The Effects of Caffeine on Sleep Following Sleep Deprivation

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ABSTRACT

Introduction: In modern day society, pressures from the business and academic worlds cause people to consistently self-impose sleep restriction in order to meet deadlines. To maintain a level of alertness during these periods, many individuals rely upon caffeinated beverages in order to heighten their awareness and repress their drive for sleep. However, caffeine's wakefulness promoting properties can negatively affect sleep quality during the following night's sleep, resulting in an individual feeling less rested upon awakening.

This thesis looked at the combined effects of sleep deprivation and caffeine on daytime recovery sleep. The study design mimics staying up all night in order to meet societal demands (e.g. tests, deadlines) and thus is directly translatable to common society.

Methods: Thirty drug free males and females ages 18 to 35 participated in this study. Subjects completed an in-laboratory study. Subjects were given an 8 h baseline polysomnographically recorded sleep opportunity on the first night. Following the baseline night, subjects were sleep deprived for 28 h. Ten subjects were assigned to a caffeine group and administered a caffeine pill at 23 h awake. A 5 h recovery sleep opportunity followed the sleep deprivation episode. Sleep records were visually scored and compared within subjects and between conditions.

Results: Sleep deprivation significantly decreased amounts of stage 2, REM and REM latency during recovery sleep in both the caffeine and placebo groups. Caffeine, in combination with sleep deprivation, significantly increased the amount of wakefulness and decreased the amount of stage 3/4 sleep when compared to placebo. These effects were seen when comparing the entire sleep episodes as well as when comparing the first 300 minutes of each sleep episode.

Conclusion: The effect of caffeine attenuates the ability to dissipate the homeostatic build-up of sleep pressure that results from extended wakefulness. This is seen as a significant interaction between caffeine and sleep deprivation in the amount of deep sleep present in the recovery sleep episode. We also see a significant increase in the percent wakefulness in the caffeine group when compared to placebo indicate that caffeine 5 hours prior to daytime recovery sleep disturbs sleep even when sleep pressure is high. The disturbed sleep findings have important implications for individuals who use caffeine to promote wakefulness at night.

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BACKGROUND

There are several models on sleep regulation; the most widely accepted is the two-process model. According to the two-process model, sleep is regulated by the balance between the homeostatic process (process S) and the circadian process (process C) [5,6]. Process S is the sleep pressure that builds up during wakefulness and dissipates during sleep [5]. This sleep pressure is aided by the clock-like circadian process that oscillates between high and low propensities for sleep throughout the 24 hour day [5,6]. Process S is susceptible to modification by behavior and exogenous stimuli. One such stimulus, caffeine, is the most widely used drug in the world and a component of one of the most popular beverages in the world; coffee. In modern times, pressure and high expectations in the business and academic worlds often require individuals to maintain wakefulness for long periods of time. In order to do so, many turn to caffeine to boost their performance and ward off fatigue. Researchers have found that a typical adult in western society consumes 200-300 mg of caffeine a day, which is roughly equivalent to a 16 oz. cup of Dunkin' Donuts coffee [11]. Although caffeine can help an individual for a short time, this alertness comes at a price. Caffeine has been reported to profoundly impact sleep in regards to both quality and quantity, even in individuals that maintain healthy sleep schedules [25]. In order to better illustrate this phenomenon, the present study examines the effects of caffeine consumption on the quality of sleep following an episode of extended wakefulness.

Two Process Model of Sleep

A circadian rhythm is defined as a biological rhythm that cycles with an approximately 24 hour period [10]. This circadian process is produced endogenously by the suprachiasmatic

nucleus (SCN) of the hypothalamus and produces physiological changes that promote sleep. Sleep regulation in the brain is influenced by the circadian system. This system promotes sleep when plasma melatonin levels are high, plasma cortisol levels are decreasing and body temperature levels are low [10,14]. This cyclic modulation by the SCN is entrained by environmental time cues and is independent of previous sleep-wakefulness history.

The homeostatic process, unlike the circadian process, is dependent on an individual's prior sleep-wakefulness history; it builds and dissipates in a non-linear fashion during wakefulness and sleep respectively [6,10]. This increasing pressure to sleep throughout wakefulness is associated with a build-up of adenosine, a nucleoside that is released during metabolism of adenosine triphosphate (ATP) and neural activity [2].

Basic Sleep Architecture

Sleep consists of two alternating states: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep [9]. NREM is further subdivided into stages 1-4, with stage 1 representing the lightest stage of sleep and 4 the deepest. The lightest stage of sleep, stage 1, is typically found during transitions from wake to sleep [28]. During an individual's initial sleep cycle, stage 1 typically lasts for a period of one to seven minutes [9,28]. Stage 1 is followed by stage 2, a deeper stage that is present throughout the sleep episode, making up half of the time asleep. Stage 2 is typically present for 10 to 25 minutes during the first cycle until it gives rise to slow-wave sleep (SWS), stages 3 and 4. SWS, the deepest sleep an individual experiences, is usually present for 20-40 minutes during the initial sleep cycle and is most prevalent during the first half of a sleep episode [9,28]. The final stage of sleep, REM sleep, is interspersed throughout the sleep episode. During the first cycle of sleep, the amount of time spent in REM is

relatively brief, lasting one to five minutes [9,28]. Duration in REM then progressively builds throughout the night with the longest periods typically in the last third of the sleep episode [28].

Sleep is composed of patterned NREM and REM sleep cycles, with each cycle typically lasting 90-100 minutes [9,27].

Sleep stages are measured and determined through polysomnography (PSG). PSG is a compilation of electrophysiological recordings of brain and muscle activity; consisting of electroencephalography (EEG), electrooculography (EOG), electromyography (EMG), and electrocardiography (ECG) [28]. PSG is recorded by placing electrodes on the scalp and face of a subject to measure the activity in the respective brain areas, muscle activity of the face, and the cornea-retinal potential of the eyes. The PSG record is scored in 30 second intervals, known as epochs. Each epoch is scored and categorized into one of the stages of sleep based upon patterned characteristics of the recorded traces; signal frequency, amplitude, and morphology.

Stage 1 of NREM sleep can be identified by waves that are relatively low in amplitude and mostly in the theta range. Theta waves fall between 2-7 cycles per second (Hz). Slow rolling eye movements are often present in the EOG recording during this stage as well [28].

Stage 2 of NREM sleep is defined by the presence of theta waves, sleep spindles and/or K complexes [28]. A sleep spindle is a burst of fast EEG activity in the 12-14 Hz sigma range; a K complex is an EEG wave form that consists of a well-formed negative sharp wave followed closely by a positive sharp wave with relatively high amplitude. Both the duration of the K complex and sleep spindle should exceed 0.5 seconds [28]. As stage 2 increases in duration throughout the beginning of the sleep period, slower delta waves, characterized by 2 Hz or less, become more prevalent [6,9,28].

When delta activity is present in 20-50% of an epoch and reaches amplitudes of 75μV or greater, the epoch is scored as stage 3 [6,9,28]. Stage 3 is typically present for a few minutes before the delta activity becomes prevalent in 50% of the epoch [9]. Once over 50% of the epoch consists of delta activity, the epoch is scored as stage 4. For this thesis, stages 3 and 4 were scored together and are referred to as SWS, which is an accepted procedure in reporting SWS.

REM sleep consists of low voltage, mixed frequency wave forms, which are similar to those found in stage 1 sleep. Rapid eye movements in the EOG traces and "sawtooth" waves in the EEG traces are common during REM sleep [28]. REM is often preceded and followed by either sleep spindles or K complexes, however, during the stage there is an absence of both [28]. REM is also characterized by low amplitude EMG trace due to muscle atonia [28].

Once a sleep episode has been scored, the entire episode can be compiled into a hypnogram to describe sleep architecture. The most common variables assessed are sleep onset latency (SOL), total sleep time (TST), sleep efficiency (TST/time in bed), wakefulness after sleep onset (WASO), amount of time spent in the various sleep stages, and the time it takes before an individual enters SWS or REM [20].

Sleep Deprivation

Three common protocols for examining the effects of sleep deprivation on physiological variables are: total sleep deprivation (TSD), sleep restriction, and sleep fragmentation [15]. The current study employs the TSD protocol and will be the focus of this literature background. In humans, a typical TSD protocol involves a level of sleep deprivation greater than 24 hours [15]. The homeostatic regulation of sleep results in a build-up of sleep pressure from one sleep episode to the next. Extended periods of wakefulness results in a greater build-up of sleep

pressure and can impact sleep architecture [6,15]. Findings from prior studies have shown increased SWS, decreased SOL, and decreased percentage of time spent in stage 1 during a recovery sleep episode following TSD [6,7,15].

The increase in slow wave activity (SWA), a marker of sleep pressure, following sleep deprivation is thought to be the result of an increase in neuronal inhibition [3,4,6, 22,27]. One possible mediator of this change in neural activity could be the activation of inhibitory adenosine receptors by the nucleoside adenosine [3,4,22].

The presence of slow-wave activity has been hypothesized to be important for the consolidation and reconsolidation of memories. Findings from several studies have demonstrated that recollection of words was higher following a period of SWS compared to REM or wakefulness [13,16].

Adenosine

Adenosine, a component of the energy molecule adenosine triphosphate (ATP), is passively and actively released by neurons [22]. Wakefulness has a ~30% greater amount of metabolic activity than NREM sleep and thus a higher rate of ATP metabolism [2,22]. This increase in metabolism results in an increase in extracellular adenosine, which is then able to bind to any of the four adenosine receptor (A_1, A_{2A}, A_{2B}, A3) located in the brain [2,22]. When adenosine binds to its receptors, it causes an inhibitory effect by hyperpolarizing the cell through increased potassium conductance [2]. Hyperpolarizing wake-promoting neurons decreases the ability for these neurons to produce action potentials. This decreased neuron activity is thought to result in increased sleep pressure that builds throughout wakefulness [2].

Receptor A_1 is expressed in the brain cortex, thalamus, hippocampus and basal ganglia; findings suggest that it is through this receptor that adenosine impacts sleep [22]. Findings from studies in rats have demonstrated that application of a selective A_1 agonist results in a dose-dependent increase in SWA, which suggests that adenosine plays a role in the homeostatic sleep process [29].

Receptor A_{2A}, found in the striatum, nucleus accumbens and olfactory bulb, also affects sleep. Methylxanthines, such as caffeine, are competitive antagonists for adenosine and, through the blocking of A_{2A} receptors, causes increased alertness [18,22]. Blocking the A_{2A} receptor using caffeine has been reported to delay SOL, increase the amount of stage 2, and decrease the amount of SWS in subjects with healthy habitual sleep [19,22,23,26]. Research has also suggested that caffeine causes increased SOL as well as fragmentation of sleep by increasing WASO [2]. Findings using knockout mice suggest that although caffeine also binds to the A_1 receptor, it does not mediate the effect of increased arousal [18].

Receptor A_{2B} is thought to be present on the pineal gland, a gland that releases the circadian controlled hormone melatonin [17]. It is hypothesized that through this receptor caffeine can alter the release of melatonin [31].

The locations of A3 receptors vary between species; in human they have been found primarily in the lungs and liver [22]. It is believed that this receptor does not directly play a role in the mediation of sleep and has greater implications mediating allergic responses [22].

Previous Studies of Caffeine and Sleep Following Sleep Deprivation

A study in rats by Wurts and Edgar conducted in 1999 investigated the effects of caffeine and sleep deprivation on the expression of NREM and REM sleep. Ten male rats had four EEG

electrodes and two EMG electrodes implanted into their brain. All rats completed both treatments of caffeine and placebo, and thus served as their own control. Baseline data was collected over a 48 h period prior to treatment, and then followed by administration of the treatment, through injection, and a 6 h sleep deprivation period. The rats were then allowed a 48 h recovery opportunity. Results from this study demonstrated that the caffeine treatment did not block the recovery of sleep loss; however, it did slow the rate of recovery in total sleep, NREM and REM duration, SWA and sleep continuity [32].

LaJambe et al. and Carrier et al., looked at the impact of caffeine on recovery sleep in humans. In the study conducted by LaJambe et al in 2005, the effects of caffeine in habitually low ($\leq$100mg d^{-1}) and high caffeine users ($\geq$400mg d^{-1}) were examined. Subjects were sleep deprived for 27 hours and received doses of 0 mg, 100 mg, or 300 mg of caffeine, three times over the 27 hour episode. Caffeine was administered through chewing gum and dose was kept constant for each subject. The last dose was given 3 hrs before the 8 hr recovery sleep opportunity [21]. LaJambe et al. found a significant difference in the TST between the high dose and low dose caffeine groups. Differences in TST between high dose and placebo were also found, and were greater, however, the difference was not significant. Concomitant to the reduction of TST, high dose caffeine significantly increased the latency to SWS when compared to both placebo and low dose caffeine [21]. In the study by Carrier et al. in 2007 the influence of caffeine on nighttime sleep following a typical day of wakefulness and on daytime sleep following 25h of sleep deprivation was examined. Caffeine was administered in 100 mg doses at 3h and 1h prior to bedtime. Caffeine increased sleep latency and the percentage of stage 1 sleep, and decreased the number of minutes in stage 2 and SWS for both the night and the daytime recovery sleep episode. Furthermore, caffeine decreased TST and REM sleep but only during

daytime recovery sleep. The authors concluded that the greater impact of caffeine during day recovery verses nighttime sleep was the combined result of the increased circadian wake propensity and dissipation of sleep pressure over the sleep daytime episode. They also concluded that the reduction of SWS, a typical marker of homeostatic sleep drive, by caffeine increased the impact of the circadian wake signal. Like in most caffeine and sleep studies where caffeine was administered before nighttime sleep episodes [23,24,25,31,32], caffeine in the studies by LaJambe et al. Carrier et al. was administered within 1 to 3 hours prior to the sleep episode following sleep deprivation. Caffeine is more commonly taken during sleep deprivation further away from bedtime to promote wakefulness. The half-life of caffeine is approximately 5-6 hours [19,25,26] and thus, we tested the influence of caffeine consumption on recovery sleep scheduled 5 hours after caffeine administration as administering caffeine 5 h prior to recovery sleep is more relevant to real world caffeine use during sleep deprivation. We hypothesized that during the recovery sleep opportunity, following 28 h TSD, both conditions will show increases in SWS. In addition, caffeine is expected to attenuate the increase in SWS caused by extended wakefulness. Caffeine is also expected to increase sleep onset latency and the amount of wakefulness during the recovery sleep episode.

METHODS

Subjects

Thirty healthy males and females subjects aged 18-32, 21.6 ± 3.5 (mean±SD), with a BMI between 18.5-27, 22.45±2.13(mean±SD) participated in the study. Subjects were recruited through flyers in the community, emails through the university (the Buff Bulletin), website, and newspapers advertisements. Participants were required to have a history free of drug and alcohol

dependence, be a non-smoker, and were drug-free (including caffeine and nicotine) for two weeks prior to the laboratory session and throughout protocol. Drug use was determined by self-report and verified by toxicology at screening and upon inpatient admission. All participants gave written informed consent and the institutional human research committee approved the procedures for the protocol. Subjects passed a rigorous health screening, conducted in the sleep lab and at the clinical translational research center (CTRC) prior to entering the study. Screening included medical history, physical exam, electrocardiogram, blood chemistries, and psychological tests. None of the subjects reported regular night work in the preceding 1-year period or crossing more than 1 time zone in the previous 3 weeks. Subjects were asked to maintain a self-selected sleep schedule of 8 h for 1 week prior to admission to the laboratory. The timing of sleep episodes were verified with sleep-wakefulness logs, call-in bed and wake times to a time stamped voice-mailbox recorder, and wrist activity and light exposure recordings through the use of Actiwatch-L (Mini Mitter, Bend, OR).

Protocol

This study was part of a larger research protocol examining the phase resetting response to bright light and caffeine. Subjects lived in the Sleep and Chronobiology Laboratory at the University of Colorado at Boulder in a special sleep suite that was an environment free from time cues for ~3.7 days. For the in laboratory portion of the protocol, the subjects arrived at the laboratory ~5 h before their habitual bedtime. Upon arrival the subject's blood pressure, heart rate, and temperature were taken and a urine drug test, breathalyzer, and pregnancy test (if female) were administered. Subjects were then given an 8 h baseline sleep opportunity. Following the baseline night subjects woke up under a constant routine protocol. During this

protocol, the room temperature was kept stable (22-24º Celsius), the light in the room was kept constant at ~1.5 lux, and the subjects were kept in a semi-recumbent seated posture (~30-40 degrees from horizontal). During this time, the subjects received scheduled meals of known caloric value, were asked to complete a number of cognition tests, and were asked to provide saliva samples to researchers. Outside of the scheduled tests, the subjects had free time during which they could engage in low-stress activities (read, watch movies, play board games with staff, etc.). Continuous electroencephalography (EEG) recordings verified wakefulness. At 23 hours of wakefulness (5 h before the recovery sleep episode), subjects in the caffeine group were administered a caffeine pill (2.9 mg/kg). At 28 h of wakefulness, subjects were given a 5 h recovery sleep opportunity, during which PSG was recorded.

Assessment of EEG Recordings

Sleep PSG recordings were obtained with Siesta digital sleep recorders (Compumedics USA Ltd, Charlotte, NC) using monopolar EEGs referenced to contra-lateral mastoids according to the International 10-20 system (C3-A2, C4-A1, O1-A2, F3-A2), right and left EOG, chin EMG and electrocardiogram (ECG). Impedences were below 5 kohms. Data were stored and sampled at a rate of 256 samples per second per channel with a 12-bit A-D board. High and low pass digital filters for EEG and EOG were set at 0.10 and 30 Hz, respectively. High and low pass digital filters for EMG were set at 10 and 100 Hz, respectively. Sleep was scored according to standard guidelines from brain region C3-A2 [28]. If the C3-A2 trace contained artifact the C4-A1 trace was used to determine the sleep stage. Sleep onset latency was defined in two ways: SOL3, defined as the time from lights out to the onset of three continuous epochs of EEG defined sleep, SOL10, defined as the time from lights out to the onset of 10 continuous minutes,

20 epochs, of EEG defined sleep. Sleep staging was scored according to standard guidelines [28].

Data Analysis

Each subject had two sleep episodes scored; the first 8 h in duration, the second 5 h in duration. Sleep episodes were scored in 30 second epochs and then analyzed within subjects as well as between groups; caffeine, placebo. Repeated measure ANOVA techniques were used to examine the effects of group (placebo, caffeine), sleep episode (baseline, recovery sleep) and the interaction between group and sleep episode for SOL (3 and 10), percentages of stage 1, stage 2, stage 3/4, REM, and wakefulness. Additionally, the same sleep measures were analyzed for the first 300 min (5 h) of the 8 h baseline sleep episode and compared directly to the 5 h recovery sleep episode. All analyses were performed with STATISTICA, version 6.0 (StatSoft Inc, Tulsa, Ok).

RESULTS

Comparison of Baseline 8h Versus Recovery 5h Sleep Episodes

ANOVA revealed significant main effects of condition for percentage of stage 3/4 ($p=0.00434$) and wakefulness ($p=0.00542$), and for sleep onset latency 10 ($p=0.02774$). Significant main effects between baseline and recovery sleep episodes were observed for percentage of stage 2 ($p<0.0001$), stage 3/4 ($p<0.0001$), and REM ($p=0.00105$), and for sleep onset latency to 3 epochs of sleep ($p=0.00685$) and 10 minutes of sleep ($p=0.00218$), and REM latency ($p<0.0001$). Lastly significant interactions between group and sleep episode were observed for percentages of stage 1 ($p=0.0434$), stage 2 ($p=0.0364$), and stage 3/4 ($p=0.0251$)

sleep with non-significant interaction for the percentage of wakefulness (p=0.0568), and sleep onset latency (SOL10 p=0.0921).

Table 1 shows sleep measures for baseline and recovery sleep episodes by group. During recovery sleep, following 28 h TSD, the placebo group exhibited significant decreases in percentages of stage 1, stage 2, REM, as well as decreases in both measures of sleep onset latency, and REM latency. The caffeine group showed significant decreases in percentages of stage 2, REM, REM latency, and a non-significant decrease in sleep onset latency to 10 minutes of sleep (p=0.0546). Both the placebo and the caffeine groups displayed an increase in percentage of slow wave sleep.

Between groups there were no differences in baseline sleep measures. Following sleep deprivation and administration of caffeine, the caffeine group had a non-significant increase in sleep onset latency (SOL3 p=0.0725), a significantly greater percentage of wakefulness, and a significantly lower percentage of slow wave sleep when compared to the placebo group (Table1).

Measure	Placebo		Caffeine	
% Recording Time	Baseline (8h)	Recovery (5h)	Baseline (8h)	Recovery (5h)
stage 1	5.0 ± 0.01	3.7±0.00	4.9 ±0.01	5.2 ± 0.01
stage 2	50.0 ± 0.01	41.5±0.02†	53.7±0.02	37.5±0.03†
stage 3/4 [a]	19.9±0.01	37.1 ± 0.02†	13.7±0.02	25.8±0.03†
REM	18.8±0.01	14.0±0.01	19.4±0.02	15.1±0.02
Wakefulness [a]	6.3±0.01	3.7±0.03	8.4±0.02	16.5±0.04
SOL 3(min)	6.1±1.21	2.1±0.61	7.8±1.72	5.5±0.87
SOL 10 (min)	8.2±2.03	2.1±0.58	12.5±2.87	6.7±0.83
REML (min)	103.7±8.85	62.7±2.73†	115.9±12.86	59.2±3.97†

Table 1: Results over full sleep period: denotes p-value = <0.05 from baseline, † denotes p-value =< 0.001 from baseline, [a] denotes significant interaction (p-value<0.01).

Comparison of first 5h of baseline versus 5h of recovery sleep

Significant main effects of condition during the first 300 min of the sleep episode were observer for percentage of stage 3/4 (p=0.0094), wakefulness (p=0.00833), and latency to 10

minutes of sleep (p=0.02774). Between the sleep episodes significant main effects were observed for the percentage of stage 2 (p<0.0001), stage 3/4 (p=0.00001), and sleep onset latency (SOL 3: p=0.00685, SOL 10: p=0.00218). During the first 300 min of the sleep episode the interaction between group and sleep episode was significant for the percentages of stage 1 (p=0.0231), stage 2 (p=0.0365), and wakefulness (p=0.0284).

Table 2 shows findings for the comparison of the first 300 min of the baseline sleep episode with the 300 min of the recovery sleep episode. The placebo group showed a significant decrease in percentage of stage 2 and a significant increase in percentage of stage 3/4 during the recovery versus baseline sleep episode when directly comparing the first 300 min of the sleep episodes. The caffeine group showed a significant decrease in percentage of stage 2 and significant increases in percentage of stages 3/4and of wakefulness.

Between groups, there was no difference between the first 300 min of the baseline sleep periods. Following sleep deprivation and caffeine administration significantly decreased the percentages of stages 3/4 and significantly increased wakefulness compared to placebo.

Measure	Placebo		Caffeine	
% Recording Time	Baseline (8h)	Recovery (5h)	Baseline (8h)	Recovery (5h)
stage 1	4.4±0.00	3.7±0.00	4.1±0.01	5.2±0.01
stage 2	49.9±0.02	41.5±0.02	55.1±0.03	37.5±0.03†
stage 3/4[a]	18.0±0.02	37.1±0.02†	21.1±0.03	25.8±0.03
REM	12.3±0.01	14.0±0.01	13.2±0.02	15.1±0.02
Wakefulness [a]	5.4±0.01	3.7± 0.03	6.4±0.01	16.5±0.04

Table 2: Results over first 300 min of sleep period: denotes p-value = <0.05 from baseline, † denotes p-value =< 0.001 from baseline, [a] denotes significant interaction (p-value<0.01).

DISCUSSION

Findings from this study demonstrate that caffeine consumption during sleep deprivation negatively affects sleep 5 hours later. Previously, it has been shown that caffeine administered

between 1 to 3 hours prior to daytime recovery sleep increased the latency to sleep[8], the latency to SWS[21] and the percentage of stage 1 sleep[8]. Caffeine also decreased the number of minutes in stage 2, SWS, TST and REM sleep [8]. Although expected, we did not see a significant difference in either of the sleep onset latency measures when comparing between caffeine and placebo conditions. Over the whole sleep episode, the caffeine condition showed a significant reduction in the amount of slow wave sleep when compared with the placebo group. This is consistent with the theory that slow-wave sleep is the result of adenosine binding to receptors. Along with the reduction of slow wave sleep, the caffeine group also displayed an increase in the amount of wakefulness, which was expected. This is consistent with the idea that caffeine decreases the depth of sleep. Although we did see an increase in stage 1 in the caffeine group, the findings were not significant. The lack of significance ($p= 0.0725$) between conditions for these measures could be resultant of the small subject population in the caffeine treatment group.

We did see a significant decrease in the sleep latencies between the two sleep episodes for both conditions as expected following extended wakefulness. Both conditions also demonstrated a decrease in the amount of stage 2 sleep during the daytime recovery sleep episode. This reduction of stage 2 is the result of the increase in the amount of stage 3/4 that is seen with an increase in homeostatic sleep pressure.

REM latency was decreased during the daytime recovery sleep episodes for both conditions. This finding is consistent with pervious literature as the recovery sleep episode was scheduled just after the circadian peak of REM sleep [14].

The current findings indicate that the common behavior of using of caffeine during sleep deprivation disturbs sleep even when caffeine is taken 5 hours prior to sleep. Future studies

should compare effects of other caffeine doses and administration at other times prior to sleep to determine if caffeine administered earlier reduces the negative influence on recovery sleep.

In summary, the effect of caffeine attenuates the ability to dissipate the homeostatic build-up of sleep pressure that results from extended wakefulness. Caffeine alters the depth of sleep and increases the amount of wakefulness during the recovery sleep episode. This sleep disruption likely hinders the restorative property of recovery sleep following sleep deprivation.

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