449	1	4.449	0.234	0.633
Task * TaskSwitch * ResponseSwitch * Alcohol * AlcSess	2.278	1	2.278	0.12	0.732
Error(Task*TaskSwitch*ResponseSwitch*Alcohol)	417.933	22	18.997		

A-8 Chapter 10 Reading

A-8.1 Chapter 10 Reading Spatial Parameters: Initial Landing Position

A-8 Table 1. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			2.709	.096	2.512	2.906
	alcohol			2.768	.079	2.607	2.929
ME Preview	legal			2.833	.08	2.67	2.996
	illegal			2.644	.088	2.464	2.824
ME Frequency	low frequency			2.733	.081	2.567	2.899
	high frequency			2.744	.086	2.568	2.92
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	2.787	.112	2.559	3.015
			alcohol	2.803	.078	2.643	2.962
		high frequency	no alcohol	2.83	.101	2.623	3.037
			alcohol	2.913	.097	2.716	3.11
	illegal	low frequency	no alcohol	2.654	.108	2.433	2.874
			alcohol	2.69	.098	2.49	2.89
		high frequency	no alcohol	2.565	.114	2.333	2.797
			alcohol	2.668	.1	2.464	2.872

A-8 Table 2. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.227	1	0.227	0.770	0.387
Error(alcohol)	9.136	31	0.295		
preview	2.281	1	2.281	18.801	0.000
Error(preview)	3.760	31	0.121		
freq	0.007	1	0.007	0.065	0.800
Error(freq)	3.417	31	0.110		
alcohol * preview	0.007	1	0.007	0.050	0.824
Error(alcohol*preview)	4.288	31	0.138		
alcohol * freq	0.072	1	0.072	0.855	0.362
Error(alcohol*freq)	2.594	31	0.084		
preview * freq	0.277	1	0.277	3.042	0.091
Error(preview*freq)	2.825	31	0.091		
alcohol * preview * freq	0.000	1	0.000	0.000	0.996
Error(alcohol*preview*freq)	3.204	31	0.103		

A-8.2 Chapter 10 Reading Spatial Parameters: Initial Landing Position by Launch Distance

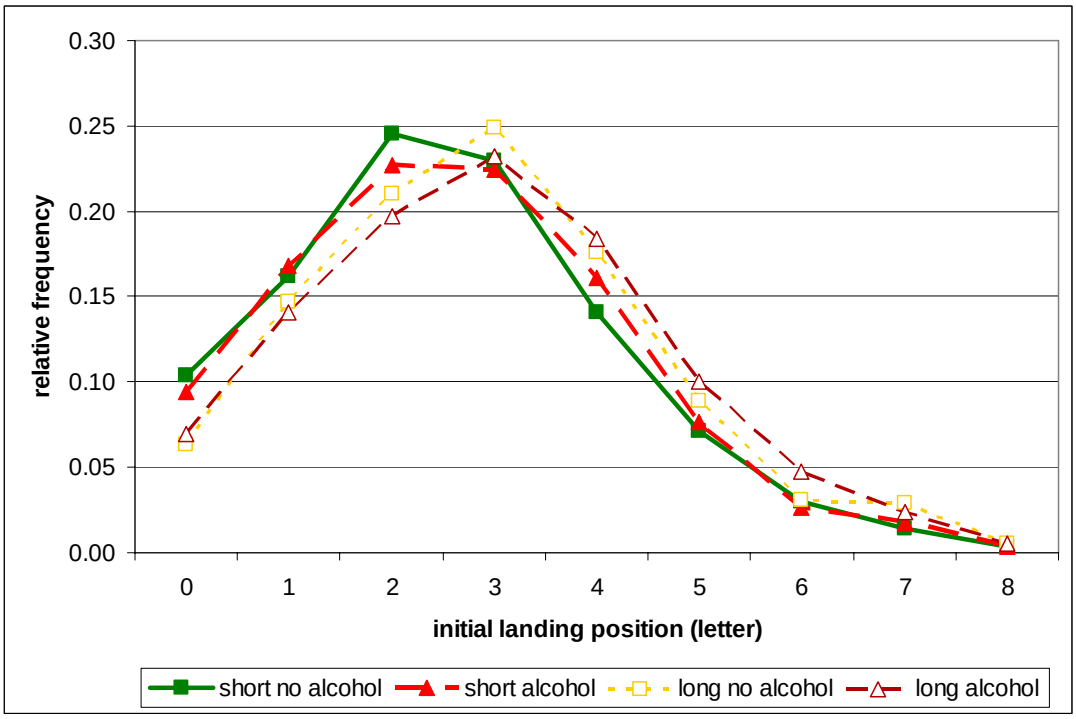
A-8 Table 3. Means and SE for ME Beverage Condition, ME Launch Distance, ME Preview and IA Launch Distance X Beverage Condition X Preview.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			2.67	.089	2.489	2.851
	alcohol			2.744	.077	2.588	2.901
ME Launch Distance	near			3.29	.102	3.082	3.498
	far			2.124	.064	1.994	2.253
ME Preview	low frequency			2.815	.078	2.656	2.973
	high frequency			2.599	.082	2.432	2.767
IA Launch Distance * Beverage Condition * Preview	near	no alcohol	low frequency	3.359	.126	3.101	3.616
			high frequency	3.135	.12	2.892	3.379
		alcohol	low frequency	3.413	.099	3.211	3.614
			high frequency	3.254	.125	3.00	3.509
	far	no alcohol	low frequency	2.197	.08	2.035	2.36
			high frequency	1.987	.088	1.807	2.168
		alcohol	low frequency	2.29	.091	2.104	2.475
			high frequency	2.021	.084	1.849	2.193

A-8 Table 4. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
dist	87.093	1	87.093	279.993	0.000
Error(dist)	9.643	31	0.311		
alc	0.357	1	0.357	1.620	0.213
Error(alc)	6.837	31	0.221		
preview	2.963	1	2.963	29.478	0.000
Error(preview)	3.116	31	0.101		
dist * alc	0.009	1	0.009	0.074	0.787
Error(dist*alc)	3.766	31	0.121		
dist * preview	0.038	1	0.038	0.210	0.650
Error(dist*preview)	5.648	31	0.182		
alc * preview	0.000	1	0.000	0.001	0.975
Error(alc*preview)	4.017	31	0.130		
dist * alc * preview	0.062	1	0.062	0.513	0.479
Error(dist*alc*preview)	3.721	31	0.120		

A-8.3 Chapter 10 Reading Spatial Parameters: Initial Landing Position by Previous Gaze Duration



A-8 Figure 1. Initial landing position in target words, with short and long gaze duration on the preceding word (N-1) for both alcohol conditions.

A-8 Table 5. Means and SE for ME Beverage Condition, ME Previous Gaze Duration, ME Frequency, ME Preview and IA Previous Gaze Duration X Preview X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			2.758	.1	2.554	2.962
	alcohol			2.801	.08	2.637	2.965
ME Previous Gaze Duration	short			2.597	.09	2.413	2.781
	long			2.962	.086	2.787	3.136
ME Frequency	low frequency			2.76	.082	2.593	2.928
	high frequency			2.799	.087	2.62	2.977
ME Preview	legal			2.86	.078	2.701	3.019
	illegal			2.699	.094	2.507	2.892
IA Previous Gaze Duration * Preview * Beverage Condition	short	legal	no alcohol	2.681	.129	2.417	2.945
			alcohol	2.719	.082	2.552	2.887
		illegal	no alcohol	2.469	.107	2.25	2.687
			alcohol	2.519	.102	2.31	2.728
	long	legal	no alcohol	2.99	.111	2.763	3.217
			alcohol	3.048	.109	2.827	3.27
		illegal	no alcohol	2.892	.143	2.6	3.183
			alcohol	2.917	.118	2.676	3.159

A-8 Table 6. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
time	17.030	1	17.030	30.200	0.000
Error(time)	17.480	31	0.564		
alc	0.237	1	0.237	0.292	0.593
Error(alc)	25.166	31	0.812		
preview	3.303	1	3.303	7.579	0.010
Error(preview)	13.511	31	0.436		
freq	0.188	1	0.188	0.656	0.424
Error(freq)	8.871	31	0.286		
dist * alc	0.000	1	0.000	0.001	0.981
Error(time*alc)	8.833	31	0.285		
dist * preview	0.269	1	0.269	0.644	0.428
Error(time*preview)	12.946	31	0.418		
alc * preview	0.003	1	0.003	0.011	0.917
Error(alc*preview)	9.569	31	0.309		
dist * alc * preview	0.016	1	0.016	0.037	0.848
Error(time*alc*preview)	13.514	31	0.436		
dist * freq	0.349	1	0.349	1.623	0.212
Error(time*freq)	6.668	31	0.215		
alc * freq	0.172	1	0.172	0.726	0.401
Error(alc*freq)	7.359	31	0.237		
dist * alc * freq	0.5	1	0.5	2.033	0.164
Error(time*alc*freq)	7.631	31	0.246		
preview * freq	0.242	1	0.242	1.505	0.229
Error(preview*freq)	4.992	31	0.161		
time * preview * freq	0.336	1	0.336	1.986	0.169
Error(time*preview*freq)	5.239	31	0.169		
alc * preview * freq	0.01	1	0.01	0.038	0.847
Error(alc*preview*freq)	8.051	31	0.26		
time * alc * preview * freq	0.245	1	0.245	0.594	0.447
Error(time*alc*preview*freq)	12.78	31	0.412		

A-8.4 Chapter 10 Reading Spatial Parameter: Saccade Amplitudes

A-8 Table 7. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			8.341	.186	7.961	8.721
	alcohol			8.408	.179	8.042	8.773
ME Preview	legal			8.587	.187	8.207	8.968
	illegal			8.162	.17	7.816	8.507
ME Frequency	low frequency			8.297	.172	7.945	8.648
	high frequency			8.452	.181	8.083	8.821
IA Preview * Frequency * Beverage Condition	legal	low	no alcohol	8.4	.213	7.966	8.833
			alcohol	8.615	.198	8.211	9.018
		high	no alcohol	8.686	.207	8.263	9.109
			alcohol	8.649	.221	8.20	9.099
	illegal	low	no alcohol	8.068	.183	7.696	8.44
			alcohol	8.105	.183	7.731	8.478
		high	no alcohol	8.211	.215	7.772	8.65
			alcohol	8.263	.183	7.89	8.637

A-8 Table 8. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.284	1	0.284	0.378	0.543
Error(alcohol)	23.326	31	0.752		
preview	11.590	1	11.590	33.941	0.000
Error(preview)	10.586	31	0.341		
freq	1.547	1	1.547	7.853	0.009
Error(freq)	6.107	31	0.197		
alcohol * preview	0.031	1	0.031	0.082	0.776
Error(alcohol*preview)	11.744	31	0.379		
alcohol * freq	0.224	1	0.224	1.697	0.202
Error(alcohol*freq)	4.091	31	0.132		
preview * freq	0.001	1	0.001	0.005	0.945
Error(preview*freq)	9.556	31	0.308		
alcohol * preview * freq	0.289	1	0.289	0.679	0.416
Error(alcohol*preview*freq)	13.190	31	0.425		

A-8.5 Chapter 10 Reading Spatial Parameter: Launch Distance

A-8 Table 9. Means and SE for ME Beverage Condition, ME Preview, ME Frequency, IA Frequency X Beverage Condition and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			-5.131	.132	-5.399	-4.862
	alcohol			-5.154	.133	-5.425	-4.884
ME Preview	legal			-5.262	.136	-5.54	-4.984
	illegal			-5.023	.115	-5.258	-4.788
ME Frequency	low frequency			-5.068	.115	-5.303	-4.834
	high frequency			-5.217	.132	-5.487	-4.947
IA Frequency * Beverage Condition	low frequency		no alcohol	-5.011	.125	-5.265	-4.757
			alcohol	-5.125	.136	-5.403	-4.847
	high frequency		no alcohol	-5.251	.154	-5.564	-4.937
			alcohol	-5.183	.136	-5.461	-4.906
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	-5.103	.167	-5.444	-4.762
			alcohol	-5.322	.169	-5.666	-4.978
		high frequency	no alcohol	-5.357	.183	-5.73	-4.984
			alcohol	-5.267	.172	-5.62	-4.917
	illegal	low frequency	no alcohol	-4.918	.138	-5.199	-4.637
			alcohol	-4.929	.13	-5.195	-4.663
		high frequency	no alcohol	-5.145	.168	-5.488	-4.801
			alcohol	-5.099	.146	-5.396	-4.802

A-8 Table 10. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.036	1	0.036	0.046	0.832
Error(alcohol)	24.133	31	0.778		
preview	3.673	1	3.673	9.402	0.004
Error(preview)	12.111	31	0.391		
freq	1.417	1	1.417	5.721	0.023
Error(freq)	7.681	31	0.248		
alcohol * preview	0.107	1	0.107	0.272	0.606
Error(alcohol*preview)	12.161	31	0.392		
alcohol * freq	0.532	1	0.532	3.156	0.085
Error(alcohol*freq)	5.223	31	0.168		
preview * freq	0.157	1	0.157	0.357	0.555
Error(preview*freq)	13.667	31	0.441		
alcohol * preview * freq	0.255	1	0.255	0.570	0.456
Error(alcohol*preview*freq)	13.853	31	0.447		

A-8.6 Chapter 10 Reading Temporal Parameters: Initial Fixation Duration

A-8 Table 11. Means and SE for ME Beverage Condition, ME Preview, ME Frequency, IA Preview X Beverage Condition and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			301.321	7.146	286.746	315.896
	alcohol			318.824	7.702	303.115	334.533
ME Preview	legal			297.766	6.298	284.922	310.611
	illegal			322.378	8.054	305.952	338.805
ME Frequency	low frequency			317.902	6.703	304.23	331.573
	high frequency			302.243	7.401	287.149	317.337
IA Preview * Beverage Condition	legal		no alcohol	289.594	6.794	275.737	303.45
			alcohol	305.939	7.596	290.447	321.43
	illegal		no alcohol	313.048	8.932	294.83	331.265
			alcohol	331.709	8.813	313.734	349.684
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	297.606	7.191	282.939	312.273
			alcohol	317.966	8.099	301.448	334.484
		high frequency	no alcohol	281.582	7.206	266.884	296.279
			alcohol	293.912	8.229	277.13	310.696
	illegal	low frequency	no alcohol	319.86	9.103	301.294	338.426
			alcohol	336.175	8.452	318.937	353.412
		high frequency	no alcohol	306.236	9.828	286.192	326.28
			alcohol	327.243	10.261	306.315	348.171

A-8 Table 12. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	19606.694	1	19606.694	10.143	0.003
Error(alcohol)	59924.533	31	1933.049		
preview	38768.387	1	38768.387	32.807	0.000
Error(preview)	36633.416	31	1181.723		
freq	15692.099	1	15692.099	27.792	0.000
Error(freq)	17503.526	31	564.630		
alcohol * preview	85.819	1	85.819	0.061	0.807
Error(alcohol*preview)	43748.384	31	1411.238		
alcohol * freq	44.561	1	44.561	0.070	0.794
Error(alcohol*freq)	19866.099	31	640.842		
preview * freq	1228.311	1	1228.311	3.548	0.069
Error(preview*freq)	10732.470	31	346.209		
alcohol * preview * freq	647.470	1	647.470	0.918	0.345
Error(alcohol*preview*freq)	21861.402	31	705.207		

A-8.7 Chapter 10 Reading Temporal Parameters: Gaze Duration

A-8 Table 13. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			393.611	14.341	364.363	422.859
	alcohol			408.923	15.047	378.234	439.613
ME Preview	legal			379.588	12.96	353.155	406.021
	illegal			422.946	15.833	390.654	455.238
ME Frequency	low frequency			439.35	17.323	404.019	474.681
	high frequency			363.184	11.619	339.486	386.882
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	409.803	15.926	377.322	442.285
			alcohol	426.677	21.886	382.039	471.314
		high frequency	no alcohol	331.803	9.498	312.432	351.174
			alcohol	350.07	11.675	326.26	373.88
	illegal	low frequency	no alcohol	453.49	20.243	412.204	494.776
			alcohol	467.431	17.923	430.877	503.984
		high frequency	no alcohol	379.346	18.047	342.539	416.154
			alcohol	391.517	17.566	355.69	427.344

A-8 Table 14. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	15006.695	1	15006.695	2.872	0.100
Error(alcohol)	161991.576	31	5225.535		
preview	120312.771	1	120312.771	34.299	0.000
Error(preview)	108741.300	31	3507.784		
freq	371281.477	1	371281.477	66.154	0.000
Error(freq)	173983.961	31	5612.386		
alcohol * preview	326.093	1	326.093	0.055	0.817
Error(alcohol*preview)	184973.757	31	5966.895		
alcohol * freq	0.568	1	0.568	0.000	0.984
Error(alcohol*freq)	41925.263	31	1352.428		
preview * freq	82.786	1	82.786	0.068	0.796
Error(preview*freq)	37685.459	31	1215.660		
alcohol * preview * freq	40.008	1	40.008	0.028	0.868
Error(alcohol*preview*freq)	44065.401	31	1421.465		

A-8.8 Chapter 10 Reading Temporal Parameters: Gaze Duration by Launch Distance

A-8 Table 15. Means and SE for ME Beverage Condition, ME Launch Distance, ME Preview and IA Launch Distance X Beverage Condition X Preview.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			408.244	17.377	372.805	443.684
	alcohol			434.253	18.45	396.624	471.882
ME Launch Distance	near			419.658	17.501	383.965	455.352
	far			422.839	16.618	388.946	456.732
ME Preview	legal			395.351	15.233	364.282	426.419
	illegal			447.147	19.456	407.466	486.827
IA Launch Distance * Beverage Condition * Preview	near	no alcohol	legal	379.267	15.922	346.793	411.741
			illegal	439.29	22.17	394.075	484.506
		alcohol	legal	398.247	18.564	360.385	436.108
			illegal	461.828	23.158	414.60	509.058
	far	no alcohol	legal	381.814	14.516	352.207	411.42
			illegal	432.606	24.489	382.661	482.551
		alcohol	legal	422.075	20.131	381.018	463.132
			illegal	454.862	19.492	415.11	494.615

A-8 Table 16. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
dist	647.648	1	647.648	0.301	0.587
Error(dist)	66652.545	31	2150.082		
alc	43292.090	1	43292.090	4.410	0.044
Error(alc)	304318.067	31	9816.712		
preview	171701.217	1	171701.217	29.854	0.000
Error(preview)	178292.447	31	5751.369		
dist * alc	1763.796	1	1763.796	0.683	0.415
Error(dist*alc)	80038.775	31	2581.896		
dist * preview	6408.392	1	6408.392	4.188	0.049
Error(dist*preview)	47437.258	31	1530.234		
alc * preview	834.953	1	834.953	0.119	0.733
Error(alc*preview)	217632.417	31	7020.401		
dist * alc * preview	1859.989	1	1859.989	1.014	0.322
Error(dist*alc*preview)	56876.833	31	1834.737		

A-8.9 Chapter 10 Reading Temporal Parameters: Total Reading Time

A-8 Table 17. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			472.49	18.654	434.445	510.534
	alcohol			480.964	20.26	439.643	522.285
ME Preview	legal			446.383	16.965	411.782	480.983
	illegal			507.071	20.58	465.098	549.044
ME Frequency	low frequency			533.846	24.538	483.801	583.892
	high frequency			419.608	13.7	391.666	447.549
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	493.524	20.706	451.295	535.754
			alcohol	509.036	28.935	450.023	568.048
		high frequency	no alcohol	386.636	12.156	361.843	411.429
			alcohol	396.335	14.693	366.37	426.303
	illegal	low frequency	no alcohol	561.811	27.202	506.333	617.289
			alcohol	571.014	29.144	511.575	630.453
		high frequency	no alcohol	447.988	22.459	402.183	493.793
			alcohol	447.472	20.686	405.282	489.661

A-8 Table 18. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	4596.059	1	4596.059	0.476	0.495
Error(alcohol)	299350.145	31	9656.456		
preview	235716.152	1	235716.152	64.954	0.000
Error(preview)	112498.089	31	3628.971		
freq	835228.498	1	835228.498	61.093	0.000
Error(freq)	423811.130	31	13671.327		
alcohol * preview	1092.197	1	1092.197	0.116	0.735
Error(alcohol*preview)	291208.439	31	9393.821		
alcohol * freq	964.793	1	964.793	0.210	0.650
Error(alcohol*freq)	142312.103	31	4590.713		
preview * freq	1264.041	1	1264.041	0.436	0.514
Error(preview*freq)	89911.821	31	2900.381		
alcohol * preview * freq	61.074	1	61.074	0.039	0.845
Error(alcohol*preview*freq)	48807.020	31	1574.420		

A-8.10 Chapter 10 Reading Temporal Parameters: Single Fixation Duration

A-8 Table 19. Means and SE for ME Beverage Condition, ME Preview IA Preview X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		316.564	8.722	298.774	334.353
	alcohol		332.075	9.104	313.507	350.643
ME Preview	legal		308.423	7.641	292.839	324.007
	illegal		340.216	9.602	320.633	359.8
IA Preview * Beverage Condition	legal	no alcohol	300.933	8.431	283.738	318.127
		alcohol	315.912	9.272	297.003	334.822
	illegal	no alcohol	332.195	10.662	310.45	353.94
		alcohol	348.238	10.601	326.62	369.859

A-8 Table 20. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	7699.229	1	7699.229	6.276	0.018
Error(alcohol)	38030.811	31	1226.800		
preview	32347.125	1	32347.125	46.854	0.000
Error(preview)	21401.986	31	690.387		
alcohol * preview	9.048	1	9.048	0.006	0.936
Error(alcohol*preview)	43244.779	31	1394.993		

A-8.11 Chapter 10 Reading Temporal Parameters: First of Two Fixation Duration

A-8 Table 21. Means and SE for ME Beverage Condition, ME Preview and IA Preview X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		275.474	7.407	260.346	290.601
	alcohol		293.359	7.837	277.354	309.365
ME Preview	legal		275.577	6.659	261.978	289.177
	illegal		293.256	8.085	276.744	309.768
IA Preview * Beverage Condition	legal	no alcohol	268.513	7.29	253.625	283.401
		alcohol	282.642	9.042	264.175	301.108
	illegal	no alcohol	282.434	10.21	261.583	303.286
		alcohol	304.077	8.305	287.12	321.039

A-8 Table 22. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	9917.028	1	9917.028	7.456	0.010
Error(alcohol)	39900.800	30	1330.027		
preview	9688.399	1	9688.399	10.505	0.003
Error(preview)	27667.839	30	922.261		
alcohol * preview	437.588	1	437.588	0.308	0.583
Error(alcohol*preview)	42589.144	30	1419.638		

A-8.12 Chapter 10 Reading Temporal Parameters: Refixation Duration (=Second of Two)

A-8 Table 23. Means and SE for ME Beverage Condition, ME Preview and IA Preview X Beverage Condition.

Effect			Mean	Std.Error	95% Confidence Interval	
					Lower Bound	Upper Bound
ME Beverage Condition	no alcohol		213.713	8.507	196.34	231.086
	alcohol		236.408	9.864	216.262	256.554
ME Preview	legal		220.112	8.624	202.499	237.725
	illegal		230.009	8.056	213.558	246.461
IA Preview * Beverage Condition	legal	no alcohol	206.356	10.158	185.61	227.102
		alcohol	233.867	10.719	211.977	255.757
	illegal	no alcohol	221.07	10.484	199.658	242.482
		alcohol	238.949	11.402	215.66	262.236

A-8 Table 24. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	15966.768	1	15966.768	4.199	0.049
Error(alcohol)	114080.901	30	3802.697		
preview	3036.806	1	3036.806	1.584	0.218
Error(preview)	57522.798	30	1917.427		
alcohol * preview	719.030	1	719.030	0.408	0.528
Error(alcohol*preview)	52837.541	30	1761.251		

A-8.13 Chapter 10 Reading Fixation Frequency Measures: Fixation Frequency

A-8 Table 25. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			1.723	.067	1.586	1.86
	alcohol			1.63	.057	1.515	1.746
ME Preview	legal			1.617	.053	1.51	1.724
	illegal			1.736	.067	1.599	1.872
ME Frequency	low frequency			1.844	.077	1.687	2.
	high frequency			1.509	.045	1.417	1.602
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	1.812	.073	1.662	1.961
			alcohol	1.733	.078	1.574	1.893
		high frequency	no alcohol	1.491	.048	1.392	1.589
			alcohol	1.434	.043	1.35	1.522
	illegal	low frequency	no alcohol	1.967	.096	1.771	2.163
			alcohol	1.862	.085	1.69	2.035
		high frequency	no alcohol	1.622	.077	1.465	1.779
			alcohol	1.491	.055	1.38	1.602

A-8 Table 26. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.552	1	0.552	5.338	0.028
Error(alcohol)	3.207	31	0.103		
preview	0.898	1	0.898	22.201	0.000
Error(preview)	1.254	31	0.040		
freq	7.149	1	7.149	54.924	0.000
Error(freq)	4.035	31	0.130		
alcohol * preview	0.041	1	0.041	0.783	0.383
Error(alcohol*preview)	1.608	31	0.052		
alcohol * freq	0.000	1	0.000	0.002	0.968
Error(alcohol*freq)	1.480	31	0.048		
preview * freq	0.037	1	0.037	1.450	0.238
Error(preview*freq)	0.790	31	0.025		
alcohol * preview * freq	0.009	1	0.009	0.361	0.552
Error(alcohol*preview*freq)	0.760	31	0.025		

A-8.14 Chapter 10 Reading Fixation Frequency Measures: Total Number of Fixations per Gaze

A-8 Table 27. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			1.403	.04	1.322	1.483
	alcohol			1.361	.033	1.293	1.428
ME Preview	legal			1.351	.032	1.286	1.415
	illegal			1.413	.039	1.333	1.493
ME Frequency	low frequency			1.493	.045	1.401	1.585
	high frequency			1.27	.028	1.212	1.328
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	1.478	.044	1.387	1.568
			alcohol	1.429	.052	1.324	1.535
		high frequency	no alcohol	1.246	.03	1.184	1.308
			alcohol	1.25	.029	1.19	1.309
	illegal	low frequency	no alcohol	1.566	.059	1.446	1.685
			alcohol	1.501	.046	1.408	1.594
		high frequency	no alcohol	1.322	.045	1.23	1.413
			alcohol	1.263	.034	1.193	1.333

A-8 Table 28. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.113	1	0.113	3.335	0.077
Error(alcohol)	1.046	31	0.034		
preview	0.249	1	0.249	11.311	0.002
Error(preview)	0.684	31	0.022		
freq	3.194	1	3.194	56.695	0.000
Error(freq)	1.746	31	0.056		
alcohol * preview	0.025	1	0.025	1.019	0.321
Error(alcohol*preview)	0.762	31	0.025		
alcohol * freq	0.014	1	0.014	1.173	0.287
Error(alcohol*freq)	0.368	31	0.012		
preview * freq	0.019	1	0.019	2.190	0.149
Error(preview*freq)	0.276	31	0.009		
alcohol * preview * freq	0.008	1	0.008	0.439	0.513
Error(alcohol*preview*freq)	0.591	31	0.019		

A-8.15 Chapter 10 Reading Fixation Frequency Measures: Refixation Frequency

A-8 Table 29. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.331	.029	.271	.39
	alcohol			.307	.026	.254	.359
ME Preview	legal			.296	.023	.248	.344
	illegal			.341	.03	.279	.403
ME Frequency	low frequency			.389	.03	.328	.45
	high frequency			.248	.025	.198	.298
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	.377	.029	.318	.435
			alcohol	.356	.038	.278	.434
		high frequency	no alcohol	.221	.026	.168	.274
			alcohol	.232	.026	0.18	.286
	illegal	low frequency	no alcohol	.433	.04	.352	.513
			alcohol	.391	.033	.325	.458
		high frequency	no alcohol	.293	.037	.216	.369
			alcohol	.248	.031	.185	.311

A-8 Table 30. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.037	1	0.037	1.771	0.193
Error(alcohol)	0.639	31	0.021		
preview	0.129	1	0.129	8.584	0.006
Error(preview)	0.467	31	0.015		
freq	1.269	1	1.269	66.873	0.000
Error(freq)	0.588	31	0.019		
alcohol * preview	0.023	1	0.023	1.081	0.306
Error(alcohol*preview)	0.663	31	0.021		
alcohol * freq	0.003	1	0.003	0.444	0.510
Error(alcohol*freq)	0.231	31	0.007		
preview * freq	0.000	1	0.000	0.006	0.937
Error(preview*freq)	0.227	31	0.007		
alcohol * preview * freq	0.005	1	0.005	0.390	0.537
Error(alcohol*preview*freq)	0.379	31	0.012		

A-8.16 Chapter 10 Reading Fixation Frequency Measures: Word Skipping

A-8 Table 31. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.034	.006	.021	.046
	alcohol			.053	.011	.03	.077
ME Preview	legal			.043	.007	.029	.056
	illegal			.044	.009	.026	.063
ME Frequency	low frequency			.044	.009	.026	.063
	high frequency			.042	.007	.028	.057
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	.037	.007	.023	.05
			alcohol	.045	.011	.023	.067
		high frequency	no alcohol	.031	.006	.018	.043
			alcohol	.059	.014	0.03	.087
	illegal	low frequency	no alcohol	.035	.013	.008	.061
			alcohol	.062	.015	.031	.092
		high frequency	no alcohol	.032	.007	.017	.048
			alcohol	.048	.012	.023	.073

A-8 Table 32. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.025	1	0.025	3.378	0.076
Error(alcohol)	0.226	31	0.007		
preview	0.000	1	0.000	0.089	0.768
Error(preview)	0.047	31	0.002		
freq	0.000	1	0.000	0.094	0.762
Error(freq)	0.084	31	0.003		
alcohol * preview	0.000	1	0.000	0.090	0.767
Error(alcohol*preview)	0.055	31	0.002		
alcohol * freq	0.000	1	0.000	0.163	0.689
Error(alcohol*freq)	0.050	31	0.002		
preview * freq	0.002	1	0.002	1.733	0.198
Error(preview*freq)	0.041	31	0.001		
alcohol * preview * freq	0.004	1	0.004	2.335	0.137
Error(alcohol*preview*freq)	0.028	31	0.001		

A-8.17 Chapter 10 Reading Fixation Frequency Measures: Regression Frequency

A-8 Table 33. Means and SE for ME Beverage Condition, ME Preview, ME Frequency and IA Preview X Frequency X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.147	.017	.112	.182
	alcohol			.112	.013	.086	.138
ME Preview	legal			.122	.013	.096	.148
	illegal			.137	.018	.101	.173
ME Frequency	low frequency			.133	.014	.103	.162
	high frequency			.126	.014	.097	.156
IA Preview * Frequency * Beverage Condition	legal	low frequency	no alcohol	.147	.018	.109	.185
			alcohol	.124	.016	.092	.156
		high frequency	no alcohol	.131	.021	.089	.173
			alcohol	.087	.012	0.06	.111
	illegal	low frequency	no alcohol	.141	.022	.097	.186
			alcohol	.118	.017	.084	.152
		high frequency	no alcohol	.168	.027	.114	.222
			alcohol	.119	.022	.075	.163

A-8 Table 34. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
alcohol	0.078	1	0.078	9.669	0.004
Error(alcohol)	0.249	31	0.008		
preview	0.014	1	0.014	1.248	0.273
Error(preview)	0.345	31	0.011		
freq	0.003	1	0.003	0.920	0.345
Error(freq)	0.088	31	0.003		
alcohol * preview	0.000	1	0.000	0.012	0.915
Error(alcohol*preview)	0.283	31	0.009		
alcohol * freq	0.009	1	0.009	1.097	0.303
Error(alcohol*freq)	0.245	31	0.008		
preview * freq	0.027	1	0.027	7.976	0.008
Error(preview*freq)	0.103	31	0.003		
alcohol * preview * freq	0.000	1	0.000	0.012	0.913
Error(alcohol*preview*freq)	0.198	31	0.006		

A-8.18 Chapter 10 Reading Fixation Frequency Measures: Regression Frequency by Initial Landing Position

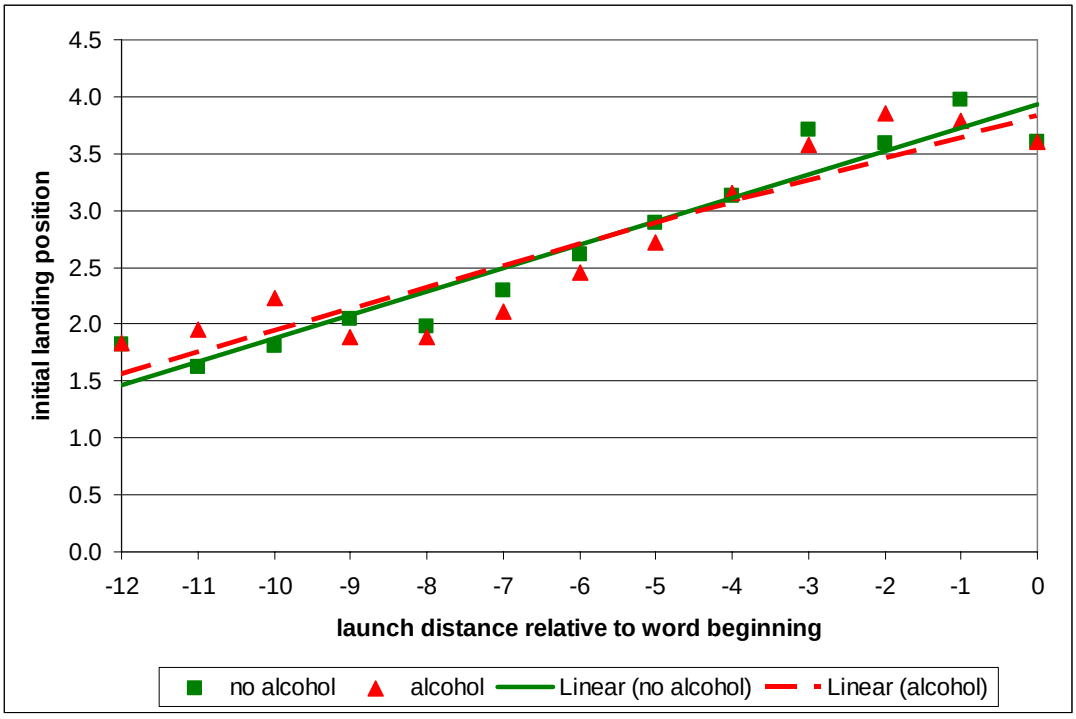
A-8 Table 35. Means and SE for ME Beverage Condition, ME Initial Landing Position, ME Preview, IA Initial Landing Position X Beverage Condition and IA Initial Landing Position X Preview X Beverage Condition.

Effect				Mean	Std.Error	95% Confidence Interval	
						Lower Bound	Upper Bound
ME Beverage Condition	no alcohol			.073	.009	.056	.091
	alcohol			.056	.006	.043	.069
ME Initial Landing Position	front			.077	.009	.059	.096
	back			.052	.006	.039	.066
ME Preview	legal			.061	.006	.048	.075
	illegal			.068	.009	.05	.086
IA Initial Landing Position * Beverage Condition	front		no alcohol	.09	.012	.065	.115
			alcohol	.064	.007	.049	.079
	back		no alcohol	.056	.007	.042	.07
			alcohol	.048	.008	.033	.064
IA Initial Landing Position * Preview * Beverage Condition	front	legal	no alcohol	.086	.013	.059	.113
			alcohol	.059	.008	.043	.075
		illegal	no alcohol	.095	.016	.062	.127
			alcohol	.069	.011	0.05	.091
	back	legal	no alcohol	.053	.009	.035	.072
			alcohol	.047	.008	.03	.065
		illegal	no alcohol	.059	.008	.043	.075
			alcohol	.049	.008	0.03	.065

A-8 Table 36. ANOVA Output.

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
liw	0.039	1	0.039	11.108	0.002
Error(liw)	0.110	31	0.004		
alc	0.019	1	0.019	9.441	0.004
Error(alc)	0.061	31	0.002		
prev	0.003	1	0.003	1.024	0.319
Error(prev)	0.086	31	0.003		
liw * alc	0.005	1	0.005	2.971	0.095
Error(liw*alc)	0.055	31	0.002		
liw * prev	0.000	1	0.000	0.214	0.647
Error(liw*prev)	0.071	31	0.002		
alc * prev	0.000	1	0.000	0.013	0.908
Error(alc*prev)	0.069	31	0.002		
liw * alc * prev	0.000	1	0.000	0.064	0.801
Error(liw*alc*prev)	0.041	31	0.001		

A-8.19 Chapter 10 Reading Fixation Frequency Measures: Landing Position Function



A-8 Figure 2. The landing position function plots the initial landing position relative to the launch distance. There were no differences between no alcohol and alcohol conditions.

Lebenslauf

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