THINK THAT’S WHAT I SLEPT: COMPARING THE DISCREPANCY BETWEEN SELF-REPORTED AND OBJECTIVELY MEASURED SLEEP AT HOME AND IN THE LABORATORY

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I THINK THAT’S WHAT I SLEPT: COMPARING THE DISCREPANCY BETWEEN SELF-REPORTED AND OBJECTIVELY MEASURED SLEEP AT HOME AND IN THE LABORATORY

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Abstract

Previous research has found that patients diagnosed with insomnia tend to over report how long it takes them to fall asleep and underreport their total sleep time as compared to objective measures of sleep, a phenomenon called sleep-state misperception. Although sleep-state misperception has been observed in home and laboratory settings, it is unclear if the magnitude of the discrepancy between self-reported and objectively measured sleep differs depending on sleep location. The purpose of the current study was to determine whether the difference between self-reported and actigraphy measured sleep varied depending on sleep location, and if these differences were due to differences in pre-sleep arousal between the home and the laboratory. Although differences in pre-sleep arousal did not predict differences between sleep-state misperception at home and in the laboratory, results demonstrated that sleep-state misperception is present in both home and laboratory settings and that estimates of sleep-state misperception based on one night of laboratory data may over-estimate the severity of the problem. Therefore, researchers and clinicians should consider how the method and location of sleep assessment they use in their studies and clinical practice impacts sleep and the results of research and clinical assessments.
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Figure 1. Arousal Self-Assessment Manikin (Arousal SAM).
Insomnia is one of the most commonly diagnosed sleep disorders. It is associated with fatigue, impaired cognition, and difficulty with mood, behavior, occupation, and interpersonal relationships. Approximately 30% of adults experience symptoms of insomnia (e.g., difficulty falling asleep, early morning awakenings, non-restorative sleep, poor quality sleep, and daytime impairment), and 5 to 10% meet criteria for an insomnia diagnosis (LeBlanc et al., 2007; Mai & Buysse, 2008; Ohayon, 2002). Although insomnia is primarily diagnosed through patient self-report, research focused on the etiology and treatment of insomnia have also utilized objective measures of sleep such as actigraphy and polysomnography (PSG) (Chesson et al., 2000; Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). Current research has increasingly focused on the discrepancy between self-reported and objectively measured sleep amongst patients diagnosed with insomnia. This study investigates differences in the perception of sleep in a naturalistic and laboratory setting amongst patients diagnosed with insomnia.

Insomnia

Insomnia is often conceptualized as insufficient sleep. However, insomnia is more specifically defined as having difficulty with initiating and maintaining sleep. The International Classification of Sleep Disorders (ICSD-2) (American Academy of Sleep Medicine, 2005) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association, 2000) criteria for insomnia specify that patients must report sleep onset latency (SOL) and wake after sleep onset (WASO) greater than 30 minutes per night. Difficulty with SOL and WASO must occur for three or more nights per week and must persist for at least one month. Difficulty with sleep occurs even when a person has plenty of opportunity to sleep (e.g.,
enough time to sleep, comfortable sleeping conditions) (Edinger et al., 2004; Mai & Buysse, 2008). Although difficulty falling asleep and experiencing many awakenings during the night are common in insomnia, they are often difficult to quantify because insomnia is primarily diagnosed through self-report.

**Subjective Versus Objective Sleep**

Studies that have utilized objective and subjective measures of sleep have found that there are often discrepancies between self-reported sleep, measured using self-report sleep diaries, and objectively measured sleep, typically measured through actigraphy or an overnight sleep evaluation using PSG, amongst participants with insomnia (Edinger & Krystal, 2003; Harvey & Tang, 2012). For example, Frankel, Coursey, Buchbinder, and Snyder (1976) compared self-report and PSG sleep data between participants with and without insomnia. They found that participants with insomnia self-reported total sleep times (TSTs) that were lower than PSG estimated TSTs and self-reported SOLs that were lower than PSG estimated SOLs. Participants without insomnia demonstrated no significant difference between self-reported and PSG measured TST and SOL. Manconi et al. (2010) arrived at similar results using similar methodology. Additionally, Bianchi, Williams, McKinney, and Ellenbogen (2013) found that participants with insomnia underreported WASO compared to PSG WASO data. These data suggest that self-reported sleep may be unreliable, and that discrepancies between subjective and objective sleep are ubiquitous amongst patients diagnosed with insomnia.

Clinically, the phenomena of sleep under and overestimation are referred to as sleep-state misperception (Edinger et al., 2004; Edinger & Krystal, 2003; Harvey & Tang, 2012; Vanable, Aikens, Tadimeti, Caruana-Montaldo, & Mendelson, 2000). Patients diagnosed with sleep-state misperception tend to underestimate TST and overestimate SOL (Edinger & Fins, 1995; Edinger
Much like insomnia, sleep-state misperception impacts patients’ psychological and physiological well-being and behavioral functioning. Patients are also at higher risk for ruminating over lack of sleep (e.g., ruminating about perceived low TST and high SOL) (Harvey & Tang, 2012). Although sleep-state misperception is considered to be a subtype of insomnia, a large body of research, including the research described previously, suggests that sleep-state misperception may be better thought of as a core feature of insomnia rather than a subtype of insomnia (Edinger & Krystal, 2003; Harvey & Tang, 2012; Salin-Pascual, Roehrs, Merlotti, Zorick, & Roth, 1992; Semler & Harvey, 2005; Vanable et al., 2000).

The underestimation of TST and overestimation of SOL and WASO is pervasive amongst patients diagnosed with insomnia. It is believed that these discrepancies between self-reported and objectively measured sleep may significantly contribute to the maintenance, worsening, and trivialization of insomnia. Therefore, researchers have made it a priority to explore factors that impact the misperception of sleep. Even though results from these studies have been robust and promising, they are not without their limitations. There has been little variance in the methodology used to explore sleep misperception. Specifically, a majority of research has examined sleep as it occurs in a laboratory setting.

**Methodological Considerations: Sleep Environment**

Few studies have examined sleep misperception as it occurs in a naturalistic sleep environment. Instead, a majority of studies have recruited participants to sleep in a laboratory for varying amounts of times (Harvey and Tang, 2012). Researchers have found that PSG studies conducted in a laboratory yield different estimates of sleep compared to PSG studies conducted at home (Edinger et al., 2001). Laboratory studies introduce measurement bias, resulting in sleep
that is either demonstrably worse (e.g., more time spent awake, shorter TST) or better (e.g., less time spent awake, greater TST) on the first night than during subsequent nights in the sleep laboratory (Agnew, Webb, & Williams, 1966; Tamaki, Nittono, Hayashi, & Hori, 2005). The term “First Night Effect” (FNE) is used to describe having poor laboratory sleep and recently, researchers have coined the term “Reverse First Night Effect” (RFNE) to describe the phenomenon of improved sleep observed during the first night in a sleep laboratory. Although the FNE is most commonly observed, both the FNE and RFNE phenomena have been documented in research participants with and without insomnia (Hirscher et al., 2015; Littner et al., 2003; McCall & McCall, 2012; Newell, Mairesse, Verbanck, & Neu, 2012; Riedel, Winfield, & Lichstein, 2001).

The determinants of FNE and RFNE are not fully understood. It is assumed that FNE is caused by the obvious discomfort and unpleasantness associated with sleeping in an unfamiliar location. However, these objective environmental characteristics do not explain all of the variance in FNE and are unlikely to be the cause of RFNE. Psychological factors such as pre-sleep arousal have been implicated as a potential cause of the FNE as well as RFNE and may also drive sleep state misperception (Agnew et al., 1966; McCall & McCall, 2012; Tang & Harvey, 2004). That is, higher levels of pre-sleep arousal may explain why participants report worse sleep their first night in a laboratory while lower levels of pre-sleep arousal may explain why participants may report sleeping better.

**Pre-Sleep Arousal**

Harvey and Tang (2012) describe arousal as a heightened sense of “being” (e.g., "activation and/or agitation"). Arousal includes both cognitive and physiological processes, such as muscular tension, increased heart rate, being “mentally alert,” and racing thoughts (Nicassio,
Mendlowitz, Fussell, & Petras, 1985). Pre-sleep arousal refers to the state of activation an individual experiences prior to falling asleep. It has been measured through electroencephalography (EEG) and self-report (Robertson, Broomfield, & Espie, 2007; Sforza, Chapotot, Pigeau, & Buguet, 2008). It is not only implicated as a precipitating factor to the FNE and RFNE phenomena, but also as a causal factor of sleep-state misperception.

Pre-sleep arousal has been found to contribute to the maintenance of insomnia and the misperception of sleep. Poor sleepers tend to report experiencing greater arousal prior going to bed (Ong, Carde, Gross, & Manber, 2011). In their review of the sleep-state misperception literature, Harvey and Tang (2012) identified pre-sleep arousal as a factor that influences the misperception of sleep such that patients with higher levels of pre-sleep arousal have greater difficulty with differentiating sleep from wakefulness. It has been found that patients with higher levels of pre-sleep arousal self-report lower TST and greater SOL compared to actigraphy measures (Maes et al., 2013; Tang & Harvey, 2004). These findings imply that the misperception of sleep may be different at home versus in a sleep laboratory due to differences in pre-sleep arousal. That is, pre-sleep arousal may differ at home versus in the laboratory and therefore the assessment of sleep misperception may differ depending on the sleep location. Specifically, Hauri and Olmstead (1989) suggest that the FNE and RFNE phenomena occur because of “conditioned arousal.” That is, patients’ sleep environments contain cues which patients associate with wakefulness. When patients change sleep environments, the arousal cues are either eliminated or, in the case of the FNE, increased and patients experience better or worse sleep.

Summary
A growing body of research has found that patients diagnosed with insomnia often provide unreliable estimates of their sleep. This is problematic because insomnia is diagnosed through self-report. Researchers have sought to determine causal factors for the discrepancy between self-reported and objectively measured sleep. A majority of the studies we reviewed utilized laboratory sleep studies. Few studies have examined the perception of sleep as it occurs in a participant’s home. Studies that have included both home and laboratory sleep data either combined sleep data from both settings or analyzed the data separately (Edinger et al., 2000; Mercer et al., 2002). Considering the breadth of research that has demonstrated that sleep is impacted by environment, it is problematic to assume that the perception of sleep in a laboratory is similar to the perception of sleep at home.

Patients’ perspectives of their well-being are important, especially when their perspectives drive treatment seeking behaviors and diagnostic decisions. This is especially true amongst patients diagnosed with insomnia. The discrepancy between self-report and objectively measured sleep is influenced by many factors. It is important to determine if environment, a factor that is not often manipulated or considered in sleep-misperception research, impacts the aforementioned discrepancy. It is beneficial to compare the misperception of sleep in a naturalistic environment to a controlled environment. Patients do not continuously sleep in laboratories for extended periods of time. Instead, they will sleep in environments that may not be free from disruption and distraction. Deriving conclusions solely from laboratory data may lead researchers and clinicians to develop treatment protocols, measurement tools, research questions, and case conceptualizations that are not relevant or reflective of a patient’s naturalistic sleep experience.
Pre-sleep arousal has been established as a causal factor of insomnia and sleep-state misperception. This has led to the development of treatment protocols aimed at reducing pre-sleep arousal. However, research has not explored the relationship among pre-sleep arousal, sleep-state misperception, and sleep locations. Exploring these relationships would allow for the further understanding of the impact of sleep location on the evaluation of sleep.

**The Current Study**

The primary objective of this study was to explore the difference between discrepancies of self-reported and objectively measured sleep at home versus in a laboratory. We hypothesized that the discrepancy between subjective and objective measures of sleep would differ depending on sleep location. A secondary objective of this study was to explore the relationship among pre-sleep arousal, sleep location, and sleep-state misperception. Specifically, we hypothesized the following:

1) We hypothesized that, on average, the discrepancy between subjective and objective measures of sleep would differ depending on sleep location. Although research has demonstrated that worse sleep is related to greater misperception of sleep, we cannot make a directional hypothesis of whether the discrepancy is greater at home or in the laboratory environment due to the FNE and RFNE. Although the FNE is more likely to be observed, we are unsure if the participants in our sample consistently experienced this effect.

2) We hypothesized that higher levels of pre-sleep arousal would be associated with greater misperception of sleep regardless of sleep location. We also hypothesized differences between home and laboratory sleep misperception would be predicted by differences in arousal. That is, home and laboratory discrepancy between subjective
and objectively measured sleep would be different due to different levels of pre-sleep arousal reported at home versus the laboratory.

A difference in sleep-state misperception depending on sleep location would indicate that the sleep environment impacts the assessment of the perception of sleep. Sleep location would thus be an important variable to consider when conducting both clinical and research work on sleep misperception and the role of pre-sleep arousal. To our knowledge, no other study has compared the perception of sleep at home to the perception of sleep in a clinical setting or explored the relationship among pre-sleep arousal, sleep location, and perception of sleep.

**Methods**

**Participants and Recruitment**

Data from this study came from the screening procedures of a treatment study examining non-pharmacological treatments for insomnia (Ong et al, 2014). Participants (N= 54) were recruited for the parent study from the community through advertisements, fliers, and clinician referrals. A majority of participants were female (74%) and White/Caucasian (67%) with a mean age of 42.9 years-old (SD= 12.2). Participants enrolled in the parent study met criteria for insomnia and endorsed at least one symptom of psychophysiological insomnia (e.g., worrying when trying to fall asleep, tension at night) as described by the International Classification of Sleep Disorders (ICSD-2) (American Academy of Sleep Medicine, 2005). At the time of data collection, the ICSD-2 was the most current edition of the ICSD. Participants did not report nor were they diagnosed with a sleep disorder co-morbid to insomnia (e.g., sleep apnea, restless leg syndrome or a psychiatric illness). Participants reported SOLs and WASOs of at least 30 minutes per night for at least three nights per week that had persisted for at least the past six months.

**Procedures**
Participants completed a phone screening to determine general eligibility. Interested and eligible participants were then invited for an in-person interview. During the in-person interview, participants were administered the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I) (First et al., 2002) and Duke Structured Interview for Sleep Disorders (Carney et al., 2008; Edinger et al. 2009). The most current edition of the SCID (i.e., Structured Clinical Interview for DSM-5) was not available during the time of data collection. Participants completed a packet of questionnaires that included demographic information and a set of self-report measures. Participants that passed the in-person interview (i.e., did not endorse a psychiatric diagnosis or sleep disorder co-morbid to insomnia) completed an overnight sleep evaluation in order to determine if a sleep disorder co-morbid with insomnia was present. During the overnight sleep evaluation, participants wore an actiwatch and completed a sleep diary. Participants were sent home with the actiwatch and a set of sleep diaries to complete. This study utilized sleep data collected from the overnight sleep evaluation and the sleep diaries.

**Measures**

**Objective sleep.** Participants wore an actiwatch (Philips Respironics, Actiwatch 2, Bend, OR) during their participation in the study. An actiwatch is a small, wrist-watch like device that records movement for a 24-hour period. Actigraphy has been used as a valid measure of objective sleep that is noninvasive with minimal participant burden (Ancoli-Isreal et al., 2003; Kushida et al., 2001). Sleep and wake intervals are calculated based on the absence or presence of movement. Respironics Actiware Software version 5.70 was used to analyze the data (Philips Respironics, Actiwatch 2, Bend, OR). Actiwatch 2 software settings were set at medium arousal and data were collected at one minute epochs (Ong et al., 2014). The medium arousal threshold setting was used to determine sleep and wake periods, where activity counts above the threshold
were considered a waking epoch and activity counts below the threshold were considered a sleep epoch.

**Subjective sleep.** Participants completed morning and evening sleep diaries for the duration of their participation in the parent study, including during the overnight sleep evaluation. Upon awakening, participants reported the time they got into bed with the intention of going to sleep, and the time they woke up and got out of bed to start their day. Participants rated the quality of their sleep and reported how long it took them to fall asleep, how many times they woke up during the night and how long the awakenings lasted, and the amount of time they slept. Before going to sleep, participants were asked to complete the Self-Assessment Manikin (SAM), and to record how many naps they had that day, the number of alcoholic drinks imbibed, any medications taken, and how fatigued and sleepy they felt.

**Pre-sleep arousal.** The Self-Assessment Manikin (SAM) is comprised of three sets of visual analogue scales. Each scale features five pictures placed across nine points that reflect a participant’s current emotional valence or feelings of dominance or arousal (Bradley & Lang, 1994). For the purpose of this study, only the arousal SAM was used (see Figure 1). The arousal SAM is anchored by one image that represents feeling calm and relaxed, and another that represents feeling excited and aroused. Bradley and Lang (1994) found that higher scores on the arousal SAM were highly correlated with adjectives that describe arousal (e.g., stimulated, excited, jittery, and wide awake), whereas lower scores were correlated with the adjectives relaxed and calm. Research has found that poor sleepers indicate greater arousal on the arousal SAM than good sleepers (Ong et al., 2011). Participants were asked to complete the SAM prior to going to sleep by rating “how they currently felt.”
**SOL and TST discrepancy.** Difference scores between self-reported and actigraphy measured SOL and TST were calculated for home and laboratory sleeps by subtracting actigraphy measured SOL and TST from self-reported SOL and TST. This method of calculating sleep discrepancy is consistent with previous research (Kay, Buysse, Germain, Hall, & Monk, 2015). Given that there were a number of extreme outliers in the data, the absolute value of each of these four discrepancy scores was used in the analyses. These absolute difference scores describe the magnitude of misperception occurring both at home and in the laboratory.

**Results**

**Data Analysis**

Data were analyzed using IBM SPSS Statistics version 20 software (IBM Corporation). Statistical significance was determined using a p-value of .01, and was adjusted for all analyses using the false discovery rate (FDR) correction as described by Benjamini and Hochberg (1995). A p-value of .01 was used in conjunction with the FDR correction because it provided a balance between type-1 and type-2 error (Benjamini & Hochberg, 1995).

**Comparing subjective and objective sleep.** Previous sleep-state misperception research compared self-reported SOL and TST to objectively measured SOL and TST to determine if on average there was a discrepancy between self-report and objectively measured sleep. Consistent with that methodology, and given the non-normal distribution of our data, we used multiple Wilcoxon Signed-Rank tests to determine if on average there was a difference between self-reported sleep and objectively measured sleep. The comparisons were:

- Home Self-Reported SOL compared to Home Actigraphy Measured SOL
- Laboratory Self-Reported SOL compared to Laboratory Actigraphy Measured SOL
- Home Self-Reported TST compared to Home Actigraphy Measured TST
Laboratory Self-Reported TST compared to Laboratory Actigraphy Measured TST

**Comparing home and laboratory sleep discrepancy.** We used two Wilcoxon Signed-Rank tests to determine if on average there was a difference in the discrepancy of SOL and TST at home versus the laboratory. SOL and TST discrepancy values were used for the following comparisons:

- Home SOL discrepancy compared to Laboratory SOL Discrepancy
- Home TST discrepancy compared to Laboratory TST Discrepancy

**Relationship between pre-sleep arousal, sleep location, and sleep discrepancy.** To explore the relationship between pre-sleep arousal and home and laboratory sleep discrepancy, we computed four Ordinary Least Square (OLS) regressions equations. For all four regression equations, the independent variable was arousal SAM scores. The dependent variables were the square root of Home and Laboratory SOL and TST discrepancy. The square roots of the dependent variables were used due to heteroscedasticity of the standard error terms. The regression equations were:

1) \( b_0 + b_1 \text{(arousal SAM)} = \text{Square root of Home SOL Discrepancy} \)
2) \( b_0 + b_1 \text{(arousal SAM)} = \text{Square root of Laboratory SOL Discrepancy} \)
3) \( b_0 + b_1 \text{(arousal SAM)} = \text{Square root of Home TST Discrepancy} \)
4) \( b_0 + b_1 \text{(arousal SAM)} = \text{Square root of Laboratory TST Discrepancy} \)

To determine if differences between home and laboratory sleep discrepancies were due to differences in pre-sleep arousal, we estimated two OLS regression equations. For both equations, the independent variable was the absolute difference between arousal SAM scores reported at home and arousal SAM scores reported in the laboratory. The dependent variable was either the square root of the absolute difference between home and laboratory SOL discrepancies or the
square root of the absolute difference between home and laboratory TST discrepancies. The dependent variables were transformed due to heteroscedasticity of the standard error terms. The regression equations for the aforementioned analyses are as follows:

1) \( b_0 + b_1 (\text{Absolute Difference between Home and Laboratory Arousal}) = \text{Square Root of Absolute Difference between Home and Laboratory SOL Discrepancy} \)

2) \( b_0 + b_1 (\text{Absolute Difference between Home and Laboratory Arousals}) = \text{Square Root Absolute Difference between Home and Laboratory TST Discrepancy} \)

**Missing Data**

Missing data were minimal. Three participants did not provide any objective sleep data because of actigraphy malfunction. Three participants did not self-report SOL or TST at home, four participants did not self-report SOL or TST in the laboratory, three participants did not report pre-sleep arousal at home, and one participant did not report pre-sleep arousal in the laboratory (Table 1). Instead of excluding these participants from the analyses (n= 8), which would have increased bias in the dataset, a multiple-imputation regression equation was used to impute missing values (Rubin, 1987). Laboratory and home ratings of arousal, mood, sleepiness, fatigue, and self-reported and actigraphy measured SOL, TST, number of awakenings, WASO, time in and out of bed, and sleep efficiency were used in the multiple-imputation regression equation. Twenty imputations were estimated. Descriptive statistics for the variables of interest (e.g., home and laboratory pre-sleep arousal, home and laboratory self-reported and actigraphy measured SOL and TST) from the original and pooled datasets are presented in Table 2. There were no statistically significant differences between means from the original and pooled datasets. The pooled statistics are reported for the subsequent analyses.

**Comparison of Self-Reported Sleep to Actigraphy Measured Sleep**
As reported in Table 3, there were differences between self-reported and actigraphy measured SOL and TST at home and in the laboratory (see Table 5 for FDR correction). Self-reported SOL at home was significantly greater than actigraphy measured SOL ($z = -4.07, p = .00$). The same relationship was also observed with laboratory self-reported and actigraphy measured SOL ($z = -5.22, p = .00$). These results indicated that participants reported SOLs that were higher than objective measures in both settings. There was also a marginally significant difference between self-reported and actigraphy measured TST in the laboratory such that self-reported TST was lower than actigraphy measures of TST ($z = -2.77, p = .01$), but this result was not found in the home environment. This indicates that in the laboratory, but not at home, participants reported TSTs that were lower than objective measures.

**Comparison of Home and Laboratory Sleep Discrepancies**

As shown in Table 4, there were differences between home and laboratory SOL and TST discrepancies (see Table 5 for FDR correction). However, the only statistically significant difference was between home and laboratory TST discrepancy ($z = -3.01, p = .00$). The discrepancy between self-reported and actigraphy measured TST at home was smaller than the discrepancy between self-reported and actigraphy measured TST in the laboratory. There was a marginally significant difference in home and laboratory SOL discrepancies again with SOL discrepancy at home being smaller than SOL discrepancy in the laboratory ($z = -2.46, p = .01$). These results suggest that participants’ self-reported SOLs and TSTs were more congruent with actigraphy measured SOLs and TSTs when they slept at home than when they slept in the laboratory.

**Relationship between Arousal, Sleep Discrepancy, and Sleep Location**
There was no statistically significant relationship between pre-sleep arousal and SOL and TST discrepancy at home or in the laboratory (Tables 6-8, see Table 9 for FDR correction). Differences between home and laboratory pre-sleep arousal also did not significantly predict differences in home and laboratory SOL and TST discrepancy.

**Discussion**

Patients diagnosed with insomnia often provide estimates of their sleep that are discrepant from objective sleep measures. Previous research has found that patients tend to over report how long it takes them to fall asleep and underreport their total sleep time. Although these phenomena have been observed in home and laboratory settings, it is unclear if the magnitude of the discrepancy between self-reported and objectively measured sleep differs depending on sleep location. The results of the current study demonstrated that sleep-state misperception is present in both home and laboratory settings, and that estimates of sleep-state misperception based on one night of laboratory data may over-estimate the severity of the problem.

The primary objective of the current study was to examine the discrepancy between self-reported and objective sleep at home and in a laboratory, and to determine if the discrepancies between self-reported and objective sleep differed between home and laboratory environments. The secondary objective was to explore the relationship between pre-sleep arousal and the discrepancy between subjective and objective sleep at home and in a laboratory, and to determine if differences between home and laboratory sleep discrepancies were due to differences in pre-sleep arousal between the home and laboratory. Our results indicated that participants reported SOLs that were greater than actigraphy measured SOLs at home and in the laboratory. Participants also self-reported TSTs that were less than actigraphy measured TSTs in the laboratory. We found that the discrepancy between self-reported and actigraphy measured
SOL was smaller at home than in the laboratory. The discrepancy between self-reported and actigraphy measured TST was also smaller at home than in the laboratory. We did not find that pre-sleep arousal predicted the difference between self-reported and actigraphy measured SOL and TST, nor did we find a significant relationship between differences in pre-sleep arousal predicting differences between home and laboratory SOL and TST sleep discrepancies.

**Methodological Considerations**

Our results replicated the findings of previous sleep-state misperception research such that we observed a difference between self-reported and objectively measured sleep at home and in the laboratory. This strongly suggests that sleep-state misperception is a phenomenon that is commonly observed in those diagnosed with insomnia. Furthermore, these results suggest that researchers and clinicians should carefully consider how they choose to measure sleep, such as through self-report, actigraphy, or using both methods. Given that insomnia is diagnosed primarily through self-report, it is important that researchers and clinicians consider whether the sleep measurement they use (e.g., actigraphy) reflects a participant or patient’s experience with disrupted sleep in their naturalistic sleep environment. Considering how and where sleep is measured could lead to insomnia treatments that are more relevant to patients diagnosed with insomnia and study outcomes that accurately reflect the etiology of insomnia.

Although we observed a discrepancy between self-reported and actigraphy measured SOL for home and laboratory sleep, we only observed a significant difference between self-reported and actigraphy measured TST for laboratory sleep observations. Self-reported and actigraphy measured TST for home sleep observations were not significantly discrepant. We may not have observed a difference between self-reported and actigraphy measured TST at home because our study used actigraphy and other studies used PSG as an objective measure of sleep
(Harvey & Tang, 2012). However, given that previous research has demonstrated that the misperception of TST is common (e.g., Bianchi, Williams, McKinney, & Ellenbogen (2013); Harvey & Tang (2012)) and that we observed misperception with SOL at home using actigraphy, we believe that the further examination of differences in self-reported and actigraphy measured TST in the laboratory and not at home is warranted. Researchers should explore differences between the naturalistic and laboratory sleep environments, such as differences in the time that participants went to bed and woke up and total sleep time in both locations, to determine if those factors play a role in why the discrepancy between self-reported and objectively measured TST was observed in the laboratory but not at home.

Not only is it important for researchers and clinicians to consider whether the sleep measurement they use reflects patients’ experiences with disrupted sleep, they should also carefully consider where the sleep assessment occurs. We determined that the magnitude of the discrepancy between self-reported and actigraphy measured sleep differed depending on sleep location. To our knowledge, no other study has demonstrated that the amount of sleep-state misperception experienced differs depending on sleep location. Basing estimates of sleep-state misperception on one night of laboratory data may lead to an overestimation of the sleep-state misperception problem. This suggests that researchers and clinicians must consider how the sleep environment, in addition to type of sleep measurement used, impacts their assessment of sleep. It also impacts the generalizability of results across and within studies that examine sleep in different locations. Researchers may need to consider including sleep location as covariate when analyzing data.

We observed a First Night Effect for sleep discrepancy in our sample (i.e., less discrepancy between subjective and objectively measured sleep at home than in the laboratory).
This phenomenon may be why we observed a difference between home and laboratory sleep discrepancies. It would be useful to compare sleep discrepancies between the first and second night in laboratory to determine if the discrepancy between subjective and objective sleep diminishes across successive nights. The present study is limited to a single night of sleep in the laboratory. To our knowledge, no other studies have compared the discrepancy between self-reported and objectively measured sleep on the first night in a laboratory to subsequent nights in the laboratory. Rather, studies have looked at either only the first night in the laboratory or an average across multiple nights (Bianchi, Williams, McKinney, & Ellenbogen, 2013; Kushida et al., 2001; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008; Manconi et al., 2010).

**Pre-Sleep Arousal**

We did not find a statistically significant relationship between pre-sleep arousal and sleep discrepancy nor between differences in pre-sleep arousal and differences in sleep discrepancy at home and in the laboratory. It is possible that we did not discover a relationship due to insufficient statistical power. It is also possible that we did not observe any statistically significant relationships due to our measure of pre-sleep arousal. We utilized the arousal self-assessment manikin to measure pre-sleep arousal, which is consistent with previous sleep research. However, pre-sleep arousal is multifaceted and it includes both cognitive and somatic complaints of arousal prior to falling asleep (Harvey, 2000). The self-assessment manikin may not have captured the full experience of pre-sleep arousal because it was not specific to the pre-sleep experience. Future studies may choose to include pre-sleep arousal measures that are specific to sleep instead of broadly measuring arousal, and should continue to examine the role of pre-sleep arousal on the discrepancy between self-reported and objectively measured sleep. Future studies may also choose to examine variables related to pre-sleep arousal, such as having
a co-morbid mood or anxiety disorder. Prior sleep-state misperception research also manipulated levels of pre-sleep arousal, such as telling participants that they would have to complete stressful tasks upon awakening and observing sleep state misperception during naps (Tang & Harvey, 2004). It is possible that inducing arousal allowed researchers to observe the relationship between pre-sleep arousal and sleep-state misperception due to levels of pre-sleep arousal being elevated beyond typical level and because shorter duration of sleep were observed (i.e., naps versus full night of sleep). Future studies may wish to explore pre-sleep arousal, as it naturally occurs, amongst those diagnosed with insomnia during normal sleep periods.

**Future Directions**

Notably, our study used actigraphy as an objective measure of sleep, while a majority of sleep-state misperception research utilizes PSG (e.g., Bianchi, Wang, & Kerman, 2012; Bianchi, Williams, McKinney, & Ellenbogen, 2013; Edinger & Fins, 1994). The results from our study indicate that sleep-state misperception is a phenomenon that is present when actigraphy measures of sleep are used. Our results suggest that even when using a different objective measure of sleep, a discrepancy between self-reported and objectively measured sleep still exists. This adds to the large body of research that has demonstrated that actigraphy is a valid method for measuring sleep that is comparable to PSG. Further investigation into discrepancies among self-report, actigraphy, and PSG measured sleep are warranted. It would be appropriate for future research to determine if the amount of discrepancy between self-reported and actigraphy measured sleep is the same or different than the amount of discrepancy between self-reported and PSG measured sleep.

Our study calculated the discrepancy between subjective and objective measures of sleep by taking the absolute difference between the two measures. We operationalized sleep-state
misperception in this manner due to previous research in this field and the presence of extreme outliers in our data. Although this is an acceptable method to operationalize sleep discrepancy, it did not allow us to determine if participants over or underestimated sleep onset latency and total sleep time. We were only able to determine whether participants estimated SOL and TST differently than what was measured by actigraphy. It may be necessary to further explore how to calculate sleep discrepancy that preserves both magnitude and direction and does not violate the assumptions of statistical analyses. Doing so would also allow us to further explore differences amongst participants that under or overestimated sleep, and possibly if those differences are related to pre-sleep arousal.

**Conclusion**

Although insomnia is primarily diagnosed through self-report, researchers and clinicians also utilize objective measures of sleep such as actigraphy to determine SOL and TST. Actigraphy is used in conjunction with self-report because of the phenomenon of sleep-state misperception, where it has been found that participants often over report SOL underreport TST as compared to objective measures of sleep. The current study demonstrated that the discrepancy between self-reported and objectively measured sleep exists in both home and laboratory settings, and that the discrepancy may be greater in the laboratory than at home. Therefore, researchers and clinicians should consider how the method and location of sleep assessment impacts sleep and the results of research and clinical assessments. Further research should continue to explore sleep-state misperception in various settings with different populations, and using multiple types of objective measures of sleep. Of particular interest is the exploration of the difference between home and laboratory TST discrepancy and the role of pre-sleep arousal in sleep-state misperception. Continuing to investigate sleep-state misperception not only adds to
our knowledge of the etiology of insomnia, but may also improve how insomnia is diagnosed and treated.
References


Frankel, B. L., Coursey, R. D., Buchbinder, R., & Snyder, F. (1976). Recorded and reported sleep in chronic primary insomnia. *Arch Gen Psychiatry, 33*(5), 615-623.


Lauderdale, D.S., Knutson, K.L., Yan, L.L., Liu, K., & Rathouz, P.J. (2008). Self-reported and measured sleep duration: how similar are they? *Epidemiology, 19*(6), 838-845. doi: 10.1097/EDE.0b013e318187a7b0


Riedel, B. W., Winfield, C. F., & Lichstein, K. L. (2001). First night effect and reverse first night effect in older adults with primary insomnia: does anxiety play a role? *Sleep Medicine, 2*(2), 125-133. doi: http://dx.doi.org/10.1016/S1389-9457(00)00054-X


### Table 1
Percentage and count of missing data

<table>
<thead>
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<th>Percent missing</th>
<th>Count</th>
</tr>
</thead>
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<td>3</td>
</tr>
<tr>
<td>Home ACT SOL</td>
<td>5.56</td>
<td>3</td>
</tr>
<tr>
<td>Home SR TST</td>
<td>5.56</td>
<td>3</td>
</tr>
<tr>
<td>Home ACT TST</td>
<td>5.56</td>
<td>3</td>
</tr>
<tr>
<td>Laboratory SR SOL</td>
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<td>4</td>
</tr>
<tr>
<td>Laboratory ACT SOL</td>
<td>7.41</td>
<td>4</td>
</tr>
<tr>
<td>Laboratory SR TST</td>
<td>7.41</td>
<td>4</td>
</tr>
<tr>
<td>Laboratory ACT TST</td>
<td>7.41</td>
<td>4</td>
</tr>
<tr>
<td>Home Arousal</td>
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<tr>
<td>Laboratory Arousal</td>
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<td>1</td>
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</table>

*Note. SR= Self-report, ACT= Actigraphy, SOL= Sleep onset latency, TST= Total sleep time.*
<table>
<thead>
<tr>
<th>Variable</th>
<th>Original Data</th>
<th></th>
<th>Pooled Data</th>
<th></th>
</tr>
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<td></td>
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<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td><strong>Laboratory</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-Sleep Arousal</td>
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<td>6.04</td>
<td>1.53</td>
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<td>53.55</td>
<td>48.70</td>
<td>53.55</td>
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<td>ACT SOL</td>
<td>13.80</td>
<td>18.95</td>
<td>15.71</td>
<td>48.05</td>
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<td>SR TST</td>
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<td>108.33</td>
<td>358.82</td>
<td>220.99</td>
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<td>ACT TST</td>
<td>406.86</td>
<td>71.05</td>
<td>408.78</td>
<td>233.13</td>
</tr>
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<td><strong>Home</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-Sleep Arousal</td>
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<td>5.79</td>
<td>2.62</td>
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<td>24.43</td>
<td>30.04</td>
<td>23.03</td>
</tr>
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<td>ACT SOL</td>
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<td>16.73</td>
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<td>99.91</td>
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<td>ACT TST</td>
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<td>400.66</td>
<td>80.39</td>
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<td></td>
<td></td>
<td></td>
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<td>15.73</td>
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<td>Home TST Discrepancy</td>
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<td>62.93</td>
<td>69.37</td>
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<td>Laboratory SOL Discrepancy</td>
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<td>51.65</td>
<td>44.04</td>
<td>70.04</td>
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<td>Laboratory TST Discrepancy</td>
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<td>130.17</td>
<td>208.41</td>
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<tr>
<td>Absolute Difference between Home and Laboratory SOL Discrepancy</td>
<td>33.31</td>
<td>48.43</td>
<td>39.94</td>
<td>65.42</td>
</tr>
<tr>
<td>Absolute Difference between Home and Laboratory TST Discrepancy</td>
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<td>84.56</td>
<td>130.34</td>
<td>206.97</td>
</tr>
<tr>
<td>Absolute Difference between Home and Laboratory Arousal</td>
<td>1.08</td>
<td>1.10</td>
<td>1.41</td>
<td>2.28</td>
</tr>
</tbody>
</table>

*Note.* SR = Self-report, ACT = Actigraphy, SOL = Sleep onset latency, TST = Total sleep time, Home SOL Discrepancy = Absolute value of Home SR SOL–Home ACT SOL, Home TST Discrepancy = Absolute value of Home SR TST–Home ACT TST, Laboratory SOL Discrepancy = Absolute value of Laboratory SR SOL–Laboratory ACT SOL, Laboratory TST Discrepancy = Absolute value of Laboratory SR TST–Laboratory ACT TST, Absolute difference between Home and Laboratory Arousal = Absolute value of Home Pre-Sleep Arousal–Laboratory Pre-Sleep Arousal.
Table 3
Comparison of actigraphy and self-reported indices of sleep

<table>
<thead>
<tr>
<th>Variable</th>
<th>Self-Report</th>
<th>Actigraphy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>M</td>
</tr>
<tr>
<td>Home SOL</td>
<td>30.04</td>
<td>16.73</td>
</tr>
<tr>
<td>Home TST</td>
<td>392.52</td>
<td>400.66</td>
</tr>
<tr>
<td>Laboratory SOL</td>
<td>48.70</td>
<td>15.71</td>
</tr>
<tr>
<td>Laboratory TST</td>
<td>358.82</td>
<td>408.78</td>
</tr>
</tbody>
</table>

*Note.* SOL = Sleep onset latency, TST = Total sleep time.
Table 4
Comparison of home and laboratory sleep discrepancy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Home</th>
<th>Laboratory</th>
<th>z</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOL Discrepancy</td>
<td>19.42</td>
<td>44.04</td>
<td>-2.46</td>
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<td>TST Discrepancy</td>
<td>62.93</td>
<td>130.17</td>
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</table>

Note. Home SOL Discrepancy = Absolute value of Home SR SOL–Home ACT SOL, Home TST Discrepancy = Absolute value of Home SR TST–Home ACT TST, Laboratory SOL Discrepancy = Absolute value of Laboratory SR SOL–Laboratory ACT SOL, Laboratory TST Discrepancy = Absolute value of Laboratory SR TST–Laboratory ACT TST.
Table 5
P-value correction for multiple Wilcoxon Signed-Rank tests using the false discovery rate (FDR) correction as described by Benjamini and Hochberg (1995).

<table>
<thead>
<tr>
<th>Rank</th>
<th>P-value</th>
<th>FDR P-value of Statistical Significance</th>
<th>P-value Less Than FDR P-value?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.00</td>
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<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>.00</td>
<td>.003</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>.00</td>
<td>.005</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
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<td>.008</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>.66</td>
<td>.010</td>
<td>No</td>
</tr>
</tbody>
</table>

*Note.* Rank = Order of p-value of each statistical test from lowest to highest, P-value = P-value of each statistical test, FDR P-value of Statistical Significance = Corrected p-value from which the significance of each statistical test is determined.
Table 6
Summary of Simple Regression Analyses of Pre-Sleep Arousal Predicting Square Root of Home and Laboratory SOL Discrepancy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Home SOL Discrepancy</th>
<th>Laboratory SOL Discrepancy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE B</td>
</tr>
<tr>
<td>Intercept</td>
<td>3.92</td>
<td>.67</td>
</tr>
<tr>
<td>Pre-Sleep Arousal</td>
<td>.03</td>
<td>.109</td>
</tr>
</tbody>
</table>

Note. Pre-Sleep Arousal=Pre-sleep arousal at home and in the laboratory, Home SOL Discrepancy = Absolute value of Home SR SOL–Home ACT SOL, Home TST Discrepancy = Absolute value of Home SR TST–Home ACT TST, Laboratory SOL Discrepancy = Absolute value of Laboratory SR SOL–Laboratory ACT SOL, Laboratory TST Discrepancy = Absolute value of Laboratory SR TST–Laboratory ACT TST.
### Table 7
Summary of Simple Regression Analyses of Pre-Sleep Arousal Predicting Square Root of the Home Laboratory TST Discrepancy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Home TST Discrepancy</th>
<th>Laboratory TST Discrepancy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$B$</td>
<td>$SE$ $B$</td>
</tr>
<tr>
<td>Intercept</td>
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<td>2.02</td>
</tr>
<tr>
<td>Pre-Sleep Arousal</td>
<td>-.05</td>
<td>.34</td>
</tr>
</tbody>
</table>

*Note.* Pre-Sleep Arousal = Pre-sleep arousal at home and in the laboratory, Home SOL Discrepancy = Absolute value of Home SR SOL–Home ACT SOL, Home TST Discrepancy = Absolute value of Home SR TST–Home ACT TST, Laboratory SOL Discrepancy = Absolute value of Laboratory SR SOL–Laboratory ACT SOL, Laboratory TST Discrepancy = Absolute value of Laboratory SR TST–Laboratory ACT TST.
Table 8
Summary of Simple Regression Analyses of Absolute Differences between Arousal at Home and Laboratory Predicting Square Root Differences in SOL and TST Discrepancy at Home and Laboratory

<table>
<thead>
<tr>
<th>Variable</th>
<th>Difference between Misperception of SOL at Home and Laboratory</th>
<th>Difference between Misperception of TST at Home and Laboratory</th>
</tr>
</thead>
<tbody>
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<td>$SE_B$</td>
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<tr>
<td>Intercept</td>
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<td>.63</td>
</tr>
<tr>
<td>Absolute Difference between Arousal at Home</td>
<td>.32</td>
<td>.34</td>
</tr>
<tr>
<td>and Laboratory</td>
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</tr>
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</table>
Table 9
P-value correction for multiple simple regression analyses using the false discovery rate correction as described by Benjamini and Hochberg (1995).

<table>
<thead>
<tr>
<th>Rank</th>
<th>P-value</th>
<th>FDR P-value</th>
<th>P-value Less Than FDR p-value?</th>
</tr>
</thead>
<tbody>
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<td>.26</td>
<td>.002</td>
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</tr>
<tr>
<td>2</td>
<td>.38</td>
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<td>No</td>
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<td>4</td>
<td>.83</td>
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</tr>
<tr>
<td>6</td>
<td>.96</td>
<td>.010</td>
<td>No</td>
</tr>
</tbody>
</table>

*Note.* Rank= Order of p-value of each statistical test from lowest to highest, P-value= P-value of each statistical test, FDR P-value of Statistical Significance= Corrected p-value from which the significance of each statistical test is determined.
Figure 1. Arousal Self-Assessment Manikin (Arousal SAM).