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Caffeine, Mental Health, and Sleep Quality in Students: A Mediation Approach

Presented to the faculty of Lycoming College in fulfillment of the requirements for
Departmental Honors in Psychology

by

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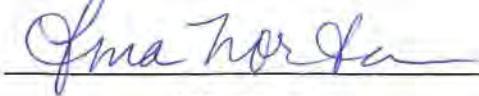
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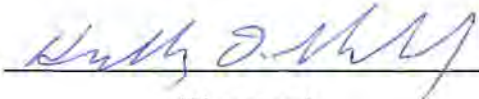
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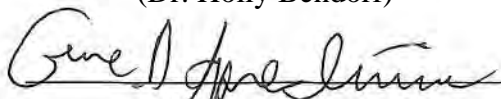
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Caffeine, Mental Health, and Sleep Quality in Students: A Mediation Approach

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Abstract

This study assessed a population of freshman students matriculating at a residential private liberal arts institution, providing a unique glimpse at a population not traditionally represented in health behaviors literature. The current study used data collected through the study entitled “Adjustment to College in Emerging Adults: Changes in Health Behaviors as Possible Mediating Factors,” to examine how sleep affects caffeine use with mental health as a mediating variable. Based on the literature review, it was predicted that the relationship between sleep duration and caffeine is mediated by the influence of depression and anxiety; that college students who report a lower sleep duration increase their caffeine consumption; that lower sleep duration increases signs of depression and anxiety; and that symptoms of depression and anxiety increase caffeine consumption. A significant relationship was found between sleep duration and caffeine consumption with depression as a mediating factor ($\Delta R^2 = 0.02$, S.E. = 0.214, $\beta = 0.27$, $p \leq 0.01$). Another marginal relationship was found between sleep duration and caffeine consumption with anxiety as a mediating factor ($\Delta R^2 = -0.01$, S.E. = 0.241, $\beta = 0.184$, $p \leq 0.1$). Sleep duration acted as a predictor variable to caffeine consumption in both models (S.E. = 0.23, $\beta = 0.197$, $p \leq 0.05$). Neither depression nor anxiety significantly predicted caffeine consumption ($p \geq 0.1$). In summary, the inclusion of depression improved prediction of caffeine consumption by sleep duration. Further, sleep duration had a direct, significant relationship with caffeine consumption. Future research should address other factors that contribute to caffeine consumption and the impact of health behavior changes on transitioning college students.

Caffeine, Mental Health, and Sleep Quality in Students: A Mediation Approach

Caffeine is considered the most widely consumed psychoactive substance available today. In the 5-18 age group, caffeine is estimated to be consumed weekly by 98% of individuals (Lee-Chiong, 2006). High caffeine intake has been associated with shorter sleep duration at night and increased sleeping during the daytime, showing a noted decline in sleep quality due to the influence of caffeine. In college students, a number of factors can increase the competition for daytime and even nighttime hours such as the demands of college work, athletics, after-school activities, newfound autonomy and working while in college (Kloss, Nash, Horsey, & Taylor, 2011). Because of these and many other factors, it was estimated that over 70% of US college students experience some sleeping difficulty on a regular basis. Consequences of sleep deprivation in this group include poor academic performance, increased risk of injury and death, behavioral problems, and increased use of drugs and other substances (including caffeine) (Lee-Chiong, 2006). Depression is often overlooked in the college student population, and connections between sleep quality and signs of depression have been found and may represent an overlooked cycle of sleep debt accumulation, self-medication through caffeine, and developing psychological symptoms of depression. A study by Whalen et al. noted that the connection between caffeine, sleep habits, and depression in youth is a crucial undertaking despite little empirical research on the topic (Whalen et al., 2007). The current study examined sleep quality and its relationship with caffeine and mental health in order to establish a mediation relationship. In order to begin to understand this relationship, one must first examine scientific literature regarding caffeine, sleep quality, and depression with particular regard to their patterns and influences on the college student population, which is the population of interest in the current study.

Caffeine

Caffeine's Neurological Influence

Caffeine is considered to be a mild stimulant to the central and autonomic nervous system and is thought to block adenosine receptors (Maisto, Galizio, & Connors, 2011). There are four types of adenosine receptors— A_1 , A_{2A} , A_{2B} , and A_3 (McKim, 2007). Caffeine and other methylxanthines seem to block the A_1 and A_{2A} types effectively (McKim, 2007). Adenosine is created naturally by the body and functions as a neuromodulator, inhibiting the release of many neurotransmitters presynaptically (Leonard, Watson, & Mohs, 1987, McKim, 2007). Caffeine prevents the inhibitory actions of adenosine by binding to the two aforementioned receptors, although it also can increase the activity of the D_2 dopamine receptor and, at very high levels, block benzodiazepine receptors (benzodiazepine functions much like adenosine) (McKim, 2007). By blocking adenosine receptors which inhibit many neurotransmitters, caffeine has a double negative effect, which means it inhibits an inhibitory factor (Levinthal, 2012). One effect of caffeine is to indirectly raise levels of epinephrine from the adrenal gland, which explains its stimulant properties (McKim, 2007). Adenosine A_{2A} receptors are found in high quantities in dopaminergic-rich areas, which provides another possible explanation for stimulant properties of caffeine (Lorist & Tops, 2003). It seems that neurological effects are consistent among the adult age groups (18+), so college students seem to be affected much the same way (Blanchard & Sawers, 1983).

Caffeine Consumption

The stimulant is most often consumed orally, and absorbed in the gastrointestinal tract. Oral caffeine consumption is also the most common in college students (James, Kristjánsson, &

Sigfúsdóttir, 2010). Caffeine quickly reaches the brain because of its ability to pass through the blood-brain barrier, but it is equally distributed through the body's total water and stays in similar concentration throughout the body because of this (Maisto, Galizio, & Connors, 2011). The maximal plasma caffeine concentration in the human body after oral consumption is 8-10 mg/L and occurs 15-120 minutes after ingestion (Nehlig, 1999). Peak effects of caffeine usually occur 15 to 45 minutes after ingestion, but this can vary based on the time the person takes the stimulant (food present in the stomach can delay absorption) as well as the source of the caffeine (Barry, Clarke, Johnstone, & Rushby, 2008, Maisto, Galizio, & Connors, 2011).

Caffeine metabolism. The metabolism of caffeine occurs primarily in the liver, where the majority of caffeine undergoes 3-methyl demethylation which converts the stimulant into paraxanthine (Arnaud, 1987). Rate of metabolism depends largely on the individual's frequency of caffeine ingestion. People who consume caffeine less frequently tend to have a slower rate of metabolism, and therefore feel the effects of caffeine over a greater period of time. Other factors can also contribute to this rate, such as smoking habits, liver disease, the use of oral contraceptives, and pregnancy (Maisto, Galizio, & Connors, 2011). A national study conducted in 2000 found that nearly one third of college students reported regularly smoking, which can largely affect the stimulant properties of caffeine (Rigotti, Lee, & Wechsler, 2000). Smokers and consistent caffeine drinkers may need to consume a higher amount of caffeine for optimal performance, whereas non-caffeine drinkers require a much lower dose (Kourtidou-Papadeli, Papadelis, Louizos, & Guiba-Tziampiri, 2002).

Caffeine as a drug. Caffeine is considered to be a safer drug than many other stimulants, and can be consumed widely and regularly without many immediate negative side effects (Giles et al., 2012, Maisto, Galizio, & Connors, 2011). Caffeine has shown to impact blood pressure in

non-caffeine consumers, where caffeine does not normally influence regular consumers' blood pressures (Kourtidou-Papadeli, Papadelis, Louizos, & Guiba-Tziampiri, 2002). Safe consumption of caffeine for healthy adults ranges between 400 and 450 mg/day, but most adults consume between 106 to 170 mg/day; widely considered a safe range (Knight, Knight, Mitchell, & Zepp, 2004). College students have been shown to drink as high as an average of over 800 mg/day, nearly twice the amount considered to be safe (McIlvain, n.d.). There are a variety of ways caffeine can be consumed including through tea, cocoa, coffee, energy drinks, and even in some pre-workout pills (Bell & McLellan, 2002). Oral consumption of caffeine is the most prevalent method in the adult and college student population.

Caffeine tolerance and withdrawal. College students are a risk population for developing caffeine tolerance as their frequency of consumption increases, and may even be open to caffeine withdrawal symptoms once their habits begin to change. Caffeine is a drug that may meet DSM-IV criteria for drug dependence in some circumstances (Maisto, Galizio, & Connors, 2011). The DSM-IV requirements for substance dependence include any three or more of the following criteria: tolerance, withdrawal, consumption in increasingly larger amounts or in a longer time frame than intended, a persistent desire or unsuccessful efforts to curb substance use, a large amount of time required to acquire, use, or recover from the effects of the substance, important social, occupational, or recreational activities are reduced because of substance use, or the substance use is continued despite knowledge of having a persistent physical or psychological problem that is likely to have been caused or exacerbated by the substance (American Psychiatric Association, 1994). People who experience caffeine withdrawal most often report headache and fatigue, although in more severe cases symptoms such as depression, increased irritability, decreased activity and energy, greater drowsiness, and decreased alertness

have been reported (Juliano & Griffiths, 2004). Caffeine tolerance seems to affect functions such as sleep, renal function, blood pressure, and heart rate, but tolerance does not seem to affect caffeine's properties as a stimulant, though research on this matter is scattered and contains some measure of experimental error. Many college students have reported feeling one or more signs of caffeine tolerance or withdrawal (McIlvain, n.d.).

Central Nervous System Effects

The central nervous system effects of caffeine have been shown to elevate mood, leaving consumers feeling confident, alert, efficient, and energized (Haskell, Kennedy, Milne, Wesnes, & Scholey, 2008). The popularity of caffeine in many cultures and ages is attributed to these effects. However, performance improvement due to caffeine consumption is unsupported and depends on the amount of caffeine consumed, the personality of the subject, and the time of day. Despite this, positive correlations with physical and mental performance show that caffeine aids the people who consume it by reducing their fatigue so that performance never drops below what is typical for that person (Maisto, Galizio, & Connors, 2011). This may be another reason for caffeine's widespread popularity. Many users (including college students) also consume caffeine for its therapeutic purposes because it reduces symptoms found in asthma, headaches, colds, and menstruation. Because of these properties, caffeine is also included in many medications to reduce possible side effects, especially reducing headaches by constricting blood vessels in the brain (Maisto, Galizio, & Connors, 2011, McKim, 2007). Due to the demands of a typical college student such as adjusting to an independent lifestyle, caffeine acts as a particularly important method of self-medication for increased concentration and wakefulness, leading to greater and more frequent consumption.

Sleep

Humans spend a large amount of time sleeping, and so sleep studies are prevalent in the biological and psychological sciences. Although sleep mechanisms have not been fully explored, research indicates that sleep is used to maintain neuronal homeostasis and that sleep is a unique brain state as found by EEG (Maloletnev, 2002). Sleep is divided into two parts: the first is referred to as NREM, or non-rapid-eye-movement sleep, and the second is REM, or rapid-eye-movement sleep. Before these stages can be further explained, it is important to know how neurologists use EEGs to measure sleep.

EEG in Sleep

The parts of a sleep cycle were discovered through the use of EEG, or electroencephalogram. There are many types of EEG waves that are associated with sleep. To begin, an individual who is awake and alert shows beta waves (Pinel, 1992). Alpha waves are associated with relaxation, typically occurring as individual experiences drowsiness. Stage 1 and 2 sleep (light sleep) are characterized by theta waves, and are differentiated by sleep spindles and K-complexes (as a person moves into stage 2). The next two stages, Stage 3 and 4, occur as the individual develops more delta waves and falls into a deeper sleep (Stage 4 occurs as delta waves become more prevalent). All of the aforementioned wave patterns are associated with NREM sleep. REM sleep involved alpha, beta, and even nonsynchronized waves. A full cycle of sleep begins with Stage 1, moves through to Stage 4, returns back through the stages until 1 is replaced by REM, then repeats. As the sleep cycle continues, REM and Stage 4 sleep is lengthened until Stage 4 stops altogether (Pinel, 1992). This cycle occurs roughly every 90 minutes, and about 4 to 6 full cycles are completed in a sleep episode (Lee-Chiong, 2006).

Importance of NREM in College Students

The vast majority of sleep is considered to be NREM, with estimates at 75-80% of sleep time. During NREM sleep, the sympathetic activity of the autonomic nervous system (ANS) is decreased and the parasympathetic activity is increased. This change in the ANS is crucial during NREM sleep in order to form REM sleep, and also for the body to change focus to repairing cells, digestion, and energy conservation (Lee-Chiong, 2006). The basis of NREM sleep is thought to be bodily rest (Stenberg, 2007). College students may benefit from the memory enhancing portion of NREM sleep. A Harvard study was conducted to find the basis of the benefits of NREM sleep. The study was test-retest formatted so the students all were asked to perform on a number of declarative memory tasks (involving conscious recall). Students were randomly assigned to two groups—one who were asked to nap for 45 minutes (considered to be a “NREM-only” nap) after the first test and another who did not get the chance to nap. The study found that the students who were allowed to nap for 45 minutes did significantly better on the second test than the students who didn’t nap if both groups exhibited mastery of the word-pairs task during the first test (Tucker & Fishbein, 2008). This study supports the idea that sleep aids in the formation of declarative memory, which is an important type of memory to college students who use declarative memory to study, perform on exams, and participate with their professors.

Importance of REM in College Students

REM sleep is considered the most interesting and important stage in sleep. The name REM comes from the associated rapid eye movement of this sleep stage, which is measured by a device called an EOG, or electrooculogram (Pinel, 1992). REM sleep is characterized by

muscular systems being nearly paralyzed, yet the brain is nearly awake. Dreaming occurs during REM sleep, and the parasympathetic nervous system “awakens” intermittently (Pinel, 1992, Lee-Chiong, 2006). REM sleep in college students has been noted to be exceptionally important for memory consolidation, especially after an intensive learning session (Buboltz, Brown, & Soper, 2001). Students who slept for at least 8 hours after learning had a significant increase in test scores the next day. In particular, the last two hours of sleep in an 8-hour cycle seem to be the most important for memory consolidation in college students.

Dangers of Poor Sleep Quality

While there is an abundance of research done on adolescent sleep patterns, there is a notable decline when it comes to college-age students and emerging adults (mean age 20). In a survey done at a private Midwestern university of 1,125 students, only about 30% of students reported getting 8 hours of sleep or more per night, which is considered to be the amount needed during this developmental phase (Lund, Reider, Whiting, & Prichard, 2009). One important thing to note that is unique among this age group is the overwhelming tendency to have restricted, inadequate sleep patterns during the school week yet have overwhelmingly sleep-intensive weekends. This is suggested to be due to the phenomena of sleep debt, which essentially states that when an individual has multiple nights lacking sleep in a row, they will require more sleep than normal to account for this deficiency (Lee-Chiong, 2006). Nearly 20% of female college students report an average weekly sleep debt of 2 hours or more, and female college students have been shown to be more at risk for poor sleep quality (Regestein et al., 2010, Tsai & Li, 2004). Lund and colleagues’ study and similar studies have indicated that most college students suffer from a chronically restricted sleep pattern (Lund, Reider, Whiting, & Prichard, 2009). There are a number of consequences connected to restricted sleep patterns in college students.

Lack of quality sleep has been correlated to an increase in alcohol drinking-related behavior, and even has been positively connected to an increase of incidence of alcohol-related consequences (Kenney, LaBrie, Hummer, & Pham, 2012). Additionally, sleep deprivation can lead to driving accidents, depression, and lower academic performance (Taylor & Bramoweth, 2010).

Depression

Criteria for a major depressive episode include at least five of the following categories representing a change from typical, with at least one being depressed mood or loss of interest or pleasure: depressed mood, loss of interest or pleasure nearly every day, significant weight loss or gain while not dieting, insomnia or hypersomnia nearly every day, psychomotor agitation or retardation nearly every day, fatigue or loss of energy nearly every day, feelings of worthlessness or excessive or inappropriate guilt nearly every day, diminished ability to think or concentrate, or indecisiveness nearly every day, or recurrent thoughts of death, recurrent suicidal ideation with or without a plan, or an attempted suicide (American Psychiatric Association, 1994). Nearly 11% of college students met these requirements for depression (Hunt & Eisenberg, 2010).

Additionally, a survey of a large public university found that 12-15% of students screened positively for depression (Zivin, Eisenberg, Gollust, & Golberstein, 2009). The overall prevalence may be higher in college students than similarly aged non-college-attending adults whose prevalence for a Major Depressive Episode is at 10.1% (Substance Abuse and Mental Health Services Administration, 2008). A 2009 NHCA survey of over 80,000 students agrees, noting that nearly 15% of college students had a diagnosis of depression within their lifetime, which may represent only a small portion of students affected by the disorder including those remaining undiagnosed (Buchanan, 2012). The World Health Organization found that mental disorders accounted for one-half the disease burden of college-age young adults ("The global

burden of disease: 2004 update," 2008). Untreated mental illness in young adults has significant implications for productivity, social relationships, academic success, and substance use (Hunt & Eisenberg, 2010). Females are at a higher risk for Major Depressive Disorder (MDD) than males (Lee-Chiong, 2006).

Depression and Higher Caffeine Consumption

Caffeine is considered a source of medication for depressed patients, particularly because it blocks the adenosine receptors and influences the dopaminergic system. Anecdotally, many patients use caffeine in their daily lives to self-medicate for depression, but caffeine's use as a prescribed treatment option is still under scrutiny (Kale, Addepalli, Bafna, & Prabhavalkar, 2010). A study done in Japan found that green tea and coffee drinkers had a significantly lower rate of depression than the normal population, which could be indicative of this self-medication (Pham et al., 2013). Another study by Bergin and Kendlar looked into the genetics of caffeine consumption and mental health and found Major Depressive Disorder (MDD) to be genetically correlated to caffeine use by a degree of 0.38, indicating depressed patient's use of caffeine (2012). MDD also correlated highly with caffeine tolerance, suggesting repeated use by those with MDD. An interesting other finding of this study was an even higher genetic correlation between Generalized Anxiety Disorder (GAD) and caffeine consumption, at 0.48, suggesting that those suffering from high anxiety may also be more influenced to use caffeine.

Current Study

The purpose of the current study was to address whether sleep predicted caffeine consumption. A second question concerned whether mental health variables mediated a relationship between sleep and caffeine. While the connection between caffeine consumption and

sleep received much attention in the literature, the inverse relationship was of interest for the current study. This literature is reviewed in the following paragraphs.

Caffeine's Effect on Sleep Quality

Caffeine's impact on sleep quality is popular in research because it is an influence that can be manufactured in a laboratory setting with minimal risk. Caffeine is linked with decreased frequency of alpha, beta, and theta waves during sleep (Barry, Clarke, Johnstone, & Rushby, 2008). However, it has also been shown that this may not be due to decreased wave frequency, but instead the attenuating effect of caffeine on EEG markers (Landolt, 2008). Caffeine also produces a significant increase in latency of sleep onset (Drake, Jefferson, Roehrs, & Roth, 2006). This research indicates that caffeine delays sleep onset in general, including the amount of time it takes a person to fall asleep. It also has been shown to increase circadian rhythms in mice, making their nighttime sleep pattern decline in total hours significantly (Oike, Kobori, Suzuki, & Ishida, 2011). In summary, caffeine seems to primarily affect the stability of wakefulness by decreasing the total sleep time of individuals and increasing the amount of time it takes to fall asleep (McKim, 2007).

Yet another way caffeine impairs sleep quality is through the reduction of the acoustic arousal threshold, which is the amount of noise it takes to wake a sleeping person. This means that an individual who consumes caffeine may find that they wake up more often during the night due to noise that would not usually rouse them (McKim, 2007).

A possible reason for caffeine's effect on sleep stems from the role of adenosine. A Harvard study indicated that adenosine is a homeostatic sleep factor, meaning that it plays a role in maintaining the body's natural sleep cycle (Basheer, Strecker, Thakkar, & McCarley, 2004).

This is likely achieved through high levels of adenosine after prolonged wakefulness, according to the study. The most important structure to this process of mediating sleep cycles is the cholinergic basal forebrain, which serves as the center of adenosine mediation through inhibition of A₁ wakefulness receptors, controlling the extent of adenosine's influence. However, A_{2A} receptors found below the rostral forebrain may also have an important role; especially in the effects of adenosine that involve the prostaglandin D₂ (PGD₂) receptors, a group of regulatory lipid receptors. These are especially interesting because the PGD₂ receptors are found in two locations; the brain, as expected, but also in mast cells. Mast cells are noted for their role in allergies because they may contain granules of histamine, however, an important role of mast cells is to repair wounds and defend against pathogens (Prussin, 2003). This connection, although not yet fully understood, may indicate an association between inhibiting adenosine and poor cell repair and weakened immune system. Studies have indicated that caffeine is linked to lesser antibody production, anti-inflammatory effects, and suppression of lymphocyte function (Horrigan, Kelly, & Connor, 2006). These findings indicate that caffeine's impact on sleep produce health problems by inhibiting proper immune functioning during sleep.

In college students, caffeine is used to self-medicate against poor sleep quality (Lund, Reider, Whiting, & Prichard, 2009). A student may stay up late to finish an assignment or a number of other factors, and in order to maintain wakefulness and concentration the next day get a cup of coffee before classes begin. This pattern of self-medication may possibly only exacerbate the key problem of lack of sleep quality in college students as a whole by decreasing their sleep quality for the next night, which increases their sleep debt. Another piece of supporting evidence claims that a significant reduction in caffeine consumption may increase

sleep quality in persons already prone to sleep pattern disturbances (Dreher, 2003). Thus, by ceasing caffeine use, sleep quality has been shown to significantly improve.

Sleep Quality's Effect on Caffeine Consumption

Due to ease of experimental methods, examining how caffeine affects sleep quality is very popular in modern literature. Unfortunately, the reverse effect, how sleep quality affects caffeine consumption, has been largely understudied. In an experiment involving middle-aged women, it was found that poor sleep quality led to significantly greater caffeine consumption (Hollander et al., 2001). In one study on college students, nearly 68% of the students who reported ever consuming an energy drink reported doing so because of insufficient sleep (Malinauskas, Aeby, Overton, Carpenter-Aeby, & Barber-Heidal, 2007). These data represent the only research that was found regarding how poor sleep quality may increase caffeine consumption. Despite this lack of research, it is often assumed that the patterns of college students involving poor sleep quality leads to greater caffeine consumption as a way to self-medicate against sleepiness. Medicating caffeine consumption begins as early as adolescence, with nearly 76% of the high school students who reported falling asleep in class revealing their use of caffeine to self-medicate for tiredness often caused by using electronic devices such as cell phones, mp3 players, and computers long into the night (Calamaro, Mason, & Ratcliffe, 2009). As aforementioned, the lifestyle of the average college student leads to the continuation of this pattern. For instance, participation in organized college activity was shown to be a predictor of increased caffeine consumption in a study where increased wakefulness was cited by participants as a main goal of consumption (McIlvain, n.d.). Due to this literature and anecdotal evidence, it was proposed that poor sleep quality may lead to higher caffeine consumption in college students, giving the current study a unique perspective on these health behaviors.

Sleep Quality's Effect on Depression

Another interest of the current study is examining the role of mental health, particularly with sleep quality. Lack of sleep is a key feature in depression. Major depressive disorder was associated with less sleep in stages 3 and 4, a prolonged stage 1, an early onset of REM sleep (usually within 60-70 minutes, whereas adults without MDD usually have an onset of REM around 80-120 minutes), a prolonged first period of REM sleep, and wakefulness after sleep onset. The most stable sleep finding regarding adolescents was prolonged sleep latency (Lee-Chiong, 2006). Daytime sleepiness caused by caffeine and other licit substances may be an important cause of negative impact on academic achievement due to substance use, as found in a survey of over 7,000 Icelandic adolescents (James, Kristjánsson, & Sigfúsdóttir, 2010). Additionally, accruing a sleep debt of two hours or more has been linked to increased melancholic symptoms, and depression was considered to be an affective symptom of sleep debt (Regestein et al., 2010). A shift in sleep cycle by as little as two hours has been documented to significantly increase depression even when the duration of sleeping time was not changed (Buboltz, Brown, & Soper, 2001).

Given demonstrated relationships between sleep quality, depression, anxiety, and caffeine use, we predicted that two different mediational models will be satisfied. The first examines the effect of sleep duration on caffeine consumption with depression as a mediating factor; the second uses the same main relationship with anxiety as a mediating factor. Thus, within these mediational models (refer to Figures 1 & 2), sleep duration serves as a predicting variable, depression and anxiety serve as mediating variables, and caffeine consumption serves as an outcome variable. Hypotheses were generated for each of the relationships of each mediational model. They are as follows: that those college students who report a lower sleep duration

increase their caffeine consumption; that lower sleep duration increases signs of depression and anxiety; and that signs of depression and anxiety increase caffeine consumption; and that the relationship between sleep duration and caffeine is mediated by the influence of depression and anxiety.

Methods

Health Behaviors Survey

The current study used information gathered in the longitudinal study entitled “Adjustment to College in Emerging Adults: Changes in Health Behaviors as Possible Mediating Factors.” This survey assesses incoming freshman students on a variety of health behaviors (including alcohol, nicotine, caffeine, mental health, and sleep), consequences of those behaviors, and college adjustment. The survey allows researchers to follow participants longitudinally over the course of their first year. The survey is conducted annually and is administered by the faculty in the Psychology Department (Dr. Gilbertson, Dr. Norton, and Dr. Beery). Participants were first evaluated during their First Weekend program prior to beginning college classes. These data provided the baseline data for the current study. Participants who opted to be contacted for further participation received an email in November with a link to a SurveyMonkey survey when Time 1 data were collected. In February, these same participants were contacted again for a re-evaluation via the same method, when Time 2 data were collected. Time 1 data were used for the purposes of this study. All participants provided written, informed consent prior to be surveyed. All forms and survey procedures were approved by the Lycoming College Institutional Review Board, and further descriptions of relevant methods follow.

Demographics Form

All participants were given a demographics questionnaire which asked for information such as age, gender, year in school, ethnicity, and participation in extracurricular activities. The extracurricular activity section of this questionnaire was modeled after Vinsel, Brown, Altman, and Foss (1980).

Caffeine Questionnaire

Participants then filled out a caffeine questionnaire which asked for their consumption patterns of a variety of caffeinated beverages including amount, frequency, and type. The different types of caffeinated beverages included coffee, espresso, tea, soft drinks, energy drinks, and chocolate/medication (such as Midol). Quantification of the frequency and quantity of caffeine consumption was modeled after a similar measure for alcohol (Cahalan, Cissin, and Crossley, 1979). Milligrams of caffeine consumption for specific caffeinated beverages (coffee, tea, espresso, soft drinks, and energy drinks) were located in the USDA National Nutrient Database (2009) and manufacturer websites.

Mental Health Inventory

The participants completed the Mental Health Inventory, a questionnaire consisting of 38 scale-based items (MHI; Veit & Ware, 1983). This measure was used to evaluate the mental health of participants and includes anxiety and depression subscales as well as an overall, global score. The MHI has been shown to assess differences between anxiety and depression, as well as overall mental health in college populations (Rosenthal et al., 1991; Fouad et al., 2006). Other subscales calculated by the MHI include total distress, overall well-being, loss of behavioral /

emotional control (shortened to emotional control), general positive affect, and emotional ties. Life satisfaction was another subscale in the MHI that was not calculated for this study.

Pittsburgh Sleep Quality Index

The Pittsburgh Sleep Quality Index was administered to participants in order to evaluate their overall sleep quality (PSQI; Buysse et al., 1989). The PSQI is one of the most widely used measures to assess sleep quality and is frequently used in college populations (Ahrberg, Dresler, Niedermaier, Steiger, & Genzel, 2012; Lund, Reider, Whiting, & Prichard, 2010). The PSQI has seven subscales which include a global overall score, sleep quality, sleep latency, sleep disturbance, daytime dysfunction, sleep efficiency, and sleep duration.

Data Analysis

Data analyses were designed to evaluate the hypotheses that depression and anxiety mediate a relationship between sleep duration and caffeine. The data analysis strategy followed that of Baron and Kenney (1986). Thus, two models (one addressing depression as a mediator, and the other addressing anxiety) were formed. Hierarchical regressions were conducted for both models using the following steps: First, a regression evaluated the main relationship for both models: that sleep duration serves as a predictor variable to caffeine consumption as an outcome variable. Second, a regression evaluated the relationship between the predictor (sleep duration) and the mediator (depression or anxiety). Third, a regression analyzed the mediator's relationship with the outcome variable (caffeine consumption). Finally, a regression reevaluated the main relationship (sleep duration on caffeine consumption) using the mediating variable to explain the relationship. The purpose of the fourth analysis was to determine if the inclusion of the mediator accounted for unique variance above that indicated in the simple regression between sleep and

caffeine. Interpretation of regression techniques addressed whether a variable can be used to predict an outcome variable. Similar to the Pearson r correlation statistic, directionality of the relationships between variables (positive or negative) can be determined via the standardized regression coefficient (β) (see Figures 1 & 2). Mediation models were evaluated for each subscale of the PSQI, as shown in Table 3, and sleep duration was selected as a significant variable. Following conventions concerning regression analyses, standardized regression coefficients were placed in figures as well as results and discussion (Norton, Gupta, Parris Stephens, Martire, & Townsend, 2005). Gender was included as a control variable in each model; however, the inclusion of gender did not alter results. All analyses were run using IBM SPSS version 19 (IBM, 2010).

Results

Descriptive Statistics

Descriptive statistics were conducted to examine both frequencies and averages for age, gender, race, residency status, and high school GPA (see Table 1). Participants were 105 (28.8% male, 71.2% female) incoming freshman students at Lycoming College whose average age was 18. As data for the current study were constrained to Time 1, analyses were conducted to compare baseline participants ($N = 203$) to those who had completed the Time 1 follow up in November ($N = 105$) (see Table 1 for demographics & Table 2 for mediational variables). As a result of these analyses, a few significant differences were found for demographics. Those who did not complete Time 1 were more likely to be slightly older (Mean Age = 18.03, $p = 0.013$), male ($p < .001$), and to commute to college ($p < .001$). No significant differences were found

between the baseline group and the Time 1 completers for the mediation modeling variables of caffeine consumption, depression, or anxiety.

Depression as a Mediator Between Sleep and Caffeine

The first mediational model assessed whether depression would mediate the relationship between sleep duration and caffeine consumption. The results of this mediation model are summarized in Figure 1 (including relationships between individual variables in model) and the overall model significance is summarized in Table 3. This model did not meet criteria for mediation. A significant relationship was found between sleep duration and caffeine consumption even with depression as a mediating factor (S.E. = 0.214, $\beta = 0.27$, $p \leq 0.01$). Sleep duration acted as a predictor variable to caffeine consumption (S.E. = 0.23, $\beta = 0.197$, $p \leq 0.05$). Sleep duration also predicted depression marginally significantly (S.E. = 0.39, $\beta = 0.17$, $p \leq 0.1$). However, no significant relationship was found between depression and caffeine consumption ($p = 0.97$). The mediating variable improved the significance of the overall model ($\Delta R^2 = 0.02$).

Anxiety as a Mediator Between Sleep and Caffeine

The second mediational model assessed whether anxiety would mediate the relationship between sleep duration and caffeine consumption. The results of this mediation model are summarized in Figure 2 (including relationships between individual variables in model) and the overall model significance is summarized in Table 3. This model did not meet criteria for mediation. A marginally significant relationship was found between sleep duration and caffeine consumption even with anxiety as a mediating factor (S.E. = 0.241, $\beta = 0.184$, $p \leq 0.1$). Sleep duration acted as a predictor variable to caffeine consumption (S.E. = 0.23, $\beta = 0.197$, $p \leq 0.05$). Sleep duration marginally acted on anxiety as a predictor variable (S. E. = 0.542, $\beta = 0.19$, $p \leq$

0.1). No significant relationship was found between anxiety and caffeine consumption ($p = 0.62$). The overall model was not improved by anxiety as a mediator ($\Delta R^2 = -0.01$).

Discussion

This study produced two models which provided many relationships counter to previous research. Both models showed significant direct relationships between sleep duration and caffeine consumption, but neither showed full mediation relationships with mental health. The directionality of the relationships and implications of these findings are further discussed below.

Overall Model Outcomes

The current study found that as sleep duration increases, caffeine consumption also increases significantly. This is counter to the hypothesis that a decrease in sleep duration would significantly increase caffeine consumption as students self-medicated against feelings of fatigue or lack of focus. This may be due to the effects of caffeine withdrawal marked by increased sleepiness and fatigue. It is possible that students who consume higher amounts of caffeine more frequently experience sleepiness after caffeine's effects wear off and therefore take more naps during the day. A study by Phillips-Bute and Lane conducted in 1997 suggests that even short-term withdrawal symptoms of caffeine can include fatigue and sleepiness, even when this expectation is methodologically controlled for. One mechanism to explain this effect is that withdrawal symptoms may be mediated by the effects of adenosine on the central nervous system. Prolonged wakefulness caused by caffeine causes the accumulation of adenosine, thus when caffeine's effects of blocking adenosine A_{2A} receptors are lessened, the impact of the accumulated adenosine, an inhibitory neurotransmitter, is greater (Lorist & Tops, 2003). Additionally, sensitization of adenosine receptors during prolonged caffeine consumption

contributes to this effect (Von Borstel, Wurtman, & Conlay, 1983). These findings maintain the prediction that caffeine withdrawal related fatigue and sleepiness may produce increases in sleep duration as affected individuals succumb to their symptoms, although research is needed.

Depression Model

The first hypothesis stated that depression would mediate the relationship between sleep duration and caffeine consumption. Inclusion of depression in the overall model did somewhat improve the ability to predict caffeine consumption from sleep duration. However, this did not produce full mediation because the relationship from depression to caffeine consumption was not significant. The direction of the relationships between individual variables within the model are discussed below.

Sleep duration significantly predicted depression in this model, which corroborates findings by Buboltz, Brown, and Soper in 2001. However, this analysis indicted that as sleep duration increased, depression also increased. One likely explanation for this finding is that depression is not caused by poor sleep duration, but that higher sleep duration may be indicative of symptoms of depression. This hypothesis is corroborated by research by Moo-Estrella, Perez-Benitez, Solis-Rodriguez, and Arankowsky-Sandoval who conducted a study on 638 college students (2005). They found that college students who exhibited depressive symptoms reported significantly higher sleep disturbances including higher sleep initiation latency, poor sleep quality, and greater amount of awakenings. Poor sleep quality overall in college students as suggested by Moo-Estrella and colleagues may increase the duration of time spent sleeping or attempting to sleep. Additionally, the study found that students who exhibited depression reported a greater number of days feeling extreme drowsiness during class, even sleeping during

class, which suggests the possibility that depressed college students may be increasing their total sleep duration through sleeping at times outside their circadian rhythms, such as during class.

Depression was not significantly related to caffeine consumption. This is counter to the research by Kale, Addepalli, Bafna, & Prabhavalkar, who in 2010 suggested that caffeine is a major source of self-medication for depression. One possible explanation for this finding is that caffeine was indeed medicating the effects of depression such that the participants reported lower depression for the purposes of this study. Regular caffeine consumption was found to be lowest in anxiety and major depression patients among a population of psychiatric inpatients with various disorders (Rihs, Müller, & Baumann, 1996). Despite this, the study found that high caffeine users showed the highest scores on depression symptoms. These findings suggest that people who are receiving medication or treatment for depression may use less caffeine to self-medicate, and that people who weren't receiving medication for mood disorders may be using caffeine to self-medicate. Caffeine has been shown to have an impact on mood even at low doses, suggesting that even as caffeine is metabolized, it can still impact mood hours later (Quinlan et al., 2000). A method to rectify the issue of caffeine impacting the mood of participants during surveying would be to test participants' blood samples for caffeine use, or potentially ask them if they are currently under the influence of caffeine when they take the survey. This would allow researchers to isolate mood-related feelings between those in a "sober state" and those under the mood-impacting influence of caffeine consumption. This is further addressed in the limitation section below.

Anxiety Model

The second hypothesis stated that anxiety would mediate the relationship between sleep duration and caffeine consumption. Inclusion of anxiety in the overall model did not significantly improve the ability to predict caffeine consumption from sleep duration. The direction of the relationships between individual variables within the model is discussed below.

Sleep duration was found to have a marginally significant effect on anxiety. Sleep disturbances that may cause lower sleep duration has been linked to greater feelings of anxiety, however, the current study found the inverse relationship (Mellinger, Balter, & Uhlenhuth, 1985). Sleep duration had a direct, strong correlation with anxiety in the current study, meaning that as sleep duration increased, anxiety increased. This finding contrasts with findings of Rosa that anxious patients had a significantly lower period of sleep overall (Rosa, 1983). Other studies report significant associations between anxiety and sleep disturbances, which also disagree with the current study's findings (Lindberg et al., 1997). One explanation for higher sleep duration in participants with higher anxiety is the use of anxiety-relieving medications such as benzodiazepines or antihistamines which induce sleep (Nussbaum & Weissberg, 2013). The precise reasons for higher sleep duration predicting greater anxiety are unknown and this is a relationship that would be valuable to reexamine in future studies.

Although the anxiety model did show not improvement with the addition of anxiety as a mediator, these results may indicate the presence of an untested relationship. This finding could be explained by the relationship between anxiety and caffeine consumption as the inverse of previously hypothesized directions (whereas instead of anxiety impacting caffeine consumption, caffeine consumption may impact anxiety). High doses of caffeine and withdrawal symptoms

can cause feelings of jitteriness that one usually associates with anxiety, indicating that caffeine consumption may impact feelings of anxiety (Smith, 2002). Caffeine is also suggested to exacerbate the symptoms of generalized anxiety disorder, and is even suggested to provide a model of generalized anxiety by some authors, further supporting an inverse direction of the caffeine-anxiety relationship (Lader & Bruce, 1986). Individuals with generalized anxiety disorder have been found to be especially sensitive to the effects of caffeine, which may further indicate this direction (Bruce, Scott, Shine, & Lader, 1992). The inverse direction of caffeine and anxiety is further suggested by the result that anxiety produced no significant effect on caffeine consumption.

Limitations

A few limitations also likely impacted this study. The nature of data collection through self-report in itself presents a few problems. For example, it is possible that participants may have been under the influence of caffeine or other mood-altering drugs which would likely impact their MHI scores. One way of reconciling this issue is to consider a laboratory-based study where more of these variables could be controlled. Measures such as the PSQI and CCQFI examined the average for each variable over time, which may have not reflected the unique sleeping habits of college students throughout the week (less sleep on weekdays and more sleep on weekends) or episodes of high or low sleep duration. Additionally, the difference between binge consumption of caffeine and daily consumption could not be accounted for through these averages. Future studies may benefit from measures which include more specific time course for these behaviors. A small sample size ($n=105$) limited the generalizability of the results. However, data analysis using regressions techniques are used in sample sizes of at least a hundred participants which this study did include (Fritz & MacKinnon, 2007). Other studies

have reported even lower return rates for additional surveys among college students (Cranford et al., 2008). Further incentives and better advertising in the future may help entice college students to participate, increasing external validity and likely improving significance. Lack of variation in sleep duration (Std Dev = 0.823) may have impacted the significance of the mediation models, although this limitation isn't readily rectified other than by increasing the sample size. Gender skews showing females as overrepresented ($F = 71.2\%$) could have impacted mental health data since females are especially likely to show symptoms of depression and anxiety (Lindberg et al., 1997). The mediational models had very low variance accounted for ($R^2 = 3-5\%$) for both models, suggesting that additional variables may have a great impact on caffeine consumption in college students. Some studies suggest that caffeine consumption may be influenced by age, tobacco use, or alcohol consumption (Hewlett & Smith, 2006). This is an area to be investigated by future researchers. Future researchers may also look into a more precise measurement of anxiety and depression, especially an instrument more aligned with the DSM-IV.

Conclusions

Findings from the current study introduced a number of implications. Further investigation of the role of mental health as a mediator of health behaviors may help develop knowledge of the role that disorders play in the daily lives of affected individuals, expanding understanding of issues these individuals may face. The high percentage of anxiety disorders in the US population (18.1%) suggest the importance of further study of anxiety and related health behaviors such as caffeine consumption, sleeping habits, and alcohol consumption (Kessler, Chiu, Demler, & Walters, 2005).

Sleep quality and caffeine consumption in college students should be further investigated as a potentially risky relationship that propagates poor academic habits such as sleeping during

class, inability to adequately consolidate information learned in classes due to poor sleeping habits, and unsafe use of caffeine-based wakefulness aids. This is of particular importance as highly caffeinated beverages marketed to college students increase in popularity. Future studies should address the directionality of the relationships between mental health, caffeine, and sleep-related factors in college populations to determine what these directions would imply about health-related behavioral changes and their impact on academic achievement and well-being.

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Table 1

Sample Demographics

Variable	Health Behaviors (N=203)				T1 Completers (N=105)				T1 Non-Completers (N=98)			
	Mean (SD)	Range	N	%	Mean (SD)	Range	N	%	Mean (SD)	Range	N	%
Age^a	18.02 (0.40)	17-20	198		18.00 (0.31)	17-19	103		18.03 (0.47)	17-20	95	
Gender^a			199				104				95	
M			79	38.9			30	28.8			49	51.6
F			120	59.1			74	71.2			46	48.4
Ethnicity			203				105				98	
Caucasian			173	85.2			89	84.8			84	85.7
African- American			11	5.4			3	2.9			8	8.2
Other Ethnicities			19	9.4			13	12.3			6	6.1
Student Type^a			203				103				95	
Resident			184	92.9			99	96.1			85	89.5
Commuters			14	7.1			4	3.9			10	10.5
HS GPA	3.49 (0.45)	2.00-4.70	195		3.53 (0.43)	2.00-4.70	98		3.45(0.47)	2.30-4.35	97	

^aSignificant differences between groups for these variables (Age $p = 0.013$, Gender $p = 0.000$, Student Type $p = 0.000$).

Table 2 - *Dependent Means*

Variable	Health Behaviors (N=203)				T1 Completers (N=105)				T1 Non-Completers (N=98)			
	Mean	Range	N	Std Dev	Mean	Range	N	Std Dev	Mean	Range	N	Std Dev
Caffeine												
Consumption BL ^a	0.69	0-7.56	131	1.15	0.70	0-7.51	70	0.14	0.67	0-7.56	61	1.19
Consumption T1					1.23	0-10.59	98	1.89				
PSQI												
Global PSQI T1					5.84	0-12	101	2.79				
Sleep Quality T1					1.13	0-3	104	0.65				
Sleep Latency T1					1.06	0-3	104	0.88				
Sleep Disturbance T1					1.11	0-3	103	0.56				
Daytime Dysfunction T1					0.90	0-3	104	0.87				
Sleep Duration T1					0.96	0-3	104	0.82				
Sleep Efficiency T1					0.36	0-3	103	0.71				
MHI												
Anxiety BL ^a	12.13	4-24	201	4.18	12.02		104	4.30	12.25		97	4.08
Depression BL ^a	8.41	4-20	181	2.91	8.53		92	3.03	8.29		89	2.80
Positive Affect BL	21.88	11-30	201	3.64	21.85		104	3.93	21.92		97	3.33
Total Distress BL	28.25	12-65	181	8.75	28.03		92	9.21	28.47		89	8.30
Well-Being BL	26.56	14-36	201	4.31	26.54		104	4.68	26.58		97	3.90
Emotional Ties BL	4.67	2-6	202	1.13	4.69		104	1.04	4.65		98	1.22
Emotional Control BL	8.38	4-22	202	3.22	8.48		105	3.48	8.27		97	2.92
Anxiety T1					11.82	4-24	102	4.53				
Depression T1					8.79	4-21	91	3.13				
Positive Affect T1					21.08	7-29	104	4.52				
Total Distress T1					28.85	14-64	88	9.85				
Well-Being T1					25.50	10-35	102	5.24				
Emotional Ties T1					4.40	1-6	102	1.23				
Emotional Control T1					9.20	4-20	102	3.60				

^aNo significant differences between groups for these variables (Caffeine Consumption $p = 0.802$, Anxiety $p = 0.748$, Depression $p = 0.849$).

Table 3

Results of Cross-Sectional Caffeine Mediation Analyses (N =105)

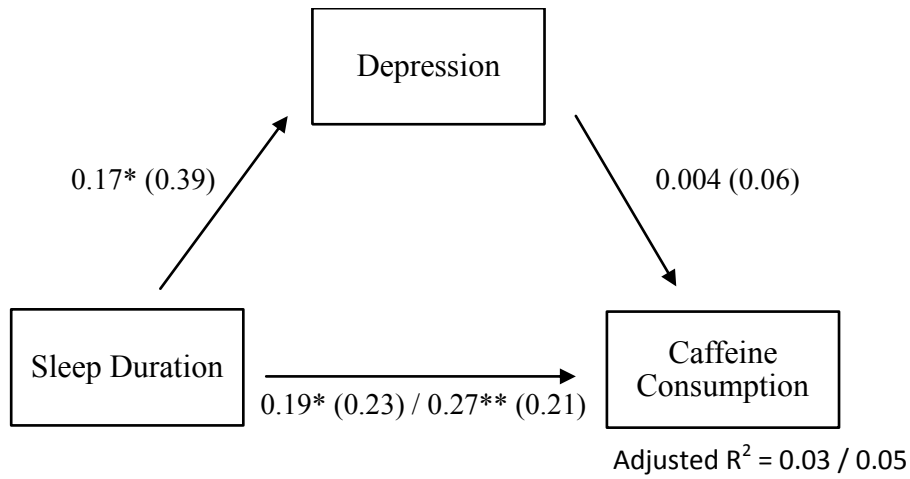
Variable	(a) Cross-Sectional with Depression			(b) Cross-Sectional with Anxiety		
	B	SE B	β	B	SE B	β
Sleep Latency	-.318	.200	-.176	-.340	.221	-.159
Sleep Disturb.	-.153	.403	-.048	.198	.381	.059
Daytime Dysfunction	-.160	.258	-.080	.006	.267	.003
Sleep Duration	.536	.214	.270**	.426	.241	.184 ^M
Global PSQI	-.029	.075	-.047	-.208	.077	-.041
Sleep Medications	-.300	.327	-.101	-.223	.346	-.067
Sleep Quality	.036	.304	.014	-.217	.307	-.076
Sleep Efficiency	-.132	.274	-.054	-.154	.303	.053

^M $p < 0.1$ * $p \leq 0.05$ ** $p \leq 0.01$ *** $p \leq 0.001$

Note: Data reflect the overall mediation relationship.

Figure 1

Mediational Analyses of Depression Model

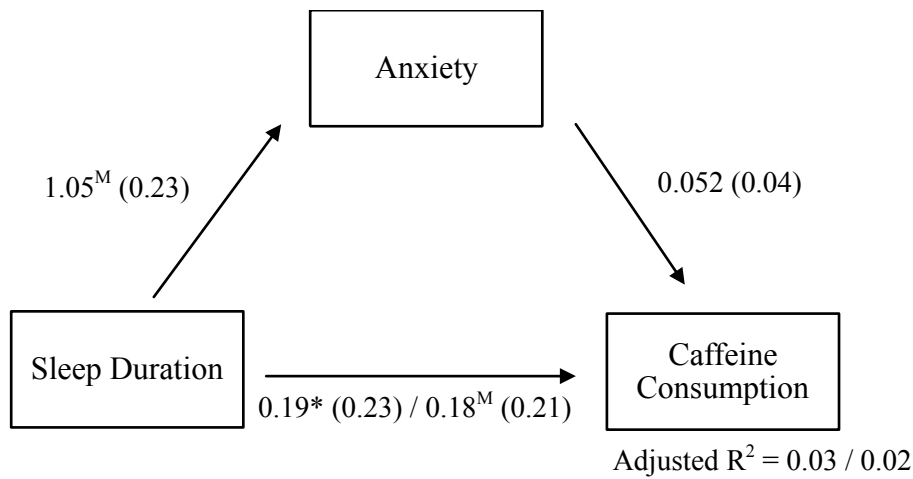


$M_p < 0.1$ $^* p \leq 0.05$ $^{**} p \leq 0.01$ $^{***} p \leq 0.001$

Note: Numbers in each pathway represent the standardized regression coefficient, showing directionality, and the standard error value: β (Std Error). Where two values are present, coefficients represent the relationship between sleep duration and caffeine consumption without the mediator/with the mediator.

Figure 2

Mediational Analyses of Anxiety Model



$^M p < 0.1$ $^* p \leq 0.05$ $^{**} p \leq 0.01$ $^{***} p \leq 0.001$

Note: Numbers in each pathway represent the standardized regression coefficient, showing directionality, and the standard error value: β (Std Error). Where two values are present, coefficients represent the relationship between sleep duration and caffeine consumption without the mediator/with the mediator.