LUMINESCENT THRESHOLD COMPARISONS, FLICKER FUSION DISCRIMINATION, AND REPETITION BLINDNESS IN RESPONSE TO TRIMETHYLXANTHINE (CAFFEINE) INGESTION

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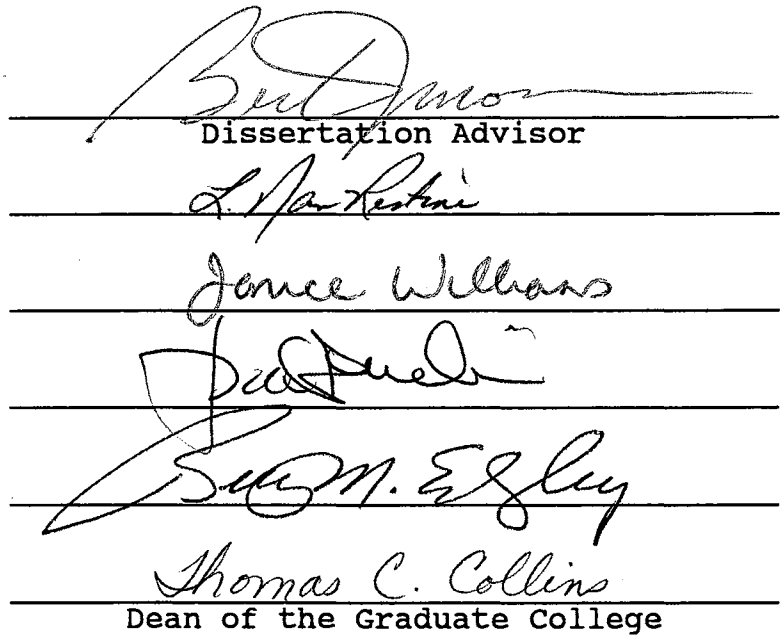

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TABLE OF CONTENTS

Chapter Page

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

Current Status of Caffeine

Caffeine, (1,3,7-trimethylxanthine), is a xanthine derivative and occurs naturally in coffee beans, tea leaves, kola nuts, and cocoa beans found throughout the world (Syed, 1976). As a result, caffeine is relatively accessible to all cultures. Caffeine has been described as the most widely consumed stimulant drug in the world (Chou, 1992). Adult intake of caffeine in the United States has been reported at approximately 2.5 milligrams per kilogram body weight (mg./kg. bwt.) or roughly 200 mg. per day (about two cups of coffee) and, for children 5 to 18 years old, the average daily caffeine intake is about 1.1 mg./kg. bwt. (Chou, 1992, p. 544). Furthermore, caffeine's stimulatory effects have been applied to both mental and physical performance (Dodd, Herb, and Powers, 1993).

Caffeine is consumed in food, beverages, prescription drugs, and over the counter (OTC) medications (Table I). For the average person, dietary caffeine intake is generally in the form of caffeinated beverages (e.g., coffee, tea, and colas) and chocolate with insignificant amounts of caffeine

TABLE I

CAFFEINE CONTENT OF SELECTED PRODUCTS

*CAFFEINE CONTENT (MG) IN A 12 OZ. SOFT DRINK

Afri-Cola	100.0 mg
Jolt	71.2
Sugar-Free Mr. Pibb	58.8
Mountain Dew	55.0 (0 in Canada)
Diet Mountain Dew	55.0
Mello Yellow	52.8
Tab	46.8
Coca-Cola	45.6
Diet Cola	45.6
Shasta Cola	44.4
Shasta Diet Cola	44.4
Mr. Pibb	40.8
Dr. Pepper	39.6
Pepsi Cola	37.2
Diet Pepsi	35.4
RC Cola	36.0
Diet RC	36.0
Canada Dry Cola	30.0
7 Up	0

* National Soft Drink Association
* Bunker, L., & McWilliams, M. (1979). Caffeine content of common beverages. Journal of the American Dietetic Association, 74, 28-32.

*CAFFEINE CONTENT (MG) IN A 7 OZ. CUP OF COFFEE/TEA:

Drip	115-175 mg
Espresso (1.5 - 2 oz.)	100
Brewed	80-135
Instant	65-100
Decaf, brewed	3-4
Decaf, instant	2-3
Tea, iced (12 oz.)	70
Tea, brewed, imported	60
Tea, brewed, U.S.	40
Tea, instant	25-150

* Bunker, L., & McWilliams, M. (1979). Caffeine content of common beverages. Journal of the American Dietetic Association, 74, 28-32.

TABLE I (Continued)

CAFFEINE CONTENT OF SELECTED PRODUCTS

*CAFFEINE CONTENT (MG) IN SELECTED FOODS:

Milk chocolate (1 oz.)	1-15 mg
Bittersweet chocolate (1 oz.)	5-35
Chocolate cake (1 slice)	20-30

* Health Letter Associates. (1990). The Daily Dose. Berkeley Wellness Letter, University of California.

*CAFFEINE CONTENT (MG) IN A STANDARD DOSE OF NONPRESCRIPTION DRUGS:

Stimulants	
Caffedrine Capsules	200 mg
NoDoz Tablets	200
Vivarin Tablets	200
Pain Relievers	
Anacin	64 mg
Excedrin	130
Midol	65
Aspirin	0
Diuretics	
Aqua-Ban	200 mg
Permathene H2Off	200
Pre-Mens Forte	100
Cold Remedies	
Coryban-D	30 mg
Dristan	32
Triaminicin	30
Weight Control Aids	
Dexatrim	200 mg
Dietac	200
Prolamine	280

* Caffeine: How to consume less. (1981, October). Consumer Reports, 597-599.

being provided in other foods flavored with coffee and chocolate (Graham, 1978).

Caffeine is widely used and very accessible to people of virtually every nation. These factors, combined with caffeine's stimulating effects, make the abuse of caffeine a potential problem for consumers of this drug. However, as E. M. Brecher and the editors of Consumer Reports (1972) point out, the majority of caffeine is consumed in such a way (e.g., dilution per serving based on preparation technique, addition of cream or milk, and/or ingestion following a meal) as to practically eliminate harmful side-effects. Therefore, serious abuse is not common.

Caffeine has no nutritional value. However, due to its widespread use in society and ease of access, sports competitors have and continue to explore caffeine's stimulatory effects on performance -- particularly improving mental outlook and reduction of fatigue (Powers & Dodd, 1985). In fact, caffeine is sometimes referred to as a "nutritional ergogenic aid" (Spriet, 1995, p.S84). Athletes, like the general population, consume caffeine in food, beverages, OTC medications, and prescription drugs (Table I). Since caffeine is consumed in many common foods, beverages, and medications, the International Olympic Committee (IOC) removed caffeine from its list of doping agents in 1972 (VanHandel, 1983). Later, however, the IOC did ban "high levels" of caffeine administered by injection or suppository

(VanHandel, 1983). Then, in 1982, caffeine was once again added to the IOC's list of doping agents with a limit of 15 micrograms per milliliter (μg./ml.) (about 10 cups of coffee). In time, this limit was lowered to 12 μg./ml. (Catin & Hatton, 1991).

Caffeine's stimulatory effects have and continue to be examined. Much emphasis has been placed on this drug's impact on motor performance and on the subject's state of alertness. However, for many occupations (e.g., pilots, air traffic controllers, professional athletes, computer programmers, and data entry) the effects of caffeine on vision may have just as big an impact. The need exists for further research to be conducted on the effects of caffeine on vision to further illuminate the potential relationship between caffeine consumption and professional, vocational, and even recreational visual performance. Caffeine's effect on luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness may provide this type of information. Despite what the results may reveal, the general public, employers, and employees have the right to benefit from such information whether from a recreational or occupational viewpoint.

Properties of the Drug

Caffeine, a xanthine derivative, is an odorless, bitter, white, crystalline powder. Its' chemical structure is

identified as 1,3,7-trimethylxanthine (Figure 1). Caffeine empties rapidly from the stomach and is absorbed by the gastrointestinal tract. The highest levels of caffeine are generally detected in the blood stream approximately one hour following ingestion with the most caffeine being delivered to tissue with high water content (e.g., muscle tissue) (Axelrod & Riechenthal, 1953). Therefore, the effects produced is directly proportional to the concentration of caffeine in various body tissues (VanHandel, 1980).

Caffeine is efficiently metabolized by the liver preventing accumulation in organs or tissues. This metabolization varies in rate evidenced by caffeine's half-life (the time required for the drug to be eliminated) ranging from 2 to 12 hours with an average half-life of 4 to 6 hours (Chou, 1992). Longer half-lives are generally experienced by pregnant women, women taking oral contraceptives, consumption of alcohol, and persons suffering from liver disease. Conversely, smokers metabolize caffeine more quickly thereby experiencing shorter half-lives. (Sawynok, 1995). Approximately 2% to 3% of the ingested caffeine is excreted unchanged in the urine (Chou, 1992).

Caffeine produces pharmacological responses in various systems of the body. Caffeine stimulates the central nervous system often resulting in heightened alertness and clarity of thought (Syed, 1976). As a result of these effects on the central nervous system, caffeine may indeed

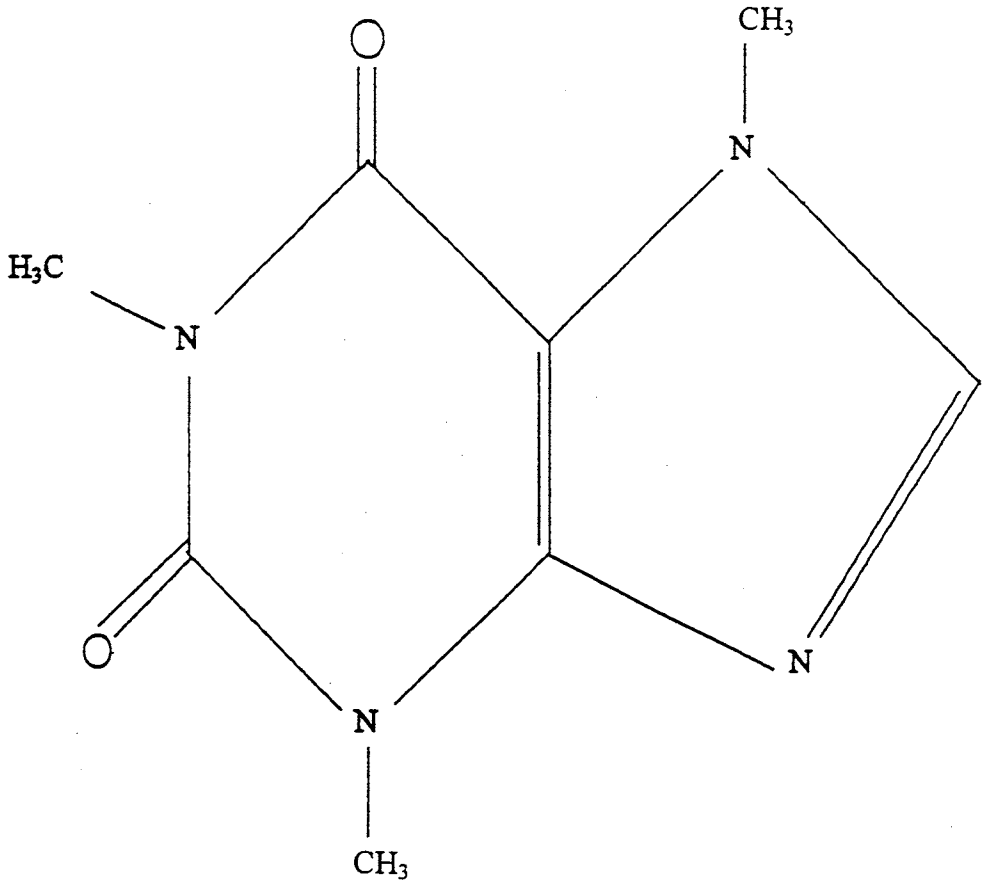


Figure 1. Chemical Structure of Caffeine

act on the muscular system by increasing motor activity. On the other hand, caffeine's effect on the central nervous system has been reported to aggravate psychiatric disease (Chou, 1992). High doses of caffeine (>600 mg. per day) produces a condition identified as "caffeinism". Caffeinism is characterized by anxiety, sleep disorders, and states of restlessness, which is often characteristic of anxiety disorders (Greden, 1974).

The stimulating effect of caffeine is apparent on the cardiovascular system as evidenced by increased heart rate. This increase, however, is generally preceded by a decrease in heart rate due to simultaneous stimulation of medullary vagal nerves (MacCornack, 1977). Increases in vasodilation, coronary circulation, and/or increased blood pressure are also common physiological responses following caffeine ingestion. Caffeine has reportedly effected vision by increasing intraocular pressure under certain conditions (Higginbotham, Kilimanjaro, Wilensky, Batenhorst & Hermann, 1989), improving visual monitoring (Putz-Anderson, Setzer & Croxton, 1981), increasing the eye's susceptibility to light (Diamond & Cole, 1970), and improving visual reaction time (Leiberman, Wurtman, Emde & Coveilla, 1987). Similarly, caffeine stimulates the kidneys causing the urinary system to increase urination. The digestive system is also activated as indicated by an increase in the secretion of gastric acids (Brecher, 1972). In addition, caffeine has been

reported to stimulate the respiratory system by increasing the rate and depth of respiration. There have also been reports of an effect on the endocrine system involving stimulation of the adrenal glands (VanHandle, 1983). All in all, caffeine has been described as increasing the body's metabolic rate.

These potential outcomes can explain the use of caffeine as an ergogenic aid despite equivocal research results. Particularly when caffeine's effects on the central nervous system, cardiovascular system, muscular system, endocrine system, and vision are considered, the potential for improved physical performance is clear. However, this ergogenic activity continues to be questioned and examined.

Caffeine does serve several therapeutic uses in the medical field today (Sawynok, 1995). Caffeine, in conjunction with other drugs, have been used to treat headaches and pain. This drug has been utilized to stimulate the respiratory systems of premature infants and to lengthen seizure duration needed in electroconvulsive therapy to treat certain types of depression. In addition, caffeine has been used to treat hypotension in the elderly following food ingestion and in the treatment of obesity. However, the ingestion of caffeine has been linked to several negative side effects as well.

Negative side effects associated with caffeine intake include: diuresis, insomnia, withdrawl headaches, diarrhea,

anxiety, tremulousness, and irritability (VanHandle, 1983). These noxious effects have been reported to occur with intake greater than 1 gram of caffeine (7 to 10 cups of coffee) (Brecher, 1972). However, such effects have also been reported with caffeine intake as low as 250 mg. (2 to 3 cups of coffee) (Sawynok, 1995). A lethal dosage has been estimated to be approximately 10 grams of caffeine or approximately 70 to 100 cups of coffee (Brecher, 1972); although death caused by caffeine intake is rare. Caffeine effects are obviously going to be dependent on the subject's sensitivity to the drug, the weight/muscle mass of the subject, the form in which caffeine is consumed, and the subject's tolerance level.

Long term use of caffeine has been the topic of much interest over the years. Caffeine intake has been linked to heart attacks, hypertension, birth defects, colorectal cancer, and pancreatic cancer. In spite of much conjecture, no significant relationship has been statistically identified between these diseased states and moderate caffeine intake (approximately 5 cups of coffee per day). The one exception reported by Sawynok (1995) is that there is an elevated risk of bladder cancer observed in coffee drinkers; however, coffee has not been identified as the cause.

Regular consumption of caffeine, like many other drugs, can produce tolerance to this stimulant. This process is still unclear (Sawynok, 1995). However, when the level of

consumption is decreased or terminated, withdrawal symptoms generally result. These symptoms include headaches, irritability, inability to concentrate, nervousness, and lethargy (Brecher, 1972). After time, these symptoms will dissipate as dependence on this stimulant dissipates. Or, if additional caffeine is consumed, the withdrawl symptoms will be postponed indefinitely.

Caffeine's effects on the central nervous system have been the focus of much research during past decades. Due to the stimulating effect caffeine has on the central nervous system, many studies have extended this application to other systems of the body including the endocrine system, muscular system, respiratory system, cardiovascular system, and even to caffeine's effects on vision. However, studies addressing the effects of caffeine on vision are not as numerous or as extensive as research dealing with caffeine and other body systems. Additional work is needed to fully examine this drug's effect on vision. As a result, caffeine's effect on luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness will be examined.

Purpose of the Study

The intent of this study was to investigate the influences of two doses of caffeine (2.5 mg./kg. bwt., and 5.0 mg./kg. bwt.) and a placebo on three aspects of vision: luminescent threshold comparisons, flicker fusion discrimi-

nation, and repetition blindness.

Hypotheses

The following hypotheses were examined in this investigation.

HO1: There will be no difference between pre-test and post-test luminescent threshold responses at the "low" luminescence setting after consumption of placebo, 2.5 mg./kg. bwt., and 5.0 mg./kg. bwt. caffeine.

HO2: There will be no difference between pre-test and post-test luminescent threshold responses at the "moderate" luminescence setting after consumption of placebo, 2.5 mg./kg. bwt., and 5.0 mg./kg. bwt. caffeine.

HO3: There will be no difference between pre-test and post-test luminescent threshold responses at the "high" luminescence setting after consumption of placebo, 2.5 mg./-kg. bwt., and 5.0 mg./kg. bwt. caffeine.

HO4: There will be no difference between pre-test and post-test flicker fusion responses after consumption of placebo, 2.5 mg./kg. bwt., and 5.0 mg./kg. bwt. caffeine.

HO5: There will be no difference between pre-test and post-test repetition blindness responses after consumption of placebo, 2.5 mg./kg. bwt., and 5.0 mg./kg. bwt. caffeine.

Delimitations

1. A total of 23 subjects (9 male and 14 female) were

used in this study.

2. All subjects were healthy college students from Oklahoma State University.

3. There were three levels of treatment administered: 2.5 mg./kg. bwt.; 5.0 mg./kg. bwt.; and placebo.

4. The subjects for this investigation were not randomly selected.

5. All subjects were classified as average caffeine consumers consuming approximately 200 mg. per day (± 200 mg.).

Limitations

1. There may have been individual sensitivity to caffeine.

2. Fluctuations in electrical voltage may have prevented the absolute value of the Light Discrimination Apparatus and the Visual Perception Control from being accurately determined.

Assumptions

1. Subjects correctly followed all instructions.

2. Subjects consumed all caffeine with tap water.

3. Subjects were honest in their initial estimation of caffeine consumption prior to the study.

4. Subjects' responses represent their best effort to correctly respond to each test.

Definition of Terms

Luminescent Threshold is the point in time at which the eye can discern the most minute difference in the intensity of light acting as a stimulus.

Flicker Fusion is the point in time where the frequency of a flickering light is no longer discernable; the light "fuses" into continuous illumination.

Repetition Blindness is the inability to identify a second exposure to a repeated word or identification of a novel word following an initial exposure to a prime word.

CHAPTER II

REVIEW OF LITERATURE

Introduction

Caffeine is one of the most commonly used drugs. It is consumed in beverages, foods, prescription drugs, and over the counter medications. As a result, it effects various systems of the body and produces both positive and negative results. Since the body is comprised of these "various systems", the effects are often cumulative and far reaching. To better understand the influence of caffeine on selected systems, this review will focus on three primary areas: neurological effects, neuromuscular effects, and the effects of caffeine on vision.

Neurological Effects

Caffeine acts as a central nervous system stimulant. After easily passing the blood/brain barrier, caffeine acts on the cerebral cortex (Syed, 1976). The cerebral cortex covers masses of nerve fibers connecting the cortex with other parts of the nervous system (Hole & Koos, 1991). So in addition to resulting in clearer thought, sharpened cognitive abilities, and a decline in drowsiness and fa-

tigue, Syed (1976) further describes caffeine as directly effecting the brain's medullary, vasomotor, respiratory, and vagal centers.

Four primary mechanisms have been hypothesized to explain caffeine's effect on the central nervous system at the cellular level (Nehlig, Duval & Debry, 1992). The first mechanism is identified as "intracellular mobilization of calcium". This intracellular mobilization was initially examined in skeletal muscle fibers (Bianchi, 1975; VanHandle, 1983). Caffeine (1 to 2 millimoles (mM)) has been reported to lower the muscle cell's excitability threshold and increases the length of the contraction by movement of calcium through the plasma membrane and from the sarcoplasmic reticulum (Bianchi, 1975). Investigating this concept with a more broad application revealed that synaptic transmission in the central and peripheral nervous systems requires neurotransmitter release which is dependent on the incursion of calcium into nerve endings (Nehlig et. al., 1992). However, a minimum caffeine concentration of 250 micromoles (μM) (2.5 to 7.5 cups of coffee) is necessary to produce these calcium shifts (Guthrie & Naylor, 1967) and pharmacological effects of caffeine are generally detected by concentrations lower than 100 μM (1 to 3 cups of coffee). As a result, it seems unlikely that "intracellular mobilization of calcium" is an integral role of caffeine on the central nervous system (Nehlig et. al., 1992).

A second hypothesis developed to explain caffeine's effect on the central nervous system was "inhibition of phosphodiesterase". Through a series of cellular reactions, adenosine 3',5'-cyclic monophosphate or cyclic AMP (cAMP) plays a vital role in controlling glycogen metabolism and peripheral lipolysis. Phosphodiesterase, an enzyme, breaks down cAMP. Caffeine (methylxanthines) prevents enzymatic breakdown of cAMP through inhibition of phosphodiesterase thereby decreasing glycogenolysis and promoting lipolysis (Beavo et al., 1970; Dodd et al., 1993). However, caffeine's effects on phosphodiesterase are produced only when the methylxanthine is present in very large quantities (1 mM); quantities that are considered toxic (Cardinali, 1980). Therefore, it is difficult to associate the "inhibition of phosphodiesterase" with concentrations of caffeine found in circulating blood.

"Antagonism of adenosine receptor sites" is a third hypothesis explaining caffeine's effect on the central nervous system. Adenosine is a normal cellular constituent and it is also commonly found in extracellular fluid. Adenosine is defined by VanHandel (1983) as being a "neurotransmitter or modulator with depressant, hypnotic, and anticonvulsant properties" (p. 133). These depressant results are quite extensive since adenosine receptors are located throughout the body. This third hypothesis, however, depicts caffeine (methylxanthines) blocking the stimu-

lation of cAMP formation by blocking adenosine receptor sites (Sattin & Rall, 1970; VanHandle, 1983). As a result, following caffeine consumption, people experience wakefulness, increased energy and increases in locomotor activity rather than lethargy, drowsiness, and decreases in activity level. Low plasma concentrations (less than 100 μM, which is equal to drinking 1-3 cups of coffee) of caffeine (methylxanthines) have been found to antagonize adenosine receptor cites (Nehlig, et. al., 1992). In addition, caffeine at such low concentrations appears to have no direct effect on metabolism of cAMP or on calcium shifts (Snyder, Katims, Annau, Bruns & Daly, 1981).

A fourth hypothesis, "interactions with benzodiazepine binding sites" has been considered. Benzodiazepines act as depressants producing minimal decreases in blood pressure and cardiac output with dose related reductions in blood flow to the brain and utilization of oxygen. Similarly, skeletal muscles experience mild relaxant effects (Stoelting, 1991). Caffeine binds to benzodiazepine receptor sites; however, it is not a strong bond (Boulenger, Patel & Marangos, 1982). Caffeine is a much stronger antagonist of adenosine receptor sites than those of benzodiazepine. As a matter of fact, the effect of caffeine on benzodiazepine receptor may actually be due to caffeine's effect on adenosine receptors. In addition, it has been proposed that caffeine's influence on benzodiazepine receptors would only

occur at toxic levels of caffeine consumption (Davies & Chow, 1984).

Four mechanisms that have been hypothesized to explain caffeine's effect on the central nervous system at the cellular level. However, of the four, only one explanation has withstood the test of time. Currently, the most commonly accepted explanation of caffeine's effect on the central nervous system is the "antagonism of adenosine receptor sites" (Daly, Bruns & Snyder, 1981; Dodd et al, 1993; Fredholm, 1995; Graham, Rush, & vanSoren, 1994).

Caffeine's effects have even broader applications. This methylxanthine may have potential effects on the central nervous system involving neurotransmitters (e.g., catecholamines, serotonin, acetylcholine, and amino acids). In addition to being dependent on calcium shifts as mentioned earlier, elevated levels of neurotransmitters are also linked with altered substrate mobilization; increased lipolysis and decreased glycolysis. A potential role of adenosine in the central nervous system is to suppress the release of numerous neurotransmitters. Consequently, adenosine antagonists (methylxanthines such as caffeine), theoretically, would be expected to increase neurotransmitter release thus altering substrate utilization (Chow, 1992; Graham et al., 1994; Nehlig et al., 1992; VanHandel, 1980).

Caffeine effects cerebral blood flow and the utilization of glucose. Caffeine precipitates changes in cerebral

energy metabolism by way of increased usage of glucose. This increased metabolism may ultimately effect spontaneous motor activity, simple and complex coordination, endurance and athletic performance, aggressiveness and mood, anxiety and sleep, and learning and memory. As mentioned earlier, caffeine is classified as a central nervous system stimulant. Methylxanthines, in general, produce vasodilation. However, rather than causing vasodilation of cerebral vasculature as would be expected of this type of stimulant, caffeine ingestion results is vasoconstriction of these vessels thus decreasing cerebral blood flow. Oddly enough, this reduction of blood flow generally occurs in areas of the brain where caffeine is simultaneously increasing metabolism. Cerebral blood flow is generally closely matched with glucose usage. When cerebral activity changes, these changes cause parallel shifts in blood flow and glucose utilization. However, counter to the effects of other methylxanthines, caffeine causes a reduction in cerebral blood flow while simultaneously increasing glucose metabolism (Nehlig et al., 1992).

All of caffeine's effects on the central nervous system are relayed to the peripheral nervous system via somatic fibers connecting to the skin and skeletal muscles and autonomic fibers connecting to the visceral organs. In addition, this autonomic nervous system further relays impulses to its two subdivisions. The sympathetic division

basically prepares the body for stress (e.g., emergencies) and the parasympathetic division is most active under ordinary conditions. In this way, the effects of caffeine on the central nervous system is transmitted to the periphery as well; thereby having the potential to effect metabolism, as well as, behavior and performance (Hole & Koos, 1991).

Neuromuscular Effects

Caffeine distribution in the body is dependent on the water content of body tissues. Therefore, skeletal muscle, possessing a high water content, generally exhibits elevated caffeine levels following ingestion. According to Dodd et al. (1993), caffeine potentially effects skeletal muscle through three possible mechanisms: increased affinity of myofilaments for calcium and/or increased calcium release from the sarcoplasmic reticulum; changes in cellular activity due to an increase in cAMP in skeletal muscle tissue; and inhibition of adenosine receptor sites in the central nervous system.

The first mechanism often considered in the explanation of caffeine's effect on skeletal muscle involves myofilaments developing a greater affinity for calcium and/or causing an increase in the calcium release from the sarcoplasmic reticulum following caffeine ingestion. The idea of caffeine enhancing the mobilization of intracellular calcium from membrane stores was very popular in the 1960's (Bian-

chi, 1961). Additional studies on isolated skeletal muscle have repeatedly shown caffeine's effects. Caffeine causes translocation of calcium through plasm and the membranes of the sarcoplasmic reticulum thereby lowering the threshold potential for excitation and extending the active period of muscle contraction (Fryer & Neering, 1989; Su & Hasselbach, 1984; VanHandel, 1983). Similarly, increased myofibrillar sensitivity to caffeine is evidenced by a greater twitch tension development at submaximal calcium concentrations (Gulati & Babu, 1985; Wendt & Stephenson, 1983).

A second mechanism developed to explain caffeine's effect on skeletal muscle involves increased levels of cAMP causing changes in the activity of the cell. Caffeine may actually alter both lipid and carbohydrate storage in skeletal muscle (VanHandel, 1983). As mentioned earlier in "Neurological Effects", caffeine increases cellular levels of cAMP. Increased cAMP has been shown to increase glycogenolysis resulting in hyperglycemia (MacCornack, 1977; Syed, 1976; VanHandel, 1983). As in the case with most activities, glycogen (carbohydrate) generally serves as the initial substrate utilized in transforming chemical energy to mechanical energy. Therefore, this initial increase in glycogenolysis serves a very useful purpose. Increased cAMP also promotes lipolysis (VanHandel, 1983). Increased lipolysis boosts the free fatty acids available as a substrate for contracting muscles; however, increases in fatty acid

oxidation generally surpass and slow the rate of carbohydrate utilization (Dodd et al., 1993; VanHandel, 1980; VanHandel, 1983). This increase of free fatty acids and sparing of glycogen during exercise could theoretically improve performance in endurance activities (Dodd et al., 1993; VanHandel, 1980).

The third mechanism explaining caffeine's effect on skeletal muscle refers to inhibition of adenosine receptor sites in the central nervous system with related effects occurring in peripheral cells (Fredholm, 1985; Zhang & Wells, 1990). Adenosine, via interaction with adenosine receptors, is involved with regulation of almost every organ system (e.g., neurotransmitter release, control of smooth and cardiac muscle tone, formation of white blood cells and platelets, and adipose tissue lipolysis) (Olson & Pearson, 1990; Ramkumar, Bumganer, Jacobson & Stiles, 1988). Initial research conducted by Sattin and Rall (1970) on slices of a guinea pig's cerebral cortex exhibited a decrease of cAMP levels following ingestion of 0.5 mM methylxanthines. Further studies conducted which resulted in blocked adenosine receptor sites in brain tissue by caffeine resulted in stimulation of behavior (Holtzman, Mante & Minneman, 1991). Similarly, Zhang and Wells (1990) reported a reduction of triglyceride content in adipocytes of caffeine treated rats inferring increased lipolysis.

It seems logical to expect much of the information pro-

vided in the previous section, "Neurological Effects", would apply to the skeletal muscles since the muscles receive stimuli from the central nervous system by way of the peripheral nervous system. For example, studies reporting increased lipolysis could, and often do, reflect alterations in substrate utilization by skeletal muscle. However, very few studies have correlated antagonism of adenosine receptors directly with skeletal muscle effects. Challis, Richards, and Budohoski (1992) conducted a study directed at examining adenosine receptors located in the skeletal muscle of rats. These findings did support the proposal that adenosine does have an insulating modulating action in skeletal muscles' transport of glucose more so than skeletal muscles' stimulation of glycogen synthesis and this activity is mediated by adenosine receptors. But, no new information concerning the importance of adenosine receptors was uncovered. Consequently, the lack of new information concerning skeletal muscle response to caffeine's antagonistic effects on adenosine receptors combined with the known effects of caffeine on the central nervous system and other organ systems leaves many questions unanswered. However, evidence does suggest that inhibition of adenosine receptors is one of the most important, if not the most important, mechanisms utilized to explain the physiological effects of caffeine at nontoxic concentrations (Dodd et al., 1993).

Visual Effects

Caffeine's stimulating effects are conveyed systemically to all parts of the body (Koetting, 1977). Since caffeine does stimulate certain medullary centers, the eye is susceptible to drugs that affect the central nervous system through systemic circulation. And, due to an ample number of vessels providing a very rich blood supply to this organ that is relatively small in mass, the eye exhibits increased sensitivity to many drugs. Adverse effects generally occur with high doses with more intense reactions occurring in individuals with heightened sensitivity and in the very young and old. (Jaanus, 1992)

The effect of caffeine on different structures in the eye has been the focus of several studies. Lotfi and Grunwald (1991) studied the effects of caffeine (200 mg.) on retinal circulation and the results point to a presumed decrease in blood flow despite an increase in diastolic blood pressure. This result led the researchers to attribute the decreased circulation to retinal vasoconstriction brought on by inhibition of endogenous adenosine. Another study conducted by Higginbotham et al. (1989) investigated the effect of drinking regular coffee on intraocular pressure in subjects with glaucoma. These authors found that caffeine (505.8 ± 19.45 $\mu g/ml$) did have a statistically significant effect on intraocular pressure when compared to caffeine-free beverages 90 minutes after ingestion but not

at the 30 and 60 minute intervals. In addition, caffeine had a statistically significant effect on diastolic pressure at both the 60 and 90 minute intervals. However, neither of these findings were found to be clinically significant. Examination of changes in pulse rate and systolic pressure were both found to be statistically insignificant. Therefore, according to these authors, there is no reason to limit/control caffeine consumption among glaucoma patients.

Adams and Brubaker (1990) also conducted a study on the eye's intraocular pressure. However, in this study, apparently healthy subjects were orally administered caffeine (400 mg.). Findings showed no statistically significant difference in aqueous humor flow measured hourly from 1 to 4 hour after caffeine ingestion. Similarly, there was no statistically significant difference in intraocular pressure measured 4 hours after caffeine ingestion. Again, there was no evidence to suggest that average or slightly above average daily caffeine consumption produces a clinically significant effect on intraocular pressure.

Studies addressing the effects of caffeine on the ability of the eye to function are also very common. As early as 1959, John Carpenter (1959) examined the effect of caffeine and alcohol on simple visual reaction time. His findings do suggest that high doses caffeine (2.94 mg./kg. bwt.) do produce significant reductions in reaction time; particularly when reaction times have been lengthened by the

consumption of alcohol. However, this particular study (Carpenter, 1959) could not substantiate this hypothesis. More recently, Leiberman et al. (1987) discovered that caffeine equivalent to that found in a single serving of cola (32 to 64 mg. of caffeine) significantly improved auditory vigilance and visual reaction time. No adverse behavioral effects were reported even at the highest dose administered (64 mg.).

Broverman and Casagrande (1982) studied the effects of caffeine (113 mg.) on tasks requiring perceptual-restructuring at different stages of practice. Caffeine tends to impair perceptual-restructuring task performance when the tasks are novel, and tends to facilitate performance of tasks that are not novel. In other words, caffeine was found to both facilitate and impair performance of these restructuring tasks. The effect was dependent on the stage of practice. A recent study (Kenemans and Lorist, 1995) examined the effects of caffeine (3 mg./kg. bwt.) on selective visual processing. The effects on visual processing was revealed utilizing five parameters: modifications in EEG indicating a change in brain state; improved performance in a simple selective attention task; increased selectivity of processing; improved task-independent discrimination of stimuli; and accelerated central motor processes. The findings included: (a) cortical activation increased following caffeine ingestion; (b) caffeine increased sensi-

tivity, or the rate at which information on the stimulus accumulates; (c) caffeine increased selectivity, in particular with respect to further processing of stimuli once selected on the basis of the primary attribute; however, these findings suggest further research is needed; (d) caffeine speeded up central or peripheral motor processes.

The effect of caffeine on visual tracking has been another topic of interest. Baker and Theologus (1972) studied the effects of caffeine (200 mg. and 400 mg.) on a protracted visual monitoring task similar to an aspect of automobile night driving. The results indicated that caffeine (both dosages) significantly inhibited response blocking or, in the case of automobile driving, attention lapses. Similarly, in a study conducted by Putz-Anderson et al. (1981) examining the effects of caffeine (3 mg./kg. bwt.), alcohol and methyl-chloride on man, the protocol included a visual vigilance task and a dual task comprised of tone-detection and eye-hand compensatory tracking. The subjects who received the caffeine showed a 4% improvement over the control group on the vigilance task. In the dual task, only the tracking activities were sensitive to the effects of caffeine treatment. When tracking activities during the caffeine treatment were compared with the pretreatment scores, tracking error decreased by an average of 10% ($p<.05$). Improved visual tracking caused by caffeine and nicotine in rats was compared to the effects of amphetamine,

cocaine, and apomorphine by Evenden, Turpin, Oliver, and Jennings (1993). In examining the caffeine-specific data generated among a wealth of statistics in this comparison, caffeine reduced the rate of responding at the highest caffeine dose (30 mg./kg.). Caffeine also increased tracking efficiency at the two highest doses (10 mg./kg. and 30 mg./kg.).

Visual vigilance is another related ability effected by caffeine. Loke and Meliska (1984) examined the effects of caffeine on protracted visual vigilance tasks. Higher caffeine users (mean = 204±84 mg./day) made significantly fewer hits, more false alarms, and responded faster than lower caffeine users (mean = 44±28 mg./day). Performance declined during the 90 minutes of vigilance testing. No improvement was found in vigilance relative to caffeine ingestion; which opposes expected results based on previous test results utilizing various methodology. Caffeine did not reverse progressively deteriorating performance relative to control. Similarly, Fine et al.(1994) examined the effects of caffeine (200 mg.) or diphenhydramine on visual vigilance. However, as opposed to Loke and Meliska'a study (1984), caffeine significantly increased the number of correct responses and decreased response times when compared to the placebo. Low habitual consumers of caffeine (<100 mg./day) and nonsmokers had more correct responses than did high habitual caffeine consumers (> 100 mg./day), but only in the

placebo condition. Fine et al. (1994) addresses the fact that these findings oppose those of Loke and Meliska (1984) and points out that the discrepancy may be due to the nature of their task and not an absence of an underlying effect.

William Paré (1961) examined the retroactive effects of caffeine (30 mg./kg. bwt.) and seconal on learning in the early 1960's. Utilizing a visual discrimination apparatus rats were required to discriminate between different colored cards after training. Results indicated that the rats who received caffeine injections manifested fewer errors on retention trails than those receiving seconal or placebo. Further, the number of errors on retention trials for these rats were positively related to the learning criterion-injection interval; caffeine injections 5 seconds after criterion made significantly fewer errors on retention trials whereas injections at 2 minutes and 1 hour following criterion did not produce significant differences.

The relationship between the effects of caffeine and fatigue is a very interesting association. Lorist was the primary investigator in two studies examining the effects of caffeine and fatigue. In the first study, Lorist, Snel, and Kok (1994) examined the effects of caffeine (200 + 50 mg., a maintenance dose) on information processing in well rested and fatigued subjects. Data showed that caffeine shortened reaction time with an accompanying interaction with stimulus degradation and time uncertainty in the majority of sub-

jects. In addition, the idea of caffeine increasing cortical arousal and perceptual sensitivity was supported. Fatigued subjects showed larger improvements in performance following caffeine consumption than did well rested subjects. These findings also indicate that the effects of caffeine were not stimulating in all subjects. Six out of 30 subjects showed no arousing effects of caffeine. In the second study, Lorist, Snel, Kok, and Mulder (1994) examined the influence of caffeine (200 + 50 mg., a maintenance dose) on selective attention utilizing a visual focused selective search task in well-rested and fatigued subjects. The findings depicted subjects reacting faster after having consumed caffeine. Reaction times in the caffeine condition were significantly shorter than those in the placebo condition ($p<.02$). Additionally, in the test focusing on target detection, caffeine's effects were dependent on the state of the subject. The author's hypothesized that the quality and speed of stimulus perception and decision to respond may be adversely effected by fatigue.

All of the information previously discussed helps to explain caffeine's effect on the body -- particularly neurologically, neuromuscularly, and visually. Specifically, this study's focus is directed at caffeine's effects on three selected visual components: luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness.

Luminescent threshold comparisons were examined by Kleman, Diamond, and Smith (1961) while observing the effects of caffeine on "foveal simultaneous contrast". The researchers utilized an apparatus that implemented a chin rest (122 cm. from the apparatus), a center baffle that separated right and left visual pathways, two different visual patterns (one to be viewed by the right eye and a different pattern for the left eye), a small circular test field surrounded by an inducing annulus, and separate light sources, 100 watts (w.) and 120 volts (v.), for each eye.

Data was collected from three subjects who were low to moderate caffeine users and who had been instructed to abstain from caffeine for 24 hours prior to testing. Testing was conducted on two separate days. On each test day, subjects were administered two levels of treatment (placebo and then caffeine) with one visual test being administered after the other. The placebo was administered to the subjects followed by a 15 minute rest period and another 15 minutes of dark adaptation (30 minutes total) prior to testing. The same procedure was then repeated with a standard 3 grains (194.4 mg.) of caffeine. The effect of caffeine was reported in log millilamberts (log mL) as a function of the inducing field luminance and plotted as a function of the log luminance.

The results of this testing showed that the subjects' responses following caffeine intake not only reduced the en-

hancement phenomenon (test field brightness being perceived as brighter when the inducing luminance is less than the test luminescence) but actually erased it so that the brightness of the test field was depressed in direct proportion to the luminance of the inducing field. Depression of the test field (perceived when the inducing luminance is equal to or greater than the test luminance) continued to occur similar to the non-drug state. A mechanism identified to possibly explain this lack of enhancement was that the "on" retinal fibers became more sensitive with caffeine and depression of the test field occurred which reduced or eliminated enhancement.

Diamond and Cole (1970) expanded on the work of Klemen et al. (1961) and examined visual threshold as a function of test area and caffeine administration. The threshold luminance of a circle was measured in relation to its' area before and after ingestion of placebo, 1.5 grains (97.2 mg.) of caffeine, and 3.0 grains (194.4 mg.) of caffeine. Three subjects were solicited for this study. The subject's average daily caffeine intake was not taken into consideration.

The test apparatus consisted of a light path (150 w., 115 v.) to the subject's right eye with an optician's trial frame fixed to the subject's head. Each test session was the same. Fifteen minutes before entering the darkened test room, the subjects were given a placebo. After this period,

the subject entered the test site and was fitted with the optician's frame and allowed to adjust to the dark. Threshold measurements were taken for 10 test-field areas. Then, the subject left for a one hour rest. Thirty minutes before reentering the darkroom the subject was given either a 1.5 grain or 3.0 grain capsule of caffeine.

The procedure described above was then repeated utilizing the remaining caffeine dose. Threshold luminance as a function of test radius was reported in log mL and threshold (in log mL) was plotted representing the respective caffeine doses. Results reported visual threshold luminescence significantly decreasing following ingestion of 1.5 grains and 3.0 grains of caffeine. These findings led the authors to intimate that caffeine causes the "on" visual pathways to become more sensitive to light and that decreases in the threshold corresponds to increases in the caffeine dose.

Flicker fusion frequency was examined in a review by Simonsons and Brozek (1952) conducted in the early 1950's. This investigation described the evolution of the flicker fusion response in man originating from the belief that flicker fusion frequency was a retinal function to a broader application incorporating cortical involvement.

Roback, Krasno, and Ivy (1952) conducted a study which addressed the effect of caffeine (30 mg.) on flicker fusion threshold. Caffeine, although a central nervous system stimulant, depressed flicker fusion frequency. This study

explored the effects of analeptic drugs on the effect of seconal and antihistamines to indicate various depressing and stimulating influences on the central nervous system with particular interest on visual mechanisms. There were 5 experimental groups receiving caffeine alone or in combination with other drugs. Subjects in each group ranged in number from 10 to 30. Subjects were seated 5 feet from the "flicker photometer" to enable the flicker to fall correctly on the fovea centralis. Other sources of light were permitted as long as the light source was not directed at the subject's eyes. The operator started the flickering light as 2900 flashes per minute (48.3 flashes per second) and reduced this speed at a constant rate until the subject reported flicker. After establishing a base line (3 identical rate times reported consecutively) the test drugs were given. Flicker fusion threshold was then measured every 15 to 30 minutes for the next 2 to 3 hours depending on the group. T-tests revealed that caffeine alone, despite being a central nervous system stimulant, depressed flicker fusion frequency.

King and Henry (1992) employed caffeine as a control in studying the effects of neuroleptic cognitive psychomotor function in healthy subjects. Critical flicker fusion threshold (CFFT) was used as one of the assessments. Twenty subjects were given single doses of caffeine (400 mg.) along with placebo and other drugs weekly in a double-blind,

randomized order. Volunteers were asked to abstain from caffeine for 12 hours before each test. CFFT was measured using the Leeds Psychomotor Tester and was reported as the mean of six runs, three with ascending and three with descending flicker frequency. Tests were repeated at 1, 2, 3, 6 and 24 hours. A principal component factor analysis revealed that caffeine (400 mg.) appeared to significantly impair CFFT at one hour ($p<0.05$).

Kelly and Wilson (1978) examined human flicker sensitivity exclusive from drug interactions from a mathematical perspective utilizing algebraic equations and progressions to address the question of whether flicker fusion discrimination was controlled by retinal mechanisms or higher visual centers. Results supported the hypothesis that flicker thresholds at high flicker frequencies are actually filtered by retinal units and then relayed to the rest of the visual system which could explain any delay or decrease in response time.

Kerr, Sherwood, and Hindmarch (1991) examined the effects of social drugs on psychomotor performance. One of the assessment techniques utilized in assessing the effects of caffeine was critical flicker fusion. Ten subjects participated in this study. Subjects with required to discriminate flicker fusion in a set of four light emitting diodes held in foveal fixation at 1 meter (almost 3 feet). Individual thresholds were determined by eliciting responses

on three ascending and three descending scales. Average daily caffeine intake was not reported nor were requirements for abstinence from caffeine prior to testing reported. The findings of a one way repeated measures analysis of variance (ANOVA) support caffeine (250 mg.) acting as a central nervous system stimulant independent of fatigue. In addition, there was no significant fluctuations in central nervous system arousal as measured by critical flicker fusion following drug ingestion. As a result, these researchers go on to postulate that caffeine may have a greater effect on information processing.

The effects of caffeine on repetition blindness is the third visual measurement that was addressed in the current study. Early studies exploring perceptual processing led to utilizing repetition blindness as one of the measurement techniques. Logsdon, Hochhaus, Williams, Rundell, and Maxwell (1984) examined the effects of secobarbital on perceptual processing (specifically choice reaction time). Secobarbital, a central nervous system depressant with sedative and hypnotic qualities, was found to adversely effect perceptual processing. Later, Marohn and Hochhaus (1988) utilized repetition blindness in accessing relative perceptual fluency and found that semantic priming (priming word paired with a related word) increases stimulus duration and repeated priming (priming word paired with itself) decreased stimulus duration. Semantic priming apparently

increased perceptual fluency. Further, Hochhaus and Mihura (1993) explored word frequency effects on repetition blindness and found that frequency effects interact with repetition blindness effects in a way that limits repetition blindness to either high frequency words or rare words made more familiar by pretraining.

No studies exist that examine caffeine's effect on repetition blindness. Examining the effects of caffeine (a stimulant) on repetition blindness would appear to be a logical step, however, since secobarbital (a depressant) negatively effected perception (Logsdon et al., 1984). If secobarbital negatively effects perception, caffeine could theoretically improve it.

Summary

The neurological effects of caffeine have been examined. At the cellular level, caffeine has been hypothesized to effect four areas: intracellular mobilization of calcium, inhibition of phosphodiesterase, antagonism of adenosine receptor sites, and interactions with benzodiazepine binding sites. Of these four potential effects, antagonism of adenosine receptor sites appears to be the theory most commonly accepted. From a more broad perspective, caffeine has been linked to increased release of neurotransmitters and a possible decrease in cerebral blood flow which have implications on substrate utilization in the central nervous

system. These plausible events, combined with the impulse conductivity that exists among the central, peripheral, autonomic nervous systems, may very well play an important role in regulating metabolism and ultimately behavior and performance.

Potential neuromuscular effects of caffeine include a decrease in the muscle fiber's excitation threshold, an increase in the length of contraction, and changes in substrate metabolism. These events seem to evolve from three possible mechanisms: increased affinity of myofilaments for calcium and/or increased calcium release from the sarcoplasmic reticulum; changes in cellular activity due to an increase in cAMP in skeletal muscle tissue; and inhibition of adenosine receptors sites in the central nervous system. This last theory, inhibition of adenosine receptors, appears to be the hypothesis most authorities prefer to explain the neuromuscular effects of caffeine. The propensity for making this choice is most understandable due to caffeine's reported inhibition of adenosine receptor sites and the relationship between the central and peripheral nervous systems.

The influence of caffeine on vision is examined from two perspectives: (1) effects on the structural components of the eye itself and; (2) from the perspective of visual performance. From a structural standpoint, caffeine has reportedly resulted in a decrease in retinal blood flow

while increasing diastolic pressure. However, caffeine had no clinically significant effect on intraocular pressure. Studies addressing caffeine's effects on visual performance examined various parameters and provided, in many cases, conflicting results. Caffeine's reported effects included both reductions and improvements of visual reaction time. Similarly, caffeine facilitated and impaired perceptual restructuring tasks. Certain parameters of selected visual processing were improved following caffeine ingestion as did visual tracking. However, decreases in response times were also reported. Studies examining caffeine's effect on visual vigilance resulted in both increases and decreases. And, the effect of caffeine on information processing of fatigued subjects were examined; reaction time decreased with results being effected by the fatigued state of the subject.

The current study was interested in the effects of caffeine on visual performance utilizing three test protocols: luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness. Previous studies testing luminescent threshold report that caffeine may reduce contrast threshold and, in some cases, erase enhancement effects. Studies examining the effect of caffeine on flicker fusion report conflicting results. Findings include a decrease in flicker fusion frequency (Roback et al., 1952) and an impairment of critical flicker fusion threshold (King

& Henry, 1992). One study (Kelly & Wilson, 1978) explains these decreases in flicker fusion frequency mathematically as occurring due to a delay in stimuli transfer from retinal units to higher visual centers. However, another study reports no significant fluctuations central nervous system arousal evidenced by a lack of a significant change in critical flicker fusion threshold (Kerr et al., 1991). As for the effects of caffeine on repetition blindness, no studies were found. However, examination of the effects of this stimulant on repetition blindness would appear called for in light of the findings of previous works examining perception.

Further caffeine research, particularly caffeine's effect on luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness, needs to be conducted. This call for further research is due to the uncertainty surrounding the results of many such studies. This fact, combined with conflicting outcomes and a lack of data, demands subsequent examination. From a practical viewpoint, additional information concerning the effect of caffeine on vision could prove beneficial for those occupations relying on sight (e.g., air traffic controllers, pilots, professional athletes, computer programmers, and data entry). Moreover, visual effects of caffeine could also impact the general population's recreational and leisure pursuits.

CHAPTER III

METHODOLOGY

Subjects

Fifteen college age students enrolled in the 1996 summer semester and fifteen college age students enrolled in the 1996 fall semester at Oklahoma State University were solicited for this study. These subjects represented a convenience sample based on availability.

Each subject voluntarily read, signed and dated an informed consent form approved by the Institutional Review Board at Oklahoma State University (Appendix A) which outlined the study's protocol, as well as, possible risks. This process occurred following a detailed verbal briefing outlining the scope of the study, purpose, procedures, and potential risks. In addition, the subject answered questions about their medical history, (Caffeine Research Questionnaire, Appendix B) describing physical conditions, medications, or disorders that may have proved problematic for the subject or hampered the study. This document (Caffeine Research Questionnaire, Appendix B) also included questions determining the subjects's caffeine consumption history and average daily intake. To inform participants,

as well as, facilitate the computation of caffeine intake, the subjects were also presented with a handout identifying the caffeine content of common beverages, foods, & medications (Appendix C).

The principal investigator verbally screened any subjects indicating elevated caffeine intake (>200 mg.) or preexisting physical conditions (e.g., heart problems, intestinal disorders, mental/emotional disorders, and high blood pressure) upon examining each subject's daily caffeine intake and medical history. Based on the responses given, subjects in question were either eliminated from the study or made fully aware of possible side-effects. Due to medical history findings (e.g., heart murmur and depression) and scheduling problems (e.g., test times conflicting with work or classes) seven of the thirty volunteers were either eliminated or were unable to participate in the study. Twenty-three subjects completed testing. All data, the informed consent form, medical history questionnaire, and all responses were held in strict confidence and filed with the principal investigator.

Preliminary Procedures

Testing was conducted in the Exercise Physiology Lab at Oklahoma State University. This location was chosen based on student accessibility, availability of adequate space, ability to provide semi-dark lighting, and relative freedom

from visual or auditory interference. On the test date, each subject was instructed to fast for five hours prior to testing. In the digestive process, food spends approximately 2 to 4 hours in the stomach (Clayman, 1989). By fasting for five hours, caffeine was absorbed more quickly and completely. Similarly, the subjects were directed to abstain from caffeine for forty-eight hours prior to testing to achieve a reduction in caffeine tolerance and to prevent any residual caffeine from interfering with the study.

The test site was semi-dark with partitions separating the three test stations. Upon arrival, the subjects were seated and asked to respond to five questions related to food and caffeine consumption and the subject's general state of well being (e.g., presence of a hangover, lack of sleep, time of last meal, and time/form of last caffeine consumed) (Vital Study Criteria on the Caffeine Research Questionnaire, Appendix B). Based on these responses, the subjects were given the opportunity to reschedule the test session. If rescheduling was not necessary, blood pressure and pulse readings were taken utilizing Lafayette model #UA-701 digitized blood pressure meter and recorded on the Caffeine Research Questionnaire (Appendix B). This data was utilized to compare pre-test and post-test blood pressure and pulse measurements following caffeine ingestion. These procedures were taken to monitor the subject's health status and identify any adverse physiological reactions to caffeine

at which time medical assistance would have been provided.

The subject's caffeine dosage was prepared by a local pharmacist based on the subject's weight as reported on the Caffeine Research Questionnaire (Appendix B). Two dosages of caffeine, 2.5 mg. per kg. bwt. and 5.0 mg. per kg. bwt., and one placebo was prepared for each subject. The caffeine, which was administered in capsule form, was Caffeine, USP, Anhydrous. Similarly, gelatin capsules filled with Sodium Bicarbonate represented the placebo.

Equipment and Testing Procedure

All tests, pre and post-tests, were repeated on three separate occasions during a three week period until all subjects had been administered the three doses of caffeine. Pre-tests and post-tests began with the investigator reading the subject instructions related to each of the three visual tests (Appendix F). Pre-tests were followed by administration of one form of the treatment (placebo, 2.5 mg./kg. bwt., or 5.0 mg./kg. bwt. of caffeine) with 118 ml. of tap water. After completion of the pre-tests, subjects were then asked to maintain their fast and refrain from additional caffeine consumption. After waiting at least one hour, but not more that three hours (caffeine's half-life), the post-tests were conducted following the same protocol described above. This weekly administration of placebo or caffeine was conducted randomly; neither the subjects nor

the technicians administering the tests were aware of the dosage. Since each subject was administered one of the three treatments (placebo, 2.5 mg./kg. bwt. caffeine, or 5.0 mg./kg. caffeine) per week for a three week period, subjects served as their own control. Test results were recorded after each testing session (both pre and post) on a data sheet (Appendix E).

The tests (pre and post) involved three modalities: luminescence threshold comparisons, flicker fusion discrimination, and repetition blindness. Luminescence threshold comparisons were measured by Lafayette model #14011 (1701), Light Discrimination Apparatus. According to Lafayette (1970), this model was designed to present two 1-3/8 in. light stimuli from a single common source, however, each stimuli could be independently varied in intensity by means of a precisely tapered aperture and finely calibrated scales on each side of the unit. The stimulus lamp was a 15 watts and 115 volts with a single contact candelabra base. Due to uncontrolled variables such as fluctuations in voltage, the absolute value of the stimulus could not be determined (Lafayette, 1970). However, relative differences between the two stimuli were reported to be highly reliable since a common light source was employed (Lafayette, 1970).

Subjects were instructed to sit directly in front of the Light Discrimination Apparatus. The distance between the unit and the subject was the subject's arm distance to

allow for adjustment of the dominant hand's dial. Three trials were conducted during each test session with the subject trying to reproduce a predetermined light stimulus with their dominant hand. The standard that the subjects attempted to reproduce included: 140 units, 290 units, and 220 units.

Flicker fusion discrimination was measured by Lafayette model #58017, Visual Perception Control, which was designed specifically for this visual test. However, Lafayette (1986) reported that the accuracy for this piece of equipment was "greater that 10%" and repeatability was "greater than 3%". Again, as with the Light Discrimination Apparatus, uncontrolled factors such as voltage fluctuation possibly served as a confounding variable (Lafayette, 1986).

Subjects were seated directly in front of the apparatus and asked to focus their attention on the two flickering lights. Each test (pre and post) consisted of three trials with the researcher starting the test at 10 flickers per second followed by a gradual increase to a maximum of 50 flickers per second. The subjects verbally indicated to the investigator the point when the flickering lights became one continuous light.

The final test measured repetition blindness. This test was conducted by using a computerized program created to measure repetition blindness by Dr. Larry Hochhaus, professor of Psychology, Oklahoma State University. Each

subject completed 40 trials to allow for the accurate depiction of repetition blindness while attempting to avoid the confounding effects of fatigue. The first eight trials were considered practice with the remaining thirty-two trials being recorded as correct and incorrect. Each trial consisted of the subject viewing a "prime" word for 300 milliseconds (ms.) displayed two lines above a pair of arrows (---> <---) which marked the location of the "target" word. The "target" was then viewed for 17 ms. The prime word was a capitalized four letter word while the target word could be either the prime word repeated or a different capitalized four letter word. The target word was immediately replaced by a 500 ms. row of six ampersands (&&&&&&). The subject was directed to repeat the target word to enable the investigator to record each response on a weekly pre/post test repetition blindness response sheet (Appendix D).

Post Procedure

Subjects were encouraged to consume a meal following the post-test. Foods high in carbohydrates and low in fats/grease were suggested to decrease the potential for stomach upset and nausea. Subjects were also directed and encouraged to inform the principal investigator of any ill feelings following the tests. Each testing session lasted approximately 1.5 hours. Therefore, the total time necessary to complete all sessions was approximately 4.5 hours.

Statistical Treatment

This study utilized a within subject research design. Caffeine doses (2.5 mg./kg. bwt. or 5.0 mg./kg. bwt.) or placebo were administered randomly to all subjects using a double-blind format. Each subject served as their own control through implementation of a pre and post-test protocol.

The data was analyzed using 2 x 3 repeated measures analyses of variance (ANOVAs) to test for significance between pre-test and post-test responses on the three visual tests (luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness). In analyzing flicker fusion discrimination and repetition blindness, difference between pre and post-test responses represented the dependent variable. In both cases, three levels of caffeine (0, 2.5, and 5.0 mg./kg. bwt.) and time of testing (pre and post) served as independent variables. In making luminescent threshold comparisons, three 2 x 3 repeated measures ANOVAs were utilized to determine significance between pre-test and post-test scores at the three different light intensity levels (140, 290 and 220 units). The difference between pre and post-test scores at these three light intensity levels represented the dependent variable in each ANOVA. And, as with flicker fusion and repetition blindness, caffeine (3 levels) and time of testing (pre and post) served as the independent variables. Newman-Keuls post hoc

analyses were conducted based on the significant findings reported by the repeated measures ANOVAs.

CHAPTER IV

RESULTS AND DISCUSSION

Results

This investigation examined the effects of three treatment levels of caffeine on three visual tests. Participant characteristics were initially analyzed utilizing descriptive statistics. The descriptive statistics included: age, gender, weight, height, and average daily caffeine consumption.

The mean age of the subjects was 23.39 (± 5.31) years. The standard error (or standard deviation of the sampling distribution of means) of ages was 1.12 years. Since most of the scores were clustered at the lower end of the distribution, the distribution could be described as being positively skewed with skewness reported at 2.70 (Table II).

This study collected data from 23 subjects. The subject pool was comprised of 14 women and 9 men (Table III).

The subjects' mean weight was 159.91 (± 50.19) pounds. Standard error of the reported weights was 10.46 pounds. A greater number of subjects' weights were located at the lower end of the range (positively skewed) with skewness reported at 1.09 (Table IV).

TABLE II

FREQUENCY DISTRIBUTION OF SUBJECTS' AGES

Years	Frequency	Percent
19	3	13.0
20	3	13.0
21	2	8.7
22	6	26.1
23	3	13.0
24	2	8.7
27	2	8.7
33	1	4.3
43	1	4.3
Total	23	100.0

Mean	23.39	Std. Error	1.11
Variance	28.20	Skewness	2.70

TABLE III

FREQUENCY DISTRIBUTION OF SUBJECTS' GENDERS

Gender	Frequency	Percent
Male	9	39.1
Female	14	60.9
Total	23	100.0

Mean	1.61	Std. Error	.10
Variance	.25	Skewness	-.48

TABLE IV

FREQUENCY DISTRIBUTION OF SUBJECTS' WEIGHTS

Pounds	Frequency	Percent
105	1	4.3
108	1	4.3
112	2	8.7
122	1	4.3
124	1	4.3
125	2	8.7
130	1	4.3
135	1	4.3
140	2	8.7
151	1	4.3
156	1	4.3
160	1	4.3
163	1	4.3
165	1	4.3
205	1	4.3
215	2	8.7
235	1	4.3
250	1	4.3
285	1	4.3
Total	23	100.0

Mean	159.91	Std. Error	10.46
Variance	2519.04	Skewness	1.10

The mean height of the subjects was 170.12 (± 9.49) centimeters. The standard error reported was 1.98 centimeters. Height was more evenly dispersed in the subject pool when compared to the other descriptive data. However, the distribution was slightly positively skewed (.09) since more of the scores were located toward the lower end of the range (Table V).

Mean caffeine consumption per day for this group of subjects was 130.8 (± 96.12) milligrams (approximately 1 cup of coffee or 2 sodas). The standard error was reported at 20.05 milligrams which represented the computed difference between caffeine actually consumed verses caffeine reported. The scores were positively skewed since most of the students had low (below the 200 mg. average intake per day) caffeine intake (Table VI).

This investigation involved examining the effects of three treatment levels of caffeine on three separate tests of vision. Five hypotheses were tested to determine if there were significant differences in visual performance on three visual tests (luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness) following ingestion of three doses of caffeine (0, 2.5, and 5.0 mg./kg. bwt.). Five 2 x 3 repeated measures analysis of variance (ANOVA) were used to analyze the treatment effect of the three levels of caffeine on the three visual tests. Newman-Keuls post hoc analyses were conducted on significant

TABLE V

FREQUENCY DISTRIBUTION OF SUBJECTS' HEIGHTS

Inches	Frequency	Percent
60	1	4.3
62	2	8.7
63	1	4.3
64	3	13.0
65	1	4.3
66	4	17.4
67	1	4.3
68	2	8.7
69	2	8.7
71	3	13.0
72	2	8.7
74	1	4.3
Total	23	100.0

Mean	66.96	Std. Error	.78
Variance	13.99	Skewness	.09

TABLE VI

FREQUENCY DISTRIBUTION OF SUBJECTS' DAILY CAFFEINE CONSUMPTION

Mg.	Frequency	Percent
.0	2	8.7
41.6	4	17.4
62.4	2	8.7
83.2	1	4.3
91.2	1	4.3
101.6	1	4.3
107.2	1	4.3
124.8	1	4.3
130.0	1	4.3
145.6	1	4.3
177.4	1	4.3
198.2	1	4.3
198.4	1	4.3
208.0	1	4.3
223.2	1	4.3
293.2	1	4.3
300.0	1	4.3
335.2	1	4.3
Total	23	100.0

Mean	130.80	Std. Error	20.05
Variance	9248.67	Skewness	.64

ANOVA results.

Hypothesis I stated that the pre to post-test difference score for luminescent threshold comparisons would remain consistent across the three treatment levels (placebo, 2.5 mg./kg. bwt. caffeine, and 5.0 mg./kg. bwt. caffeine) at the "low" luminescence setting (140 units). The repeated measures ANOVA revealed significant findings ($F = 3.619$; $p < .05$) (Appendix G). This hypothesis was rejected. There were differences in responses to "low" luminescence at the various caffeine dosages. A Newman-Keuls post hoc analysis ($p < .05$) yielded significant differences across the 2.5 mg./kg. bwt. and 5.0 mg./kg. bwt. caffeine dosages at "low" light intensity (Figure 2). See means and probabilities for the Newman-Keuls post hoc analysis in Appendix H.

Hypothesis II stated that the pre to post-test difference score for luminescent threshold comparisons would remain consistent across the three treatment levels (placebo, 2.5 mg./kg. bwt. caffeine, and 5.0 mg./kg. bwt. caffeine) at "moderate" luminescence (220 units). The repeated measures ANOVA revealed a statistically significant difference ($F = 4.362$; $p < .05$) in pre-test and post-test performance under different caffeine dosages (Appendix G). The null hypothesis was rejected. Further examination through the use of the Newman-Keuls post hoc analysis revealed that the difference was significant ($p < .05$) in responses at moderate luminescence across 2.5 mg./kg. bwt. and 5.0

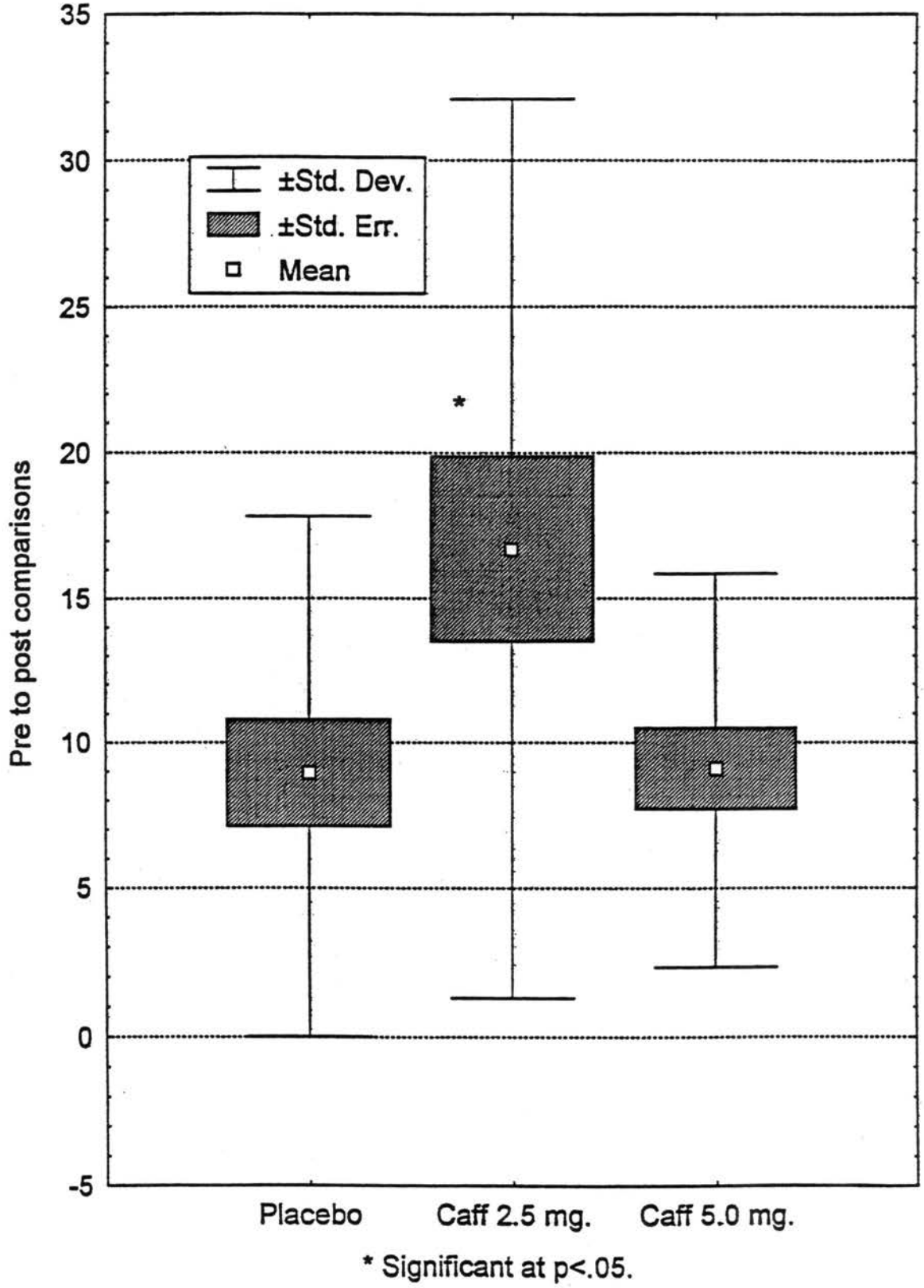


Figure 2. Group comparisons at low luminescence.

mg./kg. bwt. of caffeine and across the placebo and 5.0 mg./kg. bwt. of caffeine (Figure 3 and Appendix H).

Hypothesis III stated that the pre to post-test difference score for luminescent threshold comparisons would remain consistent across the three treatment levels (placebo, 2.5 mg./kg. bwt. caffeine, and 5.0 mg./kg. bwt. caffeine) at the "high" luminescence setting (290 units). The 2 x 3 repeated measures ANOVA conducted on data at the "high" luminescence setting (290 units) revealed no significant differences ($F = 1.864$; $p > .05$) (Figure 4, Appendix G, and Appendix H). The null hypothesis was not rejected. With luminescent threshold at the "high" setting, there appeared to be no evidence of pre to post-test pattern differences among the treatment levels.

Hypothesis IV stated that there would be no difference between pre-test and post-test flicker fusion responses after consumption of placebo, 2.5 mg./kg. bwt., and 5.0 mg./kg. bwt. caffeine. The repeated measures ANOVA revealed that there was a significant effect ($F = 7.236$; $p < .05$) (Appendix G). This hypothesis was rejected. Further application of a Newman-Keuls test of this specific hypothesis revealed a significant effect ($p < .05$) only between the pre and post-test at 2.5 mg./kg. bwt. caffeine (Figure 5 and Appendix H).

Hypothesis V stated that there would be no difference between pre-test and post-test repetition blindness respons-

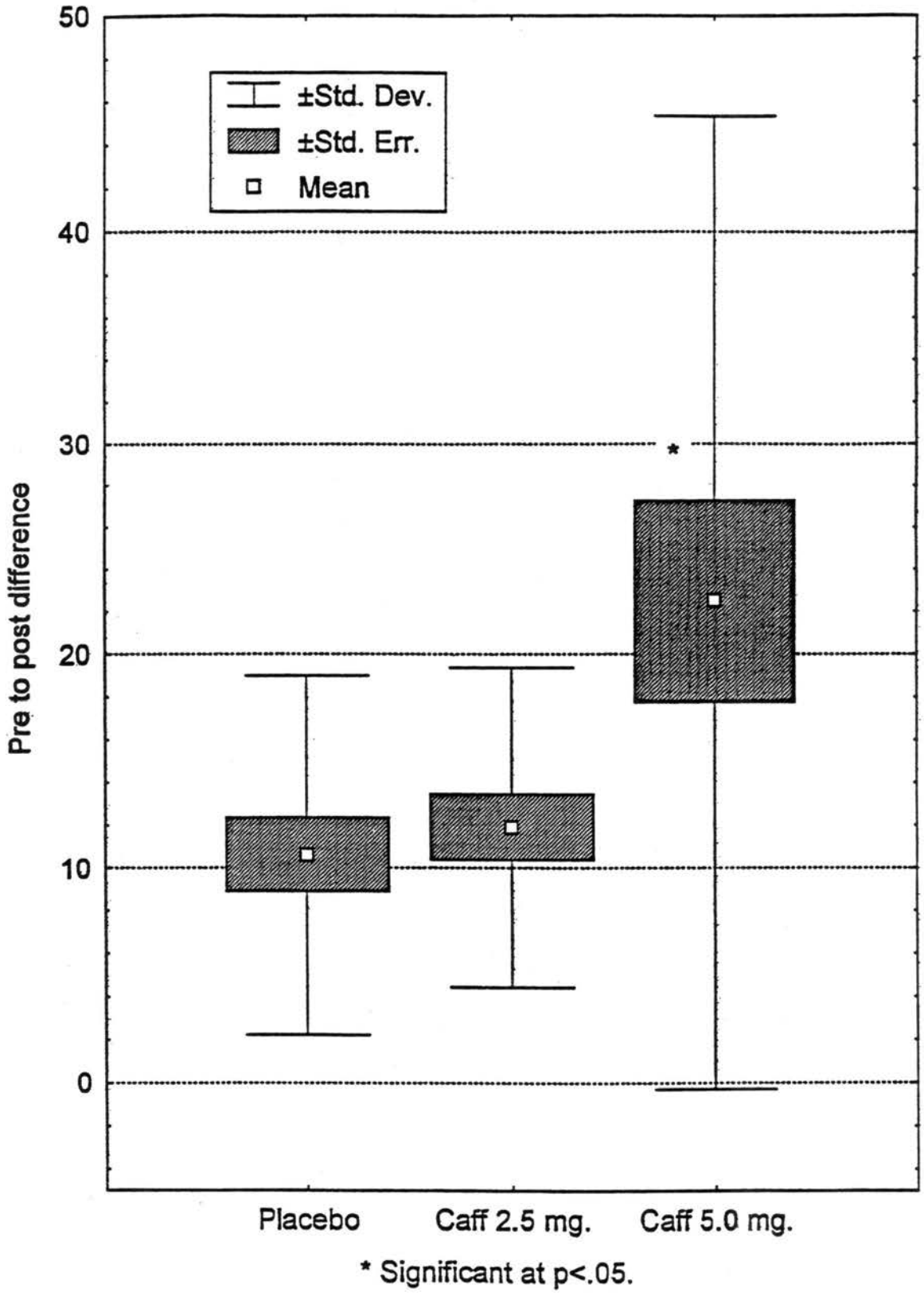


Figure 3. Group comparisons at moderate luminescence.

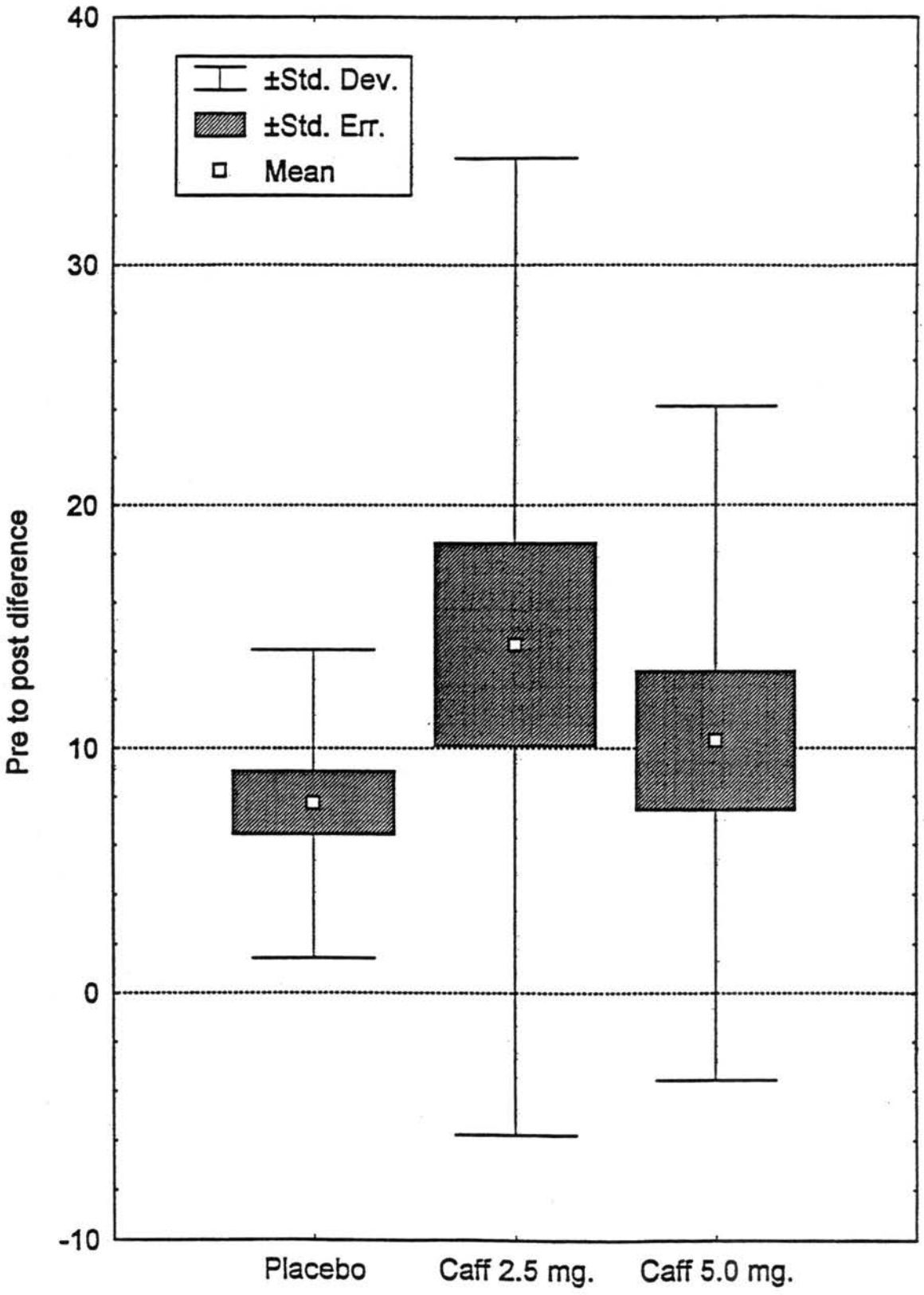
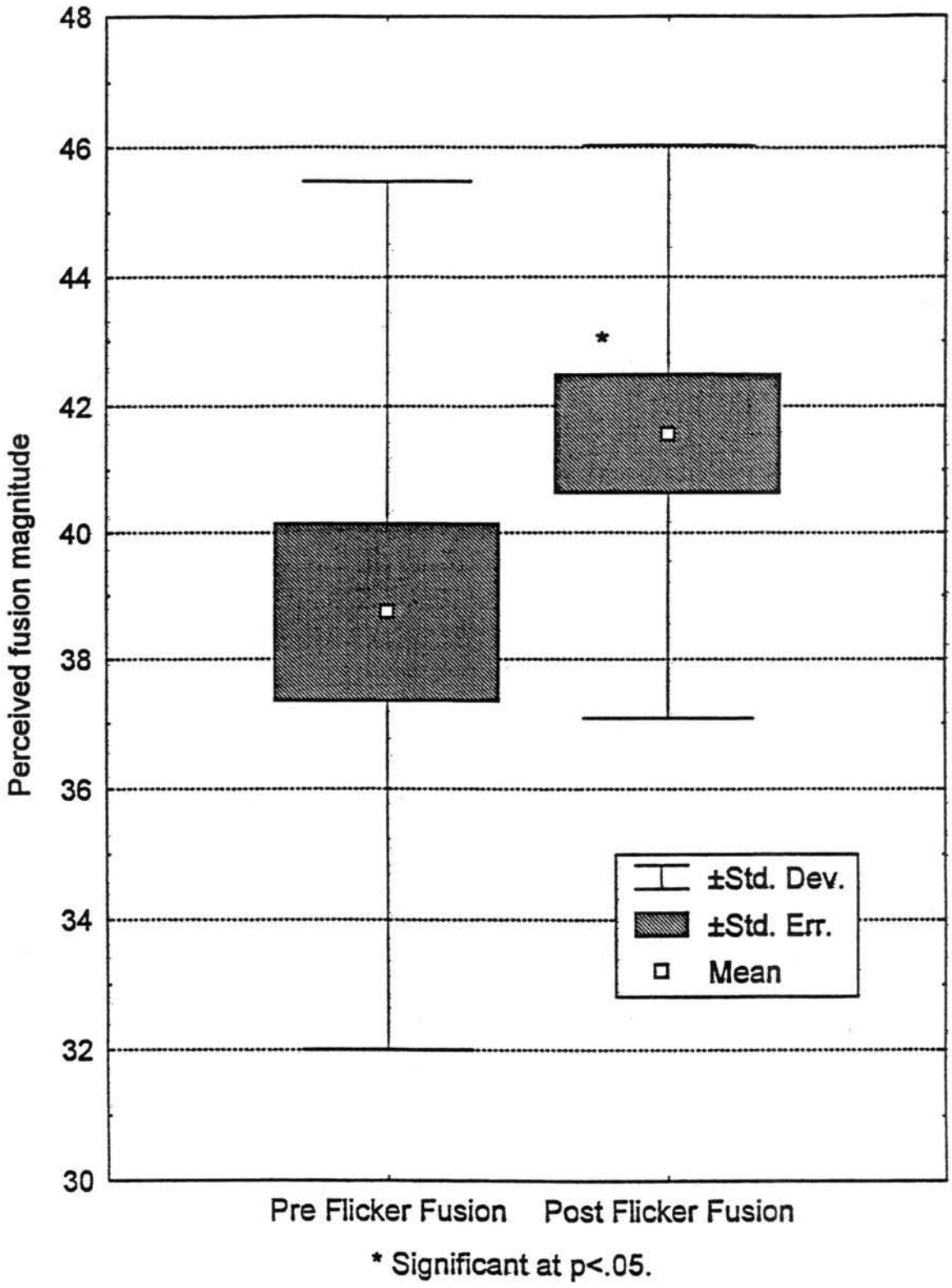


Figure 4. Group comparisons at high luminescence.



* Significant at p<.05.

Figure 5. Group flicker fusion comparisons at 2.5 mg. caffeine.

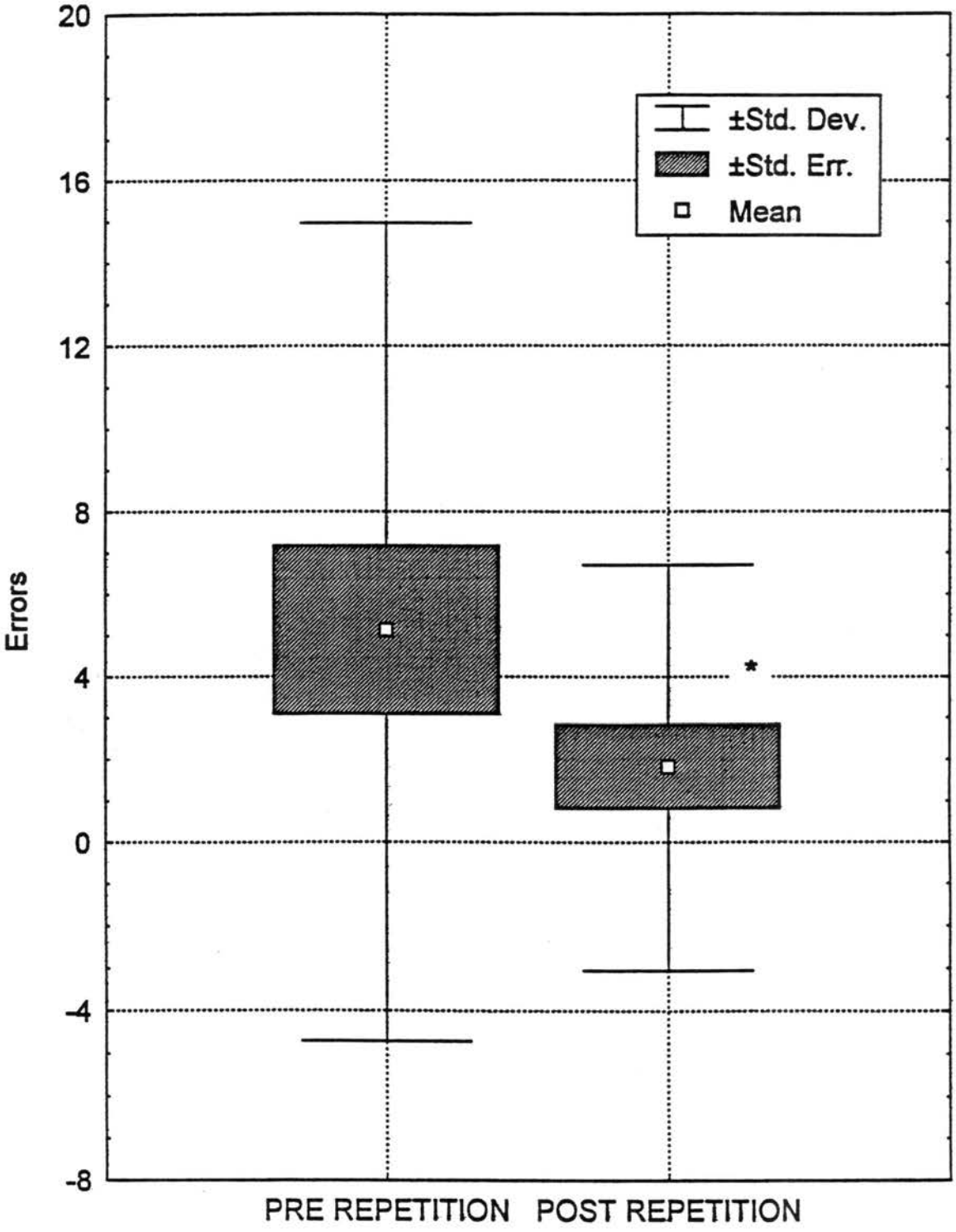
es after consumption of placebo, 2.5 mg./kg. bwt., and 5.0 mg./kg. bwt. caffeine. This hypothesis was also rejected. Statistical significance was provided by the repeated measures ANOVA ($F = 3.469$; $p < .05$) (Appendix G.) Further Newman-Keuls test revealed significance ($p < .05$) in repetition blindness scores only between the pre and post-tests at the 2.5 mg./kg. bwt. caffeine dose level (Figure 6 and Appendix H).

Discussion of Results

This study examined the effects of three doses of caffeine on three visual tests. The findings of the analyses will be discussed to clarify results. The current study will also be interpreted with respect to previous studies in order to identify additions to the existing body of knowledge, generate ideas for future research in this field, and suggest adaptations in protocol for further studies.

Analytical Findings

Repeated measures analyses of variance (ANOVAs) were the statistical analyses utilized in this study. This type of analysis is commonly used by researchers examining caffeine's effects on various physical parameters (Jacobson & Edgley, 1987; Jacobson & Thurman-Lacey, 1992; Jacobson, Webster, Claypool, & Hunt, 1992; Jacobson & Winter-Roberts, 1991; Kerr et al., 1991). Five 2 x 3 repeated measures



* Significant at p<.05.

Figure 6. Group repetition blindness comparisons at 2.5 mg. caffeine.

ANOVAs were conducted to examine the effects of three dosages of caffeine (0 mg./kg. bwt.; 2.5 mg./kg. bwt.; and 5.0 mg./kg. bwt.) on three visual tests (luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness). Of these five ANOVAs, only one (luminescent threshold comparisons at high luminescence) did not result in significance. Newman-Keuls post hoc analyses were conducted on the four significant ANOVA results. (ANOVA summary tables are located in Appendix G and Newman-Keuls results are found in Appendix H.)

Luminescent Threshold Comparisons. Post hoc analysis of low luminescence data revealed significance between the subject's pre and post-test gain score responses at the 2.5 mg./kg. bwt. and 5.0 mg./kg. bwt. caffeine doses. At this low light intensity, luminescent threshold comparisons varied a significant amount when mean responses at 2.5 mg./kg. bwt. caffeine (mean = 16.70 $\pm$ 15.39) were compared to mean responses at the 5.0 mg./kg. bwt. caffeine dose (mean = 9.09 $\pm$ 6.76). More divergent scores occurred after ingestion of 2.5 mg./kg. bwt. caffeine compared to the 5.0 mg./kg. bwt. dose. Similarly, a Newman-Keuls post hoc analysis was conducted on moderate luminescence data and two areas of significance were reported. Significance was found across the 2.5 and 5.0 mg./kg. caffeine doses and across the placebo and the 5.0 mg./kg. bwt. dose of caffeine. Luminescent threshold comparisons varied significantly at moderate

luminescence in response to 5.0 mg./kg. bwt. caffeine (mean = 22.52 ± 22.83) compared to both the 2.5 mg./kg. bwt. dose (mean = 11.91 ± 7.47) and placebo (mean = 10.61 ± 8.39). More divergent pre to post-test scores occurred following ingestion of 5.0 mg./kg. bwt. caffeine dose compared to those reported following ingestion of placebo or the 2.5 mg./kg. bwt. dose. The repeated measures ANOVA at high luminescence reported no significant effects between the treatments and vision.

Flicker Fusion Discrimination. The Newman-Keuls post hoc analysis indicated significance between pre-tests and post-tests at 2.5 mg./kg. bwt. caffeine for flicker fusion discrimination. Flicker fusion was significantly postponed after consumption of 2.5 mg./kg. bwt. caffeine (pre-test mean = 38.74 ± 6.74; post-test mean = 41.56 ± 4.47). Subjects were able to discriminate between flicker and fusion of the stimuli at greater flickers per second.

No significant findings between pre and post test responses at the other two levels of caffeine were reported by the post hoc analysis for flicker fusion discrimination. No significance was identified between pre and post-test responses after administration of the placebo. These findings were anticipated since no caffeine was administered. However, no significance was also reported between pre and post-test responses after consuming the 5.0 mg./kg. caffeine dose. Since flicker fusion was postponed with the

2.5 mg./kg. bwt. dose, further delay of flicker fusion would have conceivably been expected after consuming the higher caffeine dose.

Repetition Blindness. Improvement in performance was described when the final post hoc analysis revealed repetition blindness decreased from pre to post-test following 2.5 mg./kg. bwt. caffeine ingestion (pre-test mean = 5.13 $\pm$ 9.84; post-test mean = 1.83 $\pm$ 4.88). Subjects were able to identify the target word more frequently following consumption of 2.5 mg./kg. bwt. of caffeine compared to pre-test results.

No significant findings were identified by the Newman-Keuls post hoc analysis conducted on pre to post-test repetition blindness responses after consumption of the placebo or the 5.0 mg./kg. bwt. caffeine dose. As with flicker fusion discrimination, the lack of significant findings was expected after administration of the placebo. No stimulant (caffeine) was ingested. However, it was quite surprising that the 5.0 mg./kg. bwt. dose did not further decrease repetition blindness thereby increasing performance an even greater degree compared to the effects of the 2.5 mg./kg. bwt. dose.

Summary. Dose related responses appear to be a common theme throughout these findings. In the luminescent threshold comparisons, significant differences in pre to post-test

scores were identified across the 2.5 mg./kg. bwt. caffeine dose and the 5.0 mg./kg. bwt. dose at low luminescence. The pre to post-test differences at 2.5 mg./kg. bwt. caffeine varied to a greater degree than the results at the higher dose (5.0 mg./kg. bwt.). Conversely, at moderate luminescence, the difference in test scores at 5.0 mg./kg. bwt. caffeine varied more than both the 2.5 mg./kg. bwt. caffeine and placebo scores. And for flicker fusion comparisons and repetition blindness, the 2.5 mg./kg. bwt. caffeine dose produced a greater divergence in performance compared to both the placebo and 5.0 mg./kg. bwt. dose. These results tend to imply dose related responses (Eveden et al., 1993; Jacobson & Edgley, 1987; Jacobson & Thurman-Lacey, 1992; Jacobson, Winter-Roberts, & Gemmell, 1991; Loke & Meliska, 1984; Fine et al., 1994).

The one exception to the idea that caffeine's effects are dose related is that no significant differences between pre and post-test scores were reported in luminescent threshold comparisons at high luminescence. However, another pattern appears to have developed between caffeine and the various light intensities. The eye seems to require higher levels of caffeine to produce significant responses to the stimulus as the stimulus increases in intensity. At low luminescence, the 2.5 mg./kg. bwt. caffeine dose produced greater fluctuations in pre to post-test scores. Significant fluctuations between scores also occurred at

moderate luminescence; however, a higher caffeine dosage (5.0 mg./kg. bwt.) was required. No significance was reported at high luminescence. If this pattern was to continue, a higher level of caffeine (7.5 mg./kg. bwt.) could elicit significant results.

The discussion of results is somewhat complicated due to differences in the caffeine status of the subjects. The average caffeine intake reported by subjects in this study ranged from 0 mg. to 335.2 mg. per day with 130.8 mg. representing the subjects' mean daily intake (Table VI). Despite the fact that subjects were instructed to abstain from food (for five hours) and caffeine (for forty-eight hours) prior to testing in an attempt to diminish individual differences, caffeine tolerance could have affected the subjects with higher daily caffeine intake (5 subjects consumed >200 mg./day) and may have altered or masked results (Sawynok, 1995). On the other hand, the five subjects with higher daily intake who did follow directions and abstained from caffeine could have experienced caffeine withdrawal which may have adversely affected performance (Brecher, 1972). Even though the majority (18 subjects) consumed less than the average daily intake of 200 mg. and 10 subjects could be classified as caffeine naive (<100 mg.), the results may be slightly skewed due to the above average caffeine intake of a few of the subjects.

Comparison of the Current Study with Previous Studies

The current study was conducted to contribute to the existing body of knowledge that exists concerning the effects of caffeine on vision. To make this contribution, this study must be compared to previous studies. However, differences in statistical analyses and experimental protocols complicate these comparisons. To simplify matters, the current study's protocol and statistical analyses examining caffeine's effects on three visual tests (luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness) will be compared to those of previous research. In addition, current findings will be compared and contrasted to those of previous studies.

The current study's protocol required twenty-three subjects to abstain from caffeine for forty-eight hours and food for five hours. Each test session (both pre and post-tests) began in a semi-dark room with the subjects completing a pre-test screening followed by three visual tests. Luminescent threshold comparisons and flicker fusion discrimination were conducted separately by utilizing two apparatus designed by Lafayette. Repetition blindness was examined through the use of a computerized program. Following the pre-tests, either one dose of caffeine (2.5 mg/kg. bwt. or 5.0 mg./kg. bwt.) or the placebo was administered to the subjects. The post-test occurred at least one hour after treatment. This process was repeated on three sepa-

rate occasions during a three week period until all subjects had been administered the three doses of caffeine. Data was analyzed using 2 x 3 repeated measures ANOVAs with significant findings being further analyzed by Newman-Keuls post hoc analyses.

Luminescent Threshold Comparisons. Luminescent threshold comparisons have previously been examined using various apparatus, protocols, and statistical analyses. Kleman et al. (1961) examined the effects of caffeine on "foveal simultaneous contrast" by utilizing a small circular test field surrounded by an inducing annulus. The test apparatus separated the right and left visual pathways. A different visual pattern and a separate light source (100 w.) was provided for each eye. The study was conducted utilizing three subjects (low to moderate caffeine users) who were instructed to abstain from caffeine for 24 hours. Testing was conducted on two separate days. On each day, subjects were first administered a placebo and tested approximately 30 minutes later. This process was then repeated with 3 grains (194.4 mg.) of caffeine serving as the treatment. Results were reported in log millilamberts (log mL) as a function of the inducing field luminance and plotted as a function of the log luminance.

Diamond and Cole (1970) examined visual threshold as a function of test area and caffeine administration. Threshold luminance of a circle was measured in relation to its'

area before and after ingestion of 3 different doses of caffeine (placebo, 1.5 grains or 97.2 mg., and 3.0 grains or 194.4 mg.). The test apparatus consisted of a light path (150 w.) to the subject's right eye. Three subjects were solicited for this study without considering average daily caffeine intake. Each test session began with the placebo being administered to the subjects. After approximately 30 minutes, threshold measurements were taken for 10 test-field areas. This procedure was repeated in one hour with one of the caffeine doses (randomly administered) instead of the placebo. The remaining dose of caffeine was administered when this protocol was repeated a third time. Threshold luminance as a function of test radius was reported in log mL and threshold (in log mL) was plotted representing the respective caffeine doses.

Comparing these research studies designed to measure luminescent threshold with the current study is quite difficult due to inherent differences. The differences between the previous studies and the current study include: statistical analyses, test apparatus (e.g., monocular vs. binocular, distance of subjects from apparatus, and intensity of light source), amount of caffeine administered (97.2 mg. and 194.4 mg. vs. 2.5 mg./kg. bwt. and 5.0 mg./kg. bwt.), number of subjects, time between caffeine ingestion and post-test, and pre-test caffeine status of subjects. Despite these differences, however, there are some similarities between

the previous studies and the current study. Klemen et al. (1961) tested subjects that were classified as moderate to low caffeine users which is similar to the current study. And, Diamond and Cole (1970) administered a placebo, 1.5 grains (97.2 mg.) of caffeine, and 3.0 grains (194.4 mg.) of caffeine to their subjects. (Even though there were three levels of treatment, the dosages were generally below the amount of caffeine administered in the current study).

The previous studies found that caffeine did effect the subject's ability to discriminate between light intensity levels despite differences in apparatus and protocols. In the case of Diamond and Cole (1970), the overall decrease in luminescence grew more pronounced as the caffeine dosage increased from 1.5 grains (97.2 mg.) to 3.0 grains (194.4 mg.). These findings hint of a dose related response although a blanket dose of caffeine does not accommodate differences in the weights of subjects compared to caffeine administered according to body weight. One problem these studies have when applying currently held beliefs about caffeine is that both studies tested luminescent threshold less than one hour after caffeine ingestion. As reported by Axelrod & Riechenthal (1953), caffeine levels peak in the blood stream approximately one hour after ingestion. Both studies tested luminescent threshold before maximum caffeine levels were achieved in the subject's bloodstreams.

The present study's results do parallel previous find-

ings at low luminescence with luminescent threshold comparisons varying to a greater degree following ingestion of 2.5 mg./kg. bwt. of caffeine compared to less variation at the 5.0 mg./kg. dose. The higher dose (5.0 mg./kg. bwt.), at moderate luminescence, resulted in greater pre to post-test score variation compared to comparisons made at the lower dose (2.5 mg./kg. bwt.) or the placebo. However, unlike previous studies, no significance was found at high luminescence.

Caution should be used when comparing the current results with those of previous studies due to differences that include caffeine dosage, light intensity, and failure to test one hour after caffeine ingestion. However, the current low and moderate luminescence results do seem to infer, as did Diamond and Cole (1970), that luminescent threshold has a dose related response to caffeine when tested at different intensities. There was a significant variation in luminescent threshold comparisons at low luminescence at 2.5 mg./kg. bwt. caffeine compared to 5.0 mg./-kg. bwt. caffeine in the current study. Similarly, at moderate luminescence, significant variations in luminescent threshold comparisons were also reported. However, at moderate luminescence, comparisons of pre to post-test differences varied to a greater degree after ingestion of 5.0 mg./kg. bwt. caffeine when compared to placebo or 2.5 mg./kg. bwt. caffeine. Depending on luminescence (low or

moderate), the caffeine dose seems to have significant effects.

The lack of significance at high luminescence was somewhat unexpected considering the results of the low and moderate luminescence tests. Higher intensities of light may deter the effects of the two doses of caffeine utilized in this study or the eye's sensitivity to light may be more pronounced in response to such an intense stimuli (e.g., the eye may be more sensitive to light at high luminescence preventing caffeine from eliciting a change in luminescent threshold comparisons). In either case, if the current trend was to continue, a higher caffeine dose (e.g., 7.5 mg./kg. bwt.) might elicit a wider range of pre to post-test response differences at high luminescence.

Flicker Fusion Discrimination. Studies examining flicker fusion discrimination have varied over the years with respect to test apparatus, protocol, statistical analyses, and findings. In the current study, one of the three dosages of caffeine significantly affected flicker fusion. (The pre-test/post-test mean difference was significant at the 2.5 mg./kg. bwt. caffeine dose.) However, when comparing this study with earlier studies, the latter varied with respect to apparatus and protocol in addition to reporting conflicting results.

Roback et al. (1952) examined the effects of caffeine on flicker fusion threshold utilizing a "flicker photome-

ter". In this study, caffeine was used as a control. Five experimental groups, each comprised of 10 to 30 subjects, received caffeine (30 mg.) alone or in combination with other drugs. After establishing a base line or norm, the drugs (caffeine) was administered. Flicker fusion threshold was then measured every 15 to 30 minutes for the next 2 to 3 hours depending on the group. T-tests revealed that caffeine alone, despite being a central nervous system stimulant, depressed flicker fusion frequency.

King and Henry (1992) also employed caffeine as a control in studying the effects of neuroleptics on cognitive psychomotor function in healthy subjects. Critical flicker fusion threshold (CFFT) was used as one of the assessments. Twenty subjects were given single doses of caffeine (400 mg.), along with other drugs, weekly in a randomized order. Subjects were asked to abstain from caffeine for 12 hours before each test. CFFT was measured using the Leeds Psychomotor Tester. Tests were repeated at 1, 2, 3, 6, and 24 hour intervals. A principal component factor analysis revealed that caffeine (400 mg.) appeared to significantly impair CFFT at one hour ($p<0.05$).

Kerr et al. (1991) examined the effects of social drugs on psychomotor performance. One of the assessment techniques utilized in examining the effects of caffeine was critical flicker fusion. Ten subjects participated in this study. Subjects were required to discriminate flicker

fusion in a set of four light emitting diodes held in foveal fixation at 1 m. (almost 3 ft.). Average daily caffeine intake was not reported nor were requirements for abstinence from caffeine prior to testing reported. The findings of a one way repeated measures ANOVA support caffeine (250 mg.) acting as a central nervous system stimulant independent of fatigue. In addition, there was no significant fluctuations in central nervous system arousal as measured by critical flicker fusion following drug ingestion.

The results of the current study conflict with the findings of the three previous studies. Considering the current ANOVA and post hoc results, the 2.5 mg./kg. bwt. dose of caffeine increased flicker fusion discrimination between the pre and post-test which is contrary to the studies just cited. As with luminescent threshold comparisons, differences between the studies make it difficult to compare the current study with these three studies.

The study conducted by Kerr et al. (1991) was the only one of the three previous studies to use a repeated measures ANOVA to analyze data. All three of the apparatus in the previous studies varied from each other and from the apparatus used in the current study. Likewise, the time of testing following caffeine ingestion also varied. Roback et al. (1952) measured flicker fusion threshold every 15 to 30 minutes for 2 to 3 hours after caffeine ingestion. King and Henry (1992) determined critical flicker fusion threshold 1,

2, 3, 6 and 24 hours after caffeine ingestion. And, Kerr et al. (1991) examined subjects half an hour after caffeine ingestion. Only the studies conducted by Roback et al. (1952) and King and Henry (1992) allowed enough time after ingestion for peak caffeine levels to be reached in subject's blood plasma since caffeine reaches peak plasma levels approximately one hour after ingestion (Axelrod & Riechenthal (1953). Conversely, Chou (1992) reported caffeine's half-life (time required for the drug to be eliminated) to range from 2 to 12 hours with an average half-life of 4 to 6 hours. Therefore, the results reported by all three of the previous studies could possibly be misrepresented due to the effects of time on caffeine. Lower doses comparable to 2.5 mg./kg. bwt. were not tested. And, in the studies with comparable caffeine intake to the 5.0 mg./kg. bwt. dose, repeated measurements were taken hours after ingestion which collectively could have skewed the test results.

The dosage of caffeine ingested is another difference among the previous studies and the current study that could play a significant role in interpreting these results. In the studies previously cited (Roback et al., (1952); King & Henry (1992); and Kerr et al., 1991), researchers administered blanket doses of caffeine to subjects. Roback et al. (1952) administered a single dose of 30 mg. of caffeine. King and Henry (1992) administered 400 mg. while Kerr et al.

(1991) administered 250 mg. to each subject. In a 150 pound subject (68.18 kg.), a 2.5 mg./kg. bwt. dose of caffeine is equal to 170.5 mg. and a 5.0 mg./kg. bwt. dose is equal to 340.9 mg. of caffeine. Although the dose administered by Roback et al. (1952) represents a vary low caffeine dose, both the blanket doses of the latter two studies (Kerr et al., 1991; King & Henry, 1992) represent caffeine intake at the upper range of the current study. The dosage utilized in King and Henry's study (1992) exceeds the caffeine intake of the current study determined by body weight. Therefore, since none on the previous studies reported an increase in flicker fusion discrimination as did the current study at 2.5 mg./kg. bwt., it could be theorized that in the first study (Roback et al., 1952) the caffeine dose was too low and the caffeine dose in the latter two studies (King and Henry, 1992; Kerr et al., 1991) was too high. Again, as with luminescent threshold, flicker fusion discrimination appears to be dose specific based on the current study's results.

Variations among the studies (e.g., statistical analyses, test apparatus, time between caffeine ingestion and testing, and caffeine dosages) continue to make it difficult to compare the current study to previous studies. With further study and uniformity of protocols, more conclusive information concerning caffeine's visual effects will be discovered. Specifically, additional research is required

to clarify the effect of caffeine on flicker fusion discrimination.

Repetition Blindness. Previous studies examining the effects of caffeine on repetition blindness do not exist. In the current study, however, the Newman-Keuls post hoc analysis reported a significant decrease in repetition blindness at the 2.5 mg./kg. bwt. caffeine dose when pre-test and post-test mean scores were compared. A computerized program created by Dr. Larry Hochhaus (Hochhaus and Mihura, 1993) was utilized to test caffeine's effects on the subject's ability to identify a target word after being shown a prime word. Although Dr. Hochhaus has utilized this program to examine self-inhibition as a possible cause of repetition blindness, it has never been utilized in measuring the effect of caffeine on repetition blindness. In addition, the program (particularly the time between prime and target) was modified from its' original form for this study.

Comparisons with previous research can not be made since no studies concerning the effect of caffeine on repetition blindness are available. However, secobarbital was found to adversely effect perceptual processing (Logsdon et al., 1984); therefore, caffeine, a stimulant of the central nervous system, could theoretically improve visual performance. Keeping this in mind, along with information uncovered while exploring caffeine's effects on luminescent

threshold comparisons and flicker fusion discrimination, efforts were made to devise a way to explore the effects of caffeine on repetition blindness. Significance did occur in the repeated measures ANOVA and Newman-Keuls post hoc analysis conducted on the repetition blindness data. A significant difference was reported between the pre and post-test mean scores of the 2.5 mg./kg. bwt. caffeine dose. Repetition blindness, as with luminescent threshold and flicker fusion, appears to be dose dependent on caffeine.

CHAPTER V

SUMMARY, FINDINGS, CONCLUSIONS, AND RECOMMENDATIONS FOR FURTHER STUDY

Summary

Twenty-three undergraduate college students ranging in age from 19 to 43 were tested for the effects of selected doses of caffeine on luminescent threshold comparisons, flicker fusion discrimination, and repetition blindness. Each subject was pre-tested; given either 0 mg./kg. bwt., 2.5 mg./kg. bwt., or 5.0 mg./kg. bwt.; and post-tested on three separate occasions. Approximately 60 minutes after the ingestion of either the placebo or caffeine dose the post-test was administered repeating the pre-test procedure. A different dose of caffeine was administered following each weekly pre-test. As a result, upon completion of the three week testing period, every subject had received all three doses of caffeine.

Findings

The current study and its' findings represent an addition to the existing body of knowledge examining caffeine's effects on vision. However, it is obvious that much

work is left to be done in exploring this stimulants' visual effects. Different statistical analyses, apparatus, caffeine doses and time lapse between caffeine ingestion and post-test are just some of the reasons it is so difficult to compare the current results with previous findings. Even though caffeine is a central nervous system stimulant and has this same pharmacological effect on the rest of the body (Koetting, 1977), caffeine's effects on vision remain unclear despite years of research. Caffeine appears to impact vision by effecting the cerebral cortex, as well as, by directly effecting the eye itself. However, there continues to be many unanswered questions surrounding caffeine's effects on vision.

Repeated measures ANOVAs have customarily been the analysis of choice for exploring caffeine's effects on physiological parameters. When examining each visual test separately, the repeated measures ANOVAs and post hoc analyses identified statistically significant differences between treatments (placebo, 2.5 mg./kg. bwt. caffeine, and 5.0 mg./kg. bwt. caffeine.).

Significance was found across the 2.5 mg./kg. bwt. and 5.0 mg./kg. bwt. caffeine doses at low luminescence when making luminescent threshold comparisons. Moderate caffeine consumption (2.5 mg./kg. bwt.) at low luminescence resulted in a greater fluctuation in subjects' pre to post-test responses compared to the responses at the 5.0 mg./kg. bwt.

caffeine dose.

Findings at moderate luminescence were significant across the 2.5 and 5.0 mg./kg. bwt. caffeine levels and across the placebo and 5.0 mg./kg. bwt. caffeine dose. A higher level of caffeine (5.0 mg./kg. bwt.) resulted in greater fluctuations between pre and post-test luminescent threshold responses compared to both the placebo and the 2.5 mg./kg. bwt. dose.

Caffeine appears to have had no significant effect at high luminescence. Although significant findings were reported across treatments at both low and moderate luminescence, data collected at high luminescence did not produce significant fluctuations in pre to post-test scores at any level of treatment.

Significance was also reported at the 2.5 mg./kg. bwt. caffeine dose between pre and post-test mean scores describing flicker fusion. These findings indicate that flicker fusion discrimination increased significantly following consumption of 2.5 mg./kg. bwt. caffeine. As a result, flicker fusion was postponed after moderate caffeine consumption.

The results of the repetition blindness analyses reflect a significant difference in pre to post-test scores after consumption of the 2.5 mg./kg. bwt. caffeine dose. A greater fluctuation in responses was reported following ingestion of the moderate dose of caffeine (2.5 mg./kg. bwt.) compared to pre to post-test responses of the other

two treatments. Subjects were able to correctly identify more target words following moderate caffeine consumption (2.5 mg./kg. bwt.) compared to the other caffeine levels.

A similar pattern of significant and nonsignificant findings occurred following consumption of a particular dose of caffeine in the Newman-Keuls pot hoc analyses. Significant fluctuations in luminescent threshold comparisons occurred after consuming 2.5 mg./kg. bwt. caffeine at low luminescence and similar effects occurred at moderate luminescence after consumption of 5.0 mg./kg. bwt. caffeine. Significance was also detected between pre and post-test responses after consumption of 2.5 mg./kg. bwt. caffeine for both flicker fusion discrimination and repetition blindness. This seems to indicate that caffeine's effects may be dose related. Although significant subject variability may have influenced the outcomes to some degree, the phenomenon of the moderate caffeine dose (2.5 mg./kg. bwt.) may be the most influential across virtually all visual tests is a pattern worth noting.

Another important inference may be made concerning caffeine's effects on luminescent threshold. In making luminescent threshold comparisons, it seems as the intensity of the light stimulus increases so must the quantity of caffeine increase to result in significant differences in pre to post-test responses. At low luminescence, the 2.5 mg./kg. bwt. caffeine dose caused a significant difference

between pre and post-test responses. At moderate luminescence, the 5.0 mg./kg. bwt. dose caused significant fluctuations in scores. And, at high luminescence, no significant findings were revealed. It seems that the eye may be more sensitive to light at higher levels of luminescence which requires a higher dose of caffeine to elicit a significant response. Theoretically, it would seem that a higher dose of caffeine (e.g., 7.5 mg./kg. bwt.) would be required to elicit a significant fluctuation in pre to post-test scores as high luminance.

Conclusions

Based on the results of this study, it was concluded that moderate levels of caffeine did significantly impact vision among these subjects and, in some cases, improved visual performance. The 2.5 mg./kg. bwt. dose of caffeine was associated with significant fluctuations in pre to post-test responses when making luminescent threshold comparisons at low luminescence when contrasted to the effects of the 5.0 mg./kg. bwt. dose. Similarly, for both flicker fusion discrimination and repetition blindness, the 2.5 mg./kg. bwt. dose provided significant results. Specifically, for these two visual tests, the 2.5 mg./kg. bwt. dose was associated with improved performance; postponed flicker fusion and decreased repetition blindness. No significance was reported after ingestion of the high caffeine dose (5.0

mg./kg. bwt.) in any of the post hoc analyses which reported significance only at the 2.5 mg./kg. bwt. dose. Perhaps a dose related response exists since the higher dose of caffeine did not result in proportionate increases in fluctuations of scores (luminescent threshold comparisons) or improvements in performance (flicker fusion discrimination and repetition blindness).

A second conclusion was made based on the results of the luminescent threshold comparisons' post hoc analyses. It was concluded that higher levels of caffeine were required to elicit significant fluctuations in pre to post-test scores in response to higher intensities of luminescence. Caffeine seems to affect visual comparisons dependent on the caffeine level and the intensity of light. The eye, at brighter light intensities, appeared to be more sensitive and required more caffeine to elicit significant results. At low luminescence, the 2.5 mg./kg. bwt. dose of caffeine resulted in more diverse pre to post-test scores than the 5.0 mg./kg. dose. At moderate luminescence, the 5.0 mg./kg. bwt. dose resulted in more diverse pre to post-test scores compared to both the placebo and the 2.5 mg./kg. bwt. dose. And, at high luminescence, no significant findings were reported. At moderate luminescence, more caffeine (5.0 mg./kg. bwt.) was needed to elicit significant results compared to the caffeine (2.5 mg./kg. bwt.) associated with significant findings at low luminescence. Theo-

retically, higher doses of caffeine (e.g., 7.5 mg./kg. bwt.) may further affect luminescent threshold comparisons at high luminescence.

These conclusions should be of importance to the general public and in particular for people who maintain occupations that require and demand visual accuracy. Some occupations which would be affected by increased visual acuity (or lack there of) include: pilots, air traffic controllers, professional athletes, computer programmers, and data entry. If caffeine's effect on vision is determined by dose and/or if the intensity of light impacts caffeine's visual effects, "the public" would benefit both personally and professionally from this knowledge.

Recommendations for Further Study

Modifications are often made in methodologies, apparatus, and procedures in research designs to validate prior studies, reveal additional information, recognize relationships, and elucidate potential implications for further study. Particularly, for experimental research, this need is imperative to gain further knowledge concerning implications of such findings and more importantly for application of results. This author has several suggestions for researchers conducting future studies that examine the effects of caffeine on visual performance. These recommendations include changes involving the subjects, quantities of caf-

feine administered, procedures implemented, and the apparatus utilized in measuring caffeine's visual effects.

Modification of the subject pool may increase the potential for statistically significant differences in this study. The author suggests increasing the number of subjects in future studies. By using more subjects, the power of the statistical tests would increase which would yield a more sensitive analysis of data. Moreover, inclusion of subjects that are truly caffeine naive (<100 mg. caffeine consumed daily) would eliminate the potential for experiencing symptoms of caffeine tolerance or withdrawal which generally confound results.

Alterations in caffeine dosage may need to be included in future studies to provide adequate information to understand caffeine's effect on vision. More of a trend may develop by adding additional levels of caffeine. If the eye is more sensitive to brighter light (higher luminescence), more caffeine may be needed to elicit a significant visual response. By including at least one or possibly two more dosages of caffeine that is higher than 5.0 mg./kg. bwt. (e.g., 7.5 mg./kg. bwt. and 10 mg./kg. bwt.), more of a dose response curve and/or the saturation level in the subjects may develop which may allow researchers to detect additional differences. The addition of these extra intervals may be prohibitive, however, due to caffeine's side effects (irritability, nervousness, and tremulousness) reported for high

levels of caffeine intake (>600 mg.). As a result, a more thorough medical screening and observation of subjects following caffeine consumption by medical personnel may be warranted.

Further studies may focus on individual components of the present study given its' complexity (e.g., three levels of caffeine; three different visual tests; and one of the visual tests, luminescent threshold comparisons, tested three different intensities of light). For example, a study could be developed to assess each independent variable (e.g., caffeine dosages) separately. In this way, the study is simplified, more control is exercised on the test variables, and the power of the test increases. In addition, further study examining luminescent threshold comparisons could identify specific threshold responses to various caffeine levels.

The specific apparatus utilized in this study have not been implemented in prior studies examining caffeine's effect on vision. The author believes it would be interesting to repeat this type of testing utilizing the same apparatus to verify/clarify the current findings. Specifically, with regard to repetition blindness, the author suggests one change. By decreasing the amount of time the target word is flashed on the computer screen (e.g., from 17 ms. to 10 ms.), difficulty in identifying the target word would be increased. This would place increased demands on the subje-

cts' visual response and further accentuate the dose response curve mentioned earlier.

REFERENCES

Adams, B.A., & Brubaker, R.F. (1990). Caffeine has no clinically significant effect on aqueous humor flow in the normal human eye. Ophthalmology, 97, 1030-1031.

Axelrod, J. & Reichenthal, J. (1953). The fate of caffeine in man and a method for its estimation in biological materials. J. Pharmacol. Exp. Ther., 107, 519-523.

Baker, W.J. & Theologus, G.C. (1972). Effects of caffeine on visual monitoring. J. Appl. Physiol., 56, 422-427.

Barone, J.J. & Roberts, H.R. (1996). Caffeine consumption. Food and Chemical Toxicity, 34(1), 119-129.

Beavo, J.A., Rogers, N.L., Crofford, O.B., Hardman, J.G., Sutherland, E.S., & Newman, E.V. (1970). Effects of xanthine derivatives on lipolysis and on adenosine 3',5'-monophosphate phosphodiesterase activity. Molecular Pharmacology, 6, 597-603.

Bellet, S., Roman, L., DeCastro, O., Kim, K.E., & Kreshbaum, A. (1969). Effects of caffeine ingestion on catecholamine release. Metabolism, 18, 288-291.

Bianchi, C.P. (1961). The kinetics of radio-caffeine uptake in frog sartorius. Journal of General Physiology, 44, 845-858.

Bianchi, C.P. (1975). Cellular pharmacology of contraction of skeletal muscle. In T. Narahashi (Ed.), Cellular Pharmacology of Excitable Tissues (pp. 485-519). Springfield: Charles C. Thomas.

Boulenger, J., Patel, J., & Marangos, P.J. (1982). Effects of caffeine and theophylline on adenosine and benzodiazepine receptors in human brain. Neuroscience Letters, 30, 161-166.

Brecher, E.M., & the editors of Consumer Reports. (1972). Licit and Illicit Drugs (pp. 193-206). Boston: Little, Brown, and Company.

Broverman, D.M., & Casagrande, E. (1982). Effect of caffeine on performance of a perceptual-restructuring task at different stages of practice. Psychopharmacol. 78, 252-255.

Bunker, L., & McWilliams, M. (1979). Caffeine content of common beverages. Journal of the American Dietetic Association, 74, 28-32.

Caffeine: How to consume less. (1981, October). Consumer Reports, pp.597-599.

Caffeine: What it does. (1981, October). Consumer Reports, pp. 585-596.

Cardinali, D.P. (1980, October). Methylxanthines: possible mechanisms of action in brain. Tr. Pharmacol. Sci., pp. 405-407.

Carpenter, J.A. (1959). The effect of caffeine and alcohol on simple visual reaction time. J. Comp. Physiol. Psychol., 52, 491-495.

Catlin, D.H., & Hatton, C.K. (1991). Use and abuse of anabolic and other drugs for athletic enhancement. Advances in Internal Medicine, 36, 399-424.

Challiss, R.A.J., Richards, S.J., & Budohoski, L. (1992). Characterization of the adenosine receptor modulating insulin action in rat skeletal muscle. European Journal of Pharmacology - Molecular Pharmacology Section, 226, 121-128.

Chou, T. (1992). Wake up and smell the coffee -- caffeine, coffee, and the medical consequences. Western Journal of Medicine, 157, 544-553.

Clayman, C. (Ed.). (1989). Digestive System. In The Home Medical Encyclopedia (Vol. 1, pp. 360). New York: Random House.

Daly, J.W., Bruns, R.F., & Snyder, S.H. (1981). Adenosine receptors in the central nervous system: Relationship to the central action of methylxanthines. Life Sciences, 28, 2083-2097.

Davies, L.P., & Chow, S.C. (1984). Effect of some potent adenosine uptake inhibitors on benzodiazepine binding in the central nervous system. Neurochemistry International, 6(2), 185-189.

Diamond, A.L., & Cole, R.E. (1970). Visual threshold as a function of test area and caffeine administration. Psychon. Sci., 20, 109-111.

Dodd, S.L., Herb, R.A., & Powers, S.K. (1993). Caffeine and exercise performance: An update. Sports Medicine, 15(1), 14-23.

Evenden, J.L., Turpin, M., Oliver, L., & Jennings, C. (1993). Caffeine and nicotine improve visual tracking by rats: A comparison with amphetamine, cocaine and apomorphine. Psychopharmacology, 110, 169-176.

Fine, B.J., Kobrick, J.L., Lieberman, H.R., Marlowe, B., Riley, R.H., & Tharion, W.J. (1994). Effects of caffeine or diphenhydramine on visual vigilance. Psychopharmacology, 114, 233-238.

Fredholm, B.B. (1995). Adenosine, adenosine receptors and the actions of caffeine. Pharmacology and Toxicology, 76, 93-101.

Fredhold, B.B. (1985). On the mechanism of action of theophylline and caffeine. Acta Med. Scand., 217, 149-153.

Fryer, M.W., & Neering, I.R. (1989). Actions of caffeine on fast- and slow-twitch muscles of the rat. Journal of Physiology, 416, 435-454.

Graham, D.M. (1978). Caffeine - its identity, dietary sources, in take, and biological effects. Nutr. Rev., 36, 97-102.

Graham, T.E., Rush, J.W.E., & van Soeren, M.H. (1994). Caffeine and exercise: Metabolism and performance. Canadian Journal of Applied Physiology, 19(2), 111-138.

Graham, T.E., & Spriet, L.L. (1995). Metabolic, catecholamine, and exercise performance responses to various doses of caffeine. Journal of Applied Physiology, 78(3), 867-874.

Greden, J.F. (1974). Anxiety or caffeinism: A diagnostic dilemma. American Journal of Psychiatry, 131, 1089-1092.

Gulati, J., & Babu, A. (1985). Contraction kinetics of intact and skinned frog muscle fibers and degree of activation. J. Gen. Physiology, 86, 479-500.

Guthrie, J.R., & Nayler, W.G. (1967). Interaction between caffeine and adenosine on calcium exchangeability in mammalian atria. Arch. Int. Pharmacodyn., 170, 249-255.

Health Letter Associates (1990). The Daily Dose. Berkeley Wellness Letter, University of California.

Higginbotham, E.J., Kilimanjaro, H.A., Wilensky, J.T., Batenhorst, R.L., & Hermann, D. (1989). The effect of caffeine on intraocular pressure in glaucoma patients. Ophthalmology, 96, 624-626.

Hochhaus, L., & Mihura, J.L. (1993). Repetition blindness: Frequency effects. Unpublished research, Oklahoma State University.

Hole, J.W., & Koos, K.A. (1991). Human Anatomy (pp. 280-320). Dubuque, IA: Wm. C. Brown Publishers.

Holtzman, S.G., Mante, S., & Minneman, K.P. (1991). Role of adenosine receptors in caffeine tolerance. Journal of Pharmacology and Experimental Therapeutics, 256(1), 62-68.

Jaanus, S.D. (1992). Ocular side effects of selected system drugs. Optometry Clinics, 2, 73-96.

Jacobson, B.H., & Edgley, B.M. (1987). Effects of caffeine on simple reaction time and movement time. Aviation Space and Environmental Medicine, 58, 1153-1156.

Jacobson, B.H., & Kulling, F.A. (1989). Health and ergo genic effects of caffeine. British Journal of Sports Medicine, 23, 34-40.

Jacobson, B.H., & Thurman-Lacey, S.R. (1992). Effect of caffeine on motor performance by caffeine-naive and familiar subjects. Perceptual and Motor Skills, 74, 151-157.

Jacobson, B.H., Weber, M.D., Claypool, L., & Hunt, L.E. (1992). Effect of caffeine on maximal strength and power in elite male athletes. British Journal of Sports Medicine, 26(4), 276-280.

Jacobson, B.H., Winter-Roberts, K., & Gemmell, H.A. (1991). Influence of caffeine on selected manual manipulation skills. Perceptual and Motor Skills, 72, 1175-1181.

Kelly, D.H., & Wilson, H.R. (1978). Human flicker sensitivity: Two stages of retinal diffusion. Science, 202, 896-899.

Kenemans, J.L., & Lorist, M.M. (1995). Caffeine and selective visual processing. Pharmacology Biochemistry and Behavior, 52, 461-471.

Kerr, J.S., Sherwood, N., & Hindmarch, I. (1991). Separate and combined effects of the social drugs on psychomotor performance. Psychopharmacology, 104, 113-119.

King, D.J., & Henry, G. (1992). The effect of neuroleptics on cognitive and psychomotor function: A preliminary study in healthy volunteers. British Journal of Psychiatry, 160, 647-653.

Kleman, J.P., Diamond, A.L., & Smith, E. (1961). Effects of caffeine on enhancement in foveal simultaneous contrast. J. Exp. Psych., 61, 18-22.

Koelega, H.S. (1993). Stimulant drugs and vigilance performance: A review. Psychopharmacology, 111, 1-16.

Koetting, J.F. (1977). The use of drugs for behavior modification as it relates to the practice of optometry. Part II. Journal of the American Optometric Association, 48(2), 213-222.

Lafayette Instrument Company. (1970). Instructions for model #14011 (1701), Light Discrimination Apparatus. Lafayette, IN: Author.

Lafayette Instrument Company. (1986). Instructions for model #58017, Visual Perception Control. Lafayette, IN: Author.

Lieberman, H.R., Wurtman, R.J., Emde, G.G., Roberts, C., & Coviella, I.L.G. (1987). The effects of low doses of caffeine on human performance and mood. Psychopharmacology, 92, 308-312.

Logsdon, R., Hochhaus, L., Williams, H.L., Rundell, O.H., & Maxwell, D. (1984). Secobarbital and Perceptual Processing. Acta Psychologica, 55, 179-193.

Loke, H.W., & Meliska, C.J. (1984). Effect of caffeine use and ingestion on a protracted visual vigilance task. Psychopharm., 84, 54-57.

Lombardo, J.A. (1986). Stimulants and athletic performance: Amphetamines and caffeine (part 1 of 2). The Physician and Sports Medicine, 14(11), 128-140.

Lorist, M.M., Snel, J., & Kok, A. (1994). Influence of caffeine on information processing stages in well rested and fatigued subjects. Psychopharmacology, 113, 411-421.

Lorist, M.M., Snel, J., Kok, A., & Mulder, G. (1994). Influence of caffeine on selective attention in well-rested and fatigued subjects. Psychophysiology, 31, 525-534.

Lotfi, K., & Grunwald, J.E. (1991). The effect of caffeine on the human macular circulation. Investigative Ophthalmology and Visual Science, 32, 3028-3032.

MacCornack, R.A. (1977). The effects of coffee drinking on the cardiovascular system; experimental and epidemiological research. Prev. Med., 6, 104-119.

Marohn, K.M., & Hochhaus, L. (1987). Semantic priming increases and repetition priming decreases apparent stimulus duration. Journal of General Psychology, 115, 51-61.

Nehlig, A., & Debry, G. (1994). Caffeine and sports activity: A review. International Journal of Sports Medicine, 15, 215-223.

Nehlig, A., Duval, J., & Debry, G. (1992). Caffeine and the central nervous system: Mechanisms of action, biochemical, metabolic and psychostimulant effects. Brain Research Reviews, 17, 139-170.

Olsson, R.A., & Pearson, J.D. (1990). Cardiovascular purinoceptors. Physiological Reviews, 70(3), 761-845.

O'Neal, F.T., Hynak-Hankinson, M.T., & Gorman, J. (1986). Research and application of current topics in sports nutrition. Journal of the American Dietetic Association, 86(8), 1007-1015.

Paré, W. (1961). The effect of caffeine and seconal on a visual discrimination task. J. of Comparative and Physiological Psychology, 54, 506-509.

Powers, S.K., & Dodd, S. (1985). Caffeine and Endurance Performance. Sports Medicine, 2, 165-174.

Putz-Anderson, V., Setzer, J.V., & Croxton, J.S. (1981). Effects of alcohol, caffeine and methyl chloride on man. Psychol. Rep., 48, 715-725.

Rall, T.W. (1980). Central nervous system stimulants. In A. Goodman-Gilman, L.S. Goodman and A. Gilman (Eds.), The Pharmacological Basis of Therapeutics (6th ed.) (pp. 592-607). New York: MacMillon.

Ramkumar, V., Bumgarner, J.R., Jacobson, K.A., & Stiles, G.L. (1988). Multiple components of the A1 adenosine receptor-adenylate cyclase system are regulated in rat cerebral cortex by chronic caffeine ingestion. J. Clin. Invest., 82, 242-247.

Roback, G.S., Krasno, L.R., & Ivy, A.C. (1952). Effect of analeptic drugs on the somnifacient effect of seconal and antihistaminics as measured by the flicker fusion threshold. Journal of Applied Physiology, 4, 566-574.

Sattin, A., & Rall, T.W. (1970). The effect of adenosine and adenine nucleotides on the cyclic adenosine 3',5'-phosphate content of guinea pig cerebral cortex slices. Molecular Phamacology, 6, 13-23.

Sawynok, J. (1995). Pharmacological rationale for the clinical use of caffeine. Drugs, 49(1), 37-50.

Simonson, E., & Brozek, J. (1952). Flicker fusion frequency: Background and application. Physiological Reviews, 32, 349-378.

Snyder, S.H., Katims, J.J., Annau, Z., Bruns, R.F., & Daly, J.W. (1981). Adenosine receptors and behavioral actions of methylxanthines. Proc. Natl. Acad. Sci. USA, 78(5), 3260-3264.

Spriet, L. (1995). Caffeine and performance. International Journal of Sport Nutrition, 5, S84-S99.

Stavric, B. (1992). An update on research with coffee/caffeine (1989-1990). Food and Chemical Toxicology, 30(6), 533-555.

Stephenson, P.E. (1977). Physiologic and Psychotropic effects of caffeine on man. Journal of the American Dietetic Association, 71, 240-247.

Stoelting, R.K. (1991). Pharmacology and physiology in anesthetic practice (pp. 118-131). Philadelphia: Lippencott

Su, J.Y., & Hasselbach, W. (1984). Caffeine-induced calcium release from isolated sarcoplasmic reticulum of rabbit skeletal muscle. Pflugers Arch., 400, 17-21.

Syed, I.B. (1976). The effects of caffeine. J. Am. Pharmac. Assn., 16, 568-572.

VanHandel, P. (1980, February). Effects of caffeine on physical performance. JOPER, pp. 56-57.

VanHandle, P. (1983). Caffeine. In M.H. Williams (Ed.), Ergogenic Aids in Sports (pp. 128-163). Champaign, Ill.: Human Kinetics Publishers.

Wendt, I.R., & Stephenson, D.G. (1983). Effects of caffeine on Ca-activated force production in skinned cardiac and skeletal muscle fibers of the rat. Pflugers Arch., 389, 210-216.

What caffeine does to you. (1993, June). Consumer Reports, pp. 64-66.

Weir, R.L., & Hruska, R.E. (1983). Interaction between methylxanthines and the benzodiazepine receptor. Arch. Int. Pharmacodyn., 265, 42-48.

Work, J.A. (1991). Are java junkies poor sports? The Physician and Sportsmedicine, 19(1), 83-88.

Zhang, Y., & Wells, J.N. (1990). The effects of chronic caffeine administration on peripheral adenosine receptors. Journal of Pharmacology and Experimental Therapeutics, 254(3), 757-763.

APPENDIXES

APPENDIX A

INDIVIDUAL'S CONSENT FOR PARTICIPATION IN A RESEARCH PROJECT

Individual's Consent for Participation in a Research Project
Oklahoma State University

I, _____, voluntarily agree to participate in this study entitled: Flicker Fusion, Visual Threshold, and Repetition Blindness in Response to Trimethylxanthine (Caffeine) Ingestion.

1. **PURPOSE:** This study involves research that will be carried out under the supervision of Bert H. Jacobson, Ed.D. (principal investigator) and Darla Fent, Graduate Student. The purpose of this study will be to ascertain the effects of 2.5 mg./kg. bwt. and 5.0 mg./kg. bwt. caffeine on three aspects of visual acuity: flicker fusion, visual threshold, and repetition blindness. In the realm of many occupations such qualities are often necessary and/or vital. Given that one cup of coffee contains 100 mg. caffeine, it is safe to assume that many professionals consume up to 400 mg. caffeine prior to or during work time. However, casual consumption is not all in one dose. This study will attempt to find if deleterious effects follow a single dose of 2.5 mg./kg. bwt. and 5.0 mg./kg. bwt. caffeine consumption.

2. **STATUS OF INVESTIGATIONAL DRUG PROCEDURES:** Caffeine may alter blood pressure, heart rate, respiration and metabolic rate. Caffeine may also induce tremors, nervousness, and anxiety.

3. **DESCRIPTION OF STUDY:** This study will involve a prescreening consisting of blood pressure and heart rate. Additionally a medical history questionnaire containing the following items will be administered: oral contraceptive use, medication use, current illnesses, pregnancy, hang over, history of heart disease, etc. . . Further, a caffeine consumption questionnaire will be administered to ascertain the average amount of caffeine consumed per day and per week. Any subject indicating a blood pressure reading above 140 mm. Hg. systolic pressure and/or 90 mm. Hg. diastolic pressure and/or tachycardia will be eliminated from the study. Also, any positive response on the medical history questionnaire may result in elimination.

Subjects will be asked to fast from food for five (5) hours and fast from caffeine for 48 hours prior to testing. Subjects will be pre-tested for flicker fusion, visual threshold, and repetition blindness. Following the pre-test, each subject will ingest one of three capsules containing: 1) 0 mg. caffeine, 2) 2.5 mg./kg. bwt., or 3) 5.0 mg./kg. bwt. caffeine utilizing a double blind format. Following a one (1) hour waiting period, all subjects will be post-tested using the pre-test protocol.

The full duration of this study will take approximately one and a half (1.5) hours.

I understand that I will be given 0 mg., 2.5 mg./kg. bwt., 5.0 mg./kg. bwt. caffeine. Neither I nor the investigator will know which dosage I have been administered during each test but that information can be obtained if necessary.

4. **BENEFITS:** No direct benefit in the consumption of caffeine may be expected. However, observable physical changes may lead to a change in attitude toward caffeine consumption and a greater awareness of products containing caffeine may ensue.

5. **POSSIBLE RISKS:** Caffeine ingestion in the quantities described in this study may increase nervousness, irritability and anxiety. Respiration, blood pressure and heart rate may also be magnified. Additionally nausea may appear if the meal following caffeine consumption includes spicy and/or greasy food. STAY AWAY FROM PIZZA!

If you become nauseous or feel ill, you will be retained for observation and transported to the University Health Center.

I recognize that the primary risk is the possibility of experiencing some side effects. Those that have been observed in the past for caffeine consumption include:

Hyperactivity
Upset stomach
Nervousness

If I have any side-effects, I will report them immediately to the investigator, my physician or his/her associates. If side-effects are severe, I may be removed from the study.

6. **ALTERNATE PROCEDURES:** None

7. **SUBJECT ASSURANCES:** Whereas no assurance can be made concerning results that may be obtained (because results from investigational studies cannot be predicted with certainty), the principal investigator, will take every precaution consistent with best scientific practice.

By signing this consent form, I acknowledge that my participation in this study is voluntary. I also acknowledge that I have not waived any of my legal right or released this institution form liability for negligence.

I may revoke my consent and withdraw from this study at any time without penalty or loss of benefits. My treatment

by, and relations with the investigators and staff at Oklahoma State University, now and in the future, will not be affected in any way if I refuse to participate, or if I enter the program and later withdraw.

Records of this study will be kept confidential with respect to any written or verbal reports making it impossible to identify me individually. All records will be held in a locked file belonging to the principal investigator.

If I have any questions about my rights as a research subject, I may take them to the Office of University Research Services, 001 Life Sciences East. Phone: 744-5700.

8. **SIGNATURES:**

_____	_____
Date	Research Subject
_____	_____
Date	Witness
_____	_____
Date	Principal Investigator

Any questions regarding the research may be addressed to Bert Jacobson, Principal Investigator. 102 Colvin Center. Phone: 744-5493.

Subjects will receive a copy of this consent form following the investigation.

APPENDIX B

CAFFEINE RESEARCH QUESTIONNAIRE

CAFFEINE RESEARCH QUESTIONNAIRE

Caffeine Consumption History
Vital Statistics * Medical History

Name_____ Age____ Sex____ Wt._____ Ht._____
Date of Birth_____

	PRE	POST
HR	_____b/min	_____b/min
BP	_____/_____	_____/_____

Caffeine Consumption History

Coffee: Cups/day _____avg.
Soft Drinks (Coke, Dr. Pepper, Mt. Dew, etc.)/day _____avg.
Tea: Cups/day _____ Glasses/day _____
Other: Explain _____
How does caffeine affect you? _____

_____

Medical History

Have you experienced or know of: (Respond "YES" or "NO".)

Heart trouble	_____	Stomach disorders	_____
Intestinal disorders	_____	High Blood Pressure	_____
High Heart Rate	_____	Kidney Disorders	_____

Mental/Emotional Disorders_____
Fibrocystic Breast Disease _____

Do you wear corrective lenses or contacts? _____
Do you smoke? _____
Do you think you are pregnant? _____
Are you currently taking oral contraceptives? _____
Are your presently on medication?_____. If so, explain
_____

Vital Study Criteria

Are your suffering from a hangover? _____
Are you suffering from lack of sleep? _____
Have you fasted for 5 hours? _____
Last meal was _____hrs. ago.
Last caffeine was consumed _____hrs. ago
in the form of _____

DATE OF TESTING _____ WEEK NUMBER _____
TIME OF TESTING: Pre-test _____; Post-test _____
TIME OF CAFFEINE INGESTION FOR STUDY _____
WEEK # _____ SUBJECT # _____

APPENDIX C

CAFFEINE CONTENT

OF COMMON BEVERAGES, FOODS, AND MEDICATIONS

CAFFEINE CONTENT OF COMMON BEVERAGES, FOODS, & MEDICATIONS

---

* Caffeine Content (mg) in Selected 12 oz. Sodas:

Afri-Cola	100.0 mg (?)
Jolt	71.2
Sugar-Free Mr. Pibb	58.8
Mountain Dew	55.0 (0 in Canada)
Diet Mountain Dew	55.0
Mello Yellow	52.8
Tab	46.8
Coca-Cola	45.6
Diet Cola	45.6
Shasta Cola	44.4
Shasta Diet Cola	44.4
Mr. Pibb	40.8
Dr. Pepper	39.6
Pepsi Cola	37.2
Diet Pepsi	35.4
RC Cola	36.0
Diet RC	36.0
Canada Dry Cola	30.0
7 Up	0

* National Soft Drink Association.

* Caffeine Content (mg) in a 7 oz. cup of coffee\tea:

Drip	115-175 mg
Espresso (1.5 - 2 oz.)	100
Brewed	80-135
Instant	65-100
Decaf, brewed	3-4
Decaf, instant	2-3
Tea, iced (12 oz.)	70
Tea, brewed, imported	60
Tea, brewed, U.S.	40
Tea, instant	25-150

* Bunker, L., & McWilliams, M. (1979). Caffeine content of common beverages. Journal of the American Dietetic Association, 74, 28-32.

* Other data on caffeine (amounts expressed in mg):

Chocolate cake (1 slice)		20-30 mg
"Dristan"	(Cold relief)	30
"Anacin"	(Pain relief)	32
"Midol"	(Pain relief)	32.4
"Excedrin"	(Pain relief)	65
"Nodoz"	(Stimulant)	100
"Vivarin"	(Stimulant)	200

* Caffeine: How to consume less. (1981, October). Consumer Reports, pp. 597-599.
* Health Letter Associates (1990). The Daily Dose. Berkeley Wellness Letter, University of California.

APPENDIX D

REPETITION BLINDNESS RESPONSE SHEETS

REPETITION BLINDNESS RESPONSE SHEET

SUBJECT NAME _____ SUBJECT NUMBER _____

WEEK 1 -- PRE-TEST SUBJECT #1/DEMO.RB.A

BLOCK 1

1. ROOM R. ROOM
2. SIDE R. SIDE
3. NEED U. FOUR
4. GIVE R. GIVE
5. FORM U. CASE
6. FACE R. FACE
7. FELT U. BEST
8. EVER U. WANT

BLOCK 2

9. WITH R. WITH
10. FROM R. FROM
11. THIS R. THIS
12. HAVE R. HAVE
13. WHEN U. WHAT
14. BEEN U. SAID
15. THEY U. WILL
16. WERE U. MORE

BLOCK 3

17. INTO R. INTO
18. ONLY R. ONLY
19. THAN R. THAN
20. THEM R. THEM
21. THEN U. EVEN
22. TIME U. OVER
23. SUCH U. MOST
24. SOME U. LIKE

BLOCK 4

25. MUST R. MUST
26. WELL U. GOOD
27. YOUR U. JUST
28. MANY R. MANY
29. MUCH U. EACH
30. BACK U. DOWN
31. MADE R. MADE
32. ALSO R. ALSO

BLOCK 5

33. BOTH U. LAST
34. LIFE U. YEAR
35. LONG R. LONG
36. WORK R. WORK
37. VERY R. VERY
38. HERE U. KNOW
39. MAKE R. MAKE
40. SAME U. COME

REPETITION BLINDNESS RESPONSE SHEET (continued)

SUBJECT NAME _____ SUBJECT NUMBER _____

WEEK 1 -- POST TEST SUBJECT #2/DEMO. RB. B

BLOCK 1

1. FORM U. CASE
2. NEED U. FOUR
3. SIDE R. SIDE
4. EVER U. WANT
5. GIVE R. GIVE
6. ROOM R. ROOM
7. FACE R. FACE
8. FELT U. BEST

BLOCK 2

9. WILL U. INTO
10. BEEN U. SAID
11. THEY R. THEY
12. FROM R. FROM
13. WHEN U. WHAT
14. THIS R. THIS
15. HAVE R. HAVE
16. WERE U. MORE

BLOCK 3

17. THEM R. THEM
18. ONLY R. ONLY
19. THEN U. EVEN
20. SOME R. SOME
21. THAN R. THAN
22. TIME U. OVER
23. LIKE U. MADE
24. SUCH U. MOST

BLOCK 4

25. BACK R. BACK
26. MUST R. MUST
27. DOWN U. VERY
28. WELL U. GOOD
29. ALSO R. ALSO
30. YOUR U. JUST
31. MANY R. MANY
32. MUCH U. EACH

BLOCK 5

33. MAKE R. MAKE
34. BOTH U. LAST
35. LONG R. LONG
36. HERE R. HERE
37. WORK R. WORK
38. LIFE U. YEAR
39. KNOW U. CAME
40. SAME U. COME

REPETITION BLINDNESS RESPONSE SHEET (continued)

SUBJECT NAME _____ SUBJECT NUMBER _____

WEEK 2 -- PRE-TEST SUBJECT #3/DEMO. RB. C

BLOCK 1

1. GIVE R. GIVE
2. FORM U. CASE
3. FELT U. BEST
4. SIDE R. SIDE
5. EVER U. WANT
6. FACE R. FACE
7. NEED U. FOUR
8. ROOM R. ROOM

BLOCK 2

9. BEEN U. SAID
10. MORE U. THAN
11. FROM R. FROM
12. THEY R. THEY
13. HAVE R. HAVE
14. WILL U. INTO
15. WERE R. WERE
16. WHEN U. WHAT

BLOCK 3

17. THEM R. THEM
18. LIKE U. MADE
19. SUCH U. MOST
20. TIME R. TIME
21. OVER U. ALSO
22. SOME R. SOME
23. ONLY R. ONLY
24. THEN U. EVEN

BLOCK 4

25. DOWN U. VERY
26. YOUR U. JUST
27. BACK R. BACK
28. WELL U. GOOD
29. MUST R. MUST
30. MANY R. MANY
31. EACH U. MAKE
32. MUCH R. MUCH

BLOCK 5

33. SAME U. COME
34. LIFE U. YEAR
35. BOTH R. BOTH
36. KNOW U. CAME
37. HERE R. HERE
38. WORK R. WORK
39. LONG R. LONG
40. LAST U. USED

REPETITION BLINDNESS RESPONSE SHEET (continued)

SUBJECT NAME _____ SUBJECT NUMBER _____

WEEK 2 -- POST TEST SUBJECT #4/DEMO. RB. D

BLOCK 1

1. ROOM R. ROOM
2. EVER U. WANT
3. NEED U. FOUR
4. FELT U. BEST
5. SIDE R. SIDE
6. FACE R. FACE
7. FORM U. CASE
8. GIVE R. GIVE

BLOCK 2

9. WHEN U. WHAT
10. WILL U. INTO
11. HAVE R. HAVE
12. MORE U. THAN
13. THEY R. THEY
14. WERE R. WERE
15. SAID U. THEM
16. BEEN R. BEEN

BLOCK 3

17. EVEN U. MANY
18. TIME R. TIME
19. ONLY R. ONLY
20. SOME R. SOME
21. SUCH U. MOST
22. OVER U. ALSO
23. THEN R. THEN
24. LIKE U. MADE

BLOCK 4

25. MUST R. MUST
26. DOWN U. VERY
27. MUCH R. MUCH
28. WELL U. GOOD
29. JUST U. WORK
30. EACH U. MAKE
31. YOUR R. YOUR
32. BACK R. BACK

BLOCK 5

33. SAME U. COME
34. YEAR U. TAKE
35. LIFE R. LIFE
36. LONG R. LONG
37. LAST U. USED
38. HERE R. HERE
39. BOTH R. BOTH
40. KNOW U. CAME

REPETITION BLINDNESS RESPONSE SHEET (continued)

SUBJECT NAME _____ SUBJECT NUMBER _____

WEEK 3 -- PRE-TEST SUBJECT #5/DEMO. RB. E

BLOCK 1

1. FACE R. FACE
2. SIDE R. SIDE
3. GIVE R. GIVE
4. FELT U. BEST
5. FORM U. CASE
6. NEED U. FOUR
7. ROOM R. ROOM
8. EVER U. WANT

BLOCK 2

9. WHEN R. WHEN
10. BEEN R. BEEN
11. SAID U. THEM
12. WERE R. WERE
13. THEY R. THEY
14. WILL U. INTO
15. WHAT U. ONLY
16. MORE U. THAN

BLOCK 3

17. MOST U. MUST
18. SUCH R. SUCH
19. LIKE U. MADE
20. EVEN U. MANY
21. THEN R. THEN
22. SOME R. SOME
23. TIME R. TIME
24. OVER U. ALSO

BLOCK 4

25. BACK R. BACK
26. JUST U. WORK
27. DOWN U. VERY
28. GOOD U. LONG
29. YOUR R. YOUR
30. WELL R. WELL
31. MUCH R. MUCH
32. EACH U. MAKE

BLOCK 5

33. COME U. HOME
34. SAME R. SAME
35. KNOW U. CAME
36. BOTH R. BOTH
37. YEAR U. TAKE
38. HERE R. HERE
39. LIFE R. LIFE
40. LAST U. USED

REPETITION BLINDNESS RESPONSE SHEET (continued)

SUBJECT NAME _____ SUBJECT NUMBER _____

WEEK 3 -- POST TEST SUBJECT #6/DEMO RB. F

BLOCK 1

1. GIVE R. GIVE
2. ROOM R. ROOM
3. NEED U. FOUR
4. FELT U. BEST
5. FORM U. CASE
6. SIDE R. SIDE
7. EVER U. WANT
8. FACE R. FACE

BLOCK 2

9. SAID U. THEM
10. WERE R. WERE
11. WHAT U. ONLY
12. INTO U. SOME
13. WILL R. WILL
14. MORE U. THAN
15. WHEN R. WHEN
16. BEEN R. BEEN

BLOCK 3

17. SUCH R. SUCH
18. LIKE R. LIKE
19. TIME R. TIME
20. MOST U. MUST
21. THEN R. THEN
22. OVER U. ALSO
23. EVEN U. MANY
24. MADE U. BACK

BLOCK 4

25. YOUR R. YOUR
26. EACH U. MAKE
27. WELL R. WELL
28. JUST U. WORK
29. GOOD U. LONG
30. VERY U. HERE
31. DOWN R. DOWN
32. MUCH R. MUCH

BLOCK 5

33. KNOW R. KNOW
34. SAME R. SAME
35. COME U. HOME
36. YEAR U. TAKE
37. CAME U. WENT
38. LAST U. USED
39. LIFE R. LIFE
40. BOTH R. BOTH

APPENDIX E

RAW DATA RECORD SHEET

RAW DATA RECORD SHEET

Flicker Fusion, Visual Threshold, and Repetition Blindness in Response to Trimethylxanthine (Caffeine) Ingestion

Subject Name_____ Subject Number_____

Date of Birth_____ Week Number_____

PRE

Flicker Fusion

Setting of Response

1. _____
2. _____
3. _____

Visual Threshold

	Standard	Response
1.	140	_____
2.	290	_____
3.	220	_____

Repetition Blindness

	Correct	Incorrect
1.	_____	_____

POST

Flicker Fusion

Setting of Response

1. _____
2. _____
3. _____

Visual Threshold

	Standard	Response
1.	140	_____
2.	290	_____
3.	220	_____

Repetition Blindness

	Correct	Incorrect
1.	_____	_____

APPENDIX F

VERBAL INSTRUCTIONS FOR TESTING

VERBAL INSTRUCTIONS FOR TESTING

Flicker Fusion Discrimination

Flicker Fusion is defined as the point where the frequency of a flickering light is no longer discernable.

1. Please sit in front of the display unit and note the two stimulus windows from which light will be emitted.

2. Once the overhead lights are dimmed to facilitate visual perception, you will observe a flickering light in the display unit.

3. Your objective is to identify the point in time in which the light no longer flickers. In other words, you are identifying the earliest point at which the successive stimuli are perceived as completely fused.

4. This process will be performed three times.

Luminescent Threshold Comparisons

Luminescent Threshold is defined as the point in which the eye can discern differences in the intensity of light acting as a stimulus.

1. Please sit directly in front of the Light Discrimination Apparatus. (NOTE: IT IS IMPERATIVE THAT THE SUBJECT NOT BE ABLE TO SEE EITHER OF THE INTENSITY ADJUSTMENT DIALS.)

2. This is a light discrimination test. The light on the unit adjacent to your dominant hand is variable in intensity. The other light is fixed in intensity and is called the standard.

3. Once the lights are dimmed to facilitate visual perception, you will use your dominant hand to adjust the light intensity dial located on that side of the unit.

4. Your job is to adjust the variable light so that it LOOKS equal in intensity to the standard.

5. This process will be performed three times with the standard being set to a maximal, minimal, or median intensity for each trial.

VERBAL INSTRUCTIONS FOR TESTING (continued)

Repetition Blindness

Repetition blindness is defined as the inability to identify a second exposure to a repeated word or identification of a novel word following an initial exposure to a prime word.

1. Please sit in front of the computer screen and keyboard.

2. This exercise consists of forty trials. The first eight trials are considered "practice trials" and the data generated in these practice trials will not be considered in this study. Each trial begins when you depress the "space" bar. You may proceed at your own comfortable pace.

3. For each trial, you will see the number of the trial displayed (e.g., Trial 1). Once the space bar is depressed,you will briefly see a PRIME word, a four letter word, flashed in all caps in the center of the screen. Then, slightly below this word, a pair of arrows will immediately be displayed pointing to the TARGET word, either the same word OR a different word, also flashed in all caps (e.g., ------> GIVE <------). Each of the second, or target, words is then immediately replaced with ampersands (e.g., &&&&&&&&&&&&) and the next trial (e.g., Trial 2) is ready to begin.

4. Your objective is to verbally identify both words in each trial. You will say the trial number followed by each PRIME and TARGET word.

5. If you are unable to read either the prime or target word, verbally respond with the word "BLANK" and proceed to the next trial.

APPENDIX G

ANOVA SUMMARY TABLES

ANOVA Summary Tables

	SS	DF	MS	F	P
Low Luminescence					
Effect	905.26	2	452.63	3.62*	.04
Error	5503.23	44	125.07		
*Significant, p < .05.					
Moderate Luminescence					
Effect	1963.94	2	981.97	4.36*	.02
Error	9904.06	44	225.09		
*Significant, p < .05.					
High Luminescence					
Effect	496.55	2	248.28	1.86*	.17
Error	5859.45	44	133.17		
*Not significant, p > .05.					
Flicker Fusion					
Effect	477.15	5	95.43	7.24*	.00
Error	1450.78	110	13.19		
*Significant, p < .05.					
Repetition Blindness					
Effect	278.59	5	55.72	3.47*	.01
Error	1766.58	110	16.06		
*Significant, p. < .05.					

APPENDIX H

MEANS, STANDARD DEVIATIONS, AND PROBABILITIES FOR NEWMAN-KEULS POST HOC ANALYSES

Means, Standard Deviations, and Probabilities

for Newman-Keuls Post Hoc Analyses

Low Luminescence

Factor	Mean	SD
1) Placebo or 0 mg./kg. bwt.	8.94	8.91
2) 2.5 mg./kg. bwt.	16.70	15.39
3) 5.0 mg./kg. bwt.	9.09	6.76

Factor	Probabilities	
	1	2	3
1		.06	.96
2			.03*
3			

*Significant, $p < .05$.

Moderate Luminescence

Factor	Mean	SD
1) Placebo	10.61	8.39
2) 2.5 mg./kg. bwt.	11.91	7.47
3) 5.0 mg./kg. bwt.	22.52	22.83

Factor	Probabilities	
	1	2	3
1		.77	.03*
2			.02*
3			

*Significant, $p < .05$.

Means, Standard Deviations, and Probabilities

for Newman-Keuls Post Hoc Analyses (Continued)

High Luminescence

Factor	Mean	SD
1) Placebo or 0 mg./kg. bwt.	7.74	6.31
2) 2.5 mg./kg. bwt.	14.26	20.07
3) 5.0 mg./kg. bwt.	10.30	13.83

No significance, $p > .05$.

Flicker Fusion

	Pre-Test		Post-Test		
Caffeine	Mean	Std. Dev.	Mean	Std. Dev.	P-Value
0	44.13	4.19	44.31	5.13	.87
2.5	38.74	6.77	41.56	4.47	.01*
5.0	42.06	6.25	42.66	4.51	.58

*Significant, $p < .05$.

Repetition Blindness

	Pre-Test		Post-Test		
Caffeine	Mean	Std. Dev.	Mean	Std. Dev.	P-Value
0	1.43	4.18	1.13	4.14	.80
2.5	5.13	9.84	1.83	4.88	.01*
5.0	1.65	4.27	.96	3.95	.94

*Significant, $p < .05$.

APPENDIX I

INSTITUTIONAL REVIEW BOARD (IRB) REVIEW FORM

OKLAHOMA STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD
HUMAN SUBJECTS REVIEW

Date: 02-08-95 **IRB#:** ED-95-025

Proposal Title: FLICKER FUSION, VISUAL THRESHOLD, AND REPETITION BLINDNESS IN RESPONSE TO TRIMETHYLXANTHINE (CAFFEINE) INGESTION

Principal Investigator(s): Bert Jacobson, Darla Fent

Reviewed and Processed as: Expedited

Approval Status Recommended by Reviewer(s): Approved

APPROVAL STATUS SUBJECT TO REVIEW BY FULL INSTITUTIONAL REVIEW BOARD AT NEXT MEETING.
APPROVAL STATUS PERIOD VALID FOR ONE CALENDAR YEAR AFTER WHICH A CONTINUATION OR RENEWAL REQUEST IS REQUIRED TO BE SUBMITTED FOR BOARD APPROVAL.
ANY MODIFICATIONS TO APPROVED PROJECT MUST ALSO BE SUBMITTED FOR APPROVAL.

Comments, Modifications/Conditions for Approval or Reasons for Deferral or Disapproval are as follows:

Revisions received and approved.

Signature:  Date: February 23, 1995

Chair of Institutional Review Board

VITA

Darla Renee Fent

Candidate for the Degree of

Doctor of Education

Thesis: LUMINESCENT THRESHOLD COMPARISONS, FLICKER FUSION DISCRIMINATION, AND REPETITION BLINDNESS IN RESPONSE TO TRIMETHYLXANTHINE (CAFFEINE) INGESTION

Major Field: Higher Education

Minor Field: Health, Physical Education, and Leisure

Biographical:

Personal Data: Born in Oklahoma City, Oklahoma, November 11, 1959, the daughter of Melvin R. and Mary Ann Fent.

Education: Graduated from Luther High School, Luther, OK, in May 1978; received Bachelor of Science degree in Health and Physical Education from Oklahoma City University in 1982; received Master of Science degree in Education from Baylor University in 1983; completed the requirements for the Doctor of Education degree at Oklahoma State University in July 1997.

Professional Experience: Graduate teaching assistant, Department of HPER, Baylor University, 1982-1983; graduate teaching assistant, Department of HPER, University of Oklahoma, fall 1983; teacher/coach, Choctaw High School, Choctaw, OK, 1984-1985; teacher/coach, Heritage Hall School, Oklahoma City, OK, 1985-1988; instructor, Department of HPE, Oklahoma City University, 1989-1991; graduate teaching assistant, School of HPEL, Oklahoma State University, 1991-1993; assistant professor, Department of HPE, Oklahoma City University, 1993 to present.

Professional Memberships: AAHPERD, OAHPERD, AAUP.