EFFECT OF NICOTINE AND MUSCLE PERFORMANCE USING A
WINGATE ANAEROBIC TEST ON COLLEGIATE FOOTBALL PLAYERS

Submitted by

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Thesis Approved

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June 12, 2006

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Advisor
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Abstract of Thesis

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ABSTRACT OF THESIS

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WINGATE ANAEROBIC TEST ON COLLEGIATE FOOTBALL PLAYERS

Nicotine is a naturally occurring addictive alkaloid and in some cases, a lethal 
drug. The long-term harmful effects of nicotine have been widely documented through 
means of publications, commercials and even billboards to stop the use of nicotine in the 
form of tobacco. Even with the knowledge of these harmful side effects, thousands of 
athletes still use tobacco. In this study the use of nicotinic substances was tested during 
Wingate Anaerobic Tests (WAnT) on collegiate football athletes. These tests had three 
possible outcomes: ergogenic, ergolytic or no effect. The subjects were 12 University of 
Wisconsin – Whitewater football players between the ages of 19 - 23. They performed a 
series of two tests on a Monarch cycle ergometer for 30 seconds at a time on two separate 
days. One day post-nicotine gum administration; the other day post-placebo gum 
administration. The data received was considered significant with a p-value ≤ .05. This 
experiment showed nicotine’s effect on: peak anaerobic power (P = .34), anaerobic 
capacity (P = .92), and anaerobic fatigue percentage (P = .33) in the human body during a 
WAnT. Therefore, the data received from this experiment were concluded not to be 
statistically significant.
CHAPTER I
INTRODUCTION

The use of nicotine is prevalent in today’s society. Nicotine is a colorless and potent liquid alkaloid naturally found in all tobacco plants. It is used in drugs such as: smokeless tobacco, cigarettes and cigars (Metz, Gregersen, & Malhotra 2004). There is much research suggesting that tobacco has many harmful long-term effects. Several studies have also shown that caffeine and creatine have improved muscular performance in the human body during exercise (Racette 2003; Spriet 1995), whereas, little has been published regarding nicotine’s effect on the human body during exercise conditions. Landers et al. (1992) reported that there are less than 30 articles published articles on smokeless tobacco use. Two-thirds of these articles concentrated on the health effects of smokeless tobacco. Only one-third of these articles illustrated the physiological and exercise performance effects. There is currently no published work on the effect nicotine has on the human body during a supramaximal exercise test such as the Wingate Anaerobic Test (WAiT).

Despite the lack of publications, the question remains as to why athletes continue to use these damaging tobacco substances? Landers, Crews, Boutcher, Skinner, and Gustafsen (1992) stated that despite the Surgeon General’s warnings to the general population that the use of tobacco products can be harmful and terminally fatal, many athletes believe that smokeless tobacco enhances athletic performance. This ergogenic tobacco theory stems from the belief that performance enhancement comes from
prevention of dry mouths, improvements in reaction time and concentration abilities, and euphoric calming effects for pre-participation rituals. The effects of nicotine use in the human body vary. However, in high doses it can produce detrimental effects. This experiment was designed to show the effects of nicotine on collegiate football players during a WAnT.

Effects at Rest and Exercise

The effects of nicotine in the body vary during resting and exercising conditions. Studies by Symons and Stebbins (1996) showed that nicotine infusions during resting states caused hypertension, decreased cardiac output, increased blood pressure and decreased heart rate. Also, it increased blood levels of epinephrine and norepinephrine, decreased urine output, increased satiety, and increased systemic and regional vascular resistance (e.g., left ventricle, kidneys, splanchnic organs). During exercise nicotine caused small elevations in myocardial oxygen demand, arterial pressure, systemic and regional vascular resistance (e.g., proximal colon and pancreas). It is believed that the vasodilators in the heart trumped the effects of nicotine known during resting conditions. The findings of this study suggested that the inhibiting effects of nicotine at rest are minimized during exercise.

Tobacco and Autonomic Effects

The study by Narkiewicz, van de Borne, Hausberg, Cooley, Winniford, Davison, and Somers (1998) showed the effects of smoking increased norepinephrine and increased blood pressure. The results of an increase in blood pressure produce arterial baroreflexes that exerted a protective effect by inhibiting sympathetic activation and
tachycardia that resulted from smoking. (It is unknown if this was the result of nicotine or other chemicals found in cigarette smoke.) The drug Nitroprusside was administered to decrease the blood pressure and block the arterial baroreflexors. The result of this test proved that smoking had a powerful sympathetic excitatory effect. This finding was comparable to Wolk, Shamsuzzaman, Svatikova, Huyber, Narkiewicz, and Somers (2005). They reported that muscle sympathetic nerve activity (MSNA) and norepinephrine concentrations had no change using smokeless tobacco. However, it did show an increase in heart rate, blood pressure and epinephrine by 50% of their subjects. Despite a small population (n=16), smokeless tobacco confirmed that it is a powerful autonomic stimulus.

Van Duser and Raven (1992) suggested smokeless tobacco had strong sympathetic nervous system stimulation as well. Their findings concluded smokeless tobacco decreased endurance performance by means of an increase in heart rate and plasma lactate concentrations. When plasma lactate concentrations were high it suggested that there was a demand for glycolytic energy production, because of reduced muscle blood flow in the smokeless tobacco subjects. Therefore if lactate levels rose in cardiac muscle it could have detrimental effects on individuals at risk from congenital heart disease.

**Nicotine and Psychomotor Performance**

Landers et al. (1992) showed ergogenic effects in regards to vigilance, rapid information processing, state dependent learning and retention of paired associates. Their report stated that nicotine use had no effect on psychomotor tasks, but did have an
enhancing effect on cognitively demanding stressor tests. Also noted were elevations in heart rate and blood pressure in smokeless tobacco users compared to non-users. Escher, Tucker, Lundin, and Grabiner (1998) attempted to demonstrate the significance between smokeless tobacco, reaction time and strength in athletes. The subjects were tested on a KinCom dynamometer for reaction time, maximum voluntary force and maximum rate of force generation of the knee extensors. The results showed that tobacco did not have any effect on reaction time, but had a significant effect on decreasing strength. The experimenters were unsure if the effect was a substance in the smokeless tobacco producing an ergolytic effect, or if tobacco withdrawal stimulates an ergogenic effect. Hindmarch, Kerr, and Sherwood (1990) provided a more extensive investigation using nicotine gum on a variety of psychometric tests. These tests included: choice reaction time, memory scanning, tracking and flicker fusion threshold. The results proved that additional nicotine gum provided smokers with an ergolytic effect in speed and accuracy of motor activity. In contrast, the non-smokers demonstrated no significant findings that skewed the results.

_Tobacco and Cardiovascular Effects_

The study by Bolinger, Noren, Wahren, and De Faire (1997) determined the effect of tobacco use during a cardiovascular and pulmonary functioning test in middle-aged men. No significant differences were found between middle-aged smokeless tobacco users and non-users. However, significant data supported that smokers showed lower maximal working capacity and oxygen uptake compared to non-users. This study hypothesized that recurring exposure to nicotine directly influences the autonomic
nervous system and thus results in an increasing risk of cardiac problems related with the heart. Overall this study showed no significant difference in performance associated from the effects of nicotine. However, it should be noted that the subjects in this study were firemen and other active individuals. Siegel, Benowitz, Ernster, Grady, and Hauck (1992) attempted to perform a similar test. This study’s findings indicated agreement with Bolinger et al. (1997) that there was minimal data concerning long-term cardiovascular effects in the test subjects, who were professional baseball players and exercised regularly. The moderate to high physical fitness of these individuals may have minimized the effectiveness of tobacco on the cardiovascular system.

**Physiology**

There are many philosophies that attempt to explain the nicotine effects in the body. Landers et al. (1992) declared the multiple physiological changes linked with the use of smokeless tobacco: “vasoconstriction; decreased peripheral circulation; increased secretion of antidiuretic hormone and catecholamines; and increased levels of blood lipids, plasma glucose, glucagon, insulin, and cortisol.” Some theories were conclusive, but others were inconclusive or even contradicted one another. That is why it is imperative to look at the physiology of the human body.

In the human body, nerve stimulation produces a strength stimulus. The peripheral nerves tell the central nervous system how much stimulus is needed to produce the intended response. To produce these responses neurotransmitters are necessary to aid in the speed and strength of the stimuli. Neurotransmitters travel in the electrical form of
action potentials from dendrites to cell bodies to axons or muscle fibers. Every axon has synapses with chemical ion channels that directly affect these action potentials.

Acetylcholine (ACh) is the main neurotransmitter that affects a muscle response (Shier, Butler, and Lewis 2003). When ACh binds to its receptors, it directly or indirectly causes an opening in ion channels. In most somatic cases this produces effects of depolarization called excitatory postsynaptic potential (EPSP). In autonomic cases this will produce a hyperpolarization called an inhibitory postsynaptic potential (IPSP). Whether ACh is excitatory or inhibitory is dependant on the muscle fiber type or the organ involved (Fox 1999). In a synapse, ACh receptors can be stimulated by the toxins nicotine and muscarine. Nicotine, which is found in tobacco, stimulates receptors called nicotinic ACh receptors (nAChRs). These nAChRs produce an excitatory response of ACh on skeletal muscle cells. Muscarine (a drug found in poisonous mushrooms) stimulates muscarinic receptors that produce an inhibitory response on muscle cells. The effect of one of these drugs will produce their respective ACh receptors to open ion channels to carry out its response (McArdle, Katch, and Katch 2001).

In the case of this experiment, nicotine allows the nACh receptors to bind to the neurotransmitter ligand and allow an influx of Na⁺. This influx causes a depolarization in the postsynaptic membrane to act as an EPSP. EPSP’s stimulate the postsynaptic cell to produce action potentials. The resting membrane potential depolarizes from –70 millivolts (Na⁺ into cell) to +30 millivolts and back again (K⁺ out of cell) when repolarization takes place milliseconds later. This action potential produces a “domino
effect” for more action potentials to take place down the axon until the intended response is complete (Pranzatelli 1999).

The amplitude of action potentials is “all or none.” This means that when depolarization is below a stimulus threshold the ion channels remain closed and an action potential will not occur. However, when it does reach threshold an action potential will reach the maximum amplitude. If one stimulus is greater than another, there will be a need for a greater frequency of action potentials to complete the greater stimulus response. Stimulus strength in the nervous system is frequency modulated because action potentials are “all or none” (Pranzatelli 1999). Li and Eisenach (2002) declared that nACHRs induce norepinepherine release. This results in a “feed-forward mechanism,” because norepinepherine release stimulates more ACh, and then in turn arouses more norepinepherine release.

*Wingate Anaerobic Test*

There are many measurements and tests evaluating energy sources in the human body. In this thesis anaerobic glycolysis was the primary energy source measured by the WAnT. Ayalon, Inbar, and Bar-Or first developed the WAnT in 1974 at the Department of Research and Sport Medicine of the Wingate Institute for Physical Education and Sport in Israel (Gullstrand and Larsson 1999). The WAnT may be performed on any braked bicycle ergometer, but the Monark bicycle ergometer is widely accepted as the ergometer of choice. The WAnT can be performed to measure muscular performance in both the upper and lower extremities.
Muscular performance is calculated anaerobically by peak power, mean power and percent fatigue. The WAnT is a supramaximal exercise for 30 seconds against a predetermined force load usually 7.5% of body weight in kilograms. This means that the subjects do not pace themselves and perform at 100% of their maximal effort. Every five seconds the pedal rate is measured against the force load, giving the data for the experiment. Peak power is measured in Watts as the highest interval of five seconds for the duration of the test. Mean power is the average of all intervals throughout the full 30 seconds. Percent fatigue is the rate of fatigue calculated from the difference of peak power compared to the lowest power output or end power. Gullstrand and Larsson (1999) reported eight investigations showing a mean reliability of $r=0.94\pm3\text{SD}$. This is in agreement with studies by Bar-Or (1987). Therefore, the WAnT is proven to be a reliable test.

**Assumptions**

This study made the following assumptions:

1. The nicotine levels in the nicotine chewing gum were high enough to affect the EPSP’s in a human body.
2. The conditions of the experiment were parallel between the two test days.
3. The test subjects were honest and followed the protocol before the experiment.
4. The WAnT was a valid and reliable anaerobic test.

**Purpose of the Study**

Football is a game of explosive activity and muscular strength. A normal play usually lasts only a few seconds. These short durations of play stress the importance of
anaerobic activity, or fast twitch muscle performance. A WAnT is a good test for football athletes because it is consists of all explosive actions and fast twitch muscular activity. The WAnT also parallels football because they both last short durations of time.

Severson, Klein, Lichtenstein, Kaufman, and Orleans (2005) declared that during the mid-1980’s and the early 1990’s the use of smokeless tobacco among professional baseball players was about double the percentage of the entire population. It is not only baseball players that use smokeless tobacco. Lombardo (1986) stated that one-third of Texas varsity football and baseball players used nicotinic substances in the form of smokeless tobacco.

When will athletes realize the harmful side effects from this fatal habit? Critchley and Unal (2003) reported that there will be up to 1,000 or more nicotine related deaths in the U.S. every year due to oral cancer. This mortality rate is nothing compared to India which sadly boasts up to 10,000 or more oral cancer related deaths accredited to smokeless tobacco use alone. Since athletes still use tobacco substances today, we tested the effects of nicotine in the human body. There is minimal research concerning the effects of nicotine during exercise. All research was inconclusive based on the variety of results showing ergogenic, ergolytic and no significant statistical difference. Therefore this experiment illustrated the effects of nicotine on anaerobic exercise during a WAnT.
Subjects

Twelve University of Wisconsin-Whitewater intercollegiate football players were tested using a WAaN on two separate days (N=12). All test subjects were male and Caucasian. One day consisted of administering nicotine gum and the other day a placebo was administered. Each subject was given a one week period of recovery between tests.

Research Design and Procedure

This was a single-blind experiment (test-retest). Therefore, only the researchers knew which gum was administered for each day. The statistical analysis was performed using a paired student’s $t$-test. Testing was performed on Monark bicycle ergometers (Monark 834E). The procedures required a subject, a timer and two counters for each test. Each test subject wore a t-shirt, athletic shorts, and athletic shoes. The subject randomly was administered a piece of chewing gum by the researcher. The subject chewed the piece of gum for 30 minutes to allow optimal peak time for the possibility of 4 mg nicotine to enter the bloodstream. This is in agreement with clinical studies performed by Van Duser and Raven (1992). During the time of chewing the gum the test subject had his weight taken and subsequently the brake force set up on the Monark cycle ergometer.

The seat height was adjusted allowing a slight bend in the knee when the pedals were at the bottom of the revolution. Then the subject had a two minute warm-up cycling
with no resistance at a moderate pace. The force was then applied by dropping the weight holder. The timer began counting with a stop watch for 30 seconds and called out time intervals every five seconds. As the timer counted the subject was encouraged to keep pedaling as hard as he could by the researcher, timers and counters for the duration of the experiment. One counter counted pedal revolutions during the first, third and fifth time intervals. The other counter counted pedal revolutions during the second, fourth and sixth time intervals. After the 30 seconds were up the resistance was removed from the Monark cycle ergometer and the subject cooled down until he was comfortable with stopping. The data was used to calculate peak muscle power, average muscle power and rate of fatigue. One week later, the subject reproduced this experiment using the other piece of nicotine or placebo gum that was not administered during the first test.
CHAPTER IV
RESULTS

The purpose of this study was to determine if nicotine had a positive, negative, or no effect on muscle performance in the human body during a WAnT. The literature review showed that previous research was inconclusive. The results of this study showed the effects on non-tobacco using collegiate football players during a WAnT. The ages, height, weight and brake weight are shown in Table 1. Brake weight was the prescribed force used as resistance for the WAnT calculated by 7.5% multiplied by the body weight (kg) of the test subjects and rounded to the nearest 0.5 kg. This formula was in agreement with the experiment done by O’ Kroy (2000).

<table>
<thead>
<tr>
<th>Test Subjects (N=12)</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>20.75</td>
<td>1.356801</td>
<td>19-23</td>
</tr>
<tr>
<td>Height (in)</td>
<td>71.22917</td>
<td>2.489657</td>
<td>66-75.5</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>92.54167</td>
<td>12.33247</td>
<td>74.5-105.5</td>
</tr>
<tr>
<td>Brake Weight (kg)</td>
<td>7.083333</td>
<td>0.900337</td>
<td>6-9</td>
</tr>
</tbody>
</table>

Table 1: Demographics of the participants.

Table 1 reports the demographics of the 12 test participants. There were slight differences in the ages and brake weights of the subjects, but there were significant differences in the heights and weights of the subjects.
Figure 5.1: Results for peak anaerobic power.

Figure 5.1 presents the data collected from all twelve subjects on peak anaerobic muscle power comparing the effects of nicotine verses a placebo. Fifty percent of the Subjects (6 of 12) showed an increase in peak anaerobic power during the nicotine trial. Twenty-five percent of the subjects (3 of 12) had no difference in peak anaerobic power output. Lastly, 25% of the subjects (3 of 12) showed a decrease in muscle power during the nicotine gum experiment in comparison with the placebo gum.

Table 2 reports a wide variety of peak anaerobic power values obtained from a paired student’s t – test. The values showed that the mean scores were higher by 47 Watts in the nicotine gum trials. Standard deviation values were also more prominent in the nicotine gum with a greater difference of 14 Watts. The p-value was greater than .05 (P = .34). Therefore, one cannot claim this as statistically significant data.

Figure 5.2 presents the data collected from all twelve subjects on anaerobic
<table>
<thead>
<tr>
<th>Nicotine Gum</th>
<th>Placebo Gum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>937</td>
</tr>
<tr>
<td>95% CI</td>
<td>825.3 - 1049</td>
</tr>
<tr>
<td>SD</td>
<td>176</td>
</tr>
<tr>
<td>Median</td>
<td>882</td>
</tr>
</tbody>
</table>

| t-score | 1 | DF   | 11  | P-value | 0.34 |

Table 2: Comparison of peak anaerobic power values for nicotine and placebo trials.

![Anaerobic Capacity](image_url)

Figure 5.2: Results for anaerobic capacity.

capacity or mean anaerobic muscle power output. The effects again show the values of nicotine in comparison with a placebo. Fifty percent of the subjects (6 of 12) showed an increase in anaerobic capacity during the nicotine trial. In contrast, 50% of the football players (6 of 12) showed a decrease in anaerobic capacity during the nicotine trial.
### Table 3: Comparison of anaerobic capacity values for nicotine and placebo trials.

<table>
<thead>
<tr>
<th>Nicotine Gum</th>
<th>Placebo Gum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>Mean</td>
</tr>
<tr>
<td>696</td>
<td>694</td>
</tr>
<tr>
<td>95% CI</td>
<td>95% CI</td>
</tr>
<tr>
<td>626 – 765.7</td>
<td>617 – 771</td>
</tr>
<tr>
<td>SD</td>
<td>SD</td>
</tr>
<tr>
<td>110</td>
<td>121</td>
</tr>
<tr>
<td>Median</td>
<td>Median</td>
</tr>
<tr>
<td>679</td>
<td>686</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>t-score</th>
<th>DF</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>11</td>
<td>0.92</td>
</tr>
</tbody>
</table>

As seen in Table 3, the values for anaerobic capacity were similar. The mean scores were almost identical with nicotine scores edging placebo scores by a difference of 2 Watts. The nicotine standard deviation scores were 11 Watts smaller than the placebo. Because the resulting p-value of 0.92 was far from the 0.05 level of significance, this data was not statistically significant.

Figure 5.3 presents the data collected from all twelve subjects on anaerobic fatigue. This comparison between nicotine gum and placebo gum was based on the percentage of decline in performance from the highest to lowest five second intervals throughout the 30 second experiment. A lower percentage rate for the experiment indicated a better effect of maintaining performance; a higher percentage rate indicated decrease in muscle performance. Fifty percent of the subjects (6 of 12) showed a greater percentage loss in the nicotine gum trial. Forty-two percent of the subjects (5 of 12) showed a lower percentage from nicotine gum. Finally, eight percent of the subjects (1 of 12) showed no difference from both trials of anaerobic fatigue.

As seen in Table 4, the values are comparable. The subjects had a larger drop off
Figure 5.3: Graph results for percentage of anaerobic fatigue.

in anaerobic fatigue from highest peak interval to lowest end interval when the percent score was greater. Consequently, mean scores were higher in nicotine gum by a difference of 4%. Nicotine gum had a lower standard deviation by 2%. The resulting p – value was 0.33.

<table>
<thead>
<tr>
<th>Nicotine Gum</th>
<th>Placebo Gum</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean</strong></td>
<td><strong>Mean</strong></td>
</tr>
<tr>
<td>47%</td>
<td>43%</td>
</tr>
<tr>
<td><strong>95% CI</strong></td>
<td><strong>95% CI</strong></td>
</tr>
<tr>
<td>42% – 53%</td>
<td>35% – 50%</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td><strong>SD</strong></td>
</tr>
<tr>
<td>9%</td>
<td>11%</td>
</tr>
<tr>
<td><strong>Median</strong></td>
<td><strong>Median</strong></td>
</tr>
<tr>
<td>48%</td>
<td>40%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>t-score</th>
<th>DF</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.01</td>
<td>11</td>
<td>0.33</td>
</tr>
</tbody>
</table>

Table 4: Comparison of percentage of anaerobic fatigue in nicotine and placebo trials.
CHAPTER V
DISCUSSION

In a world where athletes are trying to gain an edge on the competition, it is apparent that athletes continue supplemental use of ergogenic aids. Ahrendt (2001) reported that 76% of college athletes currently used or had tried some form of supplement. In a survey performed in 1996, 50% of the general population had experimented with some form of supplement. It was also revealed that 100% of body builders used supplements. This data gives evidence that an athlete’s quest for obtaining an edge on competition is limitless.

Despite the warnings and potential side effects of some supplementals, athletes continue to use ergogenic aids. When will this madness end? What can be done to prevent athletes from potentially harming their bodies? Presently we see Major League Baseball beginning to put limitations on some of these substances, as well as implementing penalties for the use of these substances. San Francisco Chronicle reporters Mark Fainaru-Wada and Lance Williams wrote the book *Game of Shadows* (2006) to try and expose professional athletes and their involvement in using some of these illegal ergogenic aids. Most notably they attacked public figures Barry Bonds, Mark McGwire, Jason Giambi and Marion Jones with allegations of their illegal drug use. These public figures were portrayed as role models by the media. Unfortunately, their actions in these allegations send a horrifying message to aspiring athletes:
tomorrow.: that one must use ergogenic aids if one aspires to become an elite professional athlete.

Some athletes perceive nicotine as an ergogenic aid. Lombardo (1986) stated that the occurrence of nicotine use in sports was designed to achieve stimulation, relaxation, and weight control. Landers et al. (1992) described that athletes also believe that smokeless tobacco enhances performance by preventing dry mouths, improving concentration, improving reaction time and providing an arousal effect. If nicotine does provide athletes with these effects, should there be a ban on these nicotinic substances to provide a level playing field?

The primary purpose of this experiment was to analyze statistical significance of the effects nicotine had on muscular performance in collegiate football players. Muscular performance was identified and measured in this experiment by peak anaerobic power, anaerobic capacity, and percentage of anaerobic fatigue. A p-value of less than 0.05 was considered statistically significant. However, in these findings all three categories measured were considered not statistically significant due to p-values greater than 0.05. This data is in agreement with research done by Bolinger et. al. (1997), Hindmarch et. al. (1990), Landers et. al. (1992), and Siegel (1992). These findings are in contrast with research done by Escher et. al. (1998) who believed that smokeless tobacco decreased muscular strength. Though conflicting evidence is apparent one must look at the limitations of this experiment. Only 12 male collegiate football players from the University of Wisconsin – Whitewater were tested. The subjects may have failed to follow the research protocol properly. They also may have had physical injuries that
limited their maximal abilities. The subjects may have performed better on the second
day due to a learning effect. Finally, there may have been human error from the manual
pedal revolution counting by the researchers throughout the test.

In the future it is recommended to further investigate the effects of nicotine and
muscular performance. Recommendations for future research on nicotine and muscle
performance include:

1. Increasing the sample size. This will ensure a larger validity of the experiment.
2. Using cycle ergometers with computerized systems that automatically count
   pedal rate and determine the results to prevent human error.
3. Using a variety of subjects from different sports, races and genders.
4. Comparing the effects of nicotine users and non-users.
5. Improve validity and other factors that may influence an effect using nicotine.
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