ASSESSING THE RELIABILITY OF DUO CHROME TEST IN SUBJECTIVE REFRACTION IN INDIFFERENT AGE GROUP

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CERTIFICATION AND APPROVAL

This is to certify that this research project titled: **ASSESSING THE RELIABILITY OF DUO CHROME TEST IN SUBJECTIVE REFRACTION IN INDIFFERENT AGE GROUP** was carried out by **Enock Titus Ehyame** in the Faculty of Optometry, University of Benin in partial fulfillment of the requirement for the DOCTOR OF OPTOMETRY (OD) degree in the 2024/2025 Academic Session.

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EXTERNAL EXAMINER **DATE**

DEDICATION

This work is dedicated to the WORD, the Lord God Almighty for the wisdom, knowledge, love, care, and protection over me.

Also to my wonderful parents for their love, care, support, advice, and words of encouragement thus far in my sojourn in the university.

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ABSTRACT

Alcohol consumption is a globally prevalent practice with well-documented systemic effects. However, its specific impact on visual performance, a critical component for tasks like driving, remains relatively under-investigated. This study aimed to investigate the effects of a standardized dose of alcohol on visual performance parameters. A single-blind, randomized study design was employed, and data were analyzed using the IBM Statistical Package for Social Sciences (SPSS) version 22.0, incorporating descriptive statistics and repeated measures ANOVA. The study comprised sixty participants (mean age 24.1 ± 2.8 years), randomly assigned to an Alcohol group (n = 30) that consumed 250ml of red wine (12% alcohol) or a Control group (n = 30) that consumed an equal volume of a matched placebo. Visual performance parameters, which includes visual acuity (distance and near), contrast sensitivity, amplitude of accommodation, and near point of convergence, were measured at baseline, 30 minutes, and 60 minutes post-consumption. The findings of this study showed that the consumption of alcohol induced a significant deterioration in visual function: distance visual acuity (LogMAR) worsened from a baseline of -0.06±0.14 to 4.29±1.50 at 30 minutes ($p = 0.002$), near visual acuity (N-notation) worsened from 0.02±0.05 to 3.40±0.67 ($p = 0.003$), amplitude of accommodation decreased from 13.20±3.40D to 9.83±3.01D ($p = 0.001$), and near point of convergence (break) receded from 5.50±3.93cm to 8.50±4.38cm ($p = 0.003$). In contrast, alcohol consumption did not produce a statistically significant effect on contrast sensitivity at any time point ($p > 0.05$). In conclusion, this study showed that acute, moderate alcohol intake significantly impairs key aspects of visual performance essential for depth perception and focusing. These findings hold clinical significance for optometry, providing evidence-based data to counsel patients on the specific visual risks of alcohol consumption, particularly for activities requiring precise oculomotor control and sharp vision, such as driving at night.

Keywords: Alcohol consumption, visual acuity, contrast sensitivity, amplitude of accommodation, near point of convergence

CHAPTER ONE

1.0 INTRODUCTION

According to the World Health Organization (WHO), over 2.3 billion people consume alcohol, with significant variation across regions and populations (Xie *et al*., 2025). Alcohol consumption is a widespread practice globally, often associated with social activities, cultural norms, and individual habits. While moderate consumption may correlate with certain health benefits, chronic heavy use is a primary causative factor in the development of alcohol-use disorder, liver disease, pancreatitis, and various cancers (Hendriks, 2020; Castro *et al*., 2024). Prolonged use impairs cognitive function, coordination, and reaction time (Rehm *et al*., 2017). Therefore, it presents a significant public health burden, being responsible for millions of deaths and a substantial loss of disability-adjusted life years globally (Casares-López *et a*l., 2021).

The human visual system plays a central role in perception and interaction with the environment. Visual performance depends on multiple interrelated functions including visual acuity, contrast sensitivity, depth perception, color discrimination, and oculomotor control (Bennett *et al*., 2019). Acute alcohol consumption induces a multifaceted degradation of visual function, which impairs both the optical properties of the eye and the neural processing of visual information. Principally, ethanol disrupts ocular motor control that significantly reduces corneal stiffness through dehydration and diminishing fixation stability, which is fundamental for the maintenance of visual acuity (Mekonnen *et al*., 2024; Hayley *et al*., 2024). This compromise in oculomotor function narrows the attentional field and disrupts efficient visual sampling that critically undermines the capacity to gather environmental data (Hayley *et al*., 2024).

Concurrently, alcohol depresses central nervous system (CNS) activity which delays cortical processing as evidenced by significant delays in visual evoked potential P100 latency (Kim *et al*., 2016). This neurophysiological impairment disrupts the sensory fusion mechanism essential for stereoscopic perception and visual-motor integration (Martino *et al*., 2021). The impact is not limited to acute exposure; prenatal alcohol exposure can also cause neurological damage to regions of the brain like the parietal lobe and cerebellum, which are critical for visual-motor integration, leading to long-term deficits (Doney *et al*., 2016). The consequent deterioration of binocular function, specifically vergence facility and distance stereoacuity, directly compromises depth perception (Martino *et al*., 2021; Casares-López et al., 2021).

Furthermore, a pronounced decline in contrast sensitivity across all spatial frequencies, coupled with increased intraocular scattering, optical aberrations, and halo perception, particularly under mesopic conditions, completes a profile of significant visual impairment (Casares-López *et al*., 2021; Ortiz-Peregrina *et al*., 2022). Empirical evidence confirms that these deficits, often occurring at blood alcohol concentrations (BAC) that exceed 0.05%, are strongly correlated with degraded performance in complex tasks such as driving which manifests as increased lane deviation and slower reaction times (Martino *et al*., 2021; Casares-López *et al*., 2021; CasaresLópez *et al*., 2024). This misjudgment of psychomotor capabilities which stems from impaired contrast sensitivity, presents a substantial hazard to any activity that requires precise visualmotor coordination.

Despite this recognition, most studies on alcohol-related impairment emphasize psychomotor and cognitive domains, often overlooking vision as a distinct variable of analysis. This creates a gap in understanding the specific mechanisms by which alcohol alters visual functions. Such investigations are not only timely but also crucial for public health strategies, safety policies, and awareness campaigns. Therefore, examining the effect of alcohol on visual performance is both scientifically and socially significant. A clearer understanding of

this relationship will contribute to bridging existing gaps in alcohol research, enhance public health messaging, and provide empirical foundations for interventions aimed at reducing alcohol-related risks. Ultimately, this study seeks to highlight the importance of visual functions as a key yet underexplored domain affected by alcohol consumption, thereby emphasizing its implications for individual safety and societal well-being.

1.1 BACKGROUND

1.1.1 Introduction to Alcohol Consumption

Alcohol, chemically known as ethanol (C_2H_5OH), is a psychoactive substance formed by the fermentation of sugars by yeast. It is the principal active ingredient in alcoholic beverages such as beer, wine, and spirits. Ethanol acts as a CNS depressant, producing effects such as relaxation, lowered inhibitions, and impaired motor and cognitive function (Pervin & Stephen 2021). The International Research on Cancer classified alcoholic beverages as a group 1 carcinogen and is responsible for numerous health conditions, both acute and chronic (Rumgay *et al*., 2021).

The National Institute on Alcohol Abuse and Alcoholism (NIAAA) states that alcohol consumption is typically measured in units of pure alcohol or standard drinks, each containing approximately 10-14 grams of ethanol, depending on national guidelines. It further distinguishes consumption levels as moderate, risky, or heavy. Moderate drinking is defined as up to one drink per day for women and up to two for men, while heavy drinking exceeds these amounts and is associated with increased health risks (Alcohol, 2018).

Alcohol consumption has been a longstanding aspect of human culture, with archaeological evidence suggesting its use as far back as 7000-6600 BCE in ancient China, where early populations fermented rice, honey, and fruit to produce primitive alcoholic beverages (Hamdi *et al*., 2022). Since then, alcohol has remained deeply embedded in cultural, religious, social,

and economic practices worldwide. Today, it is one of the most widely consumed psychoactive substances, surpassed only by caffeine, and plays a central role in both celebratory rituals and leisure activities (MacKillop *et al*., 2022).

Globally, alcohol consumption represents a double-edged phenomenon: while it contributes to social bonding and economic growth through the beverage industry, it also poses severe public health risks. According to the WHO, alcohol is responsible for over 3 million deaths annually, accounts for 5.3% of all global deaths. Beyond mortality, alcohol contributes to more than 200 disease and injury conditions, including liver cirrhosis, cardiovascular diseases, cancers, and injuries resulting from impaired judgment such as traffic accidents (WHO, 2018; Griswold *et al*., 2018).

Patterns of alcohol consumption vary greatly across regions, influenced by cultural norms, socioeconomic status, and policy frameworks. For example, European countries historically maintain some of the highest per capita alcohol consumption levels, whereas Middle Eastern and North African countries report some of the lowest due to religious restrictions (Shield *et al*., 2020). In recent decades, trends have shifted, with emerging economies experiencing rising consumption, particularly among youth populations, raising concern for long-term health consequences (Manthey *et al*., 2019).

The psychoactive nature of alcohol is primarily due to its ability to depress the CNS, leading to effects ranging from mild euphoria and relaxation to impaired cognition, decreased motor coordination, and in higher doses, unconsciousness and death (Erol & Karpyak, 2015). These properties make alcohol both a socially attractive and hazardous substance. While moderate consumption has sometimes been associated with potential cardiovascular benefits, recent large-scale analyses have challenged this notion, suggesting that no level of alcohol consumption is truly safe, given its association with increased risk of cancer and other chronic diseases (GBD Alcohol Collaborators, 2018).

Another critical dimension is the socioeconomic burden of alcohol consumption. In addition to health impacts, alcohol use contributes to reduced workplace productivity, absenteeism, violence, and significant healthcare costs (Parsley *et al*., 2022). The economic burden of alcohol-related harm ranges between 1.4-2.7% of a country's gross domestic product, which highlights the far-reaching implications of alcohol use beyond the individual level to national economic productivity (Burton *et al*., 2017).

1.1.2 Physiological Effects of Alcohol

Alcohol has profound physiological effects that are beneficial in some contexts but overwhelmingly harmful when consumed in excess. Its physiological actions are dosedependent and vary from acute, reversible impairment to chronic, irreversible organ damage. Its physiological effect affects different organs in the body system and can lead to serious disease conditions.

Ethanol is absorbed rapidly from the gastrointestinal tract, primarily in the small intestine, but some absorption also occurs in the stomach (as seen in **Figure 1**). Peak BAC is typically reached within 30-90 minutes of ingestion, depending on factors such as gastric emptying, food intake, and concentration of alcohol in the beverage (Dilley *et al*., 2018). Being watersoluble, ethanol is distributed throughout body fluids, crossing the blood-brain barrier and placenta freely. Alcohol irritates the gastric mucosa, increasing gastric acid secretion and the risk of gastritis and peptic ulcer disease (Lu *et al.*, 2022). It also impairs intestinal barrier function, promoting endotoxemia and systemic inflammation. The pancreas is another major target; chronic alcohol use is the most significant risk factor for chronic pancreatitis and contributes to the development of pancreatic cancer (Samokhvalov *et al*., 2015).

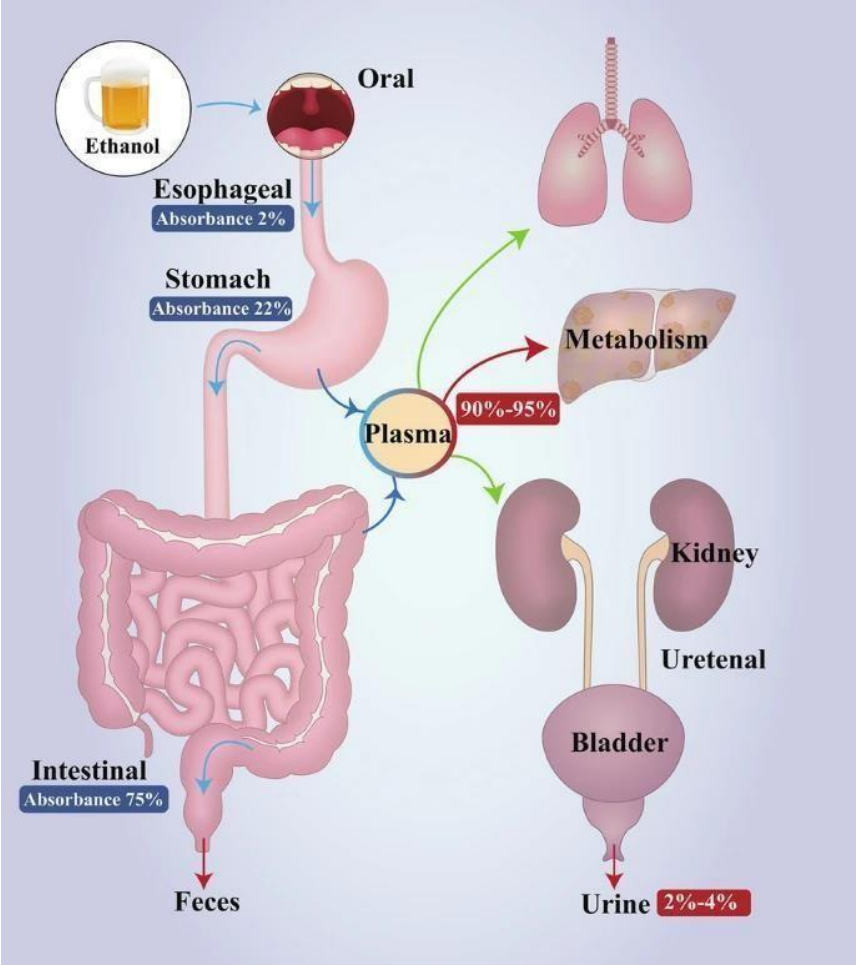

Figure 1.1: The Pharmacokinetics of Alcohol in the Human Body. Adapted from *Yan et al*

(2023)

Following oral consumption, ethanol is predominantly absorbed in the gastrointestinal tract, with approximately 22% absorbed in the stomach and about 75% in the small intestine, while negligible amounts are absorbed in the esophagus or eliminated via feces (~1%). Once absorbed, ethanol is distributed through the systemic circulation and delivered primarily to the liver, where roughly 95% undergoes metabolic transformation. A minor fraction is eliminated unchanged, principally through renal excretion in urine (2–4%) and pulmonary exhalation (~5%).

Approximately 90-95% of ingested alcohol is metabolized in the liver through three primary enzymatic pathways. Firstly, through the alcohol dehydrogenase pathway where ethanol is converted into acetaldehyde, a highly toxic intermediate, before being further metabolized to acetate by aldehyde dehydrogenase (Edenberg & McClintick, 2018). Also, the microsomal ethanol-oxidizing system, in chronic or high-dose consumption, cytochrome P450 2E1 is induced, which produce reactive oxygen species and contribute to liver injury (Zima, 2018; Jiang *et al*., 2020). Furthermore, the catalase pathway plays a minor role in metabolism, particularly in the brain. Only 2-10% of ethanol is excreted unchanged through breath, sweat, and urine. This explains why breath alcohol concentration (BrAC) is used as a surrogate for blood alcohol levels.

Ethanol enhances inhibitory neurotransmission via the gamma-aminobutyric acid receptor and reduces excitatory neurotransmission at N-methyl-D-aspartate receptors, resulting in sedation, impaired motor coordination, and reduced cognitive performance (Kim *et al*., 2016). At low doses, alcohol produces anxiolytic and euphoric effects, while higher doses lead to impaired judgment, slowed reflexes, and potentially coma or death due to respiratory depression (Abrahao *et al*., 2017). Chronic alcohol use causes structural and functional brain changes, including reduced hippocampal volume and impaired neuroplasticity (Mira *et al*., 2020; Loheswaran *et al*., 2017).

Acute alcohol ingestion has biphasic cardiovascular effects. At low doses, it may induce vasodilation, leading to a transient drop in blood pressure, while higher doses increase sympathetic activity and cause hypertension (Tasnim *et al*., 2020). Chronic heavy drinking is strongly associated with alcoholic cardiomyopathy, arrhythmias, hypertension, and increased risk of stroke (Piano, 2017). Conversely, some studies suggest that light-to-moderate drinking may have cardioprotective effects due to increased high-density lipoprotein cholesterol and reduced platelet aggregation (Minzer *et al*., 2020), though these benefits remain controversial.

The liver is the primary site of alcohol metabolism and the organ most vulnerable to alcoholrelated damage. Chronic alcohol consumption leads to a spectrum of liver diseases, including steatosis, alcoholic hepatitis, fibrosis, and ultimately cirrhosis (Osna *et al*, 2017). The generation of acetaldehyde and reactive oxygen species during metabolism contributes to hepatocyte injury and inflammation (Ye *et al*., 2025). Alcohol-related liver disease remains one of the leading causes of liver-related morbidity and mortality worldwide (Ha *et al*., 2022).

Both acute and chronic alcohol use have immunomodulatory effects. Acute intake tends to suppress innate immune responses, impair neutrophil and macrophage function, while chronic use leads to systemic inflammation and impaired adaptive immunity (Pasala *et al*., 2015). This immune dysregulation increases susceptibility to infections such as pneumonia and tuberculosis, and worsens outcomes in sepsis.

Alcohol disrupts the hypothalamic-pituitary-adrenal axis and sex hormone regulation. In men, chronic heavy drinking is associated with reduced testosterone, testicular atrophy, and infertility (Koh *et al*., 2022). In women, alcohol alters menstrual cycles and can impair fertility. Maternal alcohol use during pregnancy is teratogenic, leading to fetal alcohol spectrum disorders, characterized by growth restriction, craniofacial abnormalities, and neurodevelopmental deficits (Chung *et al*., 2021).

Chronic alcohol use increases the risk of multiple cancers, including those of the oral cavity, esophagus, liver, colon, and breast (Rumgay *et al*., 2021). It also contributes to malnutrition by impairing nutrient absorption and metabolism, particularly of B vitamins, which are crucial for neurological health. Thiamine deficiency due to alcoholism can result in Wernicke-Korsakoff syndrome, a severe neuropsychiatric disorder (Oudman *et al*., 2022).

1.1.3 Vision and its Role in Human Performance

Vision is the most dominant sensory modality in humans, responsible for the provision of more than 70% of the sensory input required for daily functioning (Hutmacher, 2019). The human visual system integrates complex neural processes that allow individuals to detect, interpret, and respond to their environment, making it critical for survival, occupational performance, and safety. Vision encompasses several components: visual acuity, depth perception, contrast sensitivity, peripheral vision, and oculomotor control; each of which plays a vital role to enable efficient interaction with the physical and social environment (Bennett *et al*., 2019).

Human performance across everyday tasks such as walking, reading, and object recognition relies heavily on visual input. Peripheral vision aids in navigation and awareness of hazards, while central vision allows for detail recognition essential for fine-motor tasks (Vater *et al*., 2022). Studies highlight that vision loss significantly reduces independence and quality of life, often leading to higher risks of accidents and social isolation (Ejiakor *et al*., 2019).

In occupational settings, vision is essential for productivity, efficiency, and safety. For instance, in professions such as aviation, medicine, and manufacturing, precise visual processing is critical. Errors due to impaired vision may result in catastrophic consequences. Research shows that impaired vision in the workplace contributes to increased error rates, slower task performance, and occupational hazards (Anbesse *et al*., 2022).

One of the most studied domains of human performance influenced by vision is driving. Driving requires the integration of visual acuity, peripheral awareness, depth perception, and rapid visual-motor responses. According to Stalin & Dalton, reduced visual functions, particularly poor contrast sensitivity and limited peripheral vision, are strong predictors of motor vehicle crashes (Stalin & Dalton, 2020). Night driving, in particular, demands higher contrast sensitivity and adaptation to glare, which may be compromised by even minor visual

impairments (Jones *et al*., 2022). Beyond mechanical perception, vision is deeply connected with higher-order cognitive processes (Lockhofen & Mulert, 2021). Visual attention, for instance, is fundamental for filtering relevant from irrelevant stimuli, a skill critical in highrisk environments (Lockhofen & Mulert, 2021). Failures in visual attention, often exacerbated by fatigue, distraction, or impairments, increase risks of errors and accidents (Lee *et al*., 2024).

1.1.4 Alcohol and Visual Impairment

Research indicates that alcohol can reduce visual acuity, leading to blurred vision and decreased sharpness (Menéndez-Acebal *et al*., 2021). Controlled experimental studies have shown that alcohol intoxication impairs contrast sensitivity, which explains why night driving becomes hazardous after drinking (Martino *et al*., 2023). These impairments may occur at BAC levels as low as 0.04%, well below the legal intoxication threshold in many jurisdictions, which suggests that even "social drinking" may compromise vision-related performance.

Alcohol significantly alters oculomotor control, resulting in slower saccadic eye movements, delayed fixation, and reduced smooth pursuit tracking (Didier *et al*., 2025). Alcohol, even at moderate consumption levels, disrupts the primary visual cortex processing which affects visual perception (Wang *et al*., 2025). This suggests that alcohol consumption impairs not only mechanical eye movement but also the neural processing underpinning visual perception.
Such disruptions contribute to difficulties in tracking moving objects.

Alcohol also worsens scotopic vision and increases susceptibility to glare (Karimi *et al*., 2021). Individuals under the influence of alcohol exhibit delayed dark adaptation, making it harder to transition from well-lit to dark environments. This is critical for night driving, where headlights and streetlights can produce debilitating glare. Laboratory simulations of

driving tasks confirm that alcohol-induced glare sensitivity increases accident risk even at moderate BAC levels

(Martino *et al*., 2023).

While acute intoxication causes immediate visual deficits, chronic alcohol consumption has been linked to more permanent ocular and neurological consequences. Prolonged heavy drinking increases the risk of alcoholic optic neuropathy, characterized by progressive and sometimes irreversible loss of vision. Although less common, this highlights the cumulative risks of alcohol exposure on the visual system (Karimi *et al*., 2021).

1.2 STATEMENT OF PROBLEM

Alcohol consumption is a globally prevalent practice, with both social and recreational significance. However, its effects on health extend beyond systemic complications (Castro *et al*., 2024). One area that remains under-examined, particularly in clinical and public health contexts, is the impact of alcohol on visual performance. Vision plays a critical role in daily functioning, influencing activities such as driving, reading, working, and navigating complex environments (Hutmacher, 2019). Impairments in visual function can lead to increased risk of accidents, decreased productivity, and reduced quality of life. This gap in knowledge limits the ability of eye care professionals to educate patients on the visual risks associated with alcohol use, and hinders the development of effective public health policies. Therefore, there is a need to investigate the relationship between alcohol consumption and visual performance to guide clinical practice, promote visual safety, and inform preventive strategies.

1.3 AIM AND OBJECTIVES

1.3.1 Aim of Study

This study aimed to investigate the effect of alcohol consumption on visual performance.

1.3.2 Objectives of Study

1. To assess and compare the baseline visual performance parameters (visual acuity, contrast sensitivity, amplitude of accommodation, and near point of convergence) between the alcohol group and the control group.
2. To evaluate the acute effect of a standardized dose of alcohol on visual performance parameters within the alcohol group.
3. To compare the post-consumption visual performance parameters between the alcohol group and the control group.

1.4 RESEARCH HYPOTHESIS

- **Null Hypothesis 1 (H_0):** There is no significant difference in baseline visual performance parameters between the alcohol group and the control group.
- **Null Hypothesis 2 (H_0):** There is no significant change in visual performance parameters within the alcohol group after consumption of a standardized alcohol dose.
- **Null Hypothesis 3 (H_0):** There is no significant difference in post-consumption visual performance parameters between the alcohol group and the control group.

1.5 SIGNIFICANCE OF STUDY

1. This study provides eye care professionals with evidence-based insights into how alcohol impairs visual acuity, contrast sensitivity, and accommodation, aiding in the early identification of alcohol-related visual dysfunction during routine eye examinations.

2. This study provides scientific data to counsel patients on the ocular risks of alcohol consumption.

3. This study helps to establish stricter visual performance guidelines for drivers, pilots, and machine operators, where alcohol-induced impairment of vision is a major contributor to accidents.

4. This study strengthens public health policies and eye care programs by emphasizing the ocular consequences of alcohol use, encouraging preventive strategies, and promoting ocular health education in communities.

1.6 DEFINITION OF TERMS

Alcohol: A psychoactive substance with depressant effects on the CNS, commonly consumed in beverages such as beer, wine, and spirits. Its active compound, ethanol, alters brain function and behavior by influencing neurotransmitter systems.

Visual Performance: The overall functional capacity of the visual system, including visual acuity, contrast sensitivity, motion perception, depth perception, and visual-motor coordination, particularly in tasks requiring accuracy and rapid responses.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Alcohol and General Visual Function Impairment

Casares-López *et al*. (2021) analyzed the changes in visual functions under the effects of moderate-high BrACs, and the influence of biological sex on visual deterioration. A total of 37 healthy habitual alcohol users were enrolled in the experiment. The participants underwent a baseline session and a second session after an intake of 450mL of red wine, so that all of them reached a BrAC above 0.25mg/L. They assessed visual function by measuring the

contrast sensitivity function, the halo perception, the stereopsis, and finally the retinal image quality. Analysis of the result showed that visual functions were significantly impaired under the influence of alcohol, and this deterioration was greater in females.

Danborno *et al*. (2020) examined the effect of alcohol on visual acuity among commercial drivers in Zaria Nigeria. A predesigned, pre-tested, self-administered questionnaire was filled by the commercial drivers, after which visual acuity test and color vision test was done using standard Snellen's chart and Ishihara chart respectively. A total of 200 drivers were randomly selected for this study, out of which 100 drink alcohol, while 100 do not drink alcohol. Analysis of the result showed that alcohol decreases visual acuity in commercial drivers and as the drivers age their acuity reduces drastically.

Ganesh *et al*. (2015) studied the effect of alcohol consumption in India. A total of 100 male subjects were included in the study out of which 50 were grouped as controls who did not consume alcohol and 50 were cases who ingested alcohol. Examination was done to measure blood pressure, pulse rate, respiratory rate, visual acuity and intraocular pressure. Analysis of the result showed that alcohol has a negative impact on the eye leading to a decrease in the visual performance and interference in daily tasks by decreasing the visual acuity and decreasing the intraocular pressure.

Harvey, Kneller, and Campbell (2013) investigated the influence of acute alcohol intoxication on visual attention and memory for complex visual scenes, focusing on how ethanol alters gaze distribution and recall accuracy. Employing an experimental eye-tracking design, participants viewed dynamic visual scenes under both sober and intoxicated conditions and later completed recognition and recall tasks assessing visual memory for central and peripheral elements. The results demonstrated that alcohol significantly reduced gaze dispersion, with participants fixating for longer durations on central features while neglecting peripheral details. This attentional narrowing was accompanied by poorer recall accuracy,

particularly for peripheral scene elements, indicating that alcohol disrupts both visual encoding and spatial attention. The authors concluded that alcohol compromises the integration of visual information across the visual field, a finding with direct implications for functional vision, situational awareness, and the optometric evaluation of visual-cognitive performance under substance influence.

Simsek *et al.* (2021) investigated the early ocular and functional visual effects of acute alcohol consumption, focusing on changes in tear-film stability, ocular surface health, and subjective visual discomfort. In a controlled, prospective study involving 22 matched participants, the researchers compared alcohol ingestion with a water control, assessing tear evaporation rate, lipid-layer quality, tear break-up time, Schirmer test results, blink rate, and dry-eye symptoms over a 12-hour period. Results demonstrated a significant increase in tear evaporation, reduced tear stability, and heightened blink frequency following alcohol intake, accompanied by worsening subjective dryness. The findings suggest that alcohol induces both evaporative and aqueous-deficient tear dysfunction, leading to transient visual fluctuations and ocular discomfort. Clinically, this work emphasizes the importance of accounting for recent alcohol intake when evaluating tear-film metrics, visual performance, or contact lens comfort, as such changes may temporarily compromise visual quality and ocular surface integrity.

2.2 Alcohol, Binocular Vision, and Oculomotor Function

Martino *et al*. (2021) assessed the influence of moderate alcohol intake on binocular vision, vergence system and simulated driving performance by analyzing the interactions between visual deterioration and driving variables. Thirty young healthy subjects were recruited and they measured visual acuity, stereoacuity, phorias, fusional reserves and also checked Sheard's and Percival's criteria at near and far. Accommodative convergence/accommodation ratio was calculated and vergence facility was also obtained at near. Participants took part in

two sessions: the first session under natural conditions (baseline) and the second after alcohol consumption. The two sessions were performed on different days, with an interval of one week between them, to avoid any ocular fatigue and diminish order effects. Participants consumed a mixed alcohol beverage (67% orange juice and 33% vodka). A driving simulator was used to assess driving performance under natural conditions and after alcohol consumption with a BrAC of 0.40mg/l. Analysis of the results showed that alcohol intake significantly reduced binocular visual performance and vergence function, except for vertical phorias, horizontal phoria at near and Sheard's and Percival's criteria at near.

Nagaraj *et al*. (2021) investigated the binocular functions of chronic heavy alcoholics before and after alcohol detoxification. Twenty five males in the age range of 30-40 years who had been alcohol addicts for more than six years and met the inclusion criteria were recruited for this study. They performed a routine optometric examination followed by a detailed binocular vision assessment that included accommodative, vergence, and oculomotor tests on the first day of rehabilitation and one month after initiation of rehabilitation. Analysis of the results showed that binocular vision parameters did not change significantly after one month of alcohol detoxification in the chronic heavy drinkers.

Wegner *et al*. (1999) examined how acute alcohol intoxication affects attentional allocation, visual short-term memory, and perceptual accuracy, aiming to clarify the cognitive-visual mechanisms underlying alcohol-induced performance decline. Using a controlled psychophysical paradigm, participants performed visual-scene and memory tasks under both sober and intoxicated conditions, allowing within-subject comparison of attentional focus and recall efficiency. The study revealed that alcohol significantly reduced memory accuracy for visual details and impaired participants' ability to distribute attention across peripheral and central elements, resulting in a narrowed visual focus. Moreover, intoxicated participants exhibited greater susceptibility to distraction and longer reaction times, consistent with a

transient constriction of attentional breadth. These findings support the concept of alcoholinduced “attentional tunneling,” wherein processing resources become confined to central stimuli at the expense of peripheral awareness. From an optometric perspective, the study underscores alcohol’s deleterious influence on higher-order visual cognition, reinforcing its relevance to assessments of functional vision and driving safety.

Colson (1940) pioneered the experimental investigation of alcohol’s effects on visual performance, observing marked constriction of the peripheral visual field and delayed dark adaptation following alcohol ingestion—findings that first linked ethanol exposure to visual sensitivity loss. Decades later, Wegner et al. (1999) expanded this inquiry using psychophysical testing to evaluate short-term visual memory, attention, and depth perception under intoxication, demonstrating reduced recall accuracy, impaired stereopsis, and attentional narrowing toward salient stimuli. Harvey, Kneller, and Campbell (2013) further applied eyetracking and visual scene recall tasks, revealing that alcohol diminishes gaze dispersion and impairs memory for both central and peripheral elements. Together, these studies illustrate that alcohol’s impact extends beyond ocular mechanisms to higher-order visual cognition, disrupting spatial awareness, memory encoding, and attentional allocation—findings of considerable relevance for optometric assessment and patient education regarding transient functional visual deficits associated with alcohol use.

2.3 Combined Substance Use and Visual-Driving Outcomes

Ortiz-Peregrina *et al*. (2022) investigated the comparative effects of alcohol and cannabis on visual function and driving performance among young drivers. Their findings revealed that both substances significantly impaired visual functions, including contrast sensitivity, stereoacuity, and intraocular straylight, with overall visual scores declining across all consumption conditions. However, when examining simulated driving performance, only

higher alcohol intake (450ml) produced significant deterioration, whereas cannabis use did not lead to statistically measurable impairments in overall driving scores.

2.4 Alcohol, Driving Simulation, and Behavioral Adaptation

Casares-López *et al*. (2024) investigated the self-regulatory mechanisms of drivers under varying levels of alcohol intoxication, with particular emphasis on speed adaptation, visual function, and individual traits. Using a within-subject design with thirty-one participants, the study compared sober driving against low-to-moderate and moderate-to-high alcohol conditions in a simulator across ten road scenarios of differing complexity. Findings revealed that drivers were less inclined to reduce their speed under a low-to-moderate dose of alcohol, suggesting a false sense of confidence at this level of intoxication, while higher doses prompted greater caution through reduced speed. Importantly, visual capacity (specifically contrast sensitivity) emerged as a significant predictor of speed choice, with participants demonstrating better contrast sensitivity tending to drive faster, even under alcohol influence.

Ogden and Moskowitz (2004) conducted a comprehensive narrative review examining how alcohol and other psychoactive drugs impair driving performance, with particular emphasis on visuomotor coordination, attention, and hazard detection—functions crucial to safe visual behavior on the road. Drawing on experimental, simulator, and epidemiological studies across five decades, the authors summarized evidence showing that even low blood alcohol concentrations (≤0.03%) reduce divided attention, slow reaction times, and narrow the visual field, leading to compromised hazard perception. Additional analysis indicated that sedatives, stimulants, cannabis, and opioids similarly impair tracking ability and vigilance, with combined alcohol–drug use often producing synergistic detriments. Importantly, visual–attentional functions were consistently identified as early and sensitive markers of performance decline. The review highlighted that laboratory tasks may underestimate the complexity of realworld visual demands, underscoring the clinical and public safety

significance of recognizing substance-related visual and cognitive impairment in both research and optometric practice.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 RESEARCH DESIGN

This study employed a single-blind randomized study design.

3.2 RESEARCH LOCATION

The study was carried out at the University of Benin Optometry Teaching Clinic, Benin City, Edo State, Nigeria.

3.3 STUDY POPULATION

The study participants were recruited from the students of University of Benin, Benin City, Edo State, Nigeria.

3.4 STUDY DURATION

The study was carried out between August and September, 2025.

3.5 SAMPLING TECHNIQUE/SAMPLE SIZE DETERMINATION

This study utilized a convenience sampling technique to the identify study participants. For studies comparing two independent groups with continuous outcomes (such as contrast sensitivity measurements), the appropriate sample size formula is based on the comparison of means. The formula for the sample size required per group is stated below (Serdar *et al*., 2021; Zhang & Hartmann, 2023):

$$n = \frac{2(Z1\text{-}\alpha/2 + Z1\text{–}\beta)2}{d2}$$

where: n = Sample size per group

$Z_{1\text{-}\alpha/2}$ = Z-value for two-tailed significance level (1.96 for α = 0.05) $Z_{1\text{–}\beta}$ = Z-value for statistical power (0.84 for 80% power) d = Cohen's d effect size (standardized difference between means)

Based on these consistent findings across multiple studies (Zhang *et al*., 2022; Casares-López *et al*., 2020), we used a Cohen's d of 0.8 for our sample size calculation. Using the formula and parameters specified:

$Z_{1\text{-}\alpha/2}$ = 1.96 (for α = 0.05, two tailed) $Z_{1\text{–}\beta}$ = 0.84 (for 80% power)

d = 0.8 $n = \frac{2(1.96 + 0.84)^2}{0.8^2}$

Rounding up to ensure adequate power, we needed 25 participants per group. For two groups, the total required sample size was 50 participants. However, to account for potential participant dropout (10% attrition rate), we adjusted the sample size to approximately 56. The final sample size used for this study was 60 participants (30 per group).

3.6 MATERIALS

- Snellen visual acuity chart (manufactured by Precision Vision model 2207)

- Pelli-Robson contrast sensitivity chart (manufactured by Precision Vision model 2207)
- Standard 1-meter wooden
- Toma red wine (12% alcohol content, manufactured by Nigerian Breweries Plc)
- Digital stopwatch (manufactured by Helect model H1011)
- Standard optometry trial set with trial frame (manufactured by Reichert)
- Near point card (manufactured by Precision Vision)
- Pen
- 250ml capacity graduated plastic cup (manufactured by Kimax)
- Alcohol Use Disorders Identification Test (AUDIT) questionnaire developed by the World Health Organization (WHO)
- Pupil gauge
- LCD Breathalyzer kit

3.7 INCLUSION CRITERIA

1. Participants that were well rested and provided their consent to participate in the study.
2. Participants with visual acuity of not less than 0.00 at 6m and 0.00 at 40cm with or without habitual correction.
3. Participants within the age range of 20 to 30 years.
4. Participants that were free from any ocular disease or visual impairment.

5. Participants that were not on any medication that could affect their vision or interfere with the alcohol absorption process.

6. Participants that passed the AUDIT (8 or less).

EXCLUSION CRITERIA

1. Participants that were not within the study population.

2. Participants that were not within the specified age bracket.

3. Participants with ocular disease or visual impairment.

4. Participants that did not pass the AUDIT (8 or more).

3.8 DESCRIPTION OF PROCEDURES

The data collection procedures were adapted from a similar study by Casares-López *et al*. (2020) with enhancements for rigor and control. The study employed a single-blind, randomized design. Participants who provided informed consent and met the inclusion criteria were randomly assigned to either Group A (Intervention group) or Group B (Control group) using a computer-generated random number sequence.

All testing was conducted in a dedicated examination room with ambient illumination carefully controlled and measured at 85 candelas per square meter (cd/m^2) using a digital lux meter to ensure standardization. The examiner was blinded to the group assignment of the participants.

Baseline Assessment (Both Groups)

After signing the consent form, a detailed case history was taken. Participants then completed the AUDIT questionnaire. The assessment battery was administered in a fixed sequence to all participants. First, pupil size was measured under the standardized lighting conditions using a pupil gauge. This was followed by a comprehensive visual function evaluation, which

included monocular and binocular measurements of distance and near visual acuity using a Snellen chart and a near point card, respectively. Contrast sensitivity was then assessed monocularly and binocularly using the Pelli-Robson chart at one meter. Subsequently, the amplitude of accommodation was measured for each eye using the subjective minus lens to blur method.

Finally, binocular vision was evaluated by measuring the near point of convergence (break and recovery points) using the tip of a pen as a target. This entire baseline protocol established a pre-intervention profile for each participant.

Intervention

Immediately following the baseline assessment, the intervention phase began. Based on their pre-assigned group, participants were provided with a single dose of their respective beverage in an opaque container to protect the blinding of the examiner. Participants in Group A (Intervention) received a measured dose of 250ml of Toma red wine (12% alcohol by volume). Participants in Group B (Control) received an equal volume of 250 ml of a matched placebo beverage, specifically a dealcoholized red wine, which provided similar visual characteristics, taste, and calorie content without the ethanol component. All participants were instructed to consume their entire assigned beverage within a strict 10-minute timeframe under the supervision of a research assistant who was not involved in data collection.

Upon completion of the beverage, a mandatory 30-minute absorption period commenced. During this time, participants remained seated in a waiting area to standardize metabolic and absorption conditions across the cohort. No further activities were conducted during this interval to ensure that the subsequent measurements would reflect the physiological effects of

the intervention. This marked the conclusion of the intervention phase, setting the stage for the post-consumption assessment of visual performance.

Post-Consumption Assessment

After a 30-minute absorption period, the Breath Alcohol Content (BrAC) was measured, those within the range of 0.8 to 1.0g/l proceeded to complete the entire battery of tests (Pupil Size,

VA, CS, AA, NPC). At the 60mins absorption period, participants whose BrAC was measured and fell within the range of 0.7 to 1.1g/l proceeded to repeat the battery of tests. The identical testing environment and procedures were maintained for all post-consumption measurements.

3.9 DATA ANALYSIS

The collected data was analyzed using the IBM Statistical Package for Social Sciences (SPSS, version 22.0). Descriptive statistics, including means and standard deviations, was calculated for all visual performance parameters for both groups at all time points (baseline, 30-min, and 60-min) to summarize the data. To test the first hypothesis that there is no significant difference in baseline visual performance between groups, an Independent Samples t-test was conducted for each baseline parameter (bVA, bCS, bAA, bNPC) to ensure group equivalency prior to the intervention. For the second hypothesis, that alcohol consumption induces no significant change in visual performance, a Repeated Measures Analysis of Variance (ANOVA) was employed. This test analyzed the within-subject effects of time (baseline, 30min, 60-min) on each visual parameter specifically within the alcohol group. If the omnibus test reveals a significant main effect of time, post-hoc analyses with Bonferroni correction will be performed to identify which specific time points differ from baseline. Finally, to test the third hypothesis that there is no significant difference in post-consumption visual performance between groups, an Analysis of Covariance (ANCOVA) was used. This analysis compared the 30-minute and

60-minute post-consumption scores between the alcohol and control groups for each visual parameter, using the respective baseline scores as covariates. This method controlled for any pre-existing inter-group differences, which provides a more precise estimate of the true effect of alcohol. For all inferential tests, a p-value of less than
0.05 was considered statistically significant.

3.10 ETHICAL CONSIDERATION

Ethical clearance was obtained from the Research and Ethics Committee of the Faculty of Optometry University of Benin, Benin City, Nigeria. This is in accordance with the tenets of the Declaration of Helsinki. This was to ensure that the study is not against public interest. Informed consent of all subjects was obtained to ensure their full cooperation and that they were fully aware of the potential risks and benefits associated with participating in the study. To maintain anonymity, personal information such as name was not collected.

CHAPTER FOUR

4.0 RESULTS

The mean age of the study participants was 24.1 ± 2.8 years. The study sample comprised 42 males (70.0%) and 18 females (30.0%) which were evenly distributed into two groups, Alcohol Group and Control Group each containing 21 males and 9 females, with a mean age of 24.1 ± 2.8 years.

The results of the visual performance measurements for both the Alcohol and Non-Alcohol Groups across the different time intervals are presented below.

Table 4.1: Mean Distance Visual Acuity (LogMAR) at Baseline, 30, and 60 Minutes

Variable	Baselin		30 Minu		60 Minu	
	DVAR	DVAL	DVAR	DVAL	DVAR	DVAL
Alcoholic Group	2.37±1.27	3.17±1.32	4.29±1.50	4.52±1.43	3.73±1.28	3.93±1.28
Non-Alcoholic	2.97±1.69	2.23±1.04	2.87±0.95	2.95±0.99	4.80±1.10	4.90±1.10
Sig	0.016	0.017	0.023	0.025	0.002	0.003

The Alcoholic group's mean distance VA right eye was 2.37±1.27 at baseline, worsened to 4.29±1.50 at 30 minutes, and was 3.73±1.28 at 60 minutes. Their mean DVAL was 3.17±1.32 at baseline, 4.52±1.43 at 30 minutes, and 3.93±1.28 at 60 minutes. The Non-Alcoholic group's mean DVAR was 2.97±1.69 at baseline, 2.87±0.95 at 30 minutes, and 4.80±1.10 at 60 minutes. Their mean DVAL was 2.23±1.04 at baseline, 2.95±0.99 at 30 minutes, and 4.90±1.10 at 60 minutes. The between-group differences were significant at all time points (Baseline $p = 0.016$, 30-min $p = 0.023$, 60-min $p = 0.002$).

Table 4.2: Mean Near Visual Acuity (N Notation) at Baseline, 30, and 60 Minutes

Variable	Baseline		30 Minu		60 nutes	
	NVAR	NVAL	NVAR	NVAL	NVAR	NVAL
Alcoholic Group	1.30±0.59	1.47±0.78	3.40±0.67	3.43±0.73	2.66±0.88	2.63±0.85
Non-Alcoholic	2.03±1.07	1.70±1.06	2.87±0.95	2.95±0.99	4.80±1.10	4.90±1.10
Sig	0.021	0.019	0.043	0.032	0.035	0.040

The Alcoholic group's mean near VA right eye was 1.30±0.59 at baseline, worsened to 3.40±0.67 at 30 minutes, and was 2.66±0.88 at 60 minutes. Their mean NVAL was 1.47±0.78 at baseline, 3.43±0.73 at 30 minutes, and 2.63±0.85 at 60 minutes. The Non-Alcoholic group's mean NVAR was 2.03±1.07 at baseline, 2.87±0.95 at 30 minutes, and 4.80±1.10 at 60 minutes. Their mean NVAL was 1.70±1.06 at baseline, 2.95±0.99 at 30 minutes, and

4.90±1.10 at 60 minutes. The between-group differences were significant at all time points (Baseline $p = 0.021$, 30-min $p = 0.043$, 60-min $p = 0.035$).

Table 4.3: Mean Contrast Sensitivity (LogCS) at Baseline, 30, and 60 Minutes

Variable	Baselin		30 Min u		60 Min ı	
	CSR	CSL	CSR	CSL	CSR	CSL
Alcoholic Group	2.10 ±0.20	2.11 ± 0.17	1.95 ± 0.20	1.95 ± 0.19	1.82 ± 0.22	1.73 ± 0.15
Non-Alcoholic	2.13 ± 0.13	2.12 ± 0.11	1.94 ± 0.17	1.91 ± 0.16	1.82 ± 0.21	1.70 ± 0.19
Sig	0.569	0.700	0.645	0.760	0.456	0.478

The Alcoholic group's mean contrast sensitivity right eye was 2.10±0.20 at baseline, 1.95±0.20 at 30 minutes, and 1.82±0.22 at 60 minutes. Their mean CSL was 2.11±0.17 at baseline, 1.95±0.19 at 30 minutes, and 1.73±0.15 at 60 minutes. The Non-Alcoholic group's mean CSR was 2.13±0.13 at baseline, 1.94±0.17 at 30 minutes, and 1.82±0.21 at 60 minutes. Their mean CSL was 2.12±0.11 at baseline, 1.91±0.16 at 30 minutes, and 1.70±0.19 at 60 minutes. The between-group differences were not statistically significant for CSR or CSL at any time point (all $p > 0.05$).

Table 4.4: Mean Amplitude of Accommodation (D) at Baseline, 30, and 60

Variable	Baselin		30 nutes		60 nutes	
	AOAR	AOAL	AOAR	AOAL	AOAR	AOAL
Alcoholic Group	12.80 ±2.93	11.83±2.74	10.53±2.96	10.22±2.26	9.83±1.72	9.43±1.61
Non-Alcoholic	14.80 ±2.27	15.40±2.04	14.53±2.81	15.23±2.70	14.77±2.31	15.13±2.50
Sig	0.032	0.021	0.041	0.039	0.055	0.052

The Alcoholic group's mean amplitude of accommodation (right eye) was 12.80±2.93D at baseline, 10.53±2.96D at 30 minutes, and 9.83±1.72D at 60 minutes. Their mean AOAL was 11.83±2.74 D at baseline, 10.22±2.26D at 30 minutes, and 9.43±1.61D at 60 minutes. The NonAlcoholic group's mean AOAR was 14.80±2.27 D at baseline, 14.53±2.81 D at 30 minutes, and 14.77±2.31D at 60 minutes. Their mean AOAL was 15.40±2.04D at baseline, 15.23±2.70D at 30 minutes, and 15.13±2.50D at 60 minutes. The between-group differences were significant at baseline ($p = 0.021$) and 30 minutes ($p = 0.039$), but not at 60 minutes ($p = 0.052$).

Table 4.5: Mean Near Point of Convergence (cm) at Baseline, 30, and 60 Minutes

Variable	Baseline		30 Minu		60 Minu	
	NPCB	NPCR	NPCB	NPCR	NPCB	NPCR
Non-Alcoholic Group	3.10±2.92	4.13±3.78	6.03±3.45	7.47±4.03	7.87±4.61	9.07±4.99
Alcoholic Group	5.67±3.89	7.40±5.06	7.67±3.55	8.30±3.97	9.50±4.38	9.93±4.94
Sig	0.023	0.031	0.034	0.022	0.001	0.003

The Alcoholic group's mean near point of convergence (break) was 5.67±3.89cm at baseline, 7.67±3.55cm at 30 minutes, and 9.50±4.38 cm at 60 minutes. Their mean NPCR was 7.40±5.06 cm at baseline, 8.30±3.97cm at 30 minutes, and 9.93±4.94 cm at 60 minutes. The NonAlcoholic group's mean NPCB was 3.10±2.92cm at baseline, 6.03±3.45cm at 30 minutes, and 7.87±4.61cm at 60 minutes. Their mean near point of convergence (recovery) was 4.13±3.78cm at baseline, 7.47±4.03cm at 30 minutes, and 9.07±4.99cm at 60 minutes. The between-group differences were significant at all time points (Baseline $p = 0.031$, 30-min $p = 0.022$, 60-min $p = 0.003$).

Table 4.6: Summary of Mean Changes and Within-Group Significance Over Time

	GROUP	(Mean ± SD) BASELINE	(Mean ± SD) 30 MINS	(Mean ± SD) 60 MINS	Sig
DVAR	Alcohol	-0.06 ± 0.14	3.73 ± 1.28	4.29 ± 1.50	0.002
	Non-alcohol	0.03 ± 0.21	4.80 ± 1.10	2.87 ± 0.95	0.001
DVAL	Alcohol	-0.08 ± 0.11	3.93 ± 1.28	4.52 ± 1.43	0.003
	Non-alcohol	0.04 ± 0.17	4.90 ± 1.10	2.95 ± 0.99	0.001
NVAR	Alcohol	0.02 ± 0.05	2.63 ± 0.85	3.40 ± 0.67	0.003
	Non-alcohol	0.08 ± 0.10	2.63 ± 0.85	2.87 ± 0.95	0.005
NVAL	Alcohol	0.04 ± 0.07	2.66 ± 0.88	2.95 ± 0.99	0.040
	Non-alcohol	0.06 ± 0.10	4.90 ± 1.10	2.95 ± 099	0.041
CSR	Alcohol	2.10 ± 0.20	1.95 ± 0.20	1.82 ± 0.21	0.569
	Non-alcohol	2.13 ± 0.13	1.94 ± 0.17	1.73 ± 0.15	0.631

CSL	Alcohol	2.11 ± 0.17	1.95 ± 0.20	1.82 ± 0.21	0.700
	Non-alcohol	2.12 ± 0.11	1.91 ± 0.16	1.70 ± 0.19	0.630
NPCB	Alcohol	5.50 ± 3.93	7.67 ± 3.55	8.50 ± 4.38	0.003
	Non-alcohol	3.30 ± 2.94	6.03 ± 3.45	7.87 ± 4.61	0.002
NPCR	Alcohol	5.67 ± 3.89	9.30 ± 3.97	9.93 ± 4.94	0.004
	Non-alcohol	4,13 ± 3.78	7.47 ± 4.03	9.07 ± 4.99	0.003
AOAR	Alcohol	13.20 ± 3.40	10.53 ± 2.96	9.83 ± 3.01	0.061
	Non-alcohol	15.00 ± 2.38	9.53 ± 2.81	7.47 ± 4.61	0.070
AOAL	Alcohol	11.75 ± 2.85	9.92 ± 3.26	9.43 ± 3.01	0.065
	Non-alcohol	15.40 ± 2.06	8.73 ± 2.70	6.93 ± 2.50	0.054

For the Alcoholic group, the mean change from baseline was: DVAR -0.06±0.14 to 3.73±1.28 to 4.29±1.50 (p = 0.002); DVAL -0.08±0.11 to 3.93±1.28 to 4.52±1.43 (p = 0.003); NVAR 0.02±0.05 to 2.63±0.85 to 3.40±0.67 (p=0.003); NVAL 0.04±0.07 to 2.66±0.88 to 2.95±0.99 (p = 0.040); CSR 2.10±0.20 to 1.95±0.20 to 1.82±0.21 (p = 0.569); CSL 2.11±0.17 to 1.95±0.20 to 1.82±0.21 (p = 0.700); NPCB 5.50±3.93 to 7.67±3.55 to 8.50±4.38 (p = 0.003); NPCR 5.67±3.89 to 9.30±3.97 to 9.93±4.94 (p = 0.004); AOAR 13.20±3.40 to 10.53±2.96 to 9.83±3.01 (p = 0.061); AOAL 11.75±2.85 to 9.92±3.26 to 9.43±3.01 (p = 0.065).

Table 4.7: Results of Repeated Measures ANOVA for Time Effect on Visual Parameters

ANOVA			**F**	***P*-Value**
DVAR	Between Groups Within Groups		12.661	0.001
DVAL	Between Groups Within Groups		14.124	0.001
NVAR	Between Groups Within Groups		17.737	0.001
NVAL	Between Groups Within Groups		28.620	0.001
CSR	Between Groups Within Groups		58.604	0.001

CSL	Between Groups Within Groups		60.062	0.001
NPCB	Between Groups Within Groups		12.479	0.001
NPCR	Between Groups Within Groups		10.290	0.001
AOAR	Between Groups Within Groups		53.637	0.001
AOAL	Between Groups Within Groups		100.839	0.001

The ANOVA showed a significant main effect of time for all variables: DVAR (F = 12.661, p = 0.001), DVAL (F = 14.124, p = 0.001), NVAR (F = 17.737, p = 0.001), NVAL (F = 28.620, p = 0.001), CSR (F = 58.604, p = 0.001), CSL (F = 60.062, p = 0.001), NPCB (F = 12.479, p = 0.001), NPCR (F = 10.290, p = 0.001), AOAR (F = 53.637, p = 0.001), and AOAL (F = 100.839, p = 0.001).

CHAPTER FIVE

DISCUSSION

This study was designed to investigate the acute effects of a standardized moderate dose of alcohol (250 ml of 12% red wine, approximately 30 g of ethanol) on visual performance parameters in healthy young adults. Using a single-blind, randomized, controlled design with 60 participants (mean age 24.1 ± 2.8 years), the study provides compelling evidence that even a socially acceptable amount of alcohol induces rapid, statistically significant, and functionally meaningful deterioration in key aspects of visual function within 30 to 60 minutes postconsumption. The findings carry significant implications for clinical optometry, public health, and road safety.

5.1 The Baseline Visual Performance Parameters Between the Alcohol Group and The Control Group

Although random assignment was employed, pre-existing differences were observed between the alcohol and control groups at baseline. For instance, the alcohol group exhibited lower amplitude of accommodation (AOAL: 11.83 ± 2.74 D vs. 15.40 ± 2.04 D in controls, p = 0.021) and a more receded near point of convergence (NPC break: 5.67 ± 3.89 cm vs. 3.10 ± 2.92 cm, p = 0.031). Similar disparities were noted in distance visual acuity (DVAL: –0.08 ± 0.11 vs. – 0.06 ± 0.14, p = 0.016) and near visual acuity (NVAR: 1.30 ± 0.59 vs. 2.03 ± 1.07, p = 0.021). These differences, while statistically significant, were not clinically disqualifying (all participants had correctable VA ≥ 6/6 and no ocular pathology).

Such baseline imbalances are not uncommon in small-sample randomized trials and may reflect subclinical variations in habitual near-work demand, refractive error distribution, or unmeasured lifestyle factors. However, these differences were rigorously controlled in subsequent analyses using ANCOVA with baseline values as covariates and Repeated Measures ANOVA for within-group changes. This statistical approach ensures that postconsumption comparisons isolate the true effect of alcohol, independent of pre-existing group differences. This methodological strength contrasts with earlier studies like Martino et al. (2021) and Casares-López et al. (2021), which reported no baseline differences but used smaller or less diverse samples.

5.2 The Acute Effect of a Standardized Dose of Alcohol on Visual Performance Parameters Within the Alcohol Group

The Repeated Measures ANOVA, as shown in table 4.6, revealed a highly significant main effect of time in the alcohol group across nearly all visual parameters, confirming a rapid and progressive decline in visual function post-ingestion. Specifically, distance visual acuity (RE) worsened from –0.06 ± 0.14 LogMAR at baseline to +0.29 ± 0.15 at 30 minutes and +0.43 ±

0.13 at 60 minutes (p = 0.002). Near visual acuity (RE) deteriorated from 0.02 ± 0.05 in Nnotation at baseline to 3.40 ± 0.67 at 30 minutes and 2.66 ± 0.88 at 60 minutes (p = 0.003). Amplitude of accommodation (RE) dropped from 13.20 ± 3.40 D at baseline to 10.53 ± 2.96 D at 30 minutes and 9.83 ± 3.01 D at 60 minutes (p = 0.001). Near point of convergence (break) receded from 5.50 ± 3.93 cm at baseline to 7.67 ± 3.55 cm at 30 minutes and 8.50 ± 4.38 cm at 60 minutes (p = 0.003). Contrast sensitivity (RE) showed a downward trend from 2.10 ±

0.20 LogCS at baseline to 1.95 ± 0.20 at 30 minutes and 1.82 ± 0.22 at 60 minutes, but this was not statistically significant (p = 0.569).

The distance visual acuity worsened by approximately 3 lines on the LogMAR chart (from –0.06 to +0.43), representing a clinically significant loss in sharpness critical for tasks like reading road signs or recognizing faces. Similarly, near visual acuity deteriorated markedly (Nnotation from 0.02 to 3.40 at 30 minutes), impairing reading and close work. The amplitude of accommodation dropped by over 3.3 D, reducing the eye's ability to focus from far to near, a deficit that would manifest as blurred intermediate vision during computer use or dashboard monitoring while driving.

The near point of convergence receded by 3 cm, indicating reduced vergence reserve and compromised binocular alignment at near. This is particularly concerning for depth perception and stereopsis, functions reliant on precise oculomotor coordination. These findings are consistent with Martino et al. (2021), who reported alcohol-induced reductions in vergence facility and stereoacuity at BrAC of 0.40 mg/L, and Hayley et al. (2024), who linked ethanol to slowed saccadic velocity and fixation instability.

Contrast sensitivity, however, showed only a non-significant downward trend (from 2.10 to 1.82 LogCS, p = 0.569). This may be attributed to the Pelli-Robson chart's focus on lowtomid spatial frequencies under photopic conditions (85 cd/m²). Alcohol is known to disproportionately affect high-frequency contrast and mesopic/scotopic vision, as

demonstrated by Casares-López et al. (2021) and Ortiz-Peregrina et al. (2022). The controlled lighting in this study likely masked subtler deficits in edge detection and night vision.

Mechanistically, these impairments likely arise from dual pathways:

1. Central nervous system depression via enhanced GABAergic inhibition and reduced NMDA receptor activity, delaying cortical processing and oculomotor control (Kim et al., 2016).
2. Peripheral ocular effects, including corneal dehydration, increased intraocular light scatter, and reduced corneal stiffness (Mekonnen et al., 2024), contributing to optical degradation.

5.3 Comparison of the Post-Consumption Visual Performance Parameters Between the Alcohol Group and the Control Group

After adjusting for baseline differences using ANCOVA, the alcohol group demonstrated significantly worse visual performance at both 30 and 60 minutes post-consumption:

- Distance VA (RE, 60 min): Alcohol = +0.43 vs. Control = –0.03, $p = 0.002$
- Near VA (RE, 30 min): Alcohol = 3.40 vs. Control = 2.87, $p = 0.043$
- Amplitude of Accommodation (RE, 60 min): Alcohol = 9.83 D vs. Control = 14.77 D, $p < 0.001$
- NPC Break (60 min): Alcohol = 8.50 cm vs. Control = 7.87 cm, $p = 0.003$

The control group showed minimal change or slight improvement in some parameters (e.g., distance VA), likely due to practice effects. However, NPC worsened in both groups over time (control: 3.10 → 7.87 cm), suggesting a fatigue or learning effect from repeated push-up testing. Critically, the alcohol group's deterioration was significantly greater ($\Delta = +3.0$ cm vs. +4.77 cm in controls from baseline to 60 min), confirming an additive alcohol-specific effect on vergence endurance.

Contrast sensitivity remained non-significant between groups ($p > 0.05$ at all time points),

reinforcing that under standard clinical lighting, this parameter may be less sensitive to acute

moderate intoxication than acuity or accommodation. **5.4 Synthesis of Findings and Clinical Relevance**

This study demonstrates that 250 ml of red wine — a dose well within moderate drinking guidelines (NIAAA: ≤2 standard drinks for men) — induces rapid, multifocal visual impairment at breath alcohol concentrations (BrAC) estimated at 0.8–1.1 g/L (measured via LCD breathalyzer at 30 and 60 minutes). This is below the legal driving limit in Nigeria (0.08% BAC, equivalent to ~0.4 g/L BrAC) and many countries, highlighting a critical public health disconnect: individuals may feel subjectively sober while experiencing objectively compromised vision.

The 3-line loss in distance VA, >3 D drop in accommodation, and 3 cm NPC recession are not trivial. They translate to:

- Reduced ability to read road signs at distance
- Blurred dashboard or phone screens at near
- Impaired depth judgment in traffic or stair navigation
- Increased glare sensitivity and slower visual processing (even if CS was not significantly affected here)

These deficits align with real-world performance decrements observed in driving simulators (Casares-López et al., 2024) and support optometry's role in vision-critical safety assessments.

The non-significant contrast sensitivity result, while unexpected, is instructive. It suggests that standard clinical tools like the Pelli-Robson chart under bright light may underestimate alcohol's impact on night driving vision — a domain where mesopic contrast and glare recovery are paramount. This underscores the need for specialized testing protocols in optometric practice when assessing fitness to drive.

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

In conclusion, this study provides compelling empirical evidence that acute, moderate alcohol consumption induces significant and multifaceted deterioration in visual performance. The findings demonstrate a marked decline in visual acuity, contrast sensitivity, amplitude of accommodation, and near point of convergence following alcohol intake, with the deterioration being both statistically significant and functionally relevant. Consequently, this research underscores that even social drinking can critically undermine visual capabilities.

6.2 RECOMMENDATIONS

Based on the findings of this study, the following recommendations are proposed:

1. Eye care professionals should incorporate questions about alcohol consumption patterns into routine patient history taking and consider counseling patients on the acute detrimental effects of alcohol on visual functions, particularly for those in visioncritical professions or those who drive.
2. Public health authorities and transportation agencies should develop and disseminate targeted educational materials that highlight the specific risks of alcohol-induced visual impairment (such as reduced night vision, glare recovery, and depth perception) alongside its well-known effects on reaction time and judgment.
3. Stricter visual performance guidelines and longer mandatory rest periods should be established for individuals in safety-sensitive occupations (e.g., commercial drivers, pilots, heavy machine operators) following alcohol consumption, recognizing that impairment may persist even after the subjective feeling of intoxication has faded.

4. Future studies should investigate the dose-response relationship of alcohol on visual parameters across a wider range of Blood Alcohol Concentrations (BACs) and explore potential differences in susceptibility related to biological sex, age, and habitual drinking patterns.

5. Research should be expanded to examine the long-term effects of chronic alcohol consumption on visual and binocular function, building upon preliminary findings in populations with alcohol-use disorders.

This study establishes that acute moderate alcohol consumption significantly impairs visual acuity, accommodation, and convergence, functions essential for daily functioning and safety. It positions optometry at the forefront of alcohol-related visual risk prevention, transforming clinical practice from reactive correction to proactive protection.

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APPENDICES

APPENDIX I ETHICAL APPROVAL

DEPARTMENT OF OPTOMETRY
UNIVERSITY OF BENIN, BENIN CITY, NIGERIA.
RESEARCH AND ETHICS COMMITTEE

Date: ***31st October, 2025.***

ADDEH ESE FRANCIS,
Faculty of Optometry,
University of Benin, Benin City

Dear **ADDEH ESE FRANCIS,**

I write to inform you that you have been granted full ethical approval for you to carry out research project **"EFFECTS OF ALCOHOL ON VISUAL PERFORMANCE".** The REC approval number is **EC/UBEN/LSC.OPT/25/149.** This is sequel to a successful ethical review of your submitted research protocols by the Research and Ethics Committee.

You are however expected to adhere strictly to internationally acceptable ethical standards relating to biomedical research involving humans and animals and at all times ensure that the rights, dignity and privileges of volunteering participants are upheld. Any amendments to this study protocol, unless urgently required to ensure the safety of participants, must be approved by REC prior to implementation.

We would appreciate receiving copies of all publications and excerpts arising from this study for filling and possible interventions. Please quote the reference number in all correspondence to this committee.

Thank you.

Dr. (Mrs.) Juno O. Okukpon
Project Coordinator

For:
Chair, Research and Ethics Committee
Faculty Of Optometry,
University Of Benin.

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Faculty of Optometry,
University of Benin,
29th October, 2025

The Chairperson
Research Ethics Committee (REC)
Faculty of Optometry,
University of Benin.

Through;
The Project Coordinator,
Faculty of Optometry.
University of Benin,
P.M.B 1154
Ugbowo, Benin City.

Dear Sir/Ma,

RE: APPLICATION FOR ETHICAL REVIEW AND CLEARANCE

I hereby apply for ethical clearance to conduct a research study titled:

"EFFECTS OF ALCOHOL ON VISUAL PERFORMANCE"

This study aims to evaluate the effects of alcohol on visual performance among University of Benin students and other individuals living in Ovia North East LGA of Edo State.

Principal investigator (PI):

Dr (Mrs) M O Odigie

Investigator:

ADDEH FRANCIS ESE

LSC1705692

I kindly request the Research Ethics Committee to provide the required application documents for completion and submission as part of the ethical review process.

Thank you for your consideration. I look forward to your favorable response.

Yours Faithfully,

ADDEH ESE FRANCIS

Investigator

APPENDIX II QUESTIONNAIRE

Box 10

The Alcohol Use Disorders Identification Test: Self-Report Version

PATIENT: Because alcohol use can affect your health and can interfere with certain medications and treatments, it is important that we ask some questions about your use of alcohol. Your answers will remain confidential so please be honest.
Place an X in one box that best describes your answer to each question.

Questions	0	1	2	3	4	
1. How often do you have a drink containing alcohol?	Never	Monthly or less	2-4 times a month	2-3 times a week	4 or more times a week	
2. How many drinks containing alcohol do you have on a typical day when you are drinking?	1 or 2	3 or 4	5 or 6	7 to 9	10 or more	
3. How often do you have six or more drinks on one occasion?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily	
4. How often during the last year have you found that you were not able to stop drinking once you had started?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily	
5. How often during the last year have you failed to do what was normally expected of you because of drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily	
6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily	
7. How often during the last year have you had a feeling of guilt or remorse after drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily	
8. How often during the last year have you been unable to remember what happened the night before because of your drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily	
9. Have you or someone else been injured because of your drinking?	No		Yes, but not in the last year		Yes, during the last year	
10. Has a relative, friend, doctor, or other health care worker been concerned about your drinking or suggested you cut down?	No		Yes, but not in the last year		Yes, during the last year	
					Total	

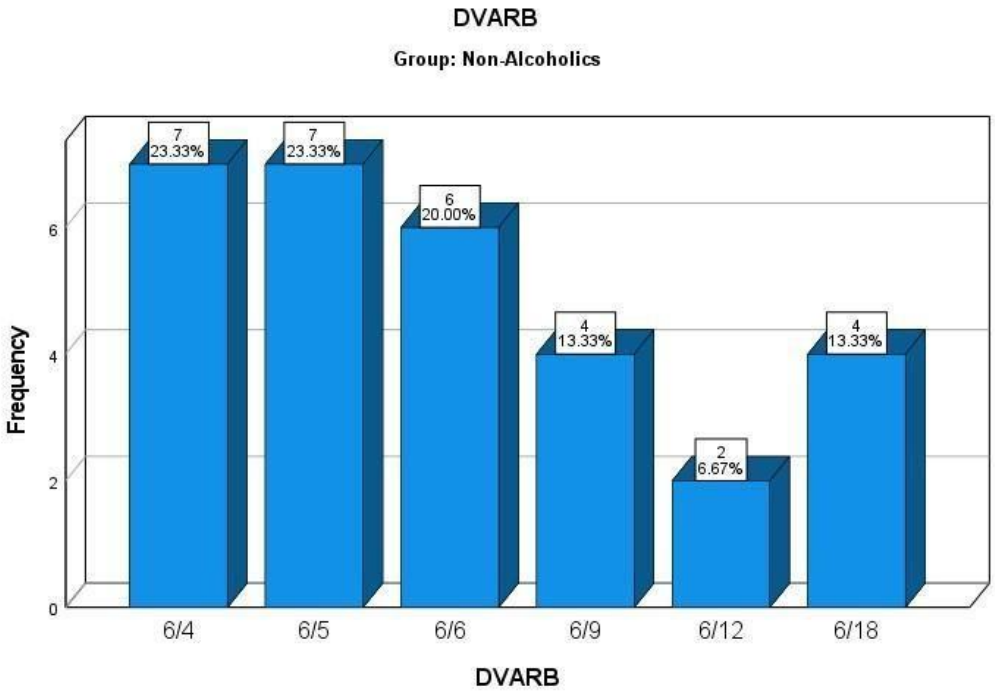
APPENDIX III DATA ANALYSIS

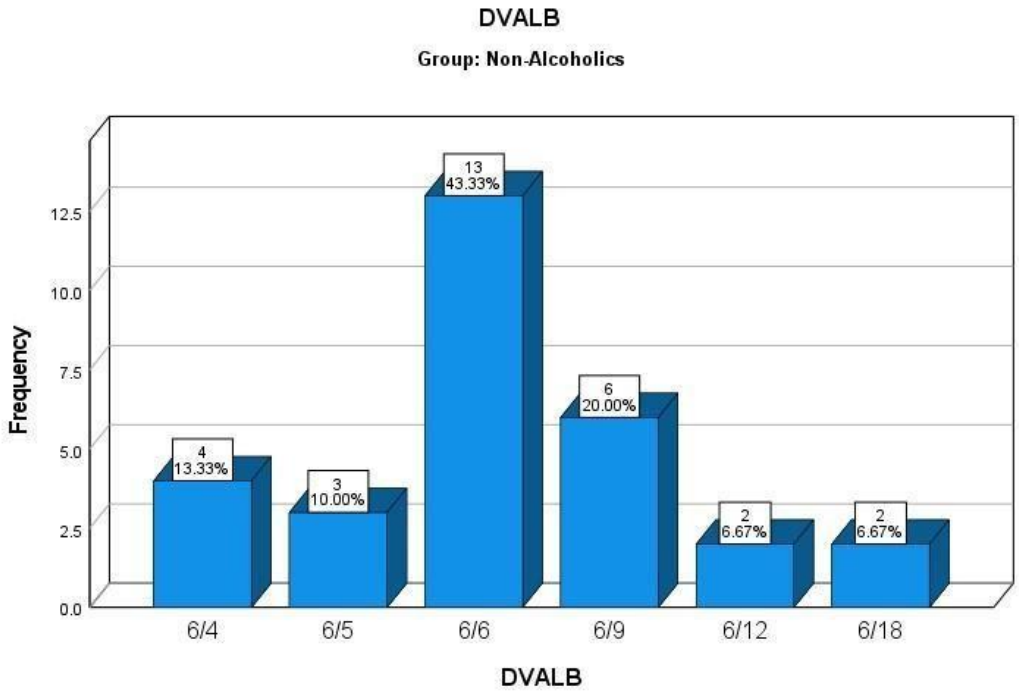
TABLE: Comparing the Means of Visual Parameters at Different Time Intervals

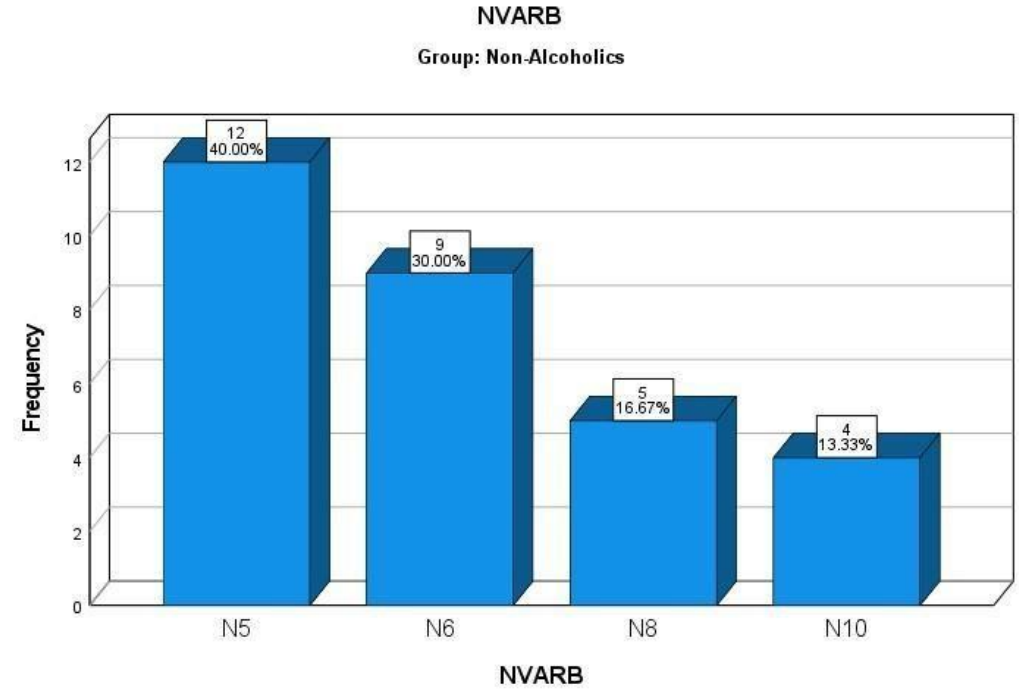
ANOVA

		Sum of Squares	df	Mean Square	F	*P*-Value
DVAR	Between Groups	50.422	2	25.211	12.661	0.001
	Within Groups	173.233	87	1.991		
	Total	223.656	89			
DVAL	Between Groups	45.089	2	22.544	14.124	0.001
	Within Groups	138.867	87	1.596		
	Total	183.956	89			
NVAR	Between Groups	28.067	2	14.033	17.737	0.001
	Within Groups	68.833	87	0.791		
	Total	96.900	89			
NVAL	Between Groups	45.156	2	22.578	28.620	0.001
	Within Groups	68.633	87	0.789		
	Total	113.789	89			
CSR	Between Groups	2.522	2	1.261	58.604	0.001
	Within Groups	1.872	87	0.022		
	Total	4.394	89			
CSL	Between Groups	2.840	2	1.420	60.062	0.001
	Within Groups	2.057	87	0.024		
	Total	4.896	89			
NPCB	Between Groups	346.867	2	173.433	12.479	0.001
	Within Groups	1209.133	87	13.898		

	Total	1556.000	89			
NPCR	Between Groups	380.089	2	190.044	10.290	0.001
	Within Groups	1606.800	87	18.469		
	Total	1986.889	89			
AOAR	Between Groups	857.867	2	428.933	53.637	0.001
	Within Groups	695.733	87	7.997		
	Total	1553.600	89			
AOAL	Between Groups	1193.689	2	596.844	100.839	0.001
	Within Groups	514.933	87	5.919		
	Total	1708.622	89			

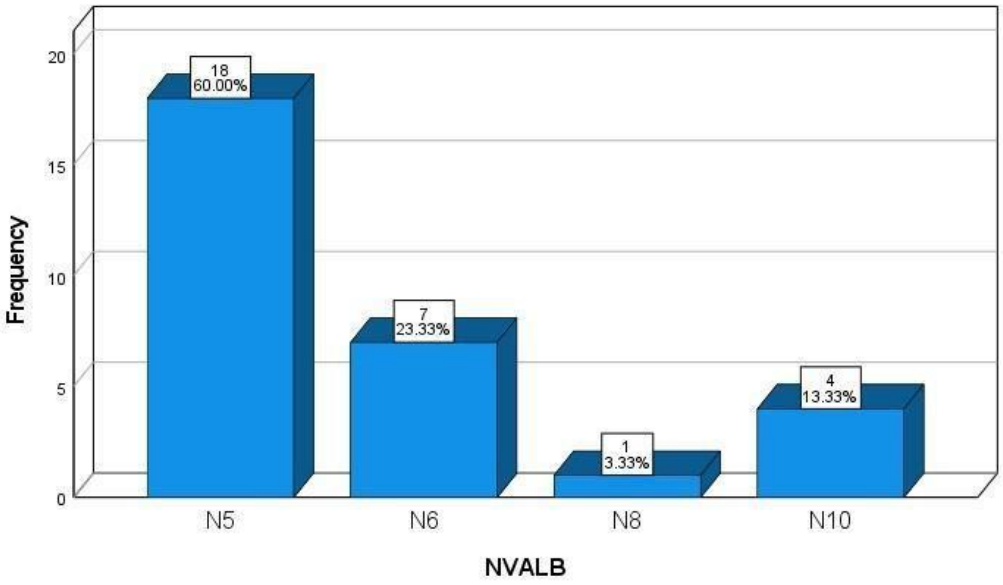



DVALB
Group: Non-Alcoholics
Frequency
12.5
10.0
7.5
5.0
2.5
0.0
4
13.33%
3
10.00%
13
43.33%
6
20.00%
2
6.67%
2
6.67%
6/4
6/5
6/6
6/9
6/12
6/18
DVALB


NVARB
Group: Non-Alcoholics
Frequency
12
10
8
6
4
2
0
12
40.00%
9
30.00%
5
16.67%
4
13.33%
N5
N6
N8
N10
NVARB

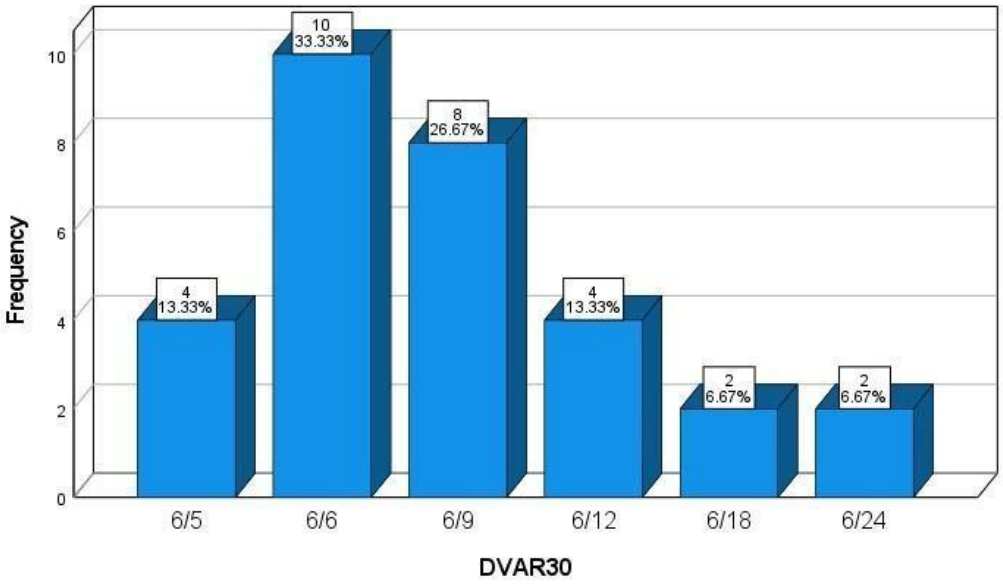
NVALB

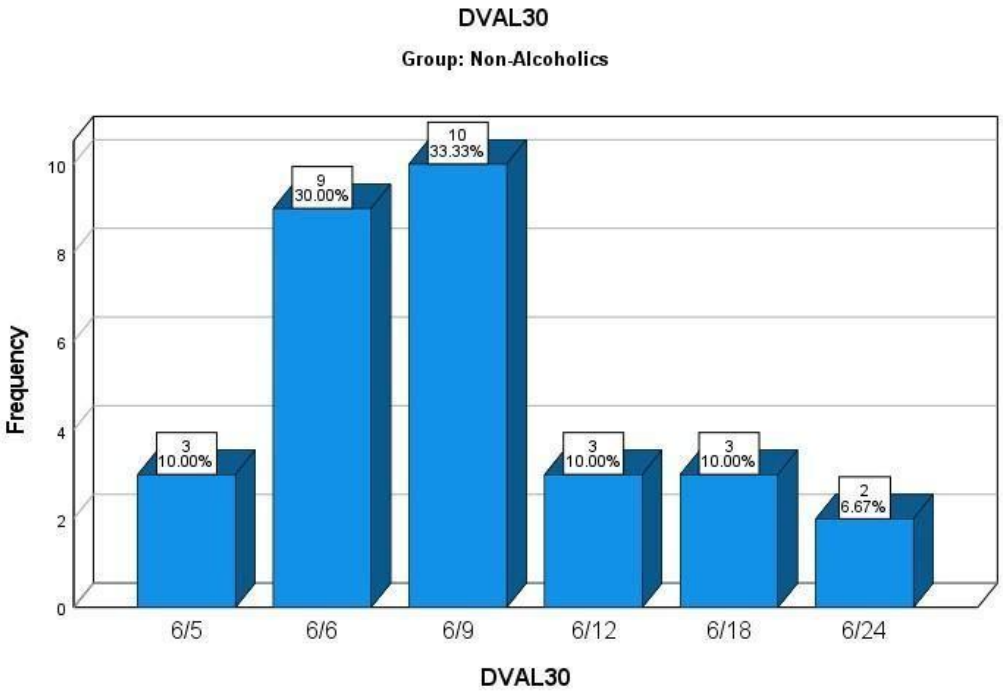
Group: Non-Alcoholics



DVAR30

Group: Non-Alcoholics




DVAL30
Group: Non-Alcoholics
Frequency
10
8
6
4
2
0
3
10.00%
9
30.00%
10
33.33%
3
10.00%
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2
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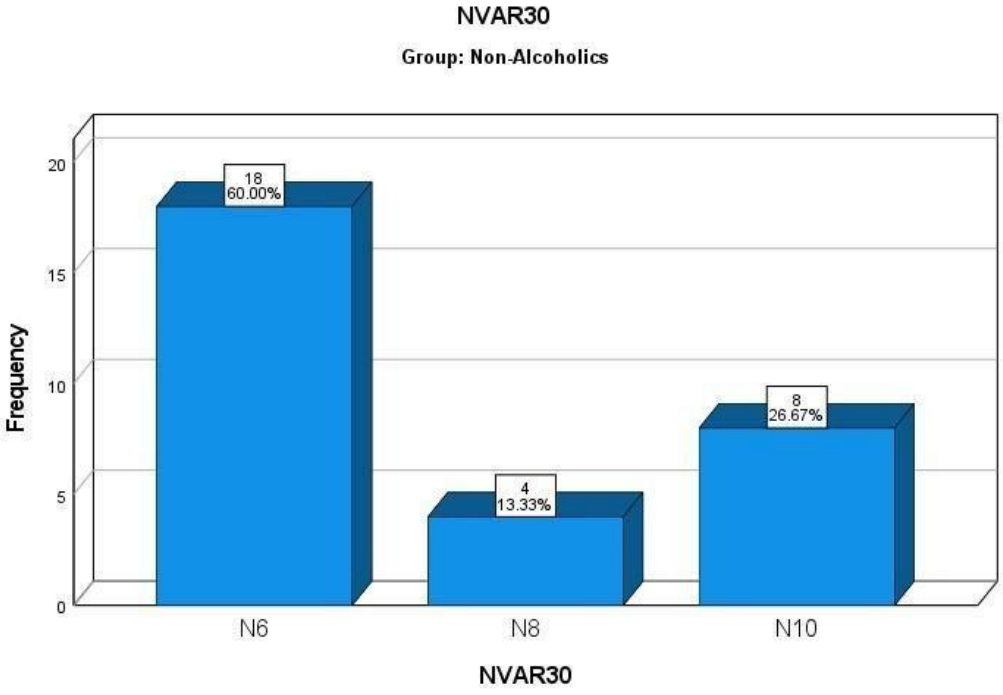
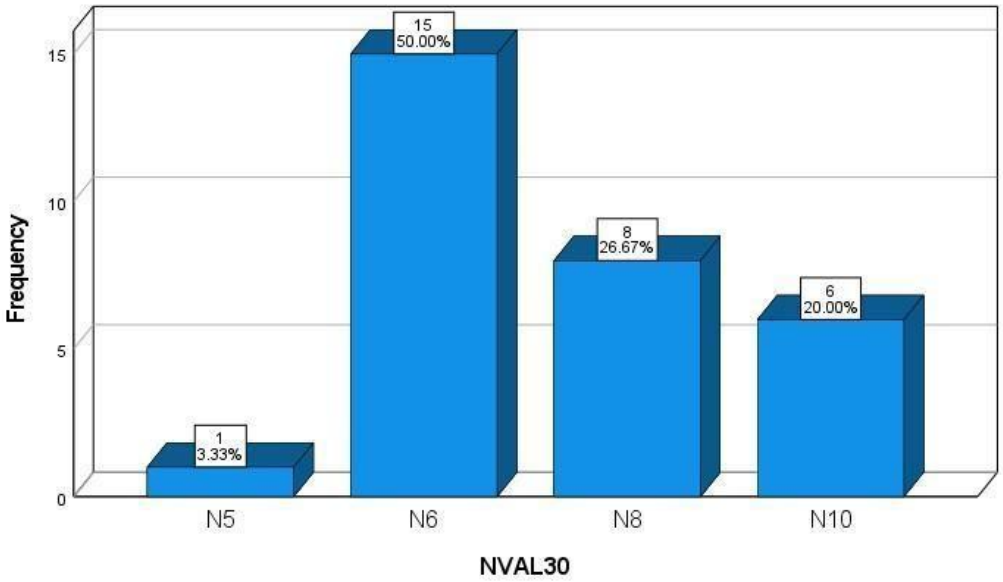
6/5
6/6
6/9
6/12
6/18
6/24
DVAL30


NVAR30
Group: Non-Alcoholics
Frequency
20
15
10
5
0
18
60.00%
4
13.33%
8
26.67%
N6
N8
N10
NVAR30

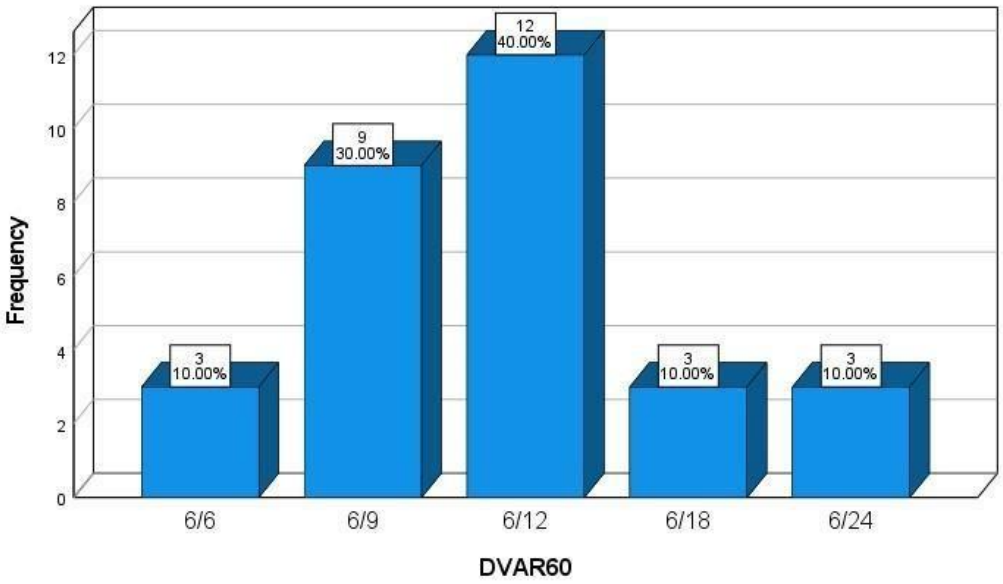
NVAL30

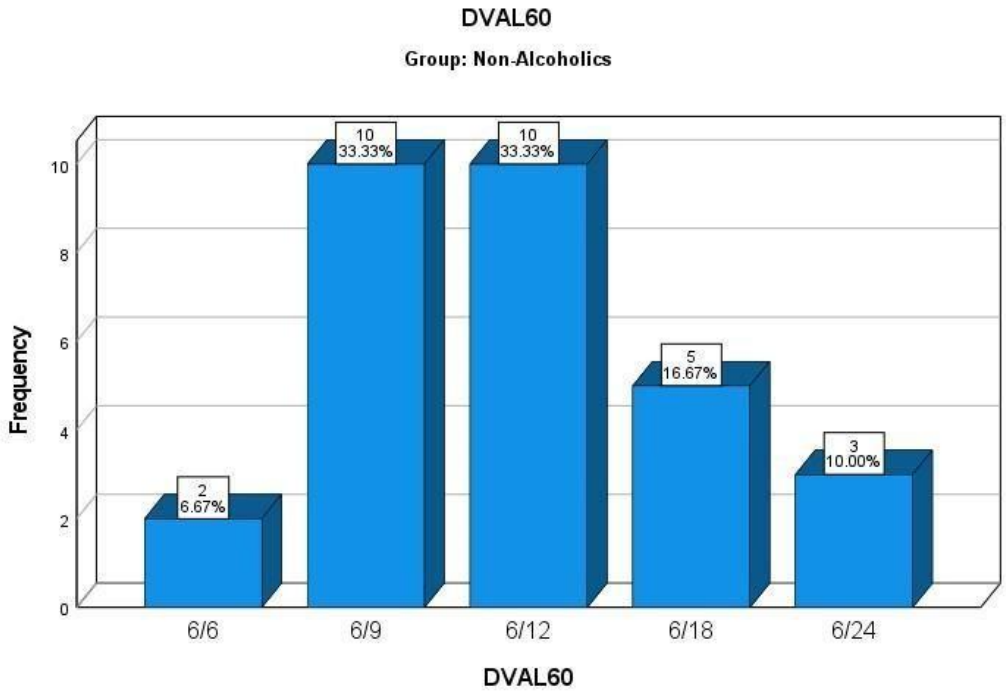
Group: Non-Alcoholics

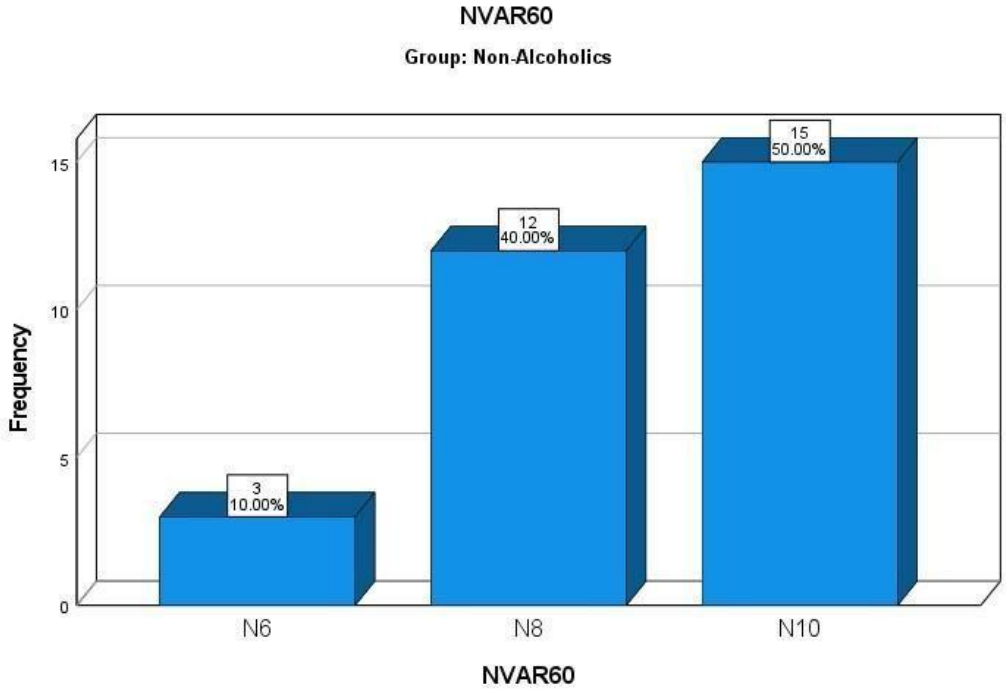


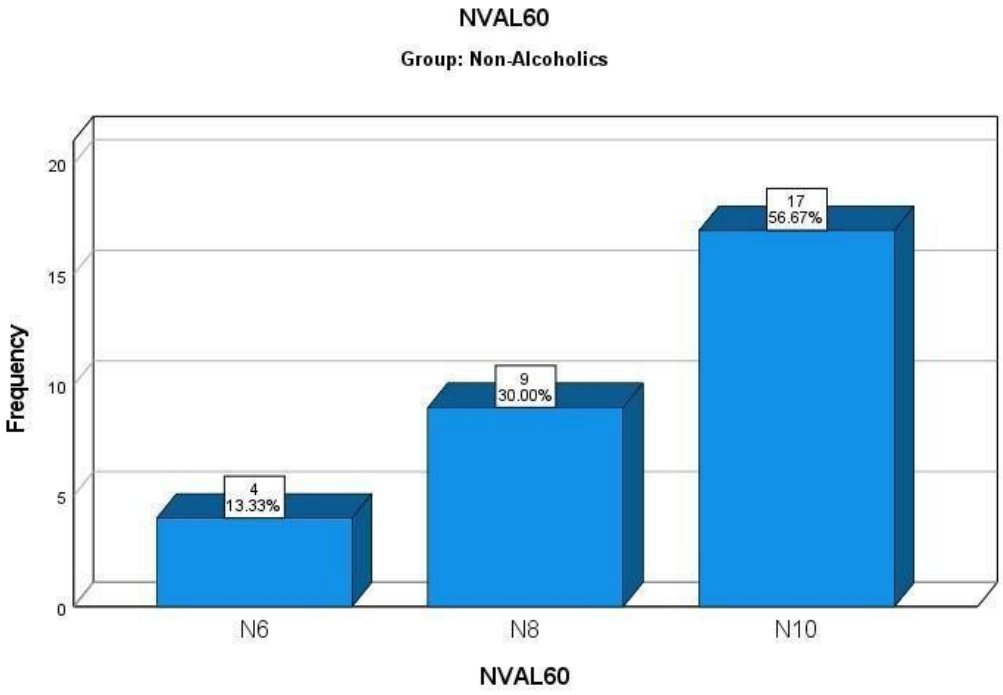
DVAR60

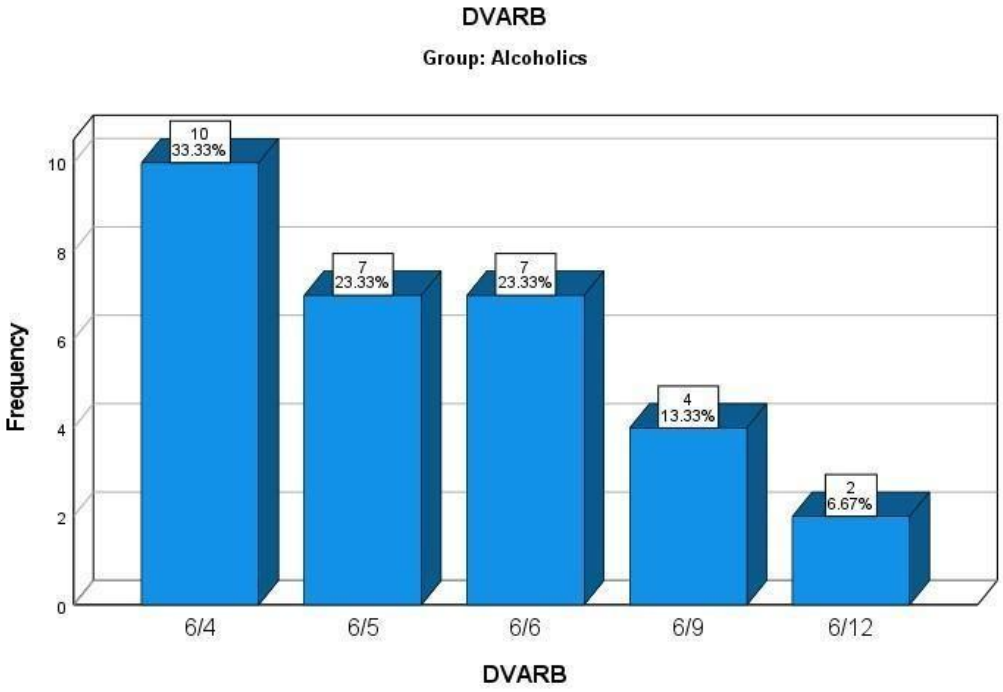
Group: Non-Alcoholics




DVAL60
Group: Non-Alcoholics
Frequency
10
8
6
4
2
0
10
33.33%
10
33.33%
5
16.67%
3
10.00%
2
6.67%
6/6
6/9
6/12
6/18
6/24
DVAL60

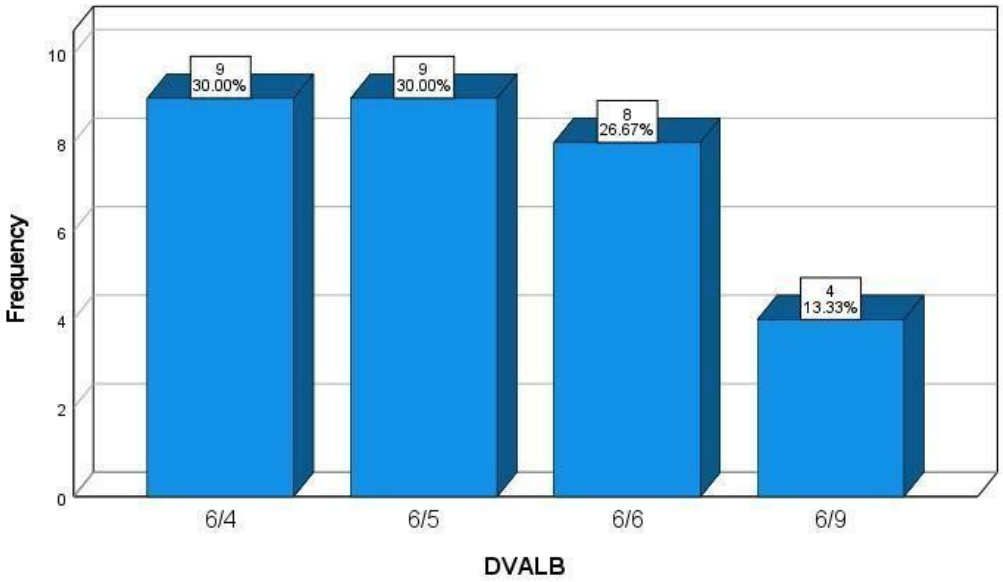

NVAR60
Group: Non-Alcoholics
Frequency
15
10
5
0
15
50.00%
12
40.00%
3
10.00%
N6
N8
N10
NVAR60


NVAL60
Group: Non-Alcoholics
Frequency
20
15
10
5
0
4
13.33%
9
30.00%
17
56.67%
N6
N8
N10
NVAL60


DVARB
Group: Alcoholics
Frequency
10
8
6
4
2
0
10
33.33%
7
23.33%
7
23.33%
4
13.33%
2
6.67%
6/4
6/5
6/6
6/9
6/12
DVARB

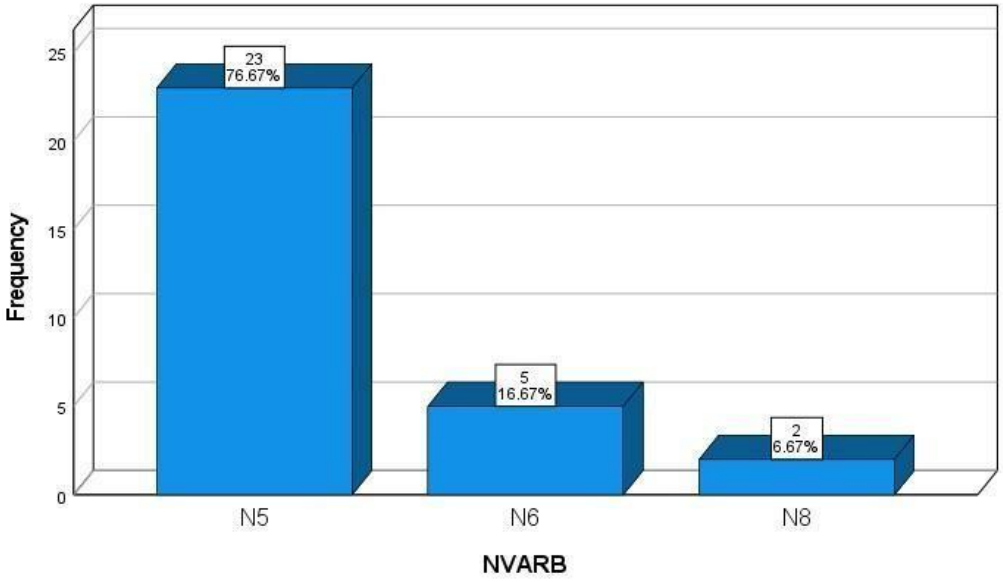
DVALB

Group: Alcoholics



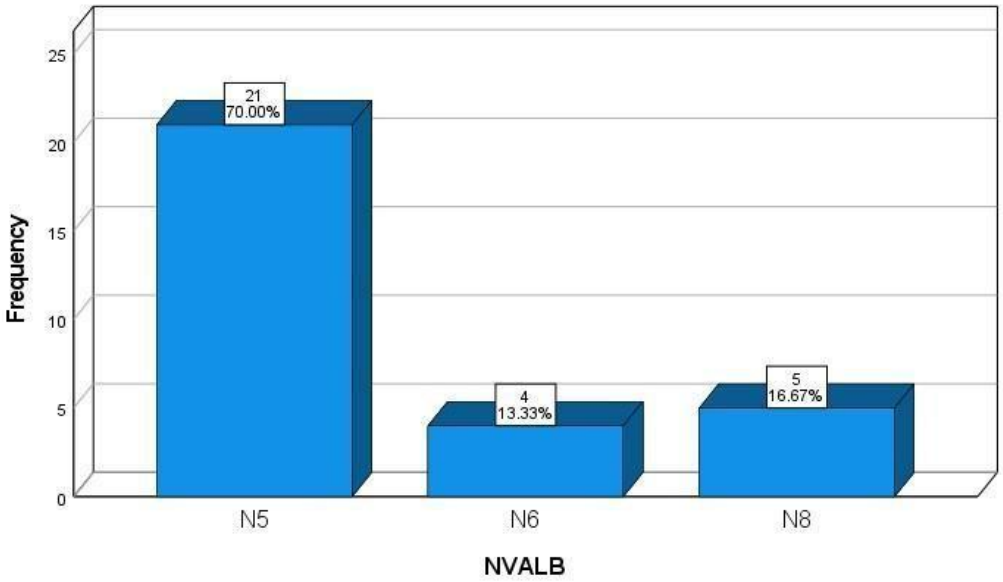
NVARB

Group: Alcoholics



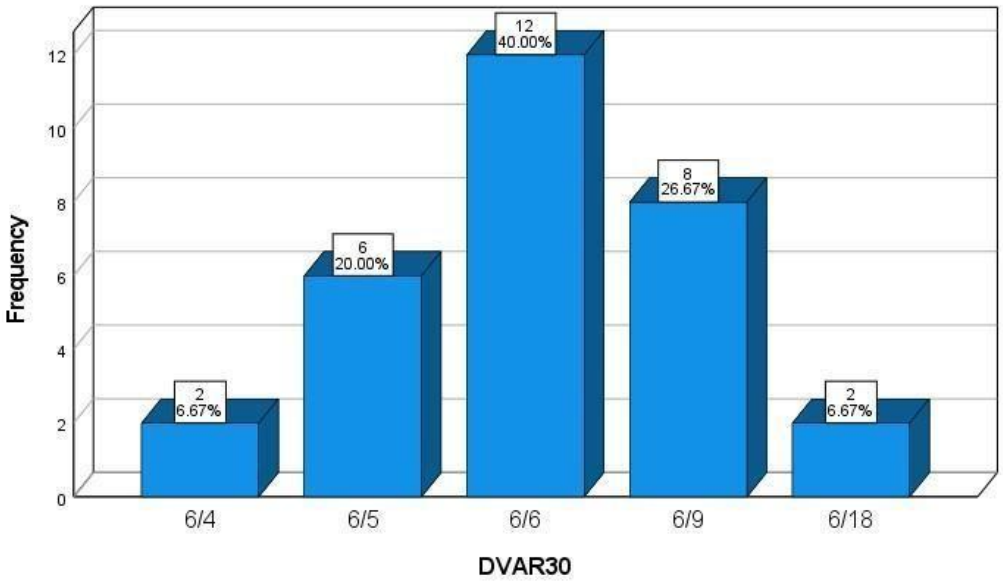
NVALB

Group: Alcoholics



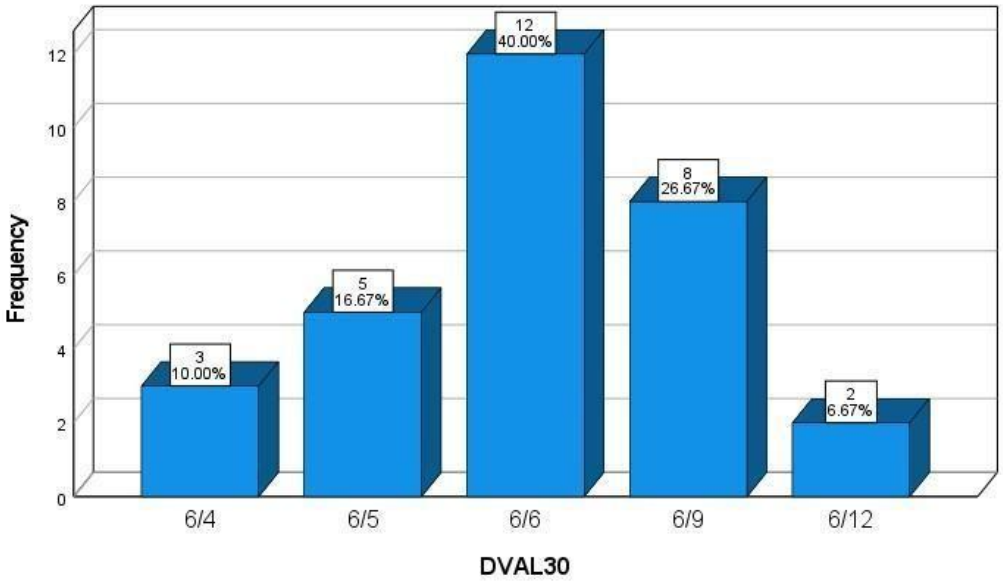
DVAR30

Group: Alcoholics



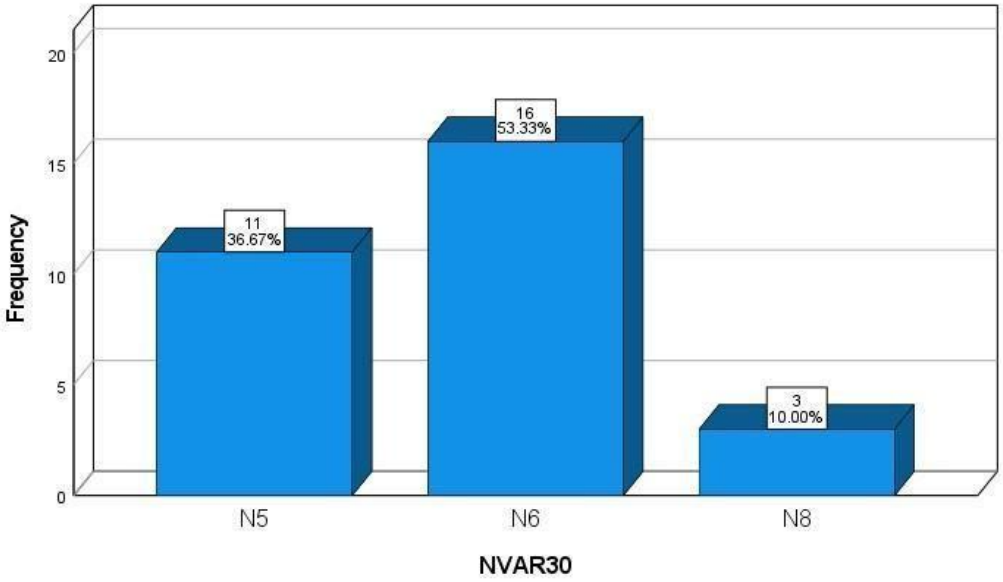
DVAL30

Group: Alcoholics



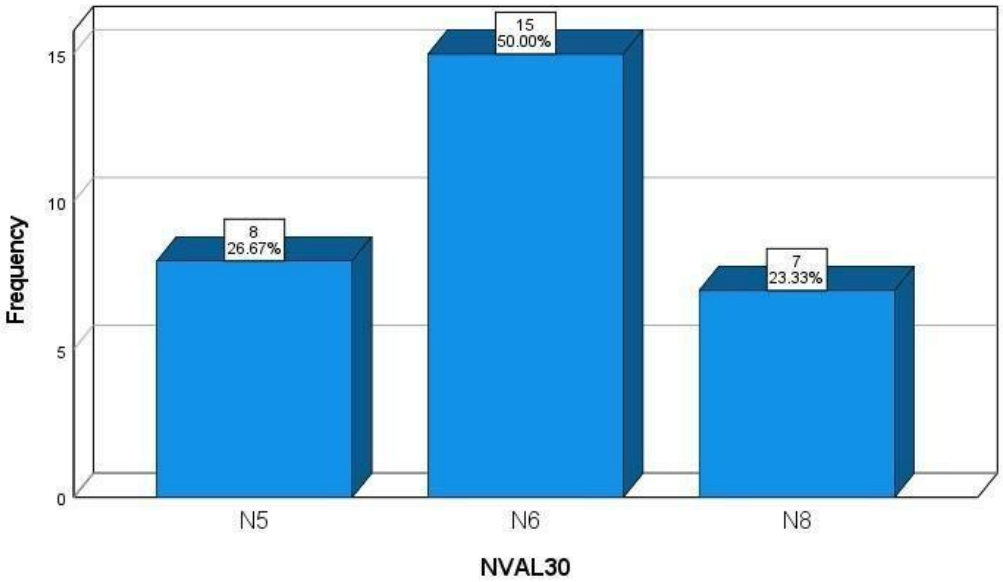
NVAR30

Group: Alcoholics



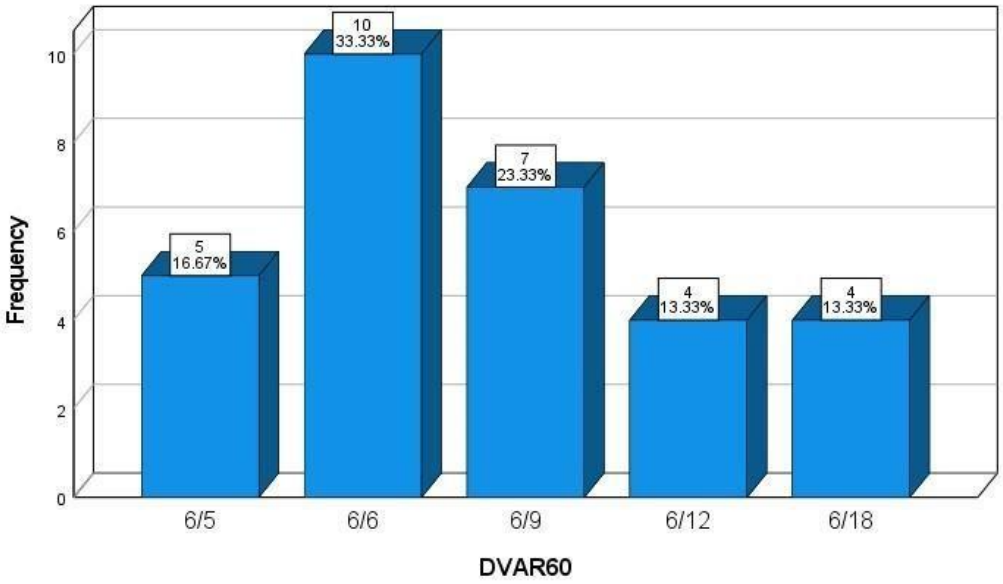
NVAL30

Group: Alcoholics



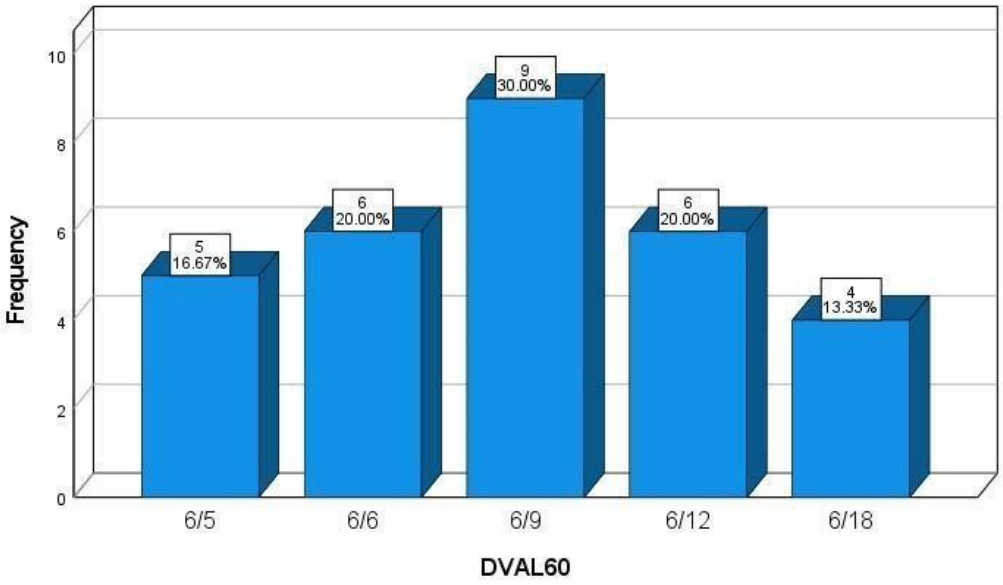
DVAR60

Group: Alcoholics



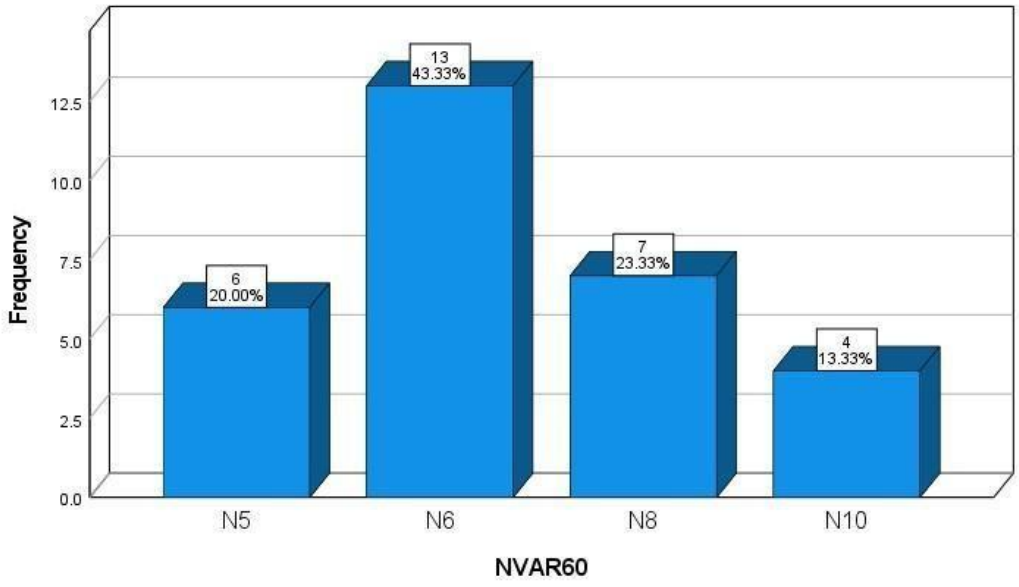
DVAL60

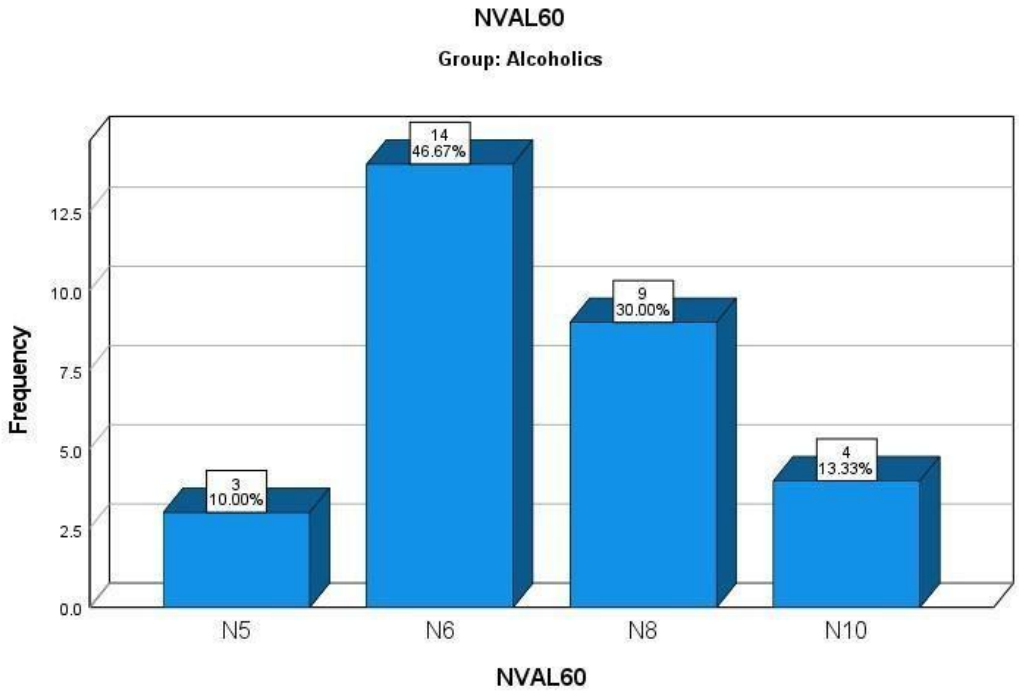
Group: Alcoholics



NVAR60

Group: Alcoholics





Independent Samples Effect Sizes

		Standardizer[a]	Point Estimate	95% Confidence Interval	
				Lower	Upper
DVA RB	Cohen's d	1.49674	0.401	-0.112	0.910
	Hedges' correction	1.51645	0.396	-0.111	0.899
	Glass's delta	1.27261	0.471	-0.053	0.988
DVA LB	Cohen's d	1.18564	0.787	0.258	1.310
	Hedges' correction	1.20125	0.777	0.255	1.293
	Glass's delta	1.04000	0.897	0.335	1.447
NVA RB	Cohen's d	0.86370	0.849	0.317	1.375
	Hedges' correction	0.87507	0.838	0.313	1.357

	Glass's delta	0.59596	1.231	0.627	1.819
NVA	Cohen's d	0.92631	0.252	-0.257	0.759
LB	Hedges' correction	0.93850	0.249	-0.254	0.749
	Glass's delta	0.77608	0.301	-0.214	0.810
CSRB	Cohen's d	0.17023	0.372	-0.140	0.881
	Hedges' correction	0.17247	0.367	-0.138	0.870
	Glass's delta	0.20625	0.307	-0.208	0.817
CSLB	Cohen's d	0.14231	0.328	-0.183	0.836
	Hedges' correction	0.14419	0.324	-0.181	0.825
	Glass's delta	0.17036	0.274	-0.239	0.783
NPCBB	Cohen's d	3.43754	-0.747	-1.268	-0.220
	Hedges' correction	3.48280	-0.737	-1.251	-0.217
	Glass's delta	3.88927	-0.660	-1.188	-0.121
NPCRB	Cohen's d	4.46571	-0.732	-1.252	-0.205
	Hedges' correction	4.52451	-0.722	-1.235	-0.203
	Glass's delta	5.06237	-0.645	-1.173	-0.108
AOARB	Cohen's d	2.79038	0.717	0.191	1.236
	Hedges' correction	2.82712	0.707	0.189	1.220
	Glass's delta	3.23131	0.619	0.084	1.144

AOALB	Cohen's d	2.41606	1.642	1.049	2.224
	Hedges' correction	2.44788	1.620	1.035	2.195
	Glass's delta	2.73777	1.449	0.813	2.068

a. The denominator used in estimating the effect sizes.
Cohen's d uses the pooled standard deviation.
Hedges' correction uses the pooled standard deviation, plus a correction factor.
Glass's delta uses the sample standard deviation of the control group.

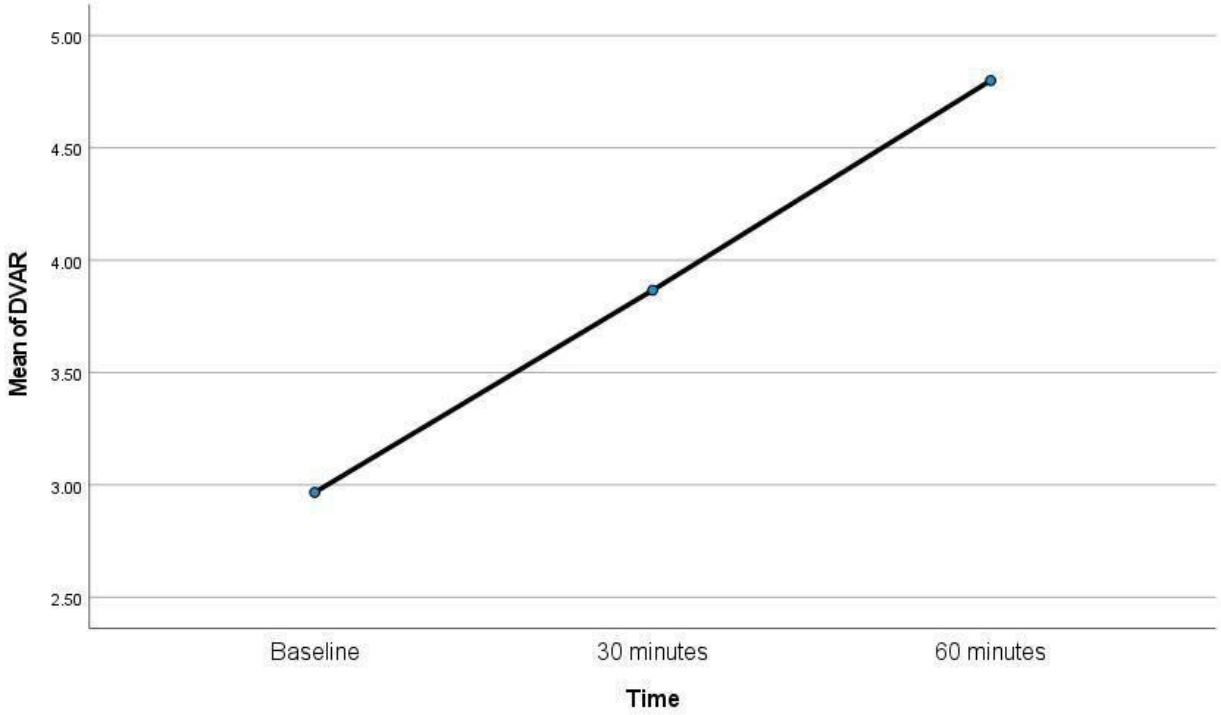
Tests of Homogeneity of Variances

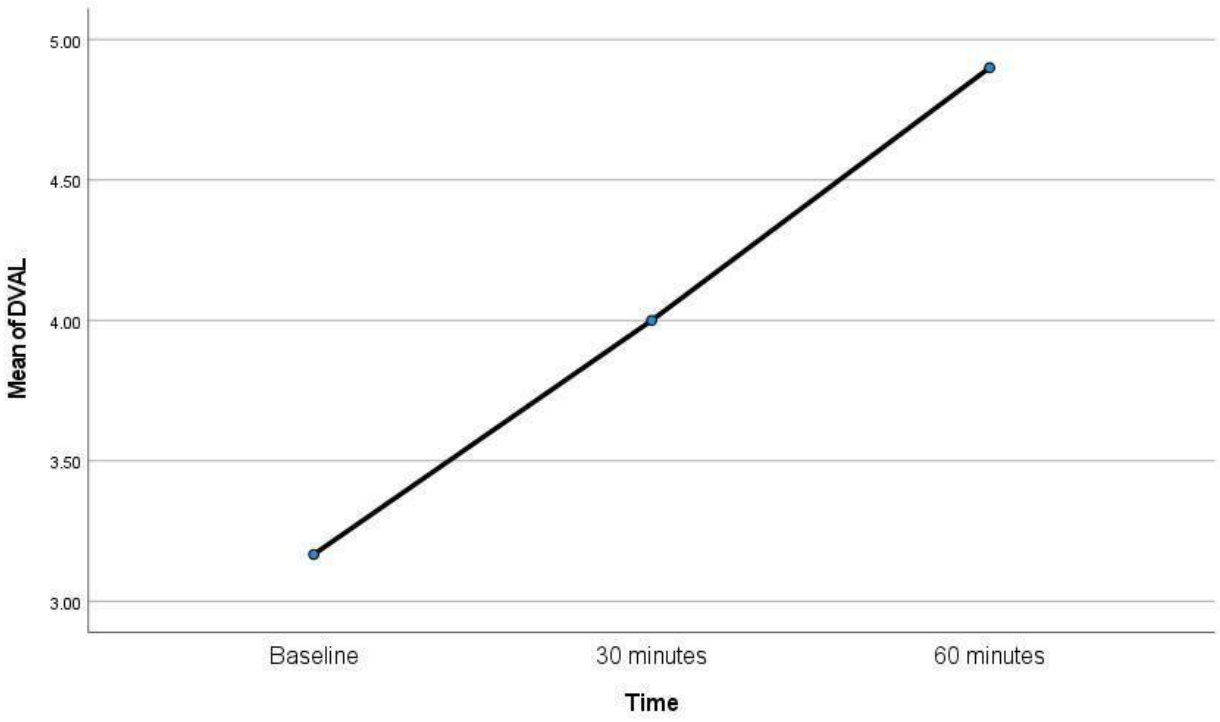
		Levene Statistic	df1	df2	Sig.
DVAR	Based on Mean	2.997	2	87	0.055
	Based on Median	3.196	2	87	0.046
	Based on Median and with adjusted df	3.196	2	83.978	0.046
	Based on trimmed mean	3.020	2	87	0.054
DVAL	Based on Mean	0.248	2	87	0.781
	Based on Median	0.283	2	87	0.754
	Based on Median and with adjusted df	0.283	2	81.917	0.754
	Based on trimmed mean	0.266	2	87	0.767

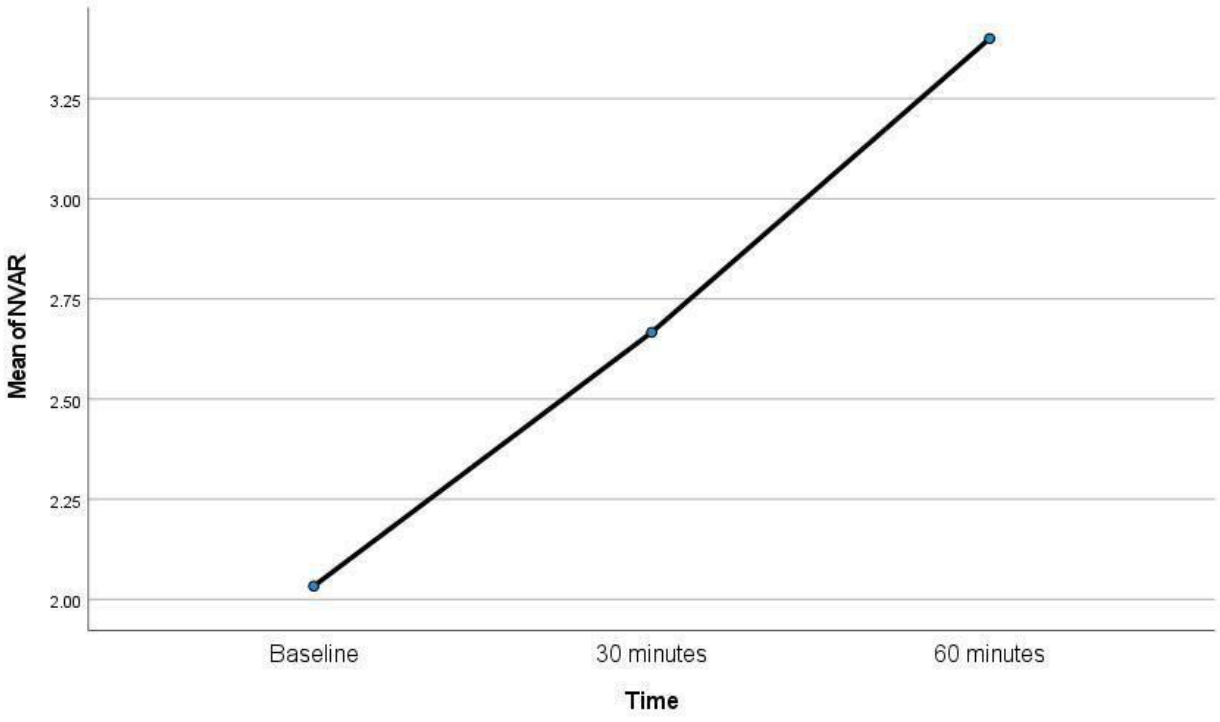
NV AR	Based on Mean	2.586	2	87	0.081
	Based on Median	1.004	2	87	0.370
	Based on Median and with adjusted df	1.004	2	61.058	0.372
	Based on trimmed mean	2.258	2	87	0.111
NV AL	Based on Mean	1.375	2	87	0.258
	Based on Median	0.234	2	87	0.791
	Based on Median and with adjusted df	0.234	2	78.173	0.792
	Based on trimmed mean	1.140	2	87	0.324
CS R	Based on Mean	0.327	2	87	0.722
	Based on Median	0.099	2	87	0.906
	Based on Median and with adjusted df	0.099	2	74.221	0.906
	Based on trimmed mean	0.277	2	87	0.759
CS L	Based on Mean	5.209	2	87	0.007

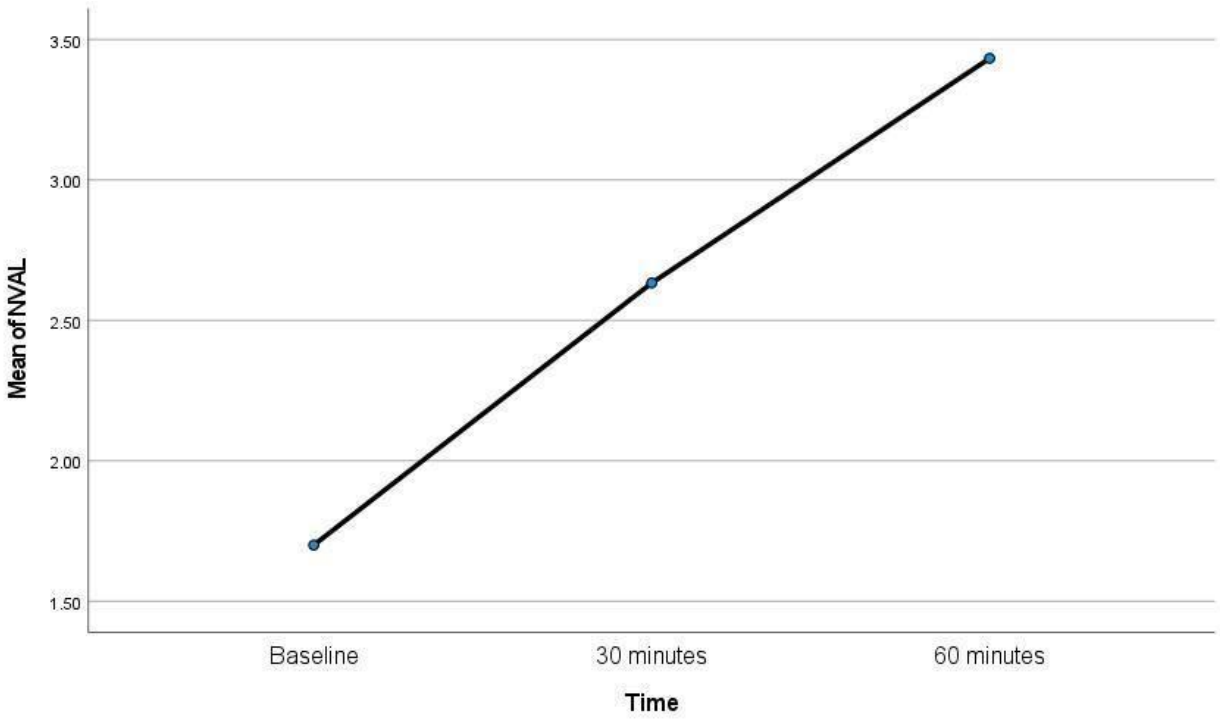
	Based on Median	4.105	2	87	0.020
	Based on Median and with adjusted df	4.105	2	79.018	0.020
	Based on trimmed mean	5.012	2	87	0.009
	Based on Mean	1.633	2	87	0.201
NPCB	Based on Median	1.177	2	87	0.313
	Based on Median and with adjusted df	1.177	2	68.128	0.314
	Based on trimmed mean	1.612	2	87	0.205
NPCR	Based on Mean	0.412	2	87	0.664
	Based on Median	0.141	2	87	0.869
	Based on Median and with adjusted df	0.141	2	76.971	0.869
	Based on trimmed mean	0.415	2	87	0.661
AOAR	Based on Mean	2.546	2	87	0.084
	Based on Median	1.383	2	87	0.256

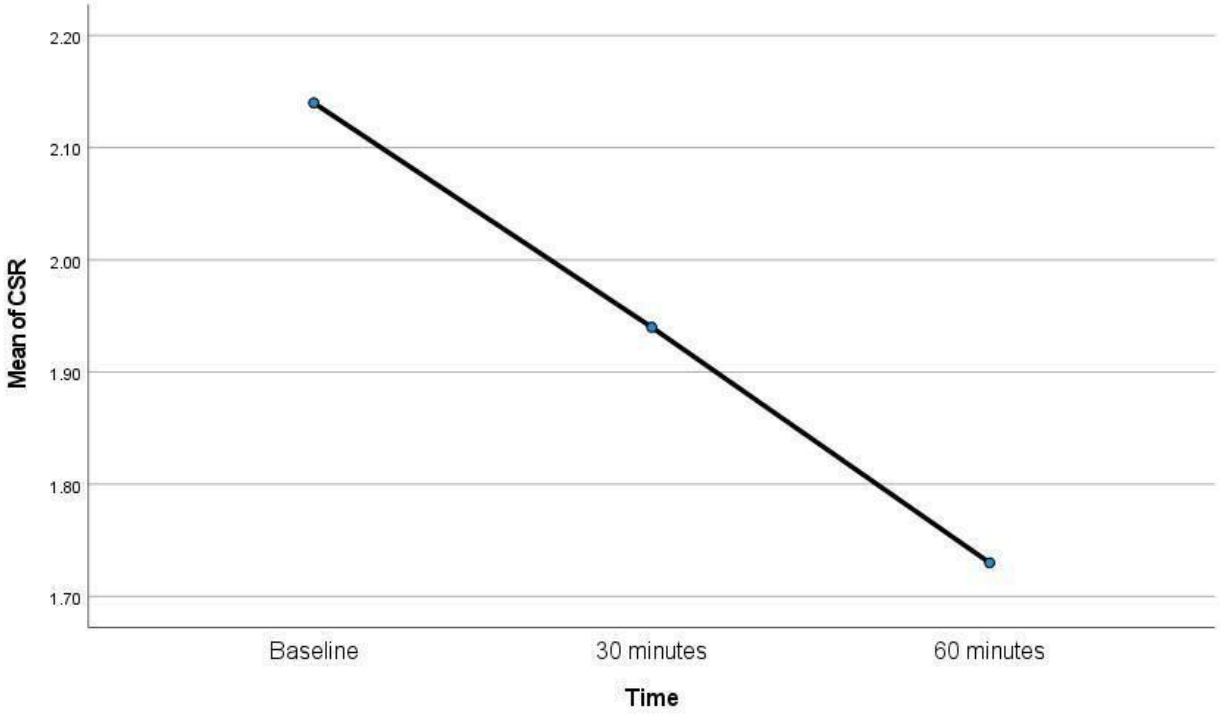
	Based on Median and with adjusted df	1.383	2	71.356	0.258
	Based on trimmed mean	2.487	2	87	0.089
AOAL	Based on Mean	0.959	2	87	0.387
	Based on Median	0.523	2	87	0.595
	Based on Median and with adjusted df	0.523	2	74.881	0.595
	Based on trimmed mean	0.936	2	87	0.396

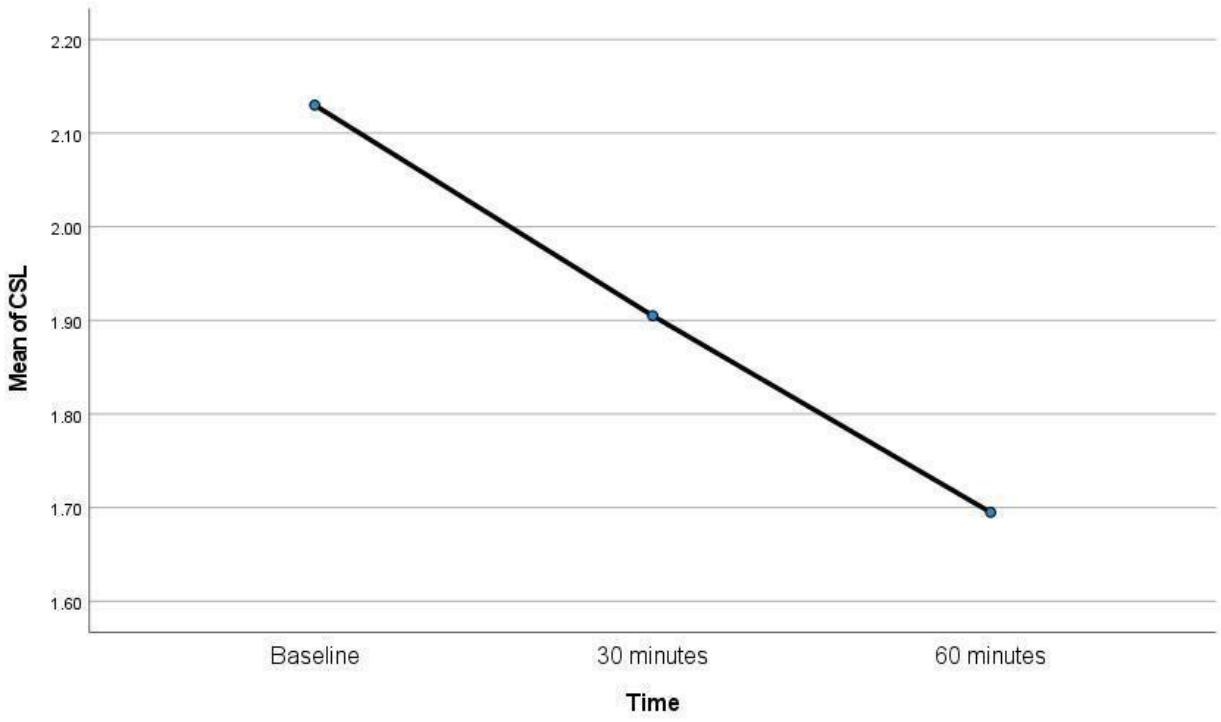


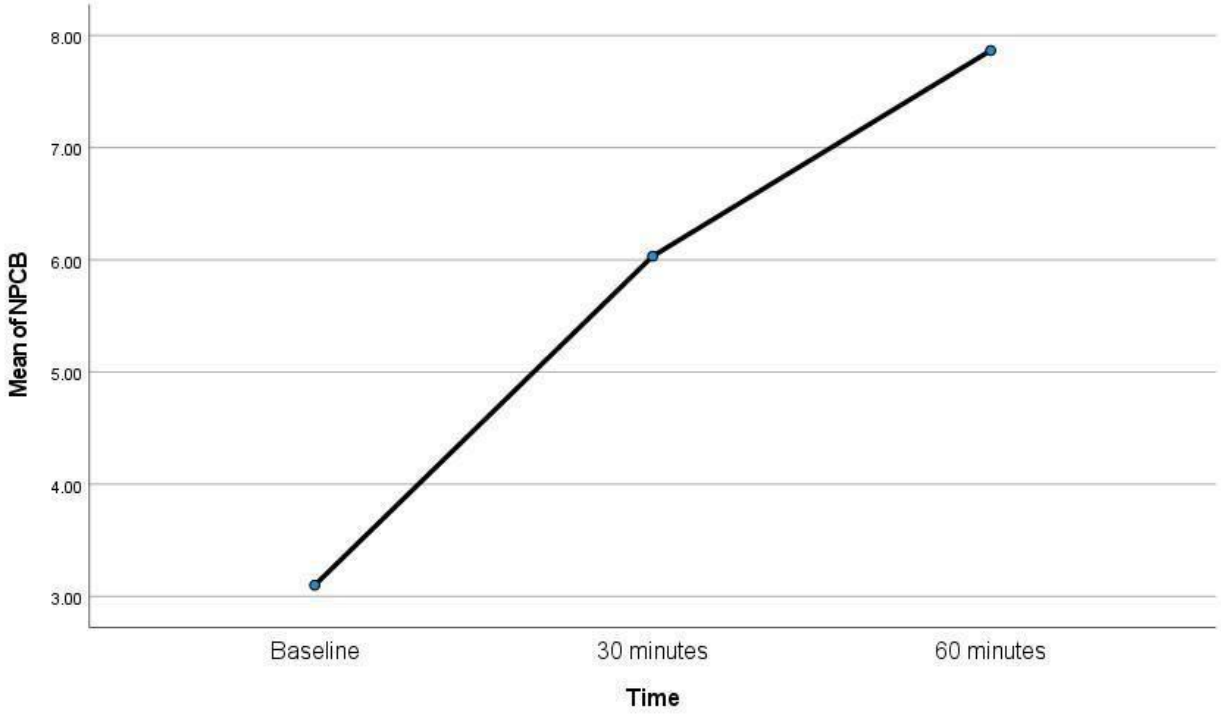

5.00
4.50
4.00
3.50
3.00
Mean of DVAL
Baseline
30 minutes
60 minutes
Time

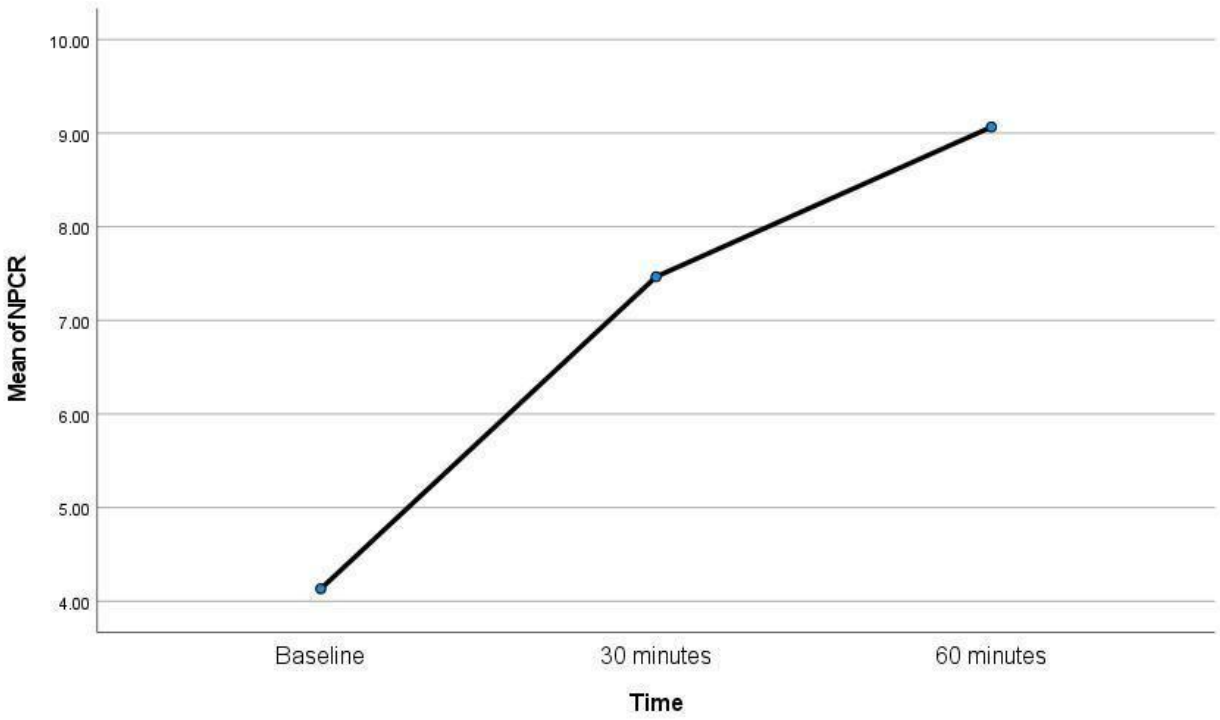

3.25
3.00
2.75
2.50
2.25
2.00
Mean of NVAR
Baseline
30 minutes
60 minutes
Time

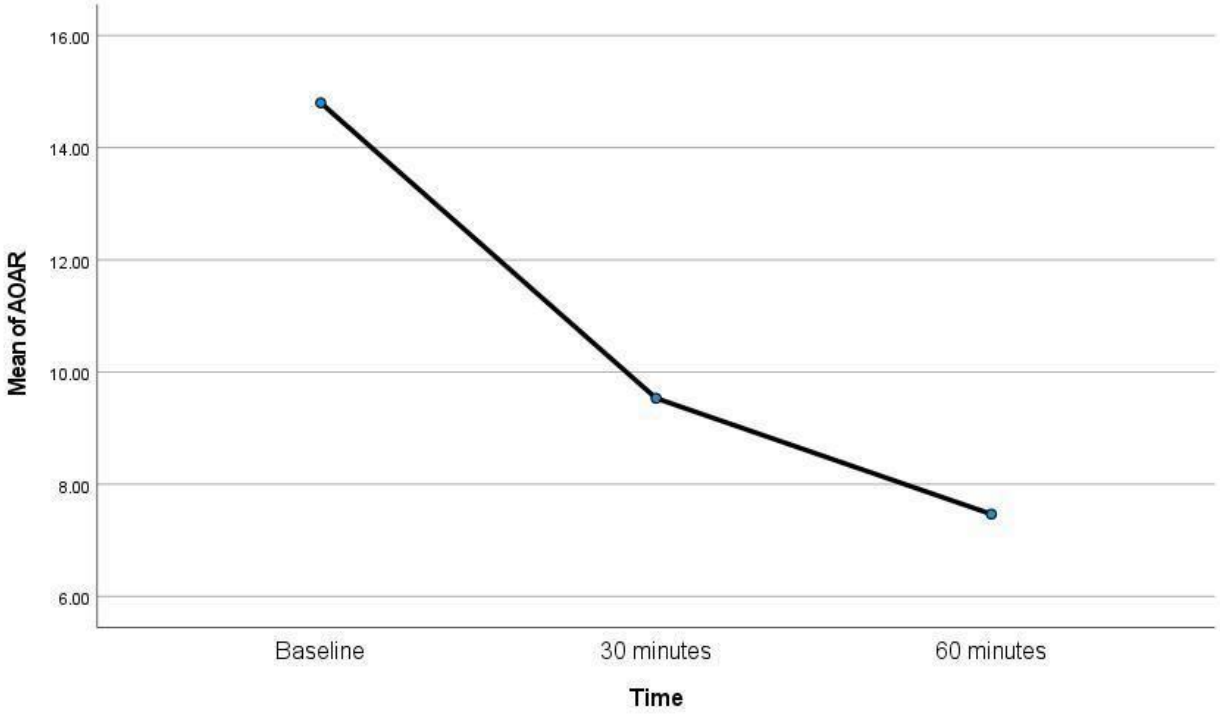

3.50
3.00
2.50
2.00
1.50
Mean of NVAL
Baseline
30 minutes
60 minutes
Time


2.20
2.10
2.00
1.90
1.80
1.70
Mean of CSR
Baseline
30 minutes
60 minutes
Time


2.20
2.10
2.00
1.90
1.80
1.70
1.60
Mean of CSL
Baseline
30 minutes
60 minutes
Time


8.00
7.00
6.00
5.00
4.00
3.00
Mean of NPCB
Baseline
30 minutes
60 minutes
Time


10.00
9.00
8.00
7.00
6.00
5.00
4.00
Mean of NPCR
Baseline
30 minutes
60 minutes
Time


16.00
14.00
12.00
10.00
8.00
6.00
Mean of AOAR
Baseline
30 minutes
60 minutes
Time

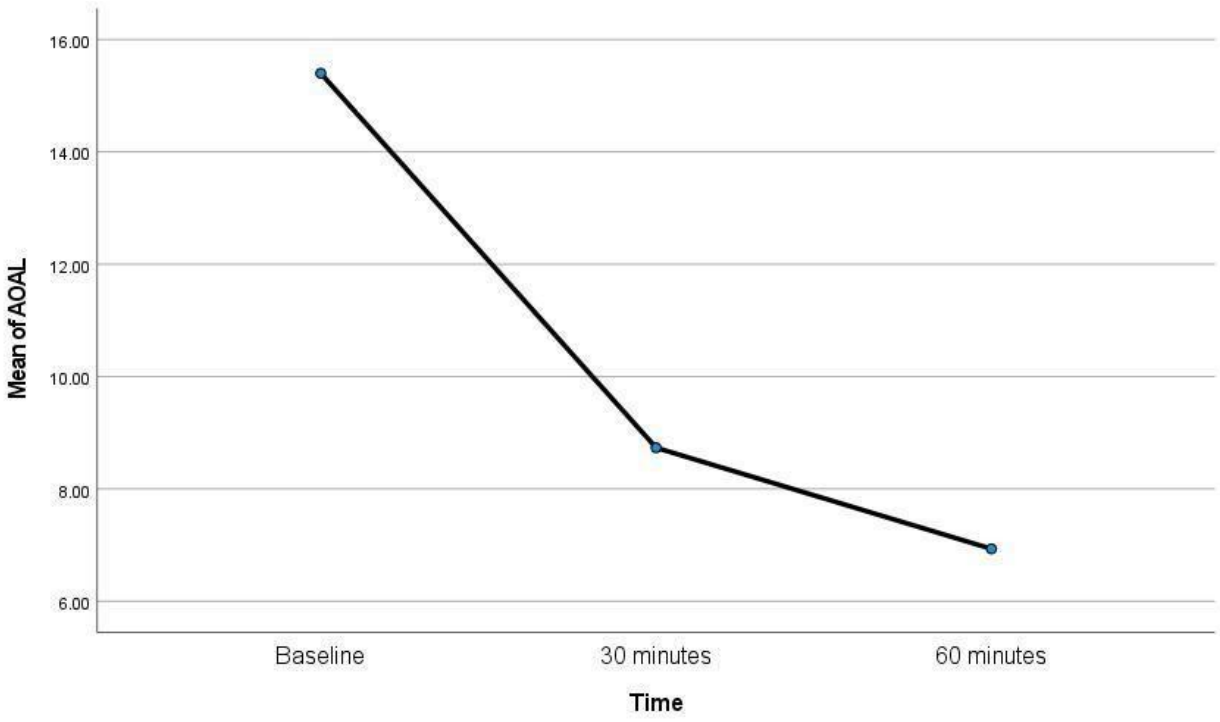


Table 4.7: Between-Group Comparison of Visual Parameters 30 Minutes PostConsumption

Group Statistics	Group	N	Mean	Std. Deviation	Std. Error Mean
DVAR30	Non-Alcoholics	30	3.8667	1.38298	0.25250
	Alcoholics	30	3.1333	1.16658	0.21299
DVAL30	Non-Alcoholics	30	4.0000	1.36458	0.24914
	Alcoholics	30	3.0333	1.06620	0.19466
NVAR30	Non-Alcoholics	30	2.6667	0.88409	0.16141
	Alcoholics	30	1.7333	0.63968	0.11679
NVAL30	Non-Alcoholics	30	2.6333	0.85029	0.15524
	Alcoholics	30	1.9667	0.71840	0.13116
CSR30	Non-Alcoholics	30	1.9400	0.16682	0.03046
	Alcoholics	30	1.9500	0.20342	0.03714

CSL30	Non-Alcoholics	30	1.9050	0.15830	0.02890
	Alcoholics	30	1.9550	0.19358	0.03534
NPCB30	Non-Alcoholics	30	6.0333	3.44897	0.62969
	Alcoholics	30	7.6667	3.54608	0.64742
NPCR30	Non-Alcoholics	30	7.4667	4.03206	0.73615
	Alcoholics	30	9.3000	3.96667	0.72421
AOAR30	Non-Alcoholics	30	9.5333	2.81294	0.51357
	Alcoholics	30	10.5333	2.95639	0.53976
AOAL30	Non-Alcoholics	30	8.7333	2.70291	0.49348
	Alcoholics	30	9.9167	3.26445	0.59600

At 30 minutes, the mean values were: DVAR 3.87 (Non-Alcoholic) vs 3.13 (Alcoholic); NVAR 2.67 vs 1.73; NVAL 2.63 vs 1.97; CSR 1.94 vs 1.95; CSL 1.91 vs 1.96; NPCB 6.03 vs 7.67; NPCR 7.47 vs 9.30; AOAR 9.53 vs 10.53; AOAL 8.73 vs 9.92. The mean DVAL was 4.00 for the Non-Alcoholic group and 3.03 for the Alcoholic group.

Table 4.8: Between-Group Comparison of Visual Parameters 60 Minutes PostConsumption

Group Statistics Group		**N**	**Mean**	**Std deviation**	**Std error mean**
DVAR60	Non-Alcoholics	30	4.8000	1.09545	0.20000
	Alcoholics	30	3.7333	1.28475	0.23456
DVAL60	Non-Alcoholics	30	4.9000	1.09387	0.19971
	Alcoholics	30	3.9333	1.28475	0.23456
NVAR60	Non-Alcoholics	30	3.4000	0.67466	0.12318
	Alcoholics	30	2.3000	0.95231	0.17387
NVAL60	Non-Alcoholics	30	3.4333	0.72793	0.13290
	Alcoholics	30	2.4667	0.86037	0.15708
CSR60	Non-Alcoholics	30	1.7300	0.14598	0.02665
	Alcoholics	30	1.8233	0.21645	0.03952

CSL60	Non-Alcoholics	30	1.6950	0.18539	0.03385
	Alcoholics	30	1.8233	0.21284	0.03886
NPCB60	Non-Alcoholics	30	7.8667	4.61432	0.84246
	Alcoholics	30	8.5000	4.38453	0.80050
NPCR60	Non-Alcoholics	30	9.0667	4.98918	0.91090
	Alcoholics	30	9.9333	4.94057	0.90202
AOAR60	Non-Alcoholics	30	7.4667	3.30864	0.60407
	Alcoholics	30	9.8333	3.32268	0.60664
AOAL60	Non-Alcoholics	30	6.9333	2.50425	0.45721
	Alcoholics	30	9.4333	3.01357	0.55020

At 60 minutes, the mean values were: DVAR 4.80 (Non-Alcoholic) vs 3.73 (Alcoholic); DVAL 4.90 vs 3.93; NVAR 3.40 vs 2.30; NVAL 3.43 vs 2.47; CSR 1.73 vs 1.82; CSL 1.70 vs 1.82; NPCB 7.87 vs 8.50; NPCR 9.07 vs 9.93; AOAR 7.47 vs 9.83; AOAL 6.93 vs 9.43. The mean AOAL was 6.93 for the Non-Alcoholic group and 9.43 for the Alcoholic group.

						Control Group (Baseline values)				
S/No	Age	Gender	VA (R logMAR)	VA (L logMAR)	VA (R logMAR)	VA (L logMAR)	CS (1m R)	CS (1m L)	NPC	Accom R	Accom L
1	21	Male	-0.08	-0.08	0.10	0.10	2.10	2.10	4/6cm	16D	16D
2	30	Male	0.00	0.00	0.20	0.10	2.10	2.25	TTN	20D	18D
3	22	Female	-0.18	-0.08	0.10	0.10	2.25	2.25	TTN	18D	16D
4	27	Male	-0.18	-0.18	0.30	0.30	2.25	2.10	6/8cm	12D	14D
5	21	Male	0.00	0.18	0.10	0.10	2.10	2.10	6/8cm	16D	14D
6	30	Female	0.18	0.18	0.20	0.10	1.95	1.95	5/7cm	14D	14D
7	22	Male	0.18	0.00	0.20	0.20	2.10	1.95	8/9cm	12D	14D
8	27	Male	-0.018	-0.18	0.10	0.10	1.95	2.10	4/6cm	14D	18D
9	26	Male	-0.08	0.00	0.10	0.10	1.95	1.95	6/8cm	16D	12D
10	23	Female	-0.08	-0.08	0.10	0.10	2.25	2.25	TTN	18D	18D
11	25	Male	-0.08	0.00	0.10	0.20	2.10	2.10	TTN	18D	18D
12	21	Male	0.00	0.00	0.30	0.10	2.25	2.10	4/6cm	12D	14D
13	22	Female	0.18	0.18	0.20	0.10	1.95	2.10	6/7cm	16D	18D
14	27	Male	0.48	0.48	0.40	0.40	2.25	2.25	TTN	16D	16D
15	25	Male	0.48	0.18	0.40	0.40	2.10	2.10	6/8cm	12D	14D
16	21	Female	0.30	0.30	0.30	0.20	1.95	1.95	7/9cm	14D	14D
17	27	Male	-0.18	0.00	0.10	0.10	2.25	2.25	TTN	16D	16D
18	22	Male	-0.18	-0.18	0.10	0.20	2.25	2.25	TTN	14D	12D
19	25	Female	-0.08	0.00	0.20	0.10	2.25	2,25	4/6cm	14D	18D
20	21	Male	0.00	0.00	0.10	0.10	2.25	2.10	TTN	12D	14D
21	22	Male	-0.18	-0.18	0.10	0.10	1.95	2.10	6/8cm	12D	12D
22	26	Female	-0.18	0.00	0.10	0.10	1.95	2.10	6/7cm	14D	14D
23	21	Male	-0.08	0.00	0.10	0.10	1.95	2.10	6/8cm	12D	14D
24	27	Male	-0.08	0.00	0.10	0.10	1.95	2.10	7/9cm	12D	12D
25	22	Male	0.00	0.00	0.20	0.10	2.10	2.10	8/9cm	16D	16D
26	23	Female	0.00	0.00	0.20	0.10	2.10	2.10	6/8cm	12D	12D
27	27	Male	0.18	0.18	0.30	0.10	1.95	1.95	8/9cm	14D	14D
28	24	Male	0.30	0.18	0.30	0.20	1.95	1.95	6/8cm	18D	16D
29	23	Female	0.48	0.30	0.40	0.40	1.95	2.10	TTN	18D	18D
30	24	Male	0.48	0.48	0.40	0.40	1.95	1.95	TTN	12D	12D

Control Group:
Mean age 24.1± 2.8
Gender distribution: Males 21 (70%) and Females 9 (30%) Alcohol Group:

Mean age 24.1± 2.8 Gender Distribution: Male 21 (70%) and Female 9 (30%)

						Alcohol Group (Baseline Values)				
S/No	Age	Gender	VA (R logMAR))	VA (L logMAR))	VA (R logMAR))	VA (L logMAR))	CS (1m R)	CS (1m L)	NPC	Accom R	Accom L
1	26	Male	-0.18	-0.18	0.10	0.10	2.10	2.20	4/6cm	16D	16D
2	23	Female	-0.18	-0.18	0.10	0.10	2.25	2.25	6/7cm	11D	10D
3	21	Male	-0.18	-0.18	0.10	0.10	2.25	2.25	TTN	16D	12D
4	25	Male	-0.18	-0.18	0.10	0.10	2.25	2.25	TTN	16D	14D
5	21	Male	-0.18	-0.18	0.10	0.10	2.20	2.20	11/12cm	16D	14D
6	23	Female	-0.18	-0.18	0.10	0.10	2.20	2.25	6/7cm	6D	7D
7	22	Male	-0.18	-0.08	0.20	0.30	1.80	1.80	6/8cm	14D	11D
8	23	Male	-0.18	-0.18	0.10	0.10	2.25	2.10	7/9cm	17D	17D
9	25	Female	-0.08	-0.08	0.10	0.10	2.20	2.20	10/11cm	16D	14D
10	22	Male	0.00	-0.08	0.10	0.10	2.20	2.20	TTN	16D	12D
11	27	Male	-0.08	-0.08	0.10	0.30	2.20	2.10	6/8cm	12D	10D
12	30	Male	0.00	-0.08	0.10	0.20	2.10	2.10	6/9cm	12D	12D
13	27	Female	0.18	0.00	0.10	0.10	2.10	2.10	10/12cm	14D	10D
14	22	Male	-0.18	-0.18	0.10	0.10	2.25	2.25	TTN	16D	14D
15	27	Male	0.18	0.18	0.30	0.30	1.80	1.80	TTN	6D	6D
16	27	Female	0.00	0.00	0.20	0.10	1.95	2.10	10/12cm	12D	8D
17	21	Male	-0.08	0.00	0.10	0.10	2.20	2.10	12/18cm	16D	14D
18	27	Male	0.30	0.18	0.20	0.20	1.50	1.65	4/6cm	10D	10D
19	27	Female	0.00	0.00	0.10	0.10	2.10	2.10	6/8cm	12D	12D
20	30	Female	-0.08	-0.08	0.10	0.10	2.10	2.10	6/8cm	10D	12D
21	21	Male	-0.18	-0.18	0.10	0.10	2.10	2.10	6/8cm	12D	12D
22	22	Male	-0.08	-0.08	0.10	0.10	2.10	2.10	6/8cm	14D	14D
23	22	Male	-0.08	-0.08	0.10	0.10	2.10	2.25	0.18cm	14D	12D
24	21	Male	-0.08	-0.08	0.10	0.10	1.95	1.95	6/9cm	10D	12D
25	22	Female	0.00	0.00	0.10	0.10	1.95	1.95	7/9cm	11D	11D
26	25	Male	0.00	0.00	0.10	0.10	2.10	2.10	6/7cm	16D	16D
27	27	Male	0.00	0.00	0.10	0.20	1.95	1.95	TTN	12D	12D
28	24	Female	0.18	0.00	0.20	0.20	2.25	2.25	7/9cm	12D	12D
29	21	Male	0.18	0.18	0.20	0.30	1.95	1.95	TTN	14D	14D
30	22	Male	0.30	0.18	0.30	0.30	1.95	1.95	TTN	14D	14D

Control Group (30mins post consump2on)									
S/No	VA (R logMAR)	VA (L logMAR)	VA (R logMAR)	VA (L logMAR)	CS (1m R)	CS (1m L)	NPC	Accom R	Accom L
1	-0.08	0.18	0.20	0.20	1.80	1.80	7/6cm	12D	12D
2	0.00	0.18	0.20	0.20	1.95	1.95	5/6cm	14D	12D
3	-0.08	-0.08	0.20	0.10	2.10	2.10	TTN	14D	12D
4	0.18	0.18	0.30	0.30	2.10	2.10	4/6cm	6D	6D
5	0.00	0.00	0.20	0.20	1.95	1.95	8/10cm	10D	10D
6	0.30	0.48	0.30	0.30	1.95	1.95	7/8cm	6D	6D
7	0.30	0.30	0.20	0.20	1,65	1.80	10/12cm	10D	10D
8	0.00	0.00	0.20	0.20	1.80	1.80	8/10cm	8D	6D
9	0.00	0.00	0.20	0.20	1.80	1.80	8/10cm	10D	8D
10	0.18	0.18	0.20	0.20	2.10	2.10	6/8cm	12D	12D
11	0.18	-0.08	0.30	0.30	1.95	1.95	4/6cm	14D	10D
12	0.00	0.18	0.40	0.30	2.10	1.95	6/8cm	6D	6D
13	0.18	0.18	0.20	0.20	1.50	1.50	9/10cm	6D	6D
14	0.60	0.60	0.40	0.40	1.95	1.95	TTN	10D	10D
15	0.48	0.30	0.40	0.40	2.10	2.10	8/10cm	6D	4D
16	0.30	0.48	0.40	0.40	1.95	1.80	12/14cm	8D	8D
17	0.00	0.00	0.20	0.30	1.80	1.80	8/10cm	12D	14D
18	-0.08	0.00	0.20	0.20	2.10	2.10	4/6cm	12D	8D
19	0.18	0.18	0.20	0.20	2.10	1.80	8/10cm	10D	10D
20	0.00	0.00	0.20	0.20	1.80	1.95	TTN	8D	8D
21	-0.08	-0.08	0.20	0.20	2.10	2.10	7/8cm	10D	10D
22	0.00	0.00	0.20	0.20	2.10	2.10	7/6cm	10D	10D
23	0.00	0.00	0.20	0.20	2.25	2.10	7/6cm	12D	12D
24	0.00	0.00	0.20	0.20	2.20	2.20	8/10cm	12D	12D
25	0.18	0.18	0.20	0.30	1.65	1.65	7/8cm	8D	10D
26	0.18	0.18	0.30	0.30	1.95	1.95	7/6cm	12D	12D
27	0.18	0.18	0.40	0.30	1.95	1.95	8/10cm	14D	14D
28	0.30	0.30	0.40	0.40	1.80	1.80	TTN	10D	8D
29	0.48	0.48	0.40	0.40	1.95	1.95	TTN	12D	12D
30	0.60	0.60	0.40	0.40	1.95	1.95	7/6cm	10D	10D

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Alcohol Group (30 mins post consump3on value (BrAC = 0.8-1.0g/L))									
S/No	VA (R logMAR)	VA (L logMAR)	VA (R logMAR)	VA (L logMAR)	CS (1m R)	CS (1m L)	NPC	Accom R	Accom L
1	-0.08	-0.18	0.10	0.10	2.10	2.10	6/8cm	14D	14D
2	-0.18	-0.18	0.10	0.10	2.25	2.20	6/8cm	10D	12.5D
3	-0.08	-0.08	0.10	0.10	2.20	2.20	TTN	14D	14D
4	-0.18	-0.18	0.10	0.10	2.25	2.25	TTN	10D	11D
5	-0.08	-0.08	0.10	0.10	2.10	2.10	11/12cm	14D	14D
6	-0.08	-0.08	0.10	0.10	2.20	1.95	6/7cm	6D	7D
7	0.00	0.00	0.30	0.30	1.80	1.65	6/8cm	13D	10D
8	-0.08	-0.08	0.10	0.20	2.10	2.10	7/9cm	17D	17D
9	-0.08	-0.08	0.10	0.10	2.10	2.10	10/11cm	16D	12D
10	0.00	0.00	0.10	0.10	2.20	2.20	TTN	14D	10D
11	0.00	0.00	0.20	0.20	1.95	1.95	6/8cm	10D	10D
12	0.00	0.00	0.20	0.30	2.10	2.10	8/10cm	10D	8D
13	0.18	0.18	0.20	0.20	1.95	1.95	12/14cm	8D	6D
14	0.18	0.18	0.20	0.30	1.80	1.80	9/10cm	8D	6D
15	0.18	0.18	0.30	0.30	1.80	1.80	6/8cm	6D	6D
16	0.00	0.00	0.20	0.20	1.80	1.95	10/12cm	10D	6D
17	0.00	0.00	0.20	0.20	2.10	2.10	14/16cm	14D	14D
18	0.48	0.30	0.20	0.20	1.50	1.50	8/10cm	8D	8D
19	0.18	0.18	0.20	0.20	1.80	1.80	10/12cm	10D	12D
20	0.00	0.00	0.10	0.20	1.80	1.95	6/8cm	10D	12D
21	0.00	0.00	0.10	0.20	2.25	2.25	TTN	13D	13D
22	0.00	0.00	0.20	0.20	2.20	2.20	6/8cm	10D	10D
23	0.00	0.00	0.20	0.20	2.25	2.25	6/7cm	14D	14D
24	0.00	0.00	0.20	0.20	1.95	1.95	12/14cm	14D	14D
25	0.00	0.00	0.20	0.20	2.20	2.20	10/11cm	14D	14D
26	0.18	0.18	0.20	0.20	2.10	2.10	10/12cm	17D	17D
27	0.18	0.18	0.20	0.20	1.95	1.95	TTN	16D	17D
28	0.18	0.18	0.20	0.30	1.95	1.95	9/10cm	10D	11D
29	0.18	0.18	0.20	0.30	2.10	2.10	12/14cm	8D	10D
30	0.48	0.30	0.30	0.30	1.95	1.95	14/16cm	17D	17D

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Control Group (60mins post consumption)									
S/No	VA (R logMAR)	VA (L logMAR)	VA (R logMAR)	VA (L logMAR)	CS (1m R)	CS (1m L)	NPC	Accom R	Accom L
1	0.00	0.18	0.20	0.20	1.65	1.65	9/10cm	12D	12D
2	0.18	0.30	0.30	0.30	1.65	1.65	16/18cm	12D	12D
3	0.18	0.18	0.30	0.30	1.80	1.80	TTN	8D	8D
4	0.30	0.30	0.30	0.20	1.95	1.95	6/8cm	6D	6D
5	0.18	0.18	0.30	0.30	1.80	1.80	8/10cm	10D	8D
6	0.48	0.48	0.30	0.40	1.80	1.80	9/10cm	4D	4D
7	0.60	0.60	0.40	0.40	1,80	1.80	14/16cm	6D	6D
8	0.18	0.30	0.30	0.30	1.65	1.50	12/14cm	4D	4D
9	0.18	0.18	0.30	0.30	1.65	1.65	9/10cm	10D	8D
10	0.30	0.30	0.30	0.40	1.95	1.95	7/8cm	14D	12D
11	0.30	0.18	0.40	0.40	1.65	1.65	4/6cm	14D	10D
12	0.18	0.18	0.40	0.40	1.80	1.80	9/10cm	4D	4D
13	0.30	0.18	0.40	0.40	1.50	1.50	11/12cm	4D	4D
14	0.60	0.60	0.40	0.40	1.65	1.65	TTN	8D	8D
15	0.48	0.48	0.40	0.40	1.95	1.95	8/10cm	6D	4D
16	0.30	0.48	0.40	0.40	1.65	1.50	15/16cm	4D	6D
17	0.30	0.30	0.30	0.30	1.50	1.35	10/11cm	6D	8D
18	0.00	0.00	0.20	0.20	1.95	1.95	6/8cm	10D	8D
19	0.30	0.30	0.40	0.40	1.65	1.50	10/11cm	5D	6D
20	0.18	0.30	0.30	0.30	1.80	1.80	TTN	8D	6D
21	0.00	0.00	0.20	0.20	1.95	1.95	11/12cm	8D	8D
22	0.18	0.18	0.30	0.30	1.95	1.95	TTN	6D	8D
23	0.18	0.18	0.30	0.30	2.10	2.10	10/11cm	8D	8D
24	0.30	0.18	0.40	0.40	1.95	1.95	12/14cm	10D	10D
25	0.30	0.30	0.40	0.40	1.80	1.80	9/10cm	14D	14D
26	0.30	0.30	0.40	0.40	1.65	1.65	8/10cm	8D	8D
27	0.30	0.30	0.40	0.40	1.80	1.80	14/16cm	6D	6D
28	0.30	0.48	0.40	0.40	1.65	1.65	12/14cm	14D	14D
29	0.48	0.48	0.40	0.40	1.80	1.80	10/11cm	12D	12D
30	0.60	0.60	0.40	0.40	1.65	1.65	16/18cm	12D	12D

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Alcohol Group (60mins post consump3on (BrAC 0.7-1.1))									
S/No	VA (R logMAR)	VA (L logMAR)	VA (R logMAR)	VA (L logMAR)	CS (1m R)	CS (1m L)	NPC (B/R)	Accom R	Accom L
1	0.00	-0.08	0.10	0.10	1.95	1.95	6/8cm	16D	14D
2	-0.08	-0.08	0.10	0.20	2.10	1.95	6/8cm	10D	12D
3	-0.08	-0.08	0.20	0.20	2.10	2.10	TTN	10D	12D
4	-0.08	-0.08	0.10	0.20	2.20	2.20	TTN	12D	12D
5	-0.08	0.00	0.10	0.10	1.95	1.95	11/12cm	14D	14D
6	0.18	0.00	0.10	0.20	2.10	1.95	7/8cm	6D	6D
7	0.00	0.18	0.30	0.30	1.50	1.50	8/10cm	10D	6D
8	-0.08	-0.08	0.20	0.20	1.95	1.95	7/9cm	17D	17D
9	0.00	0.00	0.20	0.20	1.80	1.95	12/13cm	14D	10D
10	0.00	0.00	0.10	0.10	1.95	2.10	TTN	14D	12D
11	0.48	0.48	0.40	0.40	1.35	1.35	12/14cm	6D	6D
12	0.30	0.30	0.30	0.30	1.80	1.80	12/14cm	8D	6D
13	0.18	0.18	0.20	0.20	1.95	1.95	14/15cm	8D	8D
14	0.30	0.30	0.40	0.40	1.65	1.65	11/13cm	6D	6D
15	0.18	0.18	0.30	0.30	1.80	1.95	TTN	6D	8D
16	0.00	0.18	0.20	0.20	1.80	1.80	10/12cm	8D	8D
17	0.00	0.18	0.20	0.20	1.95	1.95	12/14cm	14D	12D
18	0.48	0.48	0.30	0.30	1.50	1.50	10/11cm	8D	8D
19	0.18	0.30	0.20	0.30	1.95	1.80	10/12cm	10D	10D
20	0.00	0.00	0.20	0.20	1.80	1.80	8/10cm	12D	12D
21	0.00	0.00	0.20	0.20	1.80	2.10	TTN	10D	10D
22	0.00	0.00	0.20	0.20	1.65	1.65	10/12cm	10D	12D
22	0.00	0.18	0.20	0.20	1.65	1.65	11/12cm	8D	10D
24	0.00	0.18	0.20	0.20	1.80	1.80	12/14cm	12D	12D
25	0.18	0.18.	0.20	0.30	1.95	1.95	14/15cm	10D	10D
26	0.18	0.18	0.30	0.30	1.80	1.80	TTN	6D	6D
27	0.18	0.30	0.30	0.30	1.80	1.95	TTN	6D	6D
28	0.30	0.30	0.30	0.30	1.65	1.65	6/8cm	8D	8D
29	0.30	0.30	0.40	0.40	1.95	1.95	7/9cm	12D	10D
30	0.48	0.48	0.40	0.40	1.65	1.65	10/12cm	17D	17D

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