The Impact of Alcohol Consumption on Sleep Onset and REM Latency: A Meta-Analytic Study

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The Impact of Alcohol Consumption on Sleep Onset and REM Latency: 
A Meta-Analytic Study

A senior thesis submitted to
The Department of Psychology
College of Theology, Arts, & Sciences

In partial fulfillment of the requirements
for a Bachelor of Arts degree in Psychology

by

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The purpose of this study was to identify whether sleep onset latency (SOL) and rapid eye movement onset latency (ROL) increased or decreased after consuming alcohol. Researchers, Lobo and Tufik (1997), stated that “acute doses of ethanol significantly change the sleep of healthy volunteers” (p. 52). The current study was based on the foundation of alcohol’s effects on sleep among healthy individuals. The populations studied were alcohol-use dependent individuals and healthy individuals. The following measurements were included: Pittsburgh Sleep Questionnaire Index (PSQI), Schlaffragebogen-A/R (SF-A/R), polysomnography (PSG), and electroencephalogram (EEG). The research design for the present study was a meta-analysis of existing, experimental, and quasi-experimental studies. Overall, 23 studies were included in the data analysis and extraction. Hedge’s g was used to calculate the magnitude of effect sizes; the analyses were calculated with a random effects model with a 95% confidence interval. Overall, there was no statistical significance for the PSQI measure with SOL among the populations. Likewise, there was no statistical significance between the SOL and the ROL of healthy and alcohol-use dependent individuals. However, the ROL for healthy individuals who consumed alcohol prior to sleep was statistically significant and had a large effect size when compared to heavy alcohol users. Future studies should compare specific subgroups of healthy individuals (e.g., age, gender, & ethnicity) and include definitive blood alcohol levels (BALs).

Keywords: alcohol, ethanol, sleep latency, SOL, REM latency, ROL, consumption, healthy individuals, alcohol-use disorder
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Introduction

The purpose of this meta-analysis was to identify the amount of time in sleep latency and rapid eye movement (REM) sleep latency after consuming alcohol. According to Feige et al. (2006), “alcohol, when consumed by healthy individuals before going to sleep, shortens the amount of time needed to fall asleep, [and] reduces the amount of REM sleep…” (p. 1527). This statement was used to help guide and form the primary question of this research. This study’s goal was to gain information on alcohol consumption, the influence that alcohol had on REM sleep latency, as well as on the amount of time it took to fall asleep. This study compared the impact that alcohol consumption had on sleep latency and on REM sleep latency in healthy individuals with alcohol-use dependent individuals. During another analysis, only healthy individuals were researched to find out the length of time during sleep latency and REM sleep latency.

Background

The following researchers: (a) Anderer et al. (2005), (b) Brower (2001), (c) Brower et al. (1998); (d) Brower, Aldrich, and Hall (1998), (e) Feige et al. (2006); and (f) Roehrs and Roth (1995), were included as resources throughout this study. These researchers' studies were utilized based on the historical findings, the definitions, and the data collection processes. Brower (2001) reported significant evidence regarding alcohol intake, stating that alcohol “may cause either slowly reversible or irreversible damage to brain systems that regulate sleep” (p. 115). This statement expressed the potential for
lastling damage to occur in the anatomical structures within the brain. Brower (2001) exemplified this topic’s relevance, which was grounded in parameters, impacts, and potential effects that alcohol consumption had on sleep areas. This statement was common among various researchers, including Roehr and Roth (2001)—researchers who have studied the ethanol and sleep affects. These researchers discovered that studying the topic was difficult because the substance affected more than one area of sleep. Likewise, the information pertaining to these various areas of sleep was underdeveloped (Roehrs & Roth, 2001). Although several researchers have studied alcohol and sleep, the topic continues to develop new information.

Anderer et al. (2005) composed a study surrounding sleep fundamentals on a behavioral and biological level. As an objectively rooted study, Anderer et al. (2005) discussed trends and provided definitions pertaining to the current study’s methodology (i.e., polysomnography [PSG] & EEG). Brower (2001) and Brower et al. (1998) recorded sleep outcomes from populations who consumed alcohol. In comparison to Anderer et al. (2005), Feige et al. (2006) defined similar terminology. Roehrs and Roth (1995, 2001) were often referenced in sleep journal articles, as well as alcohol journal articles. The researcher’s credibility created the opportunity to research the findings based on researchers’ previous works; likewise, these studies were published within the last 20-years, which allowed the current study to review recently published studies.

Researchers reported sleep disturbances among alcohol-use dependent individuals. Various researchers summarized Feige et al. (2006), who reported that alcohol-use dependent individuals had an increased amount of time falling asleep and a
shortened amount of time reaching their REM sleep cycle. This finding has been debated throughout the literature. In addition, Rundell et al. (1972) stated, “late sleep following alcohol ingestion before going to bed can be ‘shallow’ and interrupted by frequent periods of wakefulness; the amount of REM sleep is increased and dreams and nightmares frequently occur” (as cited in Feige et al., 2006, p. 1527). Thus, the results have shown an increase in sleep latency and have shown a decrease in REM sleep latency.

The current study compared healthy individuals with no history of alcohol-use disorders to individuals who were diagnosed with alcohol-use disorders. The subgroup comparison outcomes were debated within the researcher’s results and discussions; thus, the need for a synthesis is required. Researcher’s studies have compared these two groups, but an updated synthesis of those studies is needed, which is why this study is a meta-analysis. For instance, Feige et al. (2006) reported that consuming alcohol prior to sleep onset could create various sleep disturbances (i.e., restlessness, perspiration, wakefulness). These are the physiological effects that alcohol has on individual’s sleep cycles. In a similar study, Landolt, Roth, Dijk, and Borbely (1996) found that “…an acute dose of ethanol can shorten sleep latency” (p. 428). Rather than focusing on the physiological effects, these researchers viewed the length of time to reach sleep from a specific measurement of alcohol (i.e., acute dose). Contrasting reports from Dijk et al. (1992) found no significant alterations in sleep among healthy participants who consumed specific blood alcohol levels (BAL; as cited in Feige et al., 2006, p. 1528). These researchers suggested, depending on BAL, that alcohol’s effects could subside
towards the end of sleep cycles. The varying results heighten the importance of original researcher that would offer a statistically comprehensive synopsis of what these researchers have been debating for many years.

These views regarding the impacts from alcohol on sleep were evaluated and were included in the literature. Roehrs, Papineau, Rosenthal, and Roth (1999) discussed the different symptoms from ingesting alcohol prior to sleep among alcohol dependent and healthy (non-alcoholic dependent) individuals. The results differed between the two populations. In fact, sleep latency decreased in healthy participants, but it increased in alcohol dependent participants (Roehrs et al., 1999). According to Allen et al. (1980), as well as Wagman and Allen (1975), “the administration of ethanol improves the sleep of the alcoholic, but only acutely” (as cited in Roehrs et al., 1999, p. 280). This statement supported earlier conclusions, regarding alcohol dependent populations. The impacts on sleep latency and REM sleep latency in non-alcoholic participants remains relevant for future studies based on the underdeveloped research pertaining to this population.

Relevance

A common misconception about alcohol use is that the substance enhances the sleep quality. Researchers found that alcohol operates conversely towards this idea; thus, alcohol does not enhance sleep quality (Feige et al., 2006). In fact, “alcohol is far from being suitable as a hypnotic, because of immediate rebound phenomena during the later part to the night that run parallel to the pharmacokinetics of alcohol” (Feige et al., 2006, p. 1536). Utilizing the evidence from researcher’s collective configuration, results displayed that alcohol ingestion initiated an impact on participants sleep, though it is not
clearly understood because of the varying populations (i.e., non-users/infrequent users versus chronic users). Alcohol’s effects, according to Roehrs and Roth (1995), have shown significant impacts on sleep stages. Thus, the topic is relevant because individuals have shown affects from alcohol.

From a neurological standpoint, researchers described ethanol’s effects to cause marked impairments during sleep. According to Landolt et al. (1996), ethanol was found to harm the quality of sleep and the variations within sleep patterns. This finding was congruent with Roehrs and Roth’s (2001) results, who stated that ethanol has the potential to cause marked distress on “sleep, sleepiness, and sleep disorders” (p. 294). The reasons that ethanol displayed these symptoms were based on its classification as a sedative, although, it was also classified as a stimulant (Roehrs & Roth, 2001).

Researchers have shown alcohol’s effects on sleep-related neurotransmitters, which were defined as, “Gamma-aminobutyric acid (GABA) [which is] one of the major inhibitory neurotransmitters and may promote sleep” (Roehrs & Roth, 1995, p. 132). If the sedative caused sleepiness, then we can assume the stimulant would increase the amount of time to fall asleep.

Researchers have shown that ethanol affected individuals’ sleep patterns, as well as other non-sleeping areas (i.e., when awake). Kleitman (1939), author of Sleep and Wakefulness, discussed the impacts that ethanol had on sleep in healthy individuals (as cited in Roehrs & Roth, 2001). Researchers have understood that the change in sleep is apparent once alcohol is consumed, but they continue to research what these changes are. Roehrs and Roth (2001) found that “ethanol affects sleep, daytime alertness,
physiological function during sleep and hence sleep disorders” (p. 287). These researchers published their studies in various years, Kleitman (1939), and Roehrs and Roth (2001), which illustrates that the debate remains ongoing. Moreover, alcohol and sleep studies have shown data throughout the century, which adds to the trends to continue studying and researching this topic.

Researchers have shown that alcohol served as a self-medicated hypnotic for different populations. According to the International Classification of Sleep Disorders (ICSD, 2005), alcohol ingestion, when intended to be an aid to falling asleep, created numerous sleep disturbances (as cited in Feige et al., 2006). For those healthy individuals, alcohol was self-medicated as a hypnotic, which generated an increase in sleep disorders, such as insomnia (Feige et al., 2006). Thus, alcohol’s effects posited an increase in developing sleep disorders. The National Sleep Foundation (1991) discovered that “28% of those who complained of insomnia reported using alcohol to help them sleep and 67% of those felt it was effective” (as cited in Roehrs et al., 1999, p. 279). These individuals were not diagnosed with sleep disorders (i.e., insomnia); however, these individuals reported sleep deprivation that equated to insomnias symptoms. Since alcohol ingestion prior to sleep has the potential to form sleep disorders among healthy individuals, investigating this topic is relevant.

Following insomnias symptoms, researchers reported sleep disorders and their side effects. According to Görtelmeyer (2011), roughly “20 percent” of adults can be diagnosed with a sleeping disorder (para. 5). Researchers posited literature pertaining toward sleep deprivations effects on brain and behavior. According to Chee and Chuah
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(2008), several researchers accumulated data regarding the cognitive aspects, as well as the health considerations in sleep. These researchers have shown that sleep deprivation affected individuals working memory (Chee & Chuah, 2008). The researcher’s findings are relevant towards this study because if REM sleep latency is increased, then individuals could report sleep deprivation, which affects working memory. Although Chee and Chuah (2008) reported their statistical findings, they also included that sleep studies require further research. Therefore, sleep deprivation has effects on working memory; however, numerous realms of sleep have yet to be researched.

A growing trend within the literature reported alcohol ingestion creating physiological impacts on sleep. Roehrs and Roth (2001) shared findings that alcohol had a significant impact on the physiological aspects in sleep, meaning that sleep deprivation, sleep restrictions, and daytime sleepiness occurred during alcohol consumption periods. Although the effects were reported, the researchers’ evidence for accumulating their conclusion was unclear. Another phenomenon between alcohol and sleep is that alcohol “may cause either slowly reversible or irreversible damage to brain systems that regulate sleep” (Brower, 2001, p. 115). Researchers have found the symptoms and impacts that alcohol has on sleep, but there has not been enough experiments conducted that are relevant to this topic.

Alcohols cost-related impact on sleep parameters was reported as a continuing trend, according to researchers who conducted a national census. The National Institute on Alcohol Abuse and Alcoholism (NIAAA, 2000) reported that in 1998, the United States had $184.6 billion cost damages due to alcohol related problems (as cited in
Brower, 2001). Of that $184.6 billion, the alcohol-related problems based on insomnia cost the United States an estimated $18.5 billion (as cited in Brower, 2001). This researcher found the cost-related impact that alcohol has on sleep, and thus, resulting in an increased amount of damages. Comparing to Brower (2001), Courtney and Polich (2009) reported, “alcohol consumption in humans is the third leading preventable cause of death in the United States” (p. 142). The cost-related evidence behind the current topic developed awareness towards individual’s lifestyles.

Sleep disorder development (with alcohol accompanied) was discussed as a prime concern throughout the literature; thus, making this subject imperative for further research. Numerous researchers discussed this topic’s relevance and the efforts for conducting future research. Furthermore, the reasons stated above were why the current study was conducted: to synthesize accumulated data to form summary understandings of the effect of alcohol on sleep parameters.

**Hypothesis and Objectives**

Evidence from researchers assisted the current study’s focus with regard to defining terminology, validating measurements, and providing relevance towards this study’s implications. While several researchers have tested alcohol and sleep, the topic is still establishing new information. Although researchers varied in their findings upon alcohol’s effects on sleep, conclusions can be drawn throughout the various studies. In this study, I hypothesized that differences would be observed between alcohol consumption and REM sleep latency, as well as sleep latency. More specifically, I hypothesized that among healthy individuals who consumed alcohol, sleep onset latency
and REM onset latency would decrease. The null hypothesis stated no differences observed across these conditions based on sleep latency, REM sleep, and alcohol intake. I hoped to reject the null hypothesis through the present meta-analysis.

This research question was pursued through a meta-analysis for two primary reasons. First, alcohol presents significant impacts on individual’s sleep. The implications differ within the various areas of sleep with regard to the measurements and alcohol levels (i.e., BALs). The current study compared healthy individuals and alcohol-use dependent individuals. Second, and more importantly, the analyses of healthy individuals sleep latency and REM onset latency, affected by alcohol, added research to the literature. Researchers have conducted comparison studies on similar populations, but have not focused primarily on healthy individuals. Instead, their focus has been on disordered individuals (e.g., people with insomnia, alcoholism). The current study observed the outcomes combining the two populations, and observed healthy individuals outcomes only. The results have the potential to benefit fellow researchers, as well as individuals who have self-medicated with alcohol to enhance their sleep quality.

**Literature Review**

Feige et al. (2006) provided a study that supplied this research with information pertaining to the measured variables. Specifically, the variables and the measurements used. Feige et al. (2006) used polysomnography to measure the sleep latency and REM sleep latency within healthy participants. Feige et al. (2006) provided a template synonymous to the current study. The researcher’s study was the *Effects of Alcohol on Polysomnographically Recorded Sleep in Healthy Subjects* (Feige et al., 2006). Among
the numerous variables researched, sleep latency appeared as a common variable, which was a key variable used in the present study. The clinicians screened the participants through medical examinations, which determined the participant’s diagnoses with or without alcohol-use disorders. The participants were given 0.03% and 0.1% BAL (distributed during two separate studies within the article) for three consecutive days and then had a withdrawal period from alcohol for two days. Feige et al. (2006) used PSG with EEGs as the objective measure and the Schlaffragebogen-A (SF-A) and the Pittsburgh Sleep Questionnaire Index (PSQI) as the subjective measures. These four measurement forms were used in the current study; therefore, evidence and literature from Feige et al. (2006) was crucial for this study. The results from the objective measurements calculated that 0.03% BAL did not affect sleep latency significantly; however, 0.1% BAL decreased the sleep latency for participants. For sleep latency, the mean and standard deviation on the third night of sleep with 0.03% BAL was “14.45 ± 7.09,” and with 0.1% BAL was “10.60 ± 5.64” (Feige et al., 2006, p. 1531). The statistical evidence from these researchers’ findings suggested that increasing BAL could decrease the amount of time to fall asleep. The results from the subjective measurements calculated “no effects of alcohol could be detected at 0.03% BAL, and only 1 parameter (exhaustion in the evening) showed an effect in the 0.1% BAL condition” (Feige et al., 2006, p. 1534). Although the self-reported evidence did not provide as much information as the PSG, the research remained pertinent because diminished sleep latency was reported in all measures.
Researchers reported a decrease in sleep latency among participants with and without sleep disorders. Similar to Feige et al. (2006), Roehrs et al. (1999) compared the breath ethanol concentrations (BEC) in healthy participants with participants who were diagnosed with insomnia. These researchers compared healthy populations to non-healthy populations; this type of design shaped this meta-analysis because these researchers assessed the differential effects of ethanol on SOL/ROL across two populations (alcoholic versus non-alcoholic). Roehrs et al. (1999) focused on participants diagnosed with insomnia; however, these participants were compared with healthy individuals, which was the current study’s focus. After numerous tests, the participants were categorized in normal and insomniac groups where they were either in the placebo group or in the ethanol group (Roehrs et al., 1999). Through PSG and EEG epochs, the normal group calculated means and standard deviations: the placebo group, 11.9 ± 11.8, and the ethanol group, 10.1 ± 7.22 (Roehrs et al., 1999). These findings illustrated a decrease in sleep latency, which corresponds with Feige et al. (2006).

**Key Definitions**

Researchers reported the symptoms, significance, the impacts, and the outcomes that alcohol has on sleep. One leading symptom from “alcohol-related sleep problems,” according to Brower (2001), is “insomnia” (p. 110). While insomnia and alcohol-related sleep problems were the main sleep disorders/problems discovered during the literature review, the populations were not included in this study. Görtelmeyer (2011), a researcher who has conducted numerous sleep studies since 1981, defined sleep as “an integral part
of the circadian rest-activity cycle” (p. 6). Alcohol’s symptoms on sleep serve as a reminder towards the potential implications.

This meta-analysis used terminology that researchers proposed, which helped comprehend the message, and helped organize the design. Beginning with the independent variable, Anderer et al. (2005) defined sleep as “...a reversible behavioral state of perceptual disengagement from and unresponsiveness to environmental stimuli” (p. 116). This definition relates to Görtelmeyer’s (2011) definition, but adds a biopsychosocial perspective.

Additional terminology related to sleep has been defined throughout the literature. Since sleep latency was the dependent variable in the present study, it is important to understand the meanings. Brower (2001) and Roehrs and Roth (1995) defined sleep latency and REM sleep latency. The terminology was important to differentiate because the definitions pose similarities and differences. For instance, sleep latency, or sleep onset latency (SOL), is calculated as the amount of time between intending on falling asleep and the beginning of sleep (Brower, 2001). Likewise, Roehrs and Roth (1995) defined sleep latency as “…the time [in minutes] between lying down and the onset of sleep” (p. 131). REM sleep latency, or REM onset latency (ROL), is a sleep stage, which occurs after sleep has commenced; it is the period between sleep onset and the initial REM sleep episode (Brower, 2001). REM sleep latency and REM onset latency are interchangeable and synonymous. These definitions were consistent throughout the literature; these terms were utilized throughout the research portion and findings. In summary, sleep latency is the amount of time it takes from initiating sleep to falling
sleep; this portion of sleep can be measured either subjectively or objectively. REM sleep latency is the amount of time it takes to get to REM sleep cycle from Nonrapid Eye Movement (NREM); this can only be measured objectively. The current study included sleep latency and REM sleep latency as the variables to be measured.

As implied above, researchers have delineated two sleep stages: REM sleep and NREM sleep. Anderer et al. (2005) defined REM sleep as a sleep stage, which consists of dreams and eye movements that occur quickly—as the name implies. “Other variables used to characterize sleep are the percentage of total time spent in REM sleep (i.e., REM %) and in [slow-wave sleep (SWS)] (i.e., SWS %), respectively,” which were not used in this study; however, the definitions are important when understanding REM sleep (Brower, 2001, p. 111). Anderer et al. (2005) defined NREM sleep, as a sleeping period, which does not include fast (or rapid) eye movement. Unlike REM sleep, NREM sleep contains four stages (although some researchers are arguing that there are only three stages): “Stages 1 and 2 are sometimes referred to as light sleep, because it is relatively easy to awaken people during these stages. Stages 3 and 4 collectively are called deep sleep, or SWS [slow-wave sleep], because it is difficult to awaken people during these stages” (Brower, 2001, p. 116). Differentiating the two main sleep stages is important to consider, because the present study focused primarily on sleep onset (i.e., before NREM) and REM sleep latency (i.e., before REM).

Lichtblau (2011), author of *Psychopharmacology Demystified*, provided definitions pertaining to the dependent variable. Alcohol is considered a drug; therefore, the effects must be categorized and understood. According to Lichtblau (2011): “Alcohol
is metabolized in the body in a two-step process. Ethanol is first metabolized by alcohol dehydrogenase to form acetaldehyde, which is subsequently metabolized by aldehyde dehydrogenase to generate acetic acid and water” (p. 102). *Ethanol* and *alcohol* are synonymous, which is why they were used interchangeably throughout this study.

Two other terms that were used interchangeably throughout this study were *alcohol consumption* and *alcohol intake*.

Few researchers recorded participant’s alcohol levels; however, alcohol concentrations are important to understand. Courtney and Polich (2009) conducted a study in which blood alcohol concentration (BAC) was described as a quantifiable measurement to define an individual’s alcohol levels. The Alcohol Policy Information System (2007) has established that 0.08% is “...the legal intoxication level in all 50 states” (as cited in Courtney & Polich, 2009, p. 146). Similar to Courtney and Polich (2009), Feige et al. (2006) reported the blood alcohol level (BAL) in their research. The BAC and BAL are used interchangeably.

One focus in this study was to compare healthy individuals with alcohol dependent individuals; therefore, clarifying the two populations should be explained and understood. There are numerous effects from alcohol can be therapeutic, as well as adverse (Lichtblau, 2011). The adverse effects that alcohol displays are classified as *addiction*, according to the American Psychiatric Association (APA) in the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition-Text Revision* (DSM-IV-TR; as cited in Lichtblau, 2011). According to Goodman (2008; as cited in Lichtblau, 2011):
...addiction is a condition in which a behavior that can function both to produce pleasure and to reduce painful affects is employed in a pattern that is characterized by two key features: (1) recurrent failure to control the behavior, and (2) continuation of the behavior despite significant harmful consequences. (p. 103)

Classifying the definition is imperative towards conceptualizing the current study because numerous researchers focused on populations with addiction. Several researchers have studied sleep within patients who have been diagnosed with alcohol as a substance use disorder (Courtney & Polich, 2009; Landolt et al., 1996; Roehrs & Roth, 2001). Understanding and being able to differentiate between the two populations (i.e., healthy versus alcohol dependent) is relevant, as these populations were the foundation for the data analyses.

Addictions operationalization is important to consider because many researchers have included this term in their participants demographic sections. The term binge drinking is similar to addiction; however, the NIAAA (2004) defined this term as “a pattern of drinking alcohol that brings [blood alcohol concentration] to 0.08-gram percent or above. For the typical adult, this pattern corresponds to consuming five or more drinks (male), or four or more drinks (female), in about two hours” (as cited in Courtney & Polich, 2009, p. 146). These researchers incorporated alcohol addiction, alcohol binging, and binge drinking. The data were extracted through comparative analyses against control groups. Binge drinking and alcohol addiction display a wide spectrum based on
how severe sleeps impact was; therefore, alcohol consumption and alcohol intake were constructed as the manipulated variable within researchers’ experiments.

**Objective Variables**

In sleep studies, objective measurements are assessments of study variables (SOL and ROL in this study) that do not allow the participants to provide self-reported outcomes, but instead use physiological measurements to avoid bias. These objective measurements are obtained primarily in laboratory settings; the objective measurements selected in the inclusion criteria for my study were polysomnography (PSG) recordings and/or electroencephalograph (EEG) recordings. As a key variable to this study, it is important to recognize how the PSG has developed throughout history. A PSG “measures numerous variables, including breathing characteristics, eye movements, leg movements, percentage of time spent in each sleep stage, sleep continuity (i.e., sleep latency, total sleep time, and sleep efficiency), and REM sleep latency” (Brower, 2001, p. 111). These measurements are recorded specifically through either “a fixed sleep-wake schedule or based on habitual sleep patterns,” according to Ohayon, Carskadon, Guilleminault, and Vitiello (2004, p. 1257). Between the 1970s and the 1980s, researchers began utilizing this sleep measurement; in fact, the patients that researchers were treating had been diagnosed as alcoholics (Brower, 2001). This standard measurement has been used for over 40 years, which creates for a valid and credible approach to testing individuals sleep as well as other variables (i.e., alcohol consumption).
Anderer et al. (2005), Carskadon and Dement (2000), and Carskadon and Rechtschaffen (2011) provided information pertaining to EEG recordings, the second objective variable. This variable coincides with the history and the functioning of the PSG. In fact, an EEG “…is considered as the ‘core measurement of polysomnography’, ” according to Anderer et al. (2005, p. 116). Data from EEGs are recorded through *epochs*, defined as a “…set of single recordings by digital averaging of [recording periods],” according to Teplan (2002, p. 4). EEGs track individuals’ patterns during their sleep; assessing these sleeping patterns occurs through “time segments of 20 or 30 [seconds], which are referred to as ‘epochs’. Thus, 8 [hours] of sleep consist of 960 30-second epochs” (Anderer et al., 2005, p. 116). A specific pattern EEGs display, according to Carskadon and Rechtschaffen (2011), are “sawtooth waves—because of their notched morphology—is fairly common during REM sleep, particularly in proximity to the eye movements, but is by no means a universal phenomenon” (p. 1205). Moreover, Carskadon and Rechtschaffen (2011) reported EEG measurement outcomes, and concluded “[the] rhythmic alpha activity (in range of 8 to 13 cps)” is when participants are “relaxed with the eyes closed” (p. 1201). This information is received centrally in the brain, but mainly in the occipital areas.

Rechtschaffen and Kales (1968) are the two frontrunners who developed the EEG paradigm in sleep recordings (as cited in Anderer et al., 2005). Rechtschaffen and Kales’ (1968) provided measures, which were referenced in research publications and manuals (as cited in Anderer et al., 2005). Anderer et al. (2005) stated, “[that] the only standard for the classification of sleep-EEG recordings that has found worldwide acceptance are
the rules published in 1968 by Rechtschaffen and Kales” (p. 115). The researchers included different sleep parameters, such as, “between wake, REM sleep, NREM sleep stages 1 to 4 and movement time” (Anderer et al., 2005, p. 116). Researchers have provided methods involving measurements that have assisted the current study’s outcomes.

Anderer et al.’s (2005) study defined EEGs fundamentals through how one operates, but Carskadon and Dement (2000) considered additional areas that required further development. When considering REM sleep, Carskadon and Dement (2000) discussed the physiological areas that EEGs detected: “[M]uscle atonia, and episodic bursts of rapid eye movements. REM sleep usually is not divided into stages, although tonic and phasic types of REM sleep are occasionally distinguished for certain purposes,” a low-voltage process (p. 18). The areas that EEGs target are important to consider when defining the objective variable. Feige et al. (2006) found that when sleeping participants BALs had decreased to undetectable areas that the EEG was able to locate the areas that alcohol could/would have inhibited. Outcomes included “low-frequency alpha activity was increased in non-REM and REM sleep; during slow-wave sleep (SWS), in addition, x and alpha activity were increased” (p. 1528). The REM sleep cycle activation is characterized through low-voltage frequencies, unlike the counterpart—NREM sleep stages—which are synchronous and activated through high-amplitude (delta) waves (Anderer et al., 2005). Carskadon and Dement (2000) reported evidence similar to Feige et al. (2006). REM sleeps low-voltage activation proposed mixed frequencies throughout EEG recordings (Carskadon & Dement, 2000). The patterns, fundamentals, validity, and
physiological representation throughout the literature are important to consider when qualifying the variability within the objective variables.

**Subjective Variables**

Subjective variables are measurements that allow the participants to record their own outcomes, rather than researchers. The subjective measurements included in the current study were (a) the PSQI (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989, see Appendix A), and (b) the SF-A/R, or Schlaffragebogen (Görtelmeyer, 2011, see Appendix B), which are questionnaires that measure sleep latency. The subjective outcomes were compared between healthy and alcohol dependent individuals, whether or not they received a placebo or ethanol.

Several researchers used the Schlaffragebogen (translated from German to English as “sleep questionnaire”), and the PSQI. Coupling the two questionnaires has provided researchers with feedback regarding sleep outcomes (Feige et al., 2006). Researchers have combined the subjective and the objective measurements to create research designs, which enclosed self-evaluated questionnaires and quantitative testing. While the researchers’ experiments that included statistical (i.e., quantitative) data are important, the interviews and the language-based (i.e., qualitative) evidence has not been overlooked. For this reason, both variables were included in the present study because the questionnaires assess the qualitative language, as well as the quantitative outcomes from the Likert scales within the questionnaires. To summarize the two scales, Voderholzer, Al-Shajlawi, Weske, Feige, and Riemann (2003) have provided literature about the Schlaffragebogen as a questionnaire that evaluated the “subjective aspects of
sleep in the preceding night” (p. 165). Likewise, Voderholzer et al. (2003) evaluated the PSQI and found that “[it] refers to the sleep during the preceding 2 weeks” (p. 165). These scales are essential to recognize because the present study included these measurement scales within the search criteria, in addition to the objective measurements.

Researchers have documented numerous sleep outcomes from the presented measurements, which is why these variables have been utilized in this study. Having been cited often, Buysse et al. (1989) assessed participants sleep quality through methods that were credible for various researchers. The motives behind the PSQIs development, according to Buysse et al. (1989) were:

   To provide a reliable, valid, and standardized measure of sleep quality; (2) to discriminate between ‘good’ and ‘poor’ sleepers; (3) to provide an index that is easy for subjects to use and for clinicians and researchers to interpret; and (4) to provide a brief, clinically useful assessment of a variety of sleep disturbances that might affect sleep quality. (p. 194)

The PSQI is important include in this meta-analysis because it provides a longitudinal representation for various sleep/wake disorders (Buysse et al., 1989). Before a polysomnographic is conducted, the PSQI must be administered to participants in order to quantitatively and to qualitatively assess and differentiate between good and poor sleepers (Buysse et al., 1989). In fact, Buysse et al. (1989) developed a study based upon the PSQI to assess the quality of sleep that patients had during a month prior to taking the questionnaire. “The PSQI consists of 19 self-related questions,” which “assess a wide variety of factors relating to sleep quality, including estimates of sleep duration and
latency and the frequency and severity of specific sleep-related problems” (Buysse et al., 1989, p. 195).

The PSQI serves as a reliable and valid measurement, according to researchers. Backhaus, Junghanns, Broocks, Riemann, and Hohagen (2002) discussed the PSQI’s resoluteness; “the Pittsburgh Sleep Quality Index (PSQI) has gained widespread acceptance as a useful tool to measure sleep quality in different patient groups” (p. 737). The populations in the current study, healthy individuals and alcohol-use dependent individuals, are considered accepted within this measurement. Backhaus et al. (2002) found the PSQI as a valid source; in addition, validity increased when participants were administered the PSQI as well as sleep logs. Thus, the PSQI remains a succinct measurement alone, but validity raises when sleep logs are included.

The second measure of subjective sleep variables that has been used in sleep studies is the Schlaffragebogen, a German Sleep Questionnaire, which was developed by Görtelmeyer (1981). The Schlaffragebogen contains two questionnaires, known as SF-A and SF-B (Görtelmeyer, 1981). However, in recent publications, Görtelmeyer (2011) created a revised version of these questionnaires, known as SF-A/R and as SF-B/R. The current study has used SF-A/R for reasons explained below.

Before explaining the revised version, the original questionnaire must be introduced. The SF-A has been used in numerous studies as a measurement variable, as well as a reference based on research from Görtelmeyer (1981, 2011). Mayer, Wyckoff, Fallgatter, Ehlis, and Strehl (2015) conducted an experiment using the SF-A before the EEG. The “23-item self-reported questionnaire that assesses sleep quality and behavior
for the previous night of sleep” was used in the researcher’s study (p. 8). In comparison to Mayer et al. (2015), Görtelmeyer (1981) developed the SF-A to subjectively report individuals sleep from the previous night (as cited in Feige et al., 2008). Feige et al. (2008) explained the SF-A as:

The questionnaire contains subjective estimates of wake times, the frequency of awakenings and five sub-scores for the items sleep quality, feeling refreshed in the morning, well-being in the evening, exhaustedness in the evening and ‘psychosomatic symptoms’ (e.g., the experience of palpitations, sweating, myalgia, etc.) during sleep. These scales can take the values 1 to 5. (p. 183)

The original questionnaires have been translated from German to English. The SF-A/R and Schlaffragebogen-B/Revised (SF-B/R) have been validated through several independent studies, according to Görtelmeyer’s (2011) revised manual, the *SF-A/R and SF-B/R Sleep Questionnaire A and B*. The English versions of the questionnaires were not available; in fact, I had to contact Görtelmeyer to receive the translated version. The SF-A/R includes 25 questions that were developed from five different sleep indications (Görtelmeyer, 2011). These “sleep indices” are rated (1-5) as followed: “[1] difficulty falling asleep, staying asleep [2] difficulty, [3] Early waking, sleeping [4] General characterization, [5] total sleep time” (Görtelmeyer, 2011, para. 2). These questionnaires were included in the International Scale of Psychiatry (1996, 2005), which validates and adds credibility for the questionnaire (as cited in Görtelmeyer, 2011). The questionnaires goal is to report the qualitative and quantitative sleep results from participants (Görtelmeyer, 2011). This questionnaire is administered to participants the morning after
the sleep; in other words, self-evaluation regarding sleep from the night prior
(Görtelmeyer, 2011). The measurement has been valuable towards the current study
because various researchers have utilized it in their own studies.

**Method**

The design—a meta-analysis—according to Gliner, Morgan, and Harmon (2003)
is known “as a research synthesis that uses a quantitative measure, effect size, to indicate
the strength of relationship between the treatments and dependent measures of studies
making up that synthesis” (as cited in Shelby & Vaske, 2008, p. 97). Therefore,
following this design’s definition, the current study used peer-reviewed journal articles as
the main source for data collection. Analyzing these studies has provided quantitative
outcomes from consuming alcohol, and the effects the substance has on sleep latency and
on REM sleep latency.

According to Stroup et al. (2000), a meta-analysis is “…a systematic approach to
identifying, appraising, synthesizing, and (if appropriate) combining the results of
relevant studies to arrive at conclusions about a body of research” (p. 2008). Likewise,
Glass (1976) characterized this design as an “analysis of analyses,” which means this
design pools “the statistical analysis of a large collection of analysis results from
individual studies for the purpose of integrating the findings” (p. 3). The process this
meta-analysis used, with the assistance from Shelby and Vaske (2008), was “(1) create
independent effect sizes for each study, (2) compute the weighted mean of effect sizes
using inverse variance weights, (3) determine the confidence interval for the mean, and
(4) analyze for homogeneity” (p. 102).
Study Selection

The inclusion and exclusion criteria used in this study were based on the following researchers materials and methods: Roehrs and Roth (1995, 2001), Roehrs et al. (1999), Feige et al. (2006), and Voderholzer et al. (2003) who have conducted similar studies. Specifically, studies included in the present meta-analysis met predefined date range, publication data, and design criteria. These criteria were as follows. The studies were peer-reviewed journal articles that were published between 1995 and 2015. The database, Google Scholar provided studies for the selection process. The studies were either experimental (randomized) or quasi-experimental (non-randomized) research. The experimenter’s manipulated variable would be alcohol. Therefore, the terminology included in the initial search were the following: (a) alcohol/ethanol ingestion, (b) alcohol/ethanol consumption, (c) alcohol/ethanol intake, (d) blood alcohol level/concentration (BAL/C), (e) electroencephalogram, (f) EEG, (g) polysomnography, (h) PSG, (i) Pittsburgh Sleep Questionnaire Index, (j) PSQI, (k) Schlaffragebogen-A/Revised, (l) SF-A/R, (m) sleep latency, and (n) REM sleep latency.

The process of developing search strings and search parameters for the data collection was the beginning stage for this method. This process followed these steps (see Appendix C): (a) identify database (i.e., Google Scholar), (b) create search string generic, (c) create database specific search strings, (d) conduct search (gather all resulting articles), (e) remove duplicate articles, (f) process remaining via title/abstract/full-text review, and (g) collect data from the remains.
Materials and Procedure

Once steps (a) through (e) were completed, a three-tiered process for screening the articles for potential inclusion was as follows:

**Title screening.** Reviewing the title of selected peer-reviewed journal articles occurred when the sources that included key terminology were collected. Article titles adhered to the required terminology and to the accurate publication date range (1995-2015) to be included for further examination.

**Abstract screening.** This process commenced after the title-screening phase was finished. This screening aspect required an in-depth look at each abstract that made it through the title-screening phase. The articles were included for further review if the information in the abstract adhered to the inclusion criteria. To be included during this phase, the abstracts must have met the following criteria: (a) the analyses were experimental and/or quantitative, (b) alcohol consumption was reported, (c) sleep outcomes were reported, and (d) the measurements used were PSQI, SF-A, PSG, and/or EEG.

**Full-text screening.** Screening the articles full text was the final stage for the collection process. Reviewing the full text required an in-depth analysis of the researcher’s experiments. To be included for data analysis, the remaining articles had to meet these guidelines: (a) participants were sub-grouped as either healthy or alcohol-use dependent, (b) a placebo/control/baseline was administered for comparison, (c) sleep latency and/or REM sleep latency outcomes were reported, and (d) studies reported either means, standard deviations, and p-value, or means and p-value.
Analysis

For the analysis, the Comprehensive Meta-Analysis (CMA) software was used for the data entry, moderator analysis, and overall effect size analysis using a random effects method (Borenstein, Hedges, Higgins, & Rothstein, 2009). In addition to Borenstein et al. (2009), Glass (1976), Hedges (1982), Shelby and Vaske (2008), and Stroup et al. (2000) provided guidance, which delivered a foundation for the method utilized in this meta-analysis. The moderators were quantitative variables “that affect the direction and/or strength of the relation between an independent or predictor variable and a dependent or criterion variable” (Baron & Kenny, 1986, p. 1174). A random effects method was used, rather than a fixed effects method, because the studies were not identical to one another, and the effect size was computed to all varying populations (Borenstein et al., 2009).

Results

Data Collection

From the initial title search (excluding citations and patents) across specific years (1995-2015), 179 articles (as well as one additional article found during the literature review process) were retrieved (see Figure 1). While one database was used, there were six duplicate articles, which were excluded. From the 174 deduplicated article pool, 18 articles were excluded due to complications upon retrieving the articles (i.e., unable to access), and two articles were not original studies. The second stage in the inclusion and exclusion process, abstract screening, then began with the remaining 154 articles. After the abstract screen 108 additional papers were omitted due to differing populations that
were being studied (i.e., Fetal Alcohol Syndrome) subjective/objective variables that were not consistent with those identified before the meta-analysis began, or both.

The remaining 46 articles were included in the full-text screening process, the last stage in the inclusion and exclusion process. In this stage, 36 articles were excluded due to the lack of human participants (i.e., animal studies), insufficient statistical data reports in a form amendable to effect size calculations, and/or having the included subjective/objective variables, but not reporting statistical data. In total, nine articles met the inclusion criteria for this study. Within the nine articles, 23 studies were embedded within them; therefore, 23 studies were analyzed.

**Random Effects Model**

The participant populations differed throughout the collected studies; therefore, the random effects model was used, rather than the fixed effects or the mixed effects models (Shelby & Vaske, 2008). A random effects model was used for this meta-analysis because the subgroups and the populations varied throughout the studies included in the data analysis. For instance, all studies in this meta-analysis assess effects that alcohol had on sleep, but the populations being sampled from varied demographically; thus, a random effects model for analysis is appropriate (Hedges & Vevea, 1998). The random effects model was appropriate to use for the current study because the subgroups were not specified. According to Higgins, Thompson, Deeks, & Altman (2003), the random effects model “is useful for comparisons of heterogeneity among subgroups, but values depend on the treatment effect scale” (p. 560). The collected studies’ subgroups ranged from different ethnicities, ages, and genders.
Therefore, the diverse populations cannot be compared together, as in a fixed effects model.

The objective and subjective variables helped explain use for the random effects method. The objective variables were found in the unbiased results from the PSG and the EEG. The subjective variables were in the group comparisons within the two surveys (i.e., SF-A/R & PSQI). After study collection and data extraction, this meta-analysis gained information regarding the average effect sizes across the studies (Hedges, 1982). The $Q$ statistic was included in the assessments from the random effects model.

Researchers, DerSimonian and Laird (1986), defined the $Q$ statistic as “the sum of squares of the treatment effect about the mean” (p. 181). Since some sample sizes were large, the $Q$ statistic was used to calculate the differences between group comparisons.

After study collection and data extraction, a $Q$ statistic revision was included in the assessment on whether or not to use a random effects model or a fixed effects model. Researchers, DerSimonian and Laird (1986), defined the $Q$ statistic as “the sum of squares of the treatment effect about the mean” (p. 181) and Higgins et al. (2003) noted that it is "a test for heterogeneity [that] examines the null hypothesis that all studies are evaluating the same effect" (p. 557). Having determined that a random effects model was appropriate to use, population effect sizes were estimated using Hedge’s $g$ with a 95% confidence interval on the forest plots (see Figures 2-6).
Figure 1. Flow chart displaying the source collection and the selection process for the data analysis.
Demographics of Populations within Studies

From the studies that were included in the quantitative analysis ($k = 19$), there were 2,595 total participants ($n = 1,315$) in control, comparison, placebo, and baseline groups; ($n = 946$ participants received alcohol as members of experimental or quasi-experimental conditions). Of those participants who received alcohol, 407 were diagnosed with alcohol-use disorder, while the remaining 539 participants were healthy individuals with no alcohol-use disorder diagnoses; however, not all of the 946 active condition participants were assessed in the same manner.

For the analysis of subjective measures, there were 351 alcohol-use disordered participants and 261 non-alcohol-use disordered participants. For the objective measurements of both REM Onset Latency (ROL) and Sleep Onset Latency (SOL), there were 167 participants treated with alcohol/ethanol, 28 of which were alcohol dependent participants.
Subjective variable outcomes. There were no studies that included the SF-A as a measurement variable. During the literature review, the SF-A was often coupled with the PSQI; however, the initial study collection did not find the SF-A. The SF-A is a German sleep questionnaire, which could have been why there were not any studies found using this measurement.

There were three studies included in the random effects model with the outcome from the Sleep Onset Latency (SOL) reported in the PSQI (see Figure 2). The SOL outcomes from the PSQI were analyzed from the following three studies: Chueh, Yang,
Across all studies included in this analysis (k = 3), the time it took participants in active conditions to enter sleep was not statistically different from participants in placebo conditions (g = 0.256, 95% CI [-0.122, 0.633]; p = 0.184; Q = 4.2, df (2), p = 0.12; see Figure 2). The difference in means between groups was 0.55 minutes. The null cannot be rejected and the use of a random effects model is appropriate. The magnitude of the effect size was relatively small, but still meaningful; the alcohol had a reported impact on the amount of time it took these self-evaluating participants to fall asleep. However, this small effect was not statistically significant (p = 0.184), which means that the null hypothesis cannot be rejected.

The results for the self-reported PSQI survey illustrate a potential trend that consuming alcohol has an impact on the amount of time it takes to fall asleep; however, these outcomes were not statistically significant.

**Objective variable outcomes.** Four different data analyses were conducted using the objective variables within studies: two ROL analyses and two SOL analyses. The first set of ROL and SOL analyses were done to assess global outcome; that is to provide a summary estimate on alcohol's effects on ROL and on SOL across samples, regardless of whether those samples were drawn either from healthy populations (non-alcoholics) or from alcohol dependent populations. The second ROL and SOL analyses were conducted only on the studies that sampled from populations of healthy individuals. This
analysis was my study’s focus; that is, people without alcohol dependence and the impact on their ROL and SOL.

**Objective analysis 1: ROL across all samples.** Across all studies included in this analysis ($k = 9$), the amount of time it took participants in active conditions (i.e., having received alcohol) to enter their first REM cycle was not statistically different from participants in placebo conditions ($g = 0.115$, 95% CI $[-0.255, 0.486]$; $p = 0.542$; $Q = 7.76$, $df (8)$, $p = 0.46$; see Figure 3). The difference in means between groups was 4.7 minutes. Thus, the null hypothesis cannot be rejected and the use of a random effects model was appropriate.

**Objective analysis 2: SOL across all samples.** Across all studies included in this analysis ($k = 9$), the amount of time it took participants in active conditions (i.e., having received alcohol) to enter their sleep cycle was not statistically different from participants in placebo conditions ($g = 0.034$, 95% CI $[-0.265, 0.333]$; $p = 0.824$; $Q = 9.017$, $df (8)$, $p = 0.34$; see Figure 4). The difference in means between groups was -0.16 minutes. Thus, the null hypothesis can be rejected and the use of a random effects model is appropriate.

**Objective analysis 3: ROL for only non-alcoholic participants.** Across all studies included in this analysis ($k = 6$), the amount of time it took participants in active conditions (i.e., having received alcohol) to enter their first REM cycle was statistically different from participants in placebo conditions ($g = 0.506$, 95% CI $[0.288, 0.724]$; $p < 0.001$; $Q = 1.505$, $df (5)$, $p = 0.91$; see Figure 5). The difference in means between groups was 16.086 minutes. Thus, the null hypothesis cannot be rejected and the use of a random effects model is appropriate. The result of this analysis showed that, when
healthy individuals consumed alcohol prior to falling asleep, the amount of time to reach
REM sleep was significantly longer compared to healthy individual’s not consuming
alcohol prior to sleep.

**Objective analysis 4: SOL for only non-alcoholic participants.** Across all
studies included in this analysis ($k = 6$), the amount of time it took participants in active
conditions (i.e., having received alcohol) to enter their sleep cycle was not statistically
different from participants in placebo conditions ($g = -0.143$, 95% CI [-0.359, 0.073]; $p =
0.196$; $Q = 4.371$, df (5), $p = 0.50$; see Figure 6). The difference in means between
groups was -0.95 minutes. Thus, the null hypothesis cannot be rejected and the use of a
random effects model is appropriate.
<table>
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<tr>
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<th>Subgroup within study</th>
<th>Comparison</th>
<th>Outcome</th>
<th>Statistics for each study</th>
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*Figure 2. Computed Meta-Analysis (CMA) of SOL-PSQI.*
### Table: Meta-analysis Results for Time to REM Sleep (ROL)

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<th>p-Value</th>
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**Random**

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**Figure 3.** CMA results for time to get to REM sleep (ROL) in healthy and alcohol dependent individuals.
**Figure 4.** CMA results for time to get to fall sleep (SOL) in healthy and alcohol dependent individuals.
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<th>Outcome</th>
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*Figure 5.* CMA results for time to get to REM sleep (ROL) in healthy individuals.
### Figure 6: CMA results for time to get to fall sleep (SOL) in healthy individuals.

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<td>Hedges' $g$</td>
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</tbody>
</table>

Note: The table above shows the results of a meta-analysis assessing the impact of alcohol consumption on sleep latency. The CMA results indicate a significant effect of alcohol on SOL in healthy individuals.
Discussion

Results and Implications

The purpose of this study was to discover the trends and patterns in the amount of time it took to fall asleep and the amount of time it took to get to REM-sleep after consuming alcohol. This research review added literature to the previously published studies; this was done through summarizing the findings and by translating those results into aggregated effect sizes. Researchers varied in their results, meaning that the outcomes were either statistically significant or not. Some researchers found that participants took a longer time falling asleep when they consumed alcohol, while others reported the opposite. The ongoing debate was the main reason as to why a meta-analysis was the experimental design.

The current study’s goal aimed to report and delineate between the varying results within the literature. For the SOL-PSQI, the results display participants reporting impacts on falling asleep from ingesting alcohol; however, the amount of participants reporting these outcomes was not statistically significant. Likewise, the ROL and the SOL results across all participants, regardless of whether they were healthy (non-dependent) or alcohol dependent, were not statistically significant, meaning that alcohol consumption did not increase sleep onset latency and/or REM sleep onset latency compared to participants who received a placebo.

The objective variables aim was to review the collection of populations (alcohol dependent and healthy individuals) within the studies and then analyzing only the healthy populations within the studies.
The result from Objective Analysis 3, which was statistically significant, indicated that healthy individuals who consumed alcohol took longer to reach their first REM sleep cycle than those who received a placebo or were in a control group. Conversely, the amount of time to fall asleep (SOL) was not lengthened when healthy individuals consumed alcohol, as compared to participants who consumed the placebo. These reports from the same amount of studies, with the same objective measurements, did not find that alcohol intake added time in sleep latency.

**Limitations**

The limitations within this study were based on the amount of studies collected, the variables included, and the lack of consideration given to specific subgrouping comparisons. There were 19 studies analyzed, which is relatively small. The ROL and SOL outcomes had six articles each for data comparison. The same six articles were used for each comparison and the total sample size was relatively small for a meta-analysis.

The PSQI subjective variable was reported in three studies, which was, again, a relatively small outcome for a meta-analysis. The SF-A variable was not found within the data collection process, which could have been for a various reasons. The survey was created in German and could have been reported in more German published articles than in English, but nonetheless, the lack of articles reporting the SF-A was a limitation of this meta-analysis.

The specificity of subgroup comparisons should have been explained in detail during the method section. The ranges within the subgroups were not a limitation to this study because this study aimed to compare alcohol consumption with no alcohol
consumption without a specific group targeted. However, the difference between alcohol-use disorder and alcohol consumption was not clearly established. The results included studies with alcohol-use disorder, which is not the same as alcohol consumption. Differentiating between the two could have created less discrepancy between the subgroups.

**Future Studies**

After conducting this research, I believe that future studies should include subgroup analyses, recording BALs, and larger sample sizes. In general, further studies published could add literature for future systematic reviews. While some analyses were inconclusive, the ROL analysis was statistically significant. This finding recognized that alcohol has a significant impact on healthy individuals’ time to reach their REM sleep. Moreover, the results show that future research should be conducted based on the varying inconclusive and significant data.

During the data extraction process, researchers included primarily college-aged students (18 to 25-years-old). Utilizing a specific age as a subgroup category could compare alcohol consumption on sleep latency and REM sleep latency within the certain age groups (e.g., college aged participants), or compare those ages to an older age range (e.g., 21-year-old participants and 50-year-old participants). Supplying an age category could add more succinct and valid results; thus, creating specific subgroups should be included in future studies.

The data collected and analyzed have demonstrated trends between SOL and ROL (after consuming alcohol); however, there are a limited number of studies, which address
the variables included in this research. For instance, the SF-A was not found during data collection, even though it was found on numerous occasions during the literature review. The SF-A was frequently paired with the PSQI, but was not collected during the initial search process in this study. While the SF-A was widely used during the literature review, the language barrier, and translation from the German variable made it unable to be collected during data collection. Therefore, the SF-A is an important measurement; if studied more, it would add credible information for future research.

Overall, this meta-analysis provided some initial clarity on an otherwise unclear question. The question was unclear because of the varied results across individual studies. The statistical outcomes add literature into the topics debate.

In addition, the study’s results contradict with other study’s results such as Rundell et al. (1972; as cited in Feige et al., 2006). Rundell et al. (1972) reported an increase in REM sleep cycles for those who consumed alcohol before falling asleep (as cited in Feige et al., 2006). The current study found the amount of time to reach REM cycle, among healthy individuals, was lengthened. The results did not include the overall period in REM sleep; moreover, the evidence from Rundell et al. (1972) is relevant. This point is relevant because the current study focused on the time to sleep and REM sleep, and therefore, Rundell et al. (1972) added another realm to which this study did not. Likewise, Feige et al. (2006) reported that alcohol ingestion in healthy individuals decreased the sleep efficiency in their REM sleep cycles. Although REM latency is lengthened, the current study did not reveal the overall sleep efficacy. *Sleep efficiency*, according to Brower (2001), “refers to the proportion of time in bed that is spent
sleeping” (p. 2). More specifically, the current study did not analyze the “total time spent in REM sleep (i.e., REM %)” (p. 2). Understanding the amount of time spent in specific sleep cycles might have added clarity to the findings within Rundell et al. (1972; as cited in Feige et al., 2006). Researchers varied among their results, which is why this meta-analysis was able to provide a synopses from the published results.

Feige et al. (2006) reported that alcohol ingestion in healthy individuals increased the amount of time to fall asleep (SOL); however, Roehrs et al. (1999) stated the opposite. Sleep latency was shortened in healthy individuals, according to Roehrs et al. (1999). Although the current study was unable to report the statistical significance from these variables, future studies should implement them. The contrasting points from researchers were noted in the literature review. During data extraction, it became apparent that there were not enough studies to draw a firm conclusion about SOL and ROL under alcohol ingestion conditions. Therefore, more studies need to be conducted that follow alcohol’s effects on healthy individuals sleep latency and REM latency.

This topic’s relevance remains succinct with what researchers have posited in their literature. For instance, it is known that alcohol has an effect on sleep (Roehrs & Roth, 1995). Alcohol’s effects on sleep, according to Brower (2001), can evolve into “irreversible damage to [the] brain systems that regulate sleep” (p. 115). Healthy individual’s self-medicating alcohol to assist falling asleep is increasing the effects that alcohol will have on the anatomical brain structures that assist sleep (Roehrs et al., 1999). Once individuals have used the substance as a hypnotic, they then began to display symptoms from various sleep disorders (i.e., insomnia; Feige et al., 2006).
Researchers have supported this topic’s relevance. The relevance is more than alcohol’s effects on sleep; in fact, the importance lies within the potential outcomes from these effects. Developing a sleep disorder and creating irreversible damage to the anatomical structures that control sleep are just two reasons as to why this topic is important for individuals who consume alcohol.
References

References marked with an asterisk indicate studies included in the meta-analysis.

Anderer, P., Gruber, G., Parapatics, S., Woertz, M., Miazhynskaia, T., Klosch, G.,…
doi:10.1159/000085205


IMPACT OF ALCOHOL CONSUMPTION ON SLEEP LATENCY


doi:10.1037/a0014414


doi:10.1111/j.1365-2869.2008.00651.x

doi:10.1111/j.1530-0277.2006.00184.x


IMPACT OF ALCOHOL CONSUMPTION ON SLEEP LATENCY


Appendix. Pittsburgh Sleep Quality Index (PSQI)

Name _________________________ ID # __________ Date __________ Age ________

**Instructions:**
The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, when have you usually gone to bed at night?
   
   USUAL BED TIME __________

2. During the past month, how long (in minutes) has it usually take you to fall asleep each night?

   NUMBER OF MINUTES __________

3. During the past month, when have you usually gotten up in the morning?

   USUAL GETTING UP TIME __________

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)

   HOURS OF SLEEP PER NIGHT __________

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you...
   
   (a) Cannot get to sleep within 30 minutes
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (b) Wake up in the middle of the night or early morning
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (c) Have to get up to use the bathroom
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (d) Cannot breathe comfortably
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (e) Cough or snore loudly
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (f) Feel too cold
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (g) Feel too hot
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (h) Had bad dreams
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______

   (i) Have pain
   
   Not during the past month ______  Less than once a week ______  twice a week ______  three or more times a week ______
IMPACT OF ALCOHOL CONSUMPTION ON SLEEP LATENCY


### Sleep questionnaire (SF-A/R)

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Today's date: ____________</td>
<td>Patient number: ____________</td>
</tr>
<tr>
<td>Age: ____________</td>
<td>Sex: m/f</td>
</tr>
<tr>
<td><strong>Good Morning!</strong></td>
<td>Please take some time to respond to the following questions about how you slept.</td>
</tr>
<tr>
<td><strong>Please tick those answers that seem to be the most appropriate for you!</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Move from one question to the next without interruption and do not omit any questions!</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Please fill out immediately on waking up in the morning!</strong></td>
<td></td>
</tr>
<tr>
<td><strong>1. At what time did you start to go to sleep last night?</strong></td>
<td>example: 11:00 p.m.</td>
</tr>
<tr>
<td></td>
<td>[ ] a.m.</td>
</tr>
<tr>
<td></td>
<td>[ ] hours : [ ] minutes</td>
</tr>
<tr>
<td><strong>2. How long did it take you to fall asleep last night?</strong></td>
<td>less than 1 minute</td>
</tr>
<tr>
<td></td>
<td>1 to 5 minutes</td>
</tr>
<tr>
<td></td>
<td>6 to 15 minutes</td>
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<td></td>
<td>16 to 30 minutes</td>
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<tr>
<td></td>
<td>more than 30 minutes</td>
</tr>
<tr>
<td><strong>3. If you could not fall asleep immediately last night, what do you think was the cause? (More than one answer possible)</strong></td>
<td>personal or work problems</td>
</tr>
<tr>
<td></td>
<td>noise inside or outside the room</td>
</tr>
<tr>
<td></td>
<td>too many thoughts on my mind</td>
</tr>
<tr>
<td></td>
<td>thoughts or worries about the coming day</td>
</tr>
<tr>
<td></td>
<td>pain</td>
</tr>
<tr>
<td></td>
<td>I had to go to the toilet</td>
</tr>
<tr>
<td></td>
<td>my thoughts were going round and round</td>
</tr>
<tr>
<td></td>
<td>strange surroundings</td>
</tr>
<tr>
<td></td>
<td>others: ____________________________</td>
</tr>
<tr>
<td><strong>4. It often happens that vivid visual images appear in the phase of falling asleep. Did this happen to you last night?</strong></td>
<td>no</td>
</tr>
<tr>
<td></td>
<td>I am not sure</td>
</tr>
<tr>
<td></td>
<td>yes, definitely</td>
</tr>
<tr>
<td><strong>5. Did you experience any muscle twitches in your arms or legs last night?</strong></td>
<td>no</td>
</tr>
<tr>
<td></td>
<td>slight</td>
</tr>
<tr>
<td></td>
<td>severe</td>
</tr>
<tr>
<td><strong>6. Did you experience a stabbing pain in your chest last night?</strong></td>
<td>no</td>
</tr>
<tr>
<td></td>
<td>slight</td>
</tr>
<tr>
<td></td>
<td>severe</td>
</tr>
</tbody>
</table>

Please turn over!
<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. Did you wake up at any time last night (including times when you felt only half-awake)?</td>
<td>no, not at all, yes, once, yes, twice, yes, three times, yes, more than three times</td>
</tr>
<tr>
<td>8. If you woke up during the night at any time, what do you think was the cause? (More than one answer possible)</td>
<td>personal or work problems, noise inside or outside the room, I had to go to the toilet, I was awoken by a dream, pain, I had not entered into a deep sleep, others:</td>
</tr>
<tr>
<td>9. If you woke up last night at any time, how long were you awake? Estimate the total time of wakefulness over the whole night.</td>
<td>less than 1 minute, 1 to 5 minutes, 6 to 15 minutes, 16 to 30 minutes, more than 30 minutes</td>
</tr>
<tr>
<td>10. Do you remember whether you dreamt last night?</td>
<td>no, I don’t remember whether I dreamt, yes, but I don’t remember the contents of my dreams, yes, and I remember the contents of my dreams</td>
</tr>
<tr>
<td>11. If you remember your dreams, what kind of dream(s) did you have?</td>
<td>pleasant dream(s), neutral dream(s), unpleasant dream(s)</td>
</tr>
<tr>
<td>12. Were you aware of sweating during the night?</td>
<td>no, slight, severe</td>
</tr>
<tr>
<td>13. Did you feel cold at any time during the night?</td>
<td>no, yes, while going to sleep, yes, during the night, yes, while waking up</td>
</tr>
</tbody>
</table>
14. At what time did you wake up this morning? example: 6:30 a.m. □ a.m. □ p.m. 

|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 15. Did you use an external source (e.g., alarm clock, radio, or other person) to wake up this morning? | no |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
|   | yes |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
|   | scheduled time: | □ a.m. | □ p.m. |
|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 16. From what depth of sleep were you awoken this morning? | I awoke gradually by myself | □ a.m. |
|   | I was awoken from a light sleep | □ a.m. |
|   | I was awoken from a deep sleep | □ a.m. |
| 17. Did you wake up too early this morning and have trouble falling back to sleep again? | no | □ a.m. |
|   | yes | □ a.m. |
| 18. Did you awake with a headache this morning? | no | □ a.m. |
|   | yes, a slight headache | □ a.m. |
|   | yes, a severe headache | □ a.m. |
| 19. Did you drink an alcohol-containing beverage (beer, wine, spirits, or others) after dinner last night? | no | □ a.m. |
|   | yes, over the evening | □ a.m. |
|   | yes, immediately before going to bed | □ a.m. |
| 20. Did you take any sleeping aids (pills, tea, or others) last night? | no | □ a.m. |
|   | yes | □ a.m. |
| 21. If yes, which sleeping aid(s)? |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 22. Was yesterday an unusually exhausting day for you? | no | □ a.m. |
|   | yes | □ a.m. |

Please check whether you have answered all questions! Please turn over!
### Directions:
On this page you will find a few expressions to describe how you felt last night before going to bed, how you slept during the night, and how you are feeling this morning.
After every expression please tick the box which is most appropriate for you.

**Please move quickly from one to the next without omissions.**

<table>
<thead>
<tr>
<th>23. How was your sleep last night?</th>
<th>not</th>
<th>little</th>
<th>medium</th>
<th>rather</th>
<th>very</th>
</tr>
</thead>
<tbody>
<tr>
<td>fitful</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>deep</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>restless</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>relaxed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>untroubled</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>good</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>long</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>24. How did you feel at the end of the day yesterday, before going to bed?</th>
<th>not</th>
<th>little</th>
<th>medium</th>
<th>rather</th>
<th>very</th>
</tr>
</thead>
<tbody>
<tr>
<td>carefree</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>exhausted</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>in need of sleep</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>stressed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>composed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>calm</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>tired</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>relaxed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>25. How do you feel this morning?</th>
<th>not</th>
<th>little</th>
<th>medium</th>
<th>rather</th>
<th>very</th>
</tr>
</thead>
<tbody>
<tr>
<td>composed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>drowsy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>energetic</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>ready to act</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>wide awake</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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</tr>
<tr>
<td>well rested</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>relaxed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

Please check whether you have answered all questions!

### Remarks/questions:

Gottingen: Hogrefe.
Appendix C

(a) Identify database. Google Scholar was the only database used in this meta-analysis. Other databases were not used because Google Scholar had the articles that other Concordia University databases had. The amount of duplicates would have increased, so instead, I used one database.

(b) Create search string generic. The articles had to be published in peer-reviewed journals, theses, or conference proceedings. The articles had to measure the outcomes related to sleep (dependent variable) and alcohol ingestion (independent variable). The specific dependent variables (under the realm of sleep) were sleep onset latency and REM onset latency.

(c) Create database specific search strings. The initial search did not include patents or citations. In addition, the articles had to be published between 1995 and 2015. The initial search string generic was as follows: “allintitle: sleep AND alcohol OR ethanol AND bac OR bal”

(d) Conduct search (gather all resulting articles). All articles that were found in the initial search string were then transferred to an online database, Zotero. From here, I was able to organize each article into inclusion and exclusion categories.

(e) Remove duplicate articles. Since only one database was used to retrieve articles, the number of duplicated articles was minimal.

(f) Process remaining via title/abstract/full-text review. This three-tiered process was conducted in a linear manner; meaning that the titles of each article were reviewed, once title searches finished, then I moved on to the abstract review, and so on. During full-text
review, the articles were screened for reporting the measurements of REM sleep latency and sleep latency (objective report [i.e., PSG and/or EEG]), and sleep latency (subjective report [i.e., PSQI and/or SF-A]).

**(g) Collect data from the remains.** Once the quantitative data was collected, the comparison of all three variable outcomes occurred. The data was imputed in the CMA software. From there, the Hedge’s $g$ and the $Q$ statistics were calculated using a random effects model. This process extracted the data, analyzed the data, and then answered the null hypotheses.