SLEEP MISPERCEPTION AMONG COMMUNITY DWELLING OLDER ADULTS

By

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To my family, especially my loving wife, thank you for your prayers and support

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Late-life insomnia is a prevalent and serious health problem. Sleep misperception (SM), overestimating time spent awake while trying to sleep, predicts insomnia onset and maintenance. Like insomnia, sleep misperception increases with age. However, research on the longitudinal patterns and correlates of sleep misperception among older adults is needed. We posit that sleep misperception results from perceptual areas of the brain remaining awake during global sleep, reflecting a form of localized sleep deprivation. Sleep misperception patterns may relate to a homeostatic sleep dysregulation, such as sleeping longer than usual. In this study four specific aims that will be investigated in a sample of community dwelling older adults. Aim 1 will determine the amount of within- to between-person variability in SM that occurs at sleep onset latency (SOL_{sm}) and that occurs during wake time after sleep onset ($WASO_{sm}$). Aim 2 will determine the amount of within- to between-person variability in SOL_{sm} and $WASO_{sm}$ by sleep complaint status. Aim 3 will be to investigate the relationship between night-to-night fluctuations in SOL_{sm} and $WASO_{sm}$. Finally, aim 4 will determine the intraindividual relationship between SOL_{sm} and $WASO_{sm}$ to objective TST (TST_o). A sample of 103 community dwelling older adults (M_{age} = 72.81, SD = 7.12) wore an Actiwatch-L® (24hrs/day/2weeks) and concurrently completed sleep diaries. Daily values for actigraphically-measured sleep onset latency (SOL) and

wake time after sleep onset (WASO) were subtracted from respective diary reports to calculate daily SM for SOL and WASO. Intraindividual variability analyses (IIV) and multilevel modeling (MLM) revealed-1) within-persons, nights that $WASO_{sm}$ was greater than usual, SOL_{sm} was less than usual, $\beta = -0.1$, $t(87.59) = -2.01$, $p < .05$. Interestingly, between-persons, those with greater SOL_{sm} had greater $WASO_{sm}$, $\beta = 0.21$, $t(98.09) = 3.98$, $p < .01$; 3) within-persons. There was a nonsignificant trend using SOL_{sm} to predict $WASO_{sm}$. ($\beta = -0.10$, $t(90.90) = -1.80$, $p = .08$); 4) nights with increased SOL_{sm} were related to longer TST_o, $\beta = 0.32$, $t(67.74) = 5.02$, $p < .01$ while nights with increased $WASO_{sm}$ were related to shorter TST_o, $\beta = -0.15$, $t(49.77) = -2.47$, $p < .05$. Between-persons, only SOL_{sm} was related to longer TST_o. Sleep misperception is ubiquitous but highly variable among older adults. Individuals with higher average SOL_{sm} had higher average $WASO_{sm}$; however, days individuals had higher $WASO_{sm}$ predicted lower SOL_{sm}. A homeostatic sleep rebound model of sleep misperception may explain the finding that increased TST_o occurred on days that SOL_{sm} was greater than usual. Understanding the variable, but predictive patterns of SM among older adults may lead new preventative and therapeutic insomnia interventions.

CHAPTER 1
INTRODUCTION

Various constructs and research variables of sleep misperception have been developed. Usually, sleep misperception is conceptualized as an individual's tendency to report objectively defined sleep as wakefulness (Adam, Tomeny, & Oswald) and to over-estimating the time spent falling asleep and underestimating total sleep time. Sleep misperception is common to insomnia and most studies on sleep misperception are based on a single night observation or an average of several nights of sleep in this patient population. These studies have yielded valuable information about the relationship between sleep misperception and insomnia; however, methodologies used in these studies are commonly based on scientifically untested assumptions. Herein, these assumptions are reviewed. This literature review also covers the scientific evidence supporting various perspectives on sleep misperception. Ultimately, this study seeks to expand a specific theory that argues that sleep misperception is a transitional state of consciousness on a continuum between wake and sleep that represents a symptom of a clinically significant sleep problem. Four secondary analyses were conducted on data collected from a sample of older adults with the aim of better understand sleep misperception. The first two analyses challenge the common assumptions that sleep misperception is a consistent pattern unique to individuals with sleep complaint. Scientifically, this study seeks to provide a clearer picture of sleep misperception with greater temporal resolution than can be obtained using traditional single night sleep recording and multiple night averaging techniques alone. The third analysis explores the relationship between sleep misperceptions that occur during sleep onset latency or SOL_{sm}, and sleep misperceptions that occur during wake time in the night or $WASO_{sm}$. The final analysis explores the possibility that sleep misperception is related to a true sleep problem predicted by longer sleep time. Will this study will not be able to directly test the theoretical involvement of

neurophysiology to sleep misperception, neurophysiologic data from previous studies were paramount in driving this investigation and the interpretation of this study.

CHAPTER 2
REVIEW OF THE LITERATURE

Broadly, sleep misperception is an individual's tendency to perceive objectively defined sleep as wakefulness. Likewise, individuals that misperceive sleep overestimate the time spent falling asleep, overestimate time spent awake during the night, and/or underestimate total sleep time. Sleep misperception is determined by comparing an individual's retrospective self-report of the sleep experience with an objective prospective measure (i.e., electroencephalograph or actigraphy) of the sleep state. While sleep misperception has been well studied, this phenomenon remains poorly understood. Research has shown significant relationships between sleep misperception and mood, arousal, and general health (Bonnet & Arand, 1997b; Carskadon et al., 1976; Chambers & Kim, 1993). In addition, sleep experts have long observed that sleep misperception is strongly associated with insomnia (Frankel, Coursey, Buchbinder, & Snyder, 1976). Many posit sleep misperception acts as a primary pathway to insomnia onset and maintenance (Borkovec, 1982; Borkovec, Lane, & VanOot, 1981; Harvey, 2002a; Lundh & Broman, 2000; Perlis, Giles, Bootzin et al., 1997). To explain the role of sleep misperception in insomnia, cognitive theorists argue that sleep misperception leads to daytime protective behaviors that exacerbate arousal, thus leading to, and perpetuating, insomnia (Harvey, Tang, & Browning, 2005). The consequences of sleep misperception in relationship to insomnia may be profound. Insomnia is among the most prevalent and costly health problems with social cost estimated between $92.5 and $107.5 billion each year (Stoller, 1994; Walsh & Engelhardt, 1999). In addition, insomnia is related to reduced cognitive functioning, reduced performance, greater utilization of health services and more missed work days (Ohayon, Caulet, Priest, & Guilleminault, 1997). Moreover, the onset of insomnia predisposes individuals to many other mental and physical health problems including, depression, anxiety, pain and substance abuse

disorders (Ford & Kamerow, 1989). Considering sleep misperception may precipitate and maintain insomnia, research aimed at gaining knowledge about sleep misperception may be a fruitful avenue leading to new preventative and therapeutic interventions for insomnia.

Sleep Misperception in Older Adults

Adults over the age of 65, often referred to as older adults, make up 13% of the total US population and have the highest rates of sleep disturbance of any age group. Older adults have more awakenings during the night, decreased sleep efficiency, more variable nocturnal total sleep time, more alpha (light), less delta (deep) sleep, more global- and micro-arousal during sleep, and increased daytime napping (Nau, McCrae, Cook, & Lichstein, 2005). Not surprisingly, older adults have higher rates of insomnia complaints (Kiley, 1999; Ohayon & Caulet, 1996) with greater associated severity and chronicity (McCrae et al., 2003). Epidemiological studies estimate that the prevalence of insomnia is roughly 65% among older adults (Newman, Enright, Manolio, Haponik, & Wahl, 1997), thus twice as prevalent as occurs in younger populations (reviewed in Nau et al., 2005; Ohayon, 2002). Interestingly, with increased age subjective accounts become less reflective of objective measures of sleep. As is the case with insomnia, sleep misperception increases with age. Additional research on the relationship between sleep misperception and the increase in sleep problems among older adults has been called for in the literature (Bonnet & Moore, 1982). Moreover, a better understanding of the relationship between objective and subjective measures in older adults is needed (Hoch et al., 1987). Sleep misperception research may yield valuable information beyond what can be gleaned from a direct relationship of either subjective or objective measures alone. The current study seeks to fill this need by investigating the patterns and correlates of sleep misperception among older adults.

Theoretical Perspectives of Sleep Misperception

Sleep Misperception as Perceptual Distortion or Deficit

The phenomenon of sleep misperception remains poorly understood (Tang & Harvey, 2005), and a ubiquitously accepted definition or unifying theory of sleep misperception is lacking. Sleep misperception has been investigated as 1. a tendency to exaggerate sleep difficulties, 2. a perceptual deficit or pathological distortion of reality related only to sleep, 3. an insomnia specific symptom, and 4. a categorical insomnia disorder subtype. Although these perspectives have never been cohesively integrated, they may be compatible. Below, several methodological and theoretical approaches of sleep misperception research are considered. The goal of this review is to contextualize the operational definition of sleep misperception tested in this study.

Sleep Misperception as Exaggeration

People with insomnia are often stereotyped as having unbelievable or seemingly exaggerated claims about sleep length or sleep difficulties. For example, patients with insomnia often report that they only sleep a few hours each night while spending 8-11 hours trying to sleep in bed each night. Moreover, for an insomnia patient to claim less than 4 hours of sleep per night while electroencephalography (EEG) records normal sleep is not uncommon. Objective measures of sleep rarely support such claims, and as a result, many physicians and sleep professionals have concluded that sleep misperception represents the patient's propensity to dramatize or exaggerate sleep difficulties (Vanable, Aikens, Tadimeti, Caruana-Montaldo, & Mendelson, 2000). However, there is no empirical evidence that sleep misperceptions represents these patients' tendency to lie or exaggerate. In fact, there is compelling evidence to the contrary. Perlis (2001) showed, for example, that the discrepancies between objective and subjective measures are not uniform across sleep parameters among insomnia patient. He argued that if

sleep misperception were a negative bias following poor sleep the patient would complain about SOL, WASO, total sleep time (TST), sleep quality, sleep efficiency, etc. However, this is not what was found. In fact, patients with sleep misperception typically misperceived only one or two aspects of their sleep.

Sleep Misperception as Psychopathology

Rather than viewing the tendency to over-estimate SOL and WASO as a form of '*mis*reporting', most sleep experts consider this tendency a type of '*mis*perception'. Sleep misperception is most commonly described as a perceptual deficit/distortion, such as an inability to estimate time or lack of ability to perceive the passing of time as others do. Though tempting to believe, sleep misperception as a global deficit in time perception has no scientific support (Rioux, Tremblay, & Bastien, 2006). Moreover, though ubiquitously maintained (e.g., Borkovec, 1982; Borkovec, Grayson, O'Brien, & Weerts, 1979; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Perlis, Merica, Smith, & Giles, 2001; Tang & Harvey, 2004; Vanable et al., 2000), there is little evidence that sleep misperception represents a perceptual distortion of time specific to the sleep experience. One study attempting to identify such a deficit was unable to induce a state specific distortion of the passing of time in insomnia patients (Tang & Harvey, 2005). Regrettably, this null finding has not dampened the fervor that such a perceptual deficit exists.

As is indicated by its moniker, 'sleep misperception' is regarded as a psychological distortion of reality, that is, that individuals who experience sleep misperception perceive the same EEG defined state differently than those who do not misperceive. An early study on sleep misperception purported to have supported this claim showing that the same EEG defined global state of sleep was perceived to be lighter by sleep complainers as compared to controls (Mendelson, Garnett, Gillin, & Weingartner, 1984). Unfortunately, given that global psychophysiological measures cannot tell the difference between joy and anger, it seems odd that

scientists should claim that the 'mis' is associated with the patient 'perception' and not in the researcher's 'measure'. Indeed, the American Sleep Disorders Association (now called the American Academy of Sleep Medicine) pointed out that standard polysomnographic (PSG) measures used in research and clinical assessment are inadequate at determining altered physiological states in insomnia (Reite, Buysse, Reynolds, & Mendelson, 1995). It is presumptive, therefore, for scientists to go as far as to say that people who experience sleep misperception "suffer from difficulty in recognizing their own state of consciousness" (Mendelson, James, Garnett, Sack, & Rosenthal, 1986, p. 268). A recent term that has emerged in the sleep literature that highlights this overconfidence in our assessment tools is "sleep quality misperception"(e.g., Neu, Mairesse, Hoffmann, Dris, Lambrecht, et al., 2007). This term suggests that there are some individuals who are incapable of determining how refreshing their sleep is and that a PSG machine knows better than the patient. Researchers have gone as far as to "teach" individuals with sleep misperception how to perceive their sleep experience (Downey & Bonnet, 1992). Although this technique may inform patients on how the clinician desires him or her to report the sleep experience, the sleep experience of these patients may not have actually changed.

There are at least four major reasons why sleep misperception should not be considered a psychopathological distortion of reality. First, sleep misperception occurs spontaneously (Sewitch, 1984) in 40-60 % of healthy sleepers (reviewed in Attarian, 2007). On this point, the claim has been made that the literature shows that good sleepers tend to correctly estimate or slightly underestimate SOL (Smith & Trinder, 2000). However, this is a qualitative assessment and is not overly descriptive. At best, the literature suggests that sleep misperception is more frequent and/or extreme among insomnia patients than controls but not that it is absent from

controls (Perlis, Smith, Andrews, Orff, & Giles, 2001). Second, sleep misperception has been induced scientifically in normal sleepers by increasing arousal, through caffeine administration before sleep onset (Bonnet & Arand, 1997b), an advanced phase shift of the sleep schedule, and the manipulation of respiratory control to induce arousal (Smith & Trinder, 2000). Third, individuals with sleep misperception are not clinically psychopathological. As a group, they do not report elevated scores on the Minnesota Personality Inventory (MMPI-II) (Mendelson et al., 1986) nor do they have significantly greater MMPI scores than normal sleepers (Bonnet & Arand, 1995). Forth, and most importantly, there is no contradictory evidence that sleep misperception reflects an accurate perception of an altered state and a true sleep disturbance commonly experienced by insomnia patients (Hauri & Olmstead, 1983).

Sleep Misperceptions as a Distinct Sleep Disorder

The International Classification of Sleep Disorders-II (ICSD-II) includes a diagnosis of 'sleep state misperception' as a distinct subgroup of insomnia. The ICSD-II outlines that individuals who meet criteria for this sleep disorder (1) complain of insomnia but lack significant EEG defined deficits that correspond to the complaints and (2) do not have mental or physical ailment that might explain the misperception. The sub-categorization of sleep misperception is unfortunate, because most research on sleep misperception has focused on the diagnostic category rather than on the actual phenomenon of sleep misperception. This problem has been associated with sleep misperception almost from its discovery. Attempts to subcategorize individuals with sleep misperception within insomnia have been made for decades and various diagnosis categories have been advocated, such as pseudoinsomnia, subjective insomnia, experimental insomnia, and sleep hypochrondriasis. Pseudoinsomnia put forth by Borkovec et al. (1979) used sleep misperception as the sole feature of the disorder. While subjective insomnia was based more on the assumption that everyone has the same sleep need and that if sleep length

was about average there is no 'real' problem with the patient's sleep. Whereas in 1988 it was argued by Trinder that, "Although over-estimation of sleep disturbance must occur in subjective insomnia, it does not appear to be uniquely associated with the clinically identified condition. Therefore, its occurrence is irrelevant to the identification, definition, or etiology of the disorder" (Trinder, 1988, p. 91). The ICSD-II seems to have compromised between subjective insomnia and pseudoinsomnia. The presence of misperception has become central to this diagnostic category in addition to the requirement that no global deficits be present (ICSD-II). By adding the construct of a "misperception" to subjective insomnia, those advocating this diagnostic category have attempted to remove the complication of individual sleep need inherent with the former construct of subjective insomnia. However, the validity of sleep state misperception as a categorical sleep disorder has been questioned on theoretical and scientific ground (Dorsey & Bootzin, 1997; Edinger & Fins, 1995; P. J. Hauri & Wisbey, 1992; McCall & Edinger, 1992; Reynolds, Kupfer, Buysse, Coble, & Yeager, 1991; Sugerman, Stern, & Walsh, 1985). Attempts to validate sleep misperception as a categorical subtype of insomnia are methodologically flawed (Salin-Pascual, Roehrs, Merlotti, Zorick, & Roth, 1992) either limited to a single night of recording (e.g., Edinger et al., 1996; Krystal, Edinger, Wohlgemuth, & Marsh, 2002; Salin-Pascual et al., 1992) or an average of several nights (e.g., Means, Edinger, Glenn, & Fins, 2003). The major problem with these approaches is discussed in detail in a section below (see Intraindividual Variability Analysis (IIV)).

Sleep Misperception: The First and Maintaining Symptom of Insomnia

There is scientific evidence to support the idea that sleep misperception represents a mild or prodromic phase of insomnia that may later develop into insomnia (Salin-Pascual et al., 1992). Individuals with sleep misperception lack objective indices of a sleep problem but have similar symptoms as insomnia patients, though less severe (Bonnet & Arand, 1997b; Salin-Pascual et al.,

1992). The major problem with the idea that sleep misperception is a prodromic phase of insomnia is that sleep misperception is so common among insomnia patients. How can sleep misperception be a prodromic phase of insomnia and a characteristic of even the most severe insomnia patients? One explanation is that individuals with sleep misperception may experience sleep onset similar to insomnia patients (Hauri & Olmstead, 1983) but have a more isolated/localized sleep problem that predisposes them to a more severe insomnia. Indeed, sleep misperception seems to be on a continuum between normal sleep and chronic insomnia. Sleep misperception patients tend to having intermediate levels of vigilance (as measured by the multiple sleep latency test (MSLT)), short term memory problems, subjectively low vigor, and subjectively low estimates of sleep quality between normal and physiological insomnia patients (Bonnet & Arand, 1995).

Sleep Misperception as a Graded Characteristic of Insomnia

There is substantial evidence that sleep misperception occurs in varying degrees of intensity and frequency in the vast majority of the general population but with increasing frequency or severity among insomnia patients (Edinger & Fins, 1995; Edinger et al., 2000; Fichten, Creti, Amsel, Bailes, & Libman, 2005; Krystal et al., 2002; Means et al., 2003; Schneider-Helmert & Kumar, 1995)**.** Moreover, it has been argued that sleep misperception is a defining feature of insomnia regardless of degree (Bonnet & Arand, 1997a; P. Hauri & Olmstead, 1983; Reynolds et al., 1991; Salin-Pascual et al., 1992; Tang & Harvey, 2004; Trinder, 1988). Although overstretching the current scientific evidence, many current sleep researchers and clinicians continue to uphold that sleep misperception occurs constantly in a large portion of insomnia patients and is a valid characterization of insomnia (e.g., Edinger & Krystal, 2003).

Intraindividual Variability Analysis (IIV)

The literature is conflicted as to whether sleep misperceptions follow a daily or occasional pattern and whether sleep misperception is a defining feature of insomnia, a sub-classification of insomnia or distinct sleep disorder. Many of the discrepancies in findings and opinions related to sleep misperception are the consequence of inherent limitations to methodology of the vast majority of sleep misperception studies. Many studies are limited to a single night of laboratory PSG recording, and other studies obtain multiple nights of observations but then average over days and weeks. Both approaches are problematic because, like objective measures of sleep, sleep estimates are not stable over time (Edinger et al., 1997b; Edinger, Marsh, McCall, Erwin, & Lininger, 1991; Wohlgemuth, Edinger, Fins, & Sullivan, 1999); nor are objective and subjective measures systematically related over time (Edinger et al., 1991; Harvey, 2000; McCrae et al., 2005). Therefore, if sleep misperception were to occur occasionally, single random night recordings are incapable of capturing sleep misperception in all insomnia participants, although all participants may experience sleep misperception several times per week (McCall & Edinger, 1992).

Researchers and clinicians attempting to glean information about sleep misperception from single night studied must use caution in interpreting the results. One larger clinic based study attempted to determine what percent of patients have been diagnosed with sleep misperception (Coleman et al., 1982). It was determined that 9.2% of 1214 diagnosed insomnia patients met criteria for sleep state misperception. The diagnostic criterion for sleep state misperception is determined by a single night of PSG recording. Thus the number of individuals diagnosed with the sleep state misperception subtype of insomnia may actually be the odds of someone with insomnia to experience sleep misperception during the night that the sleep recording took place (i.e., 1:4). Other studies suggested that between 25% and 50% of insomnia cases can be

subsumed as sleep state misperception (Dorsey & Bootzin, 1997; Mendelson, 1995c; Salin-Pascual et al., 1992; Sugerman et al., 1985). Again because these studies were based on a single night of sleep, these results could represent the odds of insomnia patients to misperceive on any given night. Studies that average sleep perceptions over several days found that between and 34.4% and 38% of the sample could be subsumed as sleep state misperception (Hauri & Wisbey, 1992; Means et al., 2003). Because these studies averaged over 3 and 6 days, respectively, they can be interpreted as the odds (i.e., 1:3) of someone with insomnia experiencing misperception more often than not or at such an extreme degree on occasional nights to not be washed out by days sleep misperception did not occur. In sum, this disorder is based on clinical judgment, unsuccessful post-hoc experimental validation attempts, and a dearth of scientific evidence. The creation of another categorical disorder centered on sleep misperception effectively represents the construct of yet another invalid ICSD-II diagnostic category driven by clinical consensus rather than scientific evidence (Edinger et al., 1996). For this reason, the current study will not limit its investigation of sleep misperception to individuals that meet criteria for the sleep disorder called sleep state misperception. Another study separated individuals into objective and subjective insomnia patients based on whether subjective complaints were validated by a single night of PSG (Krystal et al., 2002). Only individuals who lacked objective measures of sleep problems experienced high beta activity during sleep. This study may be interpreted as having shown that sleep misperception is less likely to occur on nights that objective sleep is poor or that measurement of sleep misperception on PSG measured poor sleep.

Clinically, insomnia seems to be periodic, with several days of intense insomnia followed by nights of normal sleep (Drummond, Smith, Orff, Chengazi, & Perlis, 2004; Karacan, 1972). Scientifically, insomnia is not necessarily a constant sleep pattern from night-to-night. For this

reason, averaging several days to describe the sleep of insomnia patients may be inappropriate. Likewise, it should not be assumed that averaging sleep misperceptions across days is a valid indication of the patterns of individuals sleep perceptions. For example, combining overestimates of sleep difficulties with underestimates is bound to wash out occasional patterns of misperception. While averaging techniques undoubtedly have provided greater insight to sleep misperception, the more occasional or subtle patterns of sleep misperception may be additionally important. Averaging techniques have been particularly useful in identifying individuals who consistently misperceive, that is, those individuals who fall on the extremes of sleep misperception behavior. In fact, this is exactly what Salin-Pascual et al., (1992) found in a long-term study on sleep state misperception, that is, the researchers were able to find those extreme sleep misperceives. They found that the sleep of misperceives followed a stable healthy sleep pattern, while they consistently complained of poor sleep. Single night and averaging studies load the deck, as it were, to find over and underestimating sleep as a "sleeper type" and ignores the large amount of intraindividual variability that may be occurring across days. Insomnia patients particularly have been shown to have highly variable sleep patterns (Coates et al., 1981; Edinger et al., 1997a, 1997b; Edinger et al., 1991; Frankel et al., 1976; Hauri & Wisbey, 1992; Mullaney, Kripke, & Messin, 1980; Vallieres, Ivers, Bastien, Beaulieu-Bonneau, & Morin, 2005; Vallieres & Morin, 2003) and is posited to be a central characteristic of insomnia (e.g., Espie, 2002; Frankel et al., 1976).

Given that sleep misperception is common among insomnia patients and that insomnia patients are variable in both objective and subjective sleep parameters, the assumption that misperceptions are stable among insomnia patients is preemptive and may not reflect the true patterns of sleep misperceptions. Assumptions that sleep misperception reflects "more negative

thinking" and "exaggeration" may not reflect what is actually causing sleep misperception (e.g. Vallieres et al., 2005, p.452). Sleep researchers have acknowledged many problems with single night recording due to the high variability in the sleep of insomnia patients. The problems with averaging several nights to eliminate that variability are less well recognized but gaining acceptance (e.g., Wohlgemuth et al., 1999). The relationship between variability in sleep misperception and variability in sleep among insomnia patients has not been tested longitudinally using multilevel modeling or intraindividual analyses. Such studies are needed before conclusion of "sleep estimating sleeper types" can be categorized based on single night and averaging techniques.

Most people believe that averaging eliminates error. However, intraindividual variability represents true fluctuations in peoples sleep over time and is washed out by averaging. Like other behaviors that are highly variable over time (Nesselroade, 2004; Nesselroade, Mitteness, & Thompson, 1984; Nesselroade & Salthouse, 2004), intraindividual variability analyses are essential for advancing the field of sleep. This study will employ both intraindividual analysis and multi-level modeling analysis techniques in this project.

Sleep Misperception in Context

Herein sleep misperception is defined as the difference between subjective and objective accounts of SOL and WASO. Unlike other studies, this study does not assume that sleep misperception represents a perceptual distortion, nor a distinct sleep disorder. Indeed, sleep misperception may be more the researcher's misperception of what the patient perceives than vice versa. The term 'sleep misperception' is maintained in this study because the phenomena is presumed to be the same as that which other studies have investigated. Regrettably though, sleep misperception has rarely been studied as an accurate perception of a unique physiological state that is neither wake nor sleep. Smith and Trinder aptly stated, "[O]verestimates of SOL may be a

normal response to poor sleep. If similar phenomena occur in a clinical environment, the perception of disturbed sleep might be categorized as over estimation of SOL, when in fact the conventional sleep scoring underestimates the degree of the sleep disturbance" (Smith & Trinder, 2000, p134). Given the crude measuring capability of sleep recording devices, such as actigraphy and EEG, the possibility remains that sleep misperception represents a sleep disturbance not detectible by current sleep measuring techniques. By comparing the daily phenomenon of sleep misperception to other daily sleep variables, such as total sleep time, the underlying mechanisms of sleep misperception may be better understood.

Mechanisms of Sleep Misperception

To date, research on the biopsychophysiological mechanisms involved in sleep misperception is limited to a small number of EEG, actigraphy, and sleep diary studies. More broadly, biophysiological mechanisms of insomnia are under studied and poorly understood (Drummond et al., 2004). There is substantial data to suggest that arousal is related to sleep misperception and may be a central mechanism of sleep misperception.

Hyperarousal

Many sleep experts agree that some 'type' of hyperarousal is involved in sleep misperception (Benoit & Aguirre, 1996; Bonnet & Arand, 1997b; Drummond et al., 2004; Harvey, 2002a; Perlis, Giles, Bootzin et al., 1997; Perlis, Smith et al., 2001). At least one of three 'types' of hyperarousal is endorsed by sleep researchers: cognitive (mental/emotional), physiological (somatic), and central nervous system (CNS) hyperarousal. However, the line delineating these 'types' of arousal is ambiguous and elusive. Some studies have shown a high correlation (Nicassio, Mendlowitz, Fussell, & Petras, 1985) between cognitive and physiological arousal, while others suggest relative independence of these constructs (Craske & Craig, 1984). Many researchers hold that cognitive arousal is limited to the mind, while psychological arousal

refers specifically to activation of the sympathetic nervous system (Bonnet & Arand, 1997a; Nicassio et al., 1985; Schwartz, Davidson, & Goleman, 1978; Steptoe & Kearsley, 1990). CNS arousal involves greater cognitive activation (i.e., increased beta/gamma activity) during sleep. This activity may or may not be related to cognitive or physiological arousal. (Drummond et al., 2004; Perlis, Merica et al., 2001; Perlis, Giles, Mendelson et al., 1997). The relationship of each type of hyperarousal with sleep misperception is discussed in more detail below.

Cognitive Arousal

Conceptually, cognitive arousal is posited to be the degree to which the 'mind' is agitated and has been distinguished from physiological arousal and brain activation. Scientifically, like the mind, the construct cognitive arousal is difficult to isolate. Often, cognitive arousal is thought of as a top down process that can activate the stress response but is not necessarily preceded by activation of the fight-or-flight response. Moreover, top down arousal, it is claimed, does not necessarily lead to activation of the sympathetic response. Several studies have attempted to isolate the various types of cognitive arousal and their impact on sleep misperception. Intrusive thoughts (Lichstein & Rosenthal, 1980; Mitchell, 1979; Monroe, 1967), sleep threat monitoring, clock monitoring, rumination (Freedman & Sattler, 1982) and catastrophic worry have been linked to sleep misperception (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Harvey, 2002b; Harvey & Greenall, 2003; Lichstein & Rosenthal, 1980; Morin, Blais, & Savard, 2002). From this research, two types of cognitive arousal have been constructed: neutral arousal and anxious/emotional (Bonnet & Arand, 1997a; Tang & Harvey, 2004). One study, in an attempt to contrast the impact of these two types of cognitive arousal on sleep misperception in healthy sleepers, found that participants assigned to either anxious or neutral cognitive arousal conditions overestimated SOL, but only the cognitive arousal, which involved anxiety, induced significantly lower estimates of TST than the control group during a daytime nap (Tang & Harvey, 2004).

Physiological Arousal

Tang and Harvey (2004) highlight the possibility that physiological arousal may cause sleep misperception and explain the high association between sleep misperception and insomnia. Individuals diagnosed with sleep state misperception show a variety of somatic markers of activation of the fight-or-flight response surrounding and during the sleep experience including high metabolic rate (Bonnet & Arand, 1997b). In addition, one study showed that brief global arousals lasting 3-30 seconds during sleep have been observed in patients with sleep state misperception (Smith & Trinder, 2000). This view is further strengthened by several studies which have scientifically manipulated sleep misperception. In a series of studies, Mendelson and colleagues found that benzodiazepines (i.e., Flurazepam, Zolpidem) reduced sleep misperceptions; more specifically, these drugs reduced the likelihood that insomnia patients reported EEG defined sleep as wakefulness (Mendelson, 1993, 1995a; Wallace B. Mendelson & Maczaj, 1990; Mendelson, Martin, Stephens, Giesen, & James, 1988). However, these drugs did not alter the perceptions of sleep in normal sleepers (Mendelson, 1995b). It was concluded that benzodiazepines reduced arousal in these patients and simultaneously eliminated sleep misperception. Other studies have shown that caffeine administration induces sleep misperception, increased anxiety and, somatic hyperarousal in healthy sleepers (Bonnet & Arand, 1992, 1997a; Karacan et al., 1976; Okuma, Matsuoka, Matsue, & Toyomura, 1982). Researchers concluded that caffeine directly increases arousal which then leads to sleep misperception (Bonnet & Arand, 1997b; Tang & Harvey, 2004). Collectively, these studies point to a strong relationship between physiological arousal and sleep misperception.

CNS Hyperarousal

CNS hyperarousal (as determined traditionally by increased high frequency EEG in the beta range (14-35 Hz) and more recently by high metabolic rate in positron emission topography

(PET) and blood-oxygen level and decline (BOLD) in functional magnetic resonance imagining (fMRI)) is not only claimed to be the marker of sleep misperception (Perlis, Giles, Mendelson et al., 1997) but insomnia more generally (Drummond et al., 2004; Freedman, 1986; Merica, Blois, & Gaillard, 1998; Merica & Gaillard, 1992; Perlis, Smith et al., 2001). The EEG measures brain wave activity by frequency and amplitude. High frequency EEG activity in the beta [14-35 Hz] and gamma [35-45 Hz] range represents more brain wave activity and dominates the active wake state of the brain. During healthy sleep, EEG activity dramatically decreases and high frequency waves are replaced by progressively lower frequency higher amplitude activity in the .5-4 Hz range. Higher frequency activity in the beta/gamma range is characteristic of insomnia both at sleep onset and throughout the sleep period (reviewed in Perlis, Merica et al., 1998; Merica et al., 2001). In addition, increased beta activity during the lighter stages of sleep has been directly linked to sleep misperception (Perlis, Smith et al., 2001). Science has yet to show whether the length of time that beta activity persists into sleep corresponds to insomnia patient's experience of wakefulness.

Sleep Misperception as Localized Sleep Deprivation

Because all three types of arousals mentioned above are associated with sleep misperception and because the validity of delineating hyperarousal remains hotly contested (Drummond et al., 2004; Perlis, 2001), the current study will not parse out the source of arousal nor the causal relationship between arousal and sleep misperception. Sleep misperception may represent a true sleep disorder predicted by changes in the daily expression of sleep. Given the hypothesis that isolated areas of the brain remain active, or are prevented from sleeping deeply with the rest of the brain during sleep misperception, these areas may be subjected to a form of localized sleep deprivation. Global sleep deprivation, for short and long periods of time, has been linked to reduced cognitive and physical performance, increased susceptibility to disease,

increased morbidity, reduced immune response, increased stress, and the onset of symptoms related to mental and physical health, such as depression, insomnia, and fibromyalgia. Like global sleep deprivation, localized sleep deprivation may predispose individuals to problems related to the areas of the brain that are affected in sleep misperception. Importantly, sleep misperception may represent an accurate perception of a sleep problem that is undetectable by standard EEG measures. Regardless, this phenomenon may have serious financial and health consequences.

Sleep misperception may result from the areas of the brain involved in consciousness or executive functioning remaining active, consequent of hyperarousal while the rest of the brain has entered sleep (Merica et al., 1998). This conclusion is consistent with the 'localization' theory of sleep (Krueger & Obal, 1993). They argue that sleep is not a whole brain event and that each area of the brain has its own circadian rhythm and homeostatic need. Merica and colleagues (1998), applied the local sleep theory to sleep misperception. In their neuronal transition probability model (NTP), they argue that areas of the brain that remain active during EEG sleep may represent an intermediate or transition state between sleep and wake where sleep misperception occurs (Merica et al., 1998). The subtle difference in these perspectives is that the later continues to delineate 'wake state' from 'intermediate state' from 'sleep state.' Localized theory recognizes that sleep and wake are not discrete states but can co-occur by degree. Regardless of this distinction, both perspectives highlight the possibility that whole body arousal is not necessarily required for localized areas of the brain to stay active during sleep. Only those areas of the brain involved in perception would need to be active to produce what is called sleep misperception. Although beta activity has been globally identified in conjunction with sleep misperception, the location of beta activity has not been determined. However, beta activity is

most likely occurring in only isolated areas of the brain related to attention, monitoring and self-awareness, such as the dorsal lateral prefrontal cortex, the anterior cingulate gyrus, and insula. In this study, distinct areas of the brain involved in sleep misperception were not directly investigated. Nonetheless, the understanding of sleep misperception as a localized brain event provided the theoretical foundation on which the analysis and interpretation of results were built.

Because objective changes in the sleep of patients with sleep misperceive is lacking, many have argued that misperception is an arousal problem not related to a sleep disturbance. For example, Bonnet and Arand (1995) argue that sleep misperception is related to increased arousal among individuals with longer sleep need. However, sleep requirement/length is determined by two sleep systems rather than one conceptualized in Bonnet's model. The preeminent sleep theorist and researcher Alexander Borbély (1982) posited two major brain processes essential to sleep. His theory, called "the two process model of sleep," has dominated basic sleep research for the past 2 decades and continues to influence the field. The first of his processes, the circadian process, or more simply the C-process is what most people think of when they think about sleep. The C-process is the biological drive to sleep that is unique to each species and to a lesser extent to each individual within each species. This process is what makes humans diurnal and bats nocturnal. The C-process is driven by biological clocks in the brain, which rhythmically increase and decrease the brains propensity to sleep on about a 25-hour basis. The other process in Borbély's model is called the homeostatic or S-process. This process increases the brain's propensity to sleep as a function of time awake and the intensity at which neurons are used. The idea here is that the more an area of the brain is used the more that area needs sleep. Following 24-hour sleep deprivation, the S-process intensifies the drive to sleep late in the night and into

the next day while the C-process actually begins to decrease its influence on sleep. These processes have been tied to insomnia and are applied to sleep misperception in this study.

Sleep misperception may be related to a homeostatic dysregulation. Following sleep deprivation, sleep rebounds by extending the amount of sleep. This is because it takes longer to restore sleep homeostasis following deprivation. The circadian process is not altered by sleep deprivation but is highest roughly 16 hours after waking. Tenuous evidence exists that some type of C-process dysregulation is involved in insomnia (reviewed in Pigeon & Perlis, 2006). For example, abnormal temperature rhythms have been observed in insomnia (e.g., Morris, Lack, & Dawson, 1990). However, these changes may be the result of protective behaviors often observed in insomnia patients who alter the timing of light exposure and biological rhythms, such as temperature (Pigeon & Perlis, 2006). Another study found that in relationship to sleep misperception, circadian dysregulation is most likely a consequence of sleep misperception rather than a causal factor. Nonetheless, circadian rhythms must be considered in relationship to sleep misperception because before sleep misperception could occur most of the brain must be in a sleep state. The C-process is the major force that drives the global sleep state. Sleep propensity imposed on the brain by chronobiological clocks that regulate sleep must be highly active in the presence of high arousal in perceptual centers of the brain. When arousal is relatively higher than the S-process of a given area of the brain, and the C-process is higher than both the arousal system and S-process, then global sleep will be observed but localized sleep deprivation will occur in the isolated areas. Local sleep theory would predict that if arousal in the perceptual/consciousness centers of the brain exceeds the global sleep drive (the C-process) and also the local sleep drive (S-process), that area may remain wake; thus, perceiving a wake state and the passing of time. If sleep were restricted, the area of the brain that remained awake during

global sleep would receive, potential, much less sleep than the rest of the brain. For example, if sleep were restricted to 6 hours, but the perceptual areas of the brain remained active for 1 hour, the patient would report that they had only received 5 hours in contrast to the objective 6 hours that was observed using EEG. However, it may be possible that local sleep loss, such as is posited here, may lead to sleep rebound in that area and on the whole brain. That is, if sleep were not restricted the patient may awake less during the night (or perceive that they had awoken less during the night) and sleep longer in the morning. In fact, this model would predict that even if the global brain awoke, the patient might not be aware because the perceptual areas of the brain would still be sleeping.. Aims 3 and 4 investigate the relationship between SOL_{sm}, $WASO_{sm}$ and TST_{o} from this perspective.

Summary

In summary, sleep misperception is operationally defined in this study as the discrepancy between objectively measured sleep and the participant's perceptions. The idea that sleep misperception represents a perceptual deficit or exaggeration of sleep difficulty by a select group of sleep complainers, though ubiquitously accepted, has little scientific support. That sleep misperception increases with age is well known; however, much research is needed to better understand the patterns and correlates of sleep misperception in this population. Arousal is strongly associated with sleep misperception. This study is based on the theoretical assumption that sleep misperception may be a form of localized sleep deprivation resulting in sleep deprivation-like consequences (e.g., longer total sleep time).

CHAPTER 3
STATEMENT OF THE PROBLEM

The main objective of this study is to better understand sleep misperception (SM) by investigating the distribution, patterns and correlates of subjective relative to objective measures of SOL and WASO, hereafter called SOL_{sm} and $WASO_{sm}$, in a sample of community dwelling older adults. Daily and biweekly averages of SOL_{sm} and $WASO_{sm}$ will be studied in relation to1) the amount of within- to between-person variability, 2) sleep complaint status, 3) each other, and 4) actigraphy measured total sleep time ($TST_{objective}$).

Specific Aim 1

The first specific aim is to investigate SOL_{sm} and $WASO_{sm}$ across two weeks. Aim 1 will determine the amount of within- to between-person variability in SOL_{sm} and $WASO_{sm}$ (as determined by daily diary self-report of SOL or WASO minus SOL or WASO as determined by daily actigraphy measures, respectively).

Hypothesis for Specific Aim 1

It is hypothesized that there will be greater intraindividual variability (i.e., within-person variability) than between-persons. It has been reported that people can be categorized as either positive or negative perceivers of their sleep (Means et al., 2003; Trajanovic, Radivojevic, Kaushansky, & Shapiro, 2007). However, this assertion is based on research that looks only at a single night of sleep or averages over several nights of sleep. These approaches ignore the potential confound that individual's SOL_{sm} or $WASO_{sm}$ may be highly variable across several nights. As a result these studies may only capture individuals on the extreme of a continuum of misperceiving sleep. Intraindividual variability in SOL_{sm} and $WASO_{sm}$ have not been tested to confirm or contradict the assertion that there are sleep perceiver types that consitantly either over or under perceive SOL and WASO.

Specific Aim 2

The second specific aim is to investigate SOL_{sm} and $WASO_{sm}$ among older participants who reported a sleep problem (complainers) and those that did not (non-complainers). Aim 1 will determine the amount of within-to between-person variability in SOL_{sm} and $WASO_{sm}$ that occurred among sleep complainers as compared to non-complainers.

Hypothesis for Specific Aim 2. Patients with insomnia experience high night-to-night variability in sleep. High intraindividual variability among insomnia patients has been reported for both objective and subjective measures of sleep onset latency (SOL), total sleep time (TST), waketime after sleep onset (WASO), and subjective sleep quality (SSQ) (Edinger, Marsh et al., 1991). In spite of this high degree of within-patient variability across a myriad of sleep measures, it has been reported that insomnia patients consistently misperceive the sleep experience, including overestimating SOL and WASO. Within-person variability in SOL_{sm} and $WASO_{sm}$ among individuals who complain of sleep difficulties has not been compared to individuals who do not complain of sleep difficulties. The analyses preformed in aim 2 should help determine whether sleep misperception is a consistent trait-like behavior of a subgroup of individuals or a behavior that manifests by degree among older adults.

Previously our lab showed that sleep complaint among older adults related to an occasional pattern of extreme sleep overestimating SOL and WASO that were more extreme but not more frequent among individuals with sleep complaint than those without complaint (Kay, McCrae, & Rowe, 2008). In that study, a one-way MANOVA revealed a significant group effect for the two sleep misperception dependent variables ($F_{4,89} = 7.65$, $p < .001$). Follow-up testing revealed complainers over-estimated WASO more frequently than non-complainers ($M_{\text{complainers}} = 3.67 \pm 0.74$ days versus $M_{\text{non-complainers}} = 1.27 \pm 0.22$ days; $F_{1,92} = 14.85$, $p < .001$). Frequency of SOL over-estimation did not differ by group(~7 days; $F_{1,92} = 0.01$, $p = .94$). Interestingly, on nights

that over-estimation occurred, the average amount over-estimated was greater for complainers than non-complainers for both SOL ($M_{\text{complainers}} = 22.54 \pm 2.74$; $M_{\text{non-complainers}} = 14.21 \pm 1.17$; $F_{1,92} = 9.63$, $p < .01$) and WASO ($M_{\text{complainers}} = 44.62 \pm 10.01$; $M_{\text{non-complainers}} = 14.48 \pm 3.54$; $F_{1,89} = 13.13$, $p < .001$). For both groups, SOL over-estimates did not correlate with WASO over-estimates. Importantly, over-estimating did not occur daily among complainers; however, on days over-estimating occurred, their estimates were more extreme on average than non-complainers.

Intraindividual analyses which compare the amount of within- to between-person variability by complaint status can be used to help determine if SM is a between-persons trait or a highly variable pattern within-persons. Aim 2 will test the hypothesis that SOL_{sm} and $WASO_{sm}$ will be highly variable within individuals regardless of sleep complaint status.

Specific Aim 3

To investigate the relationship between day-to-day fluctuations in SOL_{sm} to fluctuations in $WASO_{sm}$ in older adults.

Hypothesis for Specific Aim 3. Most studies of sleep misperception have focused on SOL. Perlis et al. (1997) argued that because high frequency EEG activity is central to both sleep onset and sleep misperception, SM should be more prominent during that period. Moreover, Mendelson (1998) found that sleep perceptions where more congruent with PSG during the third sleep cycle as compared to cycles 1 and 2 among insomnia patients. However, SM can and does occur during WASO also. Sleep maintenance problems that contribute to WASO event are much rarer in younger adults than older adults, even among insomnia populations (Bliwise, 2005). In late life, nighttime awakenings become more frequent and present greater opportunity to study sleep misperception than occur spontaneously during the night. Traditionally, sleep researchers have combined SOL_{sm} and $WASO_{sm}$ across days to create a more robust SM variable (Means et

al., 2003). Because combining multiple nights of SOL_{sm} and $WASO_{sm}$ into one variable creates a more robust variable, it is likely that these variables co-occur on some level. However, it remains unclear whether day-to-day fluctuations in SOL_{sm} and $WASO_{sm}$ vary together or fluctuate independently from one another. That is, on days that $WASO_{sm}$ are high does that predict that SOL_{sm} will also be high. The positive relationship between SOL_{sm} and $WASO_{sm}$ has only been demonstrated on the average but not the daily level. For example, one study that investigated SM in insomnia patients showed that participants perceived sleep as wakefulness during all lighter stages of sleep (i.e., stages 1 and 2) throughout the night (Mercer, Bootzin, & Lack, 2002). However, the averaging techniques used in that study prevented the researchers from concluding that those who misperceived SOL were the same individuals who misperceived WASO. Ultimately, they showed only that both SOL_{sm} and $WASO_{sm}$ are common among insomnia patients. The question remains, are perceptions of SOL and WASO independent from or dependent on each other. Studies that investigate the night-to-night relationship between SOL_{sm} and $WASO_{sm}$ are needed to determine their longitudinal relationship.

Many people believe that sleep misperception represents a negative bias imposed on sleep or an exaggeration of sleep difficulties. This assumption would predict that a negative bias would be imposed consistently on all aspects of sleep. While the consistency in average sleep misperception is well established, consistency as it relates to night-to-night consistency and variable-to-variable (e.g., SOL_{sm} to $WASO_{sm}$) consistency has not been tested. This assumption would be greatly weakened if it can be shown that there is no relationship between SOL_{sm} and $WASO_{sm}$ on a night-to-night basis. It is hypothesized that $WASO_{sm}$ and SOL_{sm} will not vary together night-to-night. More specifically, it is hypothesized that, even though on an average

SOL_{sm} and $WASO_{sm}$ are positively related, greater SOL_{sm} will not necessitate greater $WASO_{sm}$ and vice versa on a daily level.

Specific Aim 4

To determine the intraindividual daily relationship between SOL_{sm} and $WASO_{sm}$ to objectively measured TST_o (actigraphy).

Hypothesis for Specific Aim 4. It is hypothesized that within-person fluctuation in SOL_{sm} and $WASO_{sm}$ will share a systematic relationship to fluctuation in TST_o. Bonnet's model of sleep posits that SM is the interaction between the sleep and arousal systems, such that longer sleep requirement coupled with chronic hyperarousal is the formula for SM. Indeed, insomnia patients who sleep longer than others are more likely to be identified as having sleep misperception. Moreover, our lab has previously shown that older adults who complained of hyperarousal related health conditions, such as chronic pain and depression, have greater SM than non-complainers regardless of whether there was a sleep complaint or not (Kay, McCrae, & Rowe, 2008). Bonnet and Arand (1995) assumed that sleep requirement is a trait-like characteristic of sleepers. However, sleep requirement may vary significantly day-to-day. Increased TST is a marker of an S-process sleep dysregulation. Sleep deprivation is the most salient way to disrupt the S-process. During sleep deprivation, sleep promoting substances accumulate in the brain and exert their effects once sleep is obtained. This disruption to the S-Process through sleep deprivation can induce increased TST. We posit that SM represents a form of localized sleep deprivation to perceptual areas of the brain. While global sleep is obtained, the areas responsible for conscious awareness remain active and are, thus, prevented from obtaining the localized sleep need. As a result, SM as a localized sleep deprivation may predict a similar sleep rebound in TST as global sleep deprivation. Based on this theory, we predict that TST_o will increase with increased SOL_{sm} and $WASO_{sm}$ from night-to-night. Aim 4 will test the hypothesis that, within-

persons, on days that individuals have greater SOL_{sm} or $WASO_{sm}$ than usual, his/her TST_o will be longer than usual.

CHAPTER 4
METHODS

Participants

This study involved secondary analyses on a database compiled by McCrae and Rowe (2003) in a study on sleep in older adults. A convenience sample of 116 community-dwelling older adults (*Mean* age=72.81, *SD*=7.12) were recruited from the North Florida area. A variety of recruitment techniques were employed including media advertisements, community groups, and flyers. Recruitment materials described the research as a study of sleep patterns in the elderly. Participants were compensated $30 for their participation. Interested individuals were screened in two phases to determine if they met the criteria for inclusion. Phase one consisted of a brief telephone interview (15-20 minutes), and phase two involved an in-person interview either in the individual's home (76%) or at a local continuing care retirement center (24%).

Individuals were excluded on the basis of six exclusionary criteria: 1) age younger than 60 years; 2) self-report of sleep disorder diagnoses other than insomnia (e.g., sleep apnea or narcolepsy); 3) self-report of sleep symptoms indicative of sleep diagnoses other than insomnia (e.g., heavy snoring, gasping for breath, leg jerks, daytime sleep attacks); 4) presence of severe psychiatric disorders (e.g., thought disorders or depression); 5) cognitive impairment, scoring in the impaired range on three or more subtests of the Neurobehavioral Cognitive Status Examination (Cognistat; Mueller, Kiernan, & Langstron, 2001); 6) use of psychotropic or other medications know to alter sleep (e.g., beta-blockers); and 7) medical conditions that impaired independent daily functioning (McCrae et al., 2005). Participants who entered the study were gave informed consent in accordance with the standards of the University of Florida Institutional Review Board.

Of the 116 individuals recruited, 103 were enrolled in the study. Thirteen individuals were ineligible to participate in the study due to age, dementia, medication, and sleep apnea diagnosis. The mean age of the participants was 72.81 years (*SD* = 7.12). The majority of participants were European Caucasian (96%), female (66%), college educated (75%; *M* = 16.34 years, *SD* = 2.92), and married (59%). All of the participants lived in their own homes during the study.

Procedures

Data was collected at three periods during the study: baseline, end of first week, and end of second week. During the initial 1- 1½ hour interview, participants read and signed an informed consent form approved by the University of Florida Institutional Review Board. Once consent was obtained, the Cognistat, and the demographics and health survey were administered by a member of the research team. At this time, both the sleep diaries and the Actiwatch-L® (ACT-L; Mini Mitter, Inc.) were explained to the participants. The participants were advised to complete the sleep diaries and wear the Actiwatch-L® continuously for 14 days. At the end of the first week, the sleep diaries were collected from the participants and the data was downloaded from the Actiwatch-L®. At the end of the second week, the final week of sleep diaries and Actiwatch-L® data were collected. The Beck Depression Inventory-Second Edition (BDI-II Beck, Steer, & Garbin, 1996) was also completed at this time.

Participants wore the Actiwatch-L® wrist device both day and night for two weeks to determine objective total sleep time (TST_o), sleep onset latency (SOL_o) and wake-time after sleep onset ($WASO_o$). While wearing the Actiwatch-L®, participants also completed daily sleep diaries, recording their subjective estimates of, sleep onset latency (SOL_s) and wake time after sleep onset ($WASO_s$).

Measures

Overviews of the sleep variables, the demographics and health survey, the Cognistat, and the measures of daytime functioning are presented below.

Objective Sleep Variables

Objective sleep was measured using the Actiwatch-L®(Mini Mitter Co., 2001). Within the Actiwatch-L®, data is sampled 32 times per second over a 30 second epoch using an omnidirectional, piezoelectric accelerometer with a sensitivity of $\geq$ 0.01 g-force. A sum of the peak activity counts for each 30 second epoch is downloaded to a PC and then analyzed by Actiware-Sleep vol. 3.3. (Mini Mitter Co. Inc., 2001). Three sensitivity settings (high, medium, and low) are provided by the software for detecting wake/sleep periods. A high sensitivity setting was used in the current study since it provides high correlations with PSG measured total sleep time (.95) for healthy older adults (Colling et al., 2000) and for total sleep time (.73) and sleep onset latency (.93) for individuals with insomnia (Cook et al., 2004). Additionally, actigraphy has valid criterion-validity when compared to PSG (.80) and high test-retest reliability (0.92; (Ancoli-Israel et al., 2003; de Souza et al., 2003; Hauri & Wisbey, 1992). A validated algorithm is used to identify the activity of each epoch as wake or sleep (Oakley, 1997). With the high sensitivity setting, the threshold for wake is 20 activity counts. If the peak activity count for an epoch is $\geq$ 20, the epoch will be scored as wake. If the peak activity count for an epoch is < 20, the wake/sleep determination is made based on the activity that occurs in the two-minute period surrounding the epoch. The wake/sleep determination if the activity count is < 20 is made based on following equation:

Total Activity for Epoch A = E_{A-4} (.04) + E_{A-3} (.04) + E_{A-2} (.20) + E_{A-1} (.20) + E (2) + E_{A+1} (.20) + E_{A+2} (.20) + E_{A+3} (.04) + E_{A+4} (.04)

where A = # of activity counts for the epoch being scored; $E_{A +/- 1-4}$ = # of activity counts in adjacent epochs. If the Total Activity for Epoch A (weighted sum of activity counts) exceeded the threshold value of 20, then Epoch A is scored as wake; otherwise, it is scored as sleep (McCrae et al., 2005). Using the Actiware-Sleep vol. 3.3 software (Mini Mitter Co. Inc., 2001), a number of sleep parameters are derived from the data including sleep onset latency, total sleep time, and wake after sleep onset. The definitions of the objective sleep variables used in this study are: sleep onset latency$_o$ (interval between bedtime and sleep start); total sleep time$_o$ (time in bed minus SOL_o, $WASO_o$, and time spent in bed between awakening in the morning and getting out of bed); and wake time after sleep onset$_o$ (time spent awake after initial sleep onset until last awakening). The subscripts "s" and "o" are used to distinguish between the subjective and objective sleep variables.

Actigraphy is less reliable at determining WASO than it is at identifying sleep onset or TST. Compared to PSG, actigraphy systematically underestimated WASO (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992). This is explained by the relatively lower resolution of actigraphy at detecting wake compared to detecting sleep. In field studies, however, actigraphy remains a good measure of detecting WASO in comparison to PSG, particularly when the WASO event lasts longer than 15 seconds. Actigraphy detects 88% of such events (Horne, Pankhurst, Reyner, Hume, & Diamond, 1994).

Subjective Sleep Variables

Sleep diary. Subjective sleep quantity was measured using sleep diaries. The sleep diary (Lichstein, Riedel, & Means, 1999) was completed by each participant each morning for 14 days (see Appendix A). Two subjective sleep measures were obtained from the sleep diary data: sleep onset latency (SOL_s; initial time from lights out until sleep onset), and wake time after sleep onset ($WASO_s$; time spent awake after initial sleep onset until last awakening). Sleep diaries are

commonly used in research, have been validated in several studies, and are accepted as a scientifically useful measures of the subjective sleep experience (Espie, Inglis, Tessier, & Harvey, 2001).

Sleep Misperception Variables (SOL_{sm} and $WASO_{sm}$)

In this study, the difference between objective and subjective measures are called sleep misperception. Raw daily subjective reports of SOL and WASO were compared to respective daily actigraphy measures to compute daily SOL_{sm} and $WASO_{sm}$ (subscript '*sm*' denotes sleep misperception). The equation for each variable is listed below:

$$SOL_{sm} = SOL_s - SOL_o$$

$$WASO_{sm} = WASO_s - WASO_o$$

Daytime Functioning Measure

Beck Depression Inventory – Second Edition (BDI-II)

Depression was measured using the Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Garbin, 1996). This is a 21-item measure with a scale ranging from 0-3 measuring the severity of depressive symptoms (3 being the most severe). Scores range from 0 – 63. Scores within the 0 – 13 range indicate none or minimal depression, 14 to 19 indicate mild depression, 20 to 28 indicate moderate depression, and 29 to 63 indicate severe depression. Participants were asked to respond to the questions based on the previous two weeks to match the two-week sleep and actigraphy recording period. The BDI-II has demonstrated sufficient internal consistency reliability (.90) and concurrent validity (.69 - .76; Storch, Roberti, & Roth, 2004).

Demographics and Health Survey

This survey consists of 13 items collecting information on demographics, sleep disorder symptoms, physical health, and mental health (see Appendix A; Lichstein et al., 2004). Health

conditions were assessed as the number of conditions selected from the following list: heart attack, other heart problems, cancer, AIDS, hypertension, neurological disorder (e.g., seizures, Parkinson's), breathing disorder (e.g., asthma, emphysema, allergies), urinary problems (e.g., kidney disease, prostate problems), diabetes, pain (e.g., arthritis, back pain, migraines), and gastrointestinal disorders (e.g., stomach, irritable bowels, ulcers, gastric reflux). Self-report sleep questions on the survey contained information on whether the participant had a sleep problem and if they or a bed partner noticed heavy snoring, difficulty breathing or gasping for breath, frequent leg jerks, restlessness before sleep onset, sleep attacks during the day, or paralysis at sleep onset. Two health survey variables were used in this study; sleep complaint and the total number of health complaints.

Sleep Complaints: Determined by yes/no report to the question: "Do you have a sleep problem? yes or no"

Number of Health Conditions: The number of health conditions that the participants complained of, except sleep and pain) were summed. Scores could range from 0-10 complaints.

Data Analyses

The current study applied several statistical techniques to investigate the four specific aims of this study including intraindividual analyses and multilevel modeling (MLM). The analytical techniques used for each aim are explained and described below.

Specific Aim 1

The first specific aim is to investigate SOL_{sm} and $WASO_{sm}$ across two weeks. Aim 1 will determine the amount of within-to between-person variability in SOL_{sm} and $WASO_{sm}$.

Data analysis. Intraindividual variability (IIV) analyses will be used to determine the relative amount if within- to between-person variability in SOL_{sm} and $WASO_{sm}$. To control for potential systemic growth in the data, all variables will be de-trended prior to calculation of

indexes of within- and between-person variability. To de-trend the data, linear regression will be run with SOL_{sm} and $WASO_{sm}$ as the independent variables. The subsequent unstandardized residual values resulting from the regressions will then be saved and used as time independent values. Using the residual values computed above, an index of between-person variability (Sample Standard Deviation, SD) and within-person variability (Individual Standard Deviation, ISD) will be computed. These values will then be compared by dividing the ISD by the SD to get the proportion of between-person variability that is found within-persons.

Specific Aim 2

The second specific aim is to compare the amount of within- to between-person variability in SOL_{sm} and $WASO_{sm}$ among sleep complainers with that among sleep noncomplainers.

Data analysis. Intraindividual variability analyses will be used to determine the relative amount if within- to between-person variability in SOL_{sm} and $WASO_{sm}$ within each of the sleep groups (i.e., sleep complaining and sleep non-complaining). The de-trended data obtained following aim one will be separated into the respective two groups. Two separate linear regressions (one for each group) will be run with SOL_{sm} and $WASO_{sm}$ as the independent variables. The subsequent unstandardized residual values resulting from the regressions will then be saved and used as time independent values. Using the residual values computed above, an index of between-person variability (Sample Standard Deviation, SD) and within-person variability (Individual Standard Deviation, ISD) will be computed for each group. For each group of data, these values will then be compared by dividing the ISD by the SD to get the proportion of between-person variability that is found within-persons.

Specific Aim 3

To investigate the relationship between day-to-day fluctuations in SOL_{sm} to fluctuations in $WASO_{sm}$.

Data analysis. A multilevel modeling (MLM) approach was used to predict within-person and between-person relationships between SOL_{sm} and $WASO_{sm}$. MLM, also referred to as hierchical linear modeling (HLM; Bryk & Rauenbush, 1992) is an extension of the general linear model and is especially suited for daily data, such as sleep, because of their autoregressive nature and hierarchical structure with daily observations nested within each participant (Singer, Davidson, Graham, & Davidson, 1998; Singer, Fuller, Keiley, & Wolf, 1998; Singer & Willett, 2003). Because of the hierarchical nature of the data used in this study (14 days consecutive days nested within 103 participants) and in order to increase the precision of predicting fluctuations in SOL_{sm} with fluctuation in $WASO_{sm}$), the data will be modeled with a MLM approach. This provides the opportunity to examine the relationship between these variables on two levels: a within-(level 1) and a between (level 2)-persons. Level 1 analysis addresses two questions: First,"On days in which a person reports above-average SOL_{sm}, does s/he also report above-average $WASO_{sm}$?" and second, "On days in which a person reports above-average $WASO_{sm}$, does s/he also report above-average SOL_{sm}?" This level of analysis is concerned with questions of atypical days within an individual and what variables fluctuate, within-persons, on these atypical days. Level 2 analyses examine the question: "Do people who generally have high $WASO_{sm}$ also report high SOL_{sm} and vica versa?" $WASO_{sm}$ will be used to predict SOL_{sm} and vice versa in two separate models, each using a three step MLM approach.

In the first model, Step 1 (Tables 5-3, Row 1), the null (baseline) model, will estimate only a fixed and random intercept for $WASO_{sm}$(Bryk & Raudenbush, 1992). This model will specify that $WASO_{sm}$ for person *j* is a function of the overall group-average $WASO_{sm}$, a between-person random error term, and a within-person random residual component. This step provides a comparison for the step 3 model, which includes SOL_{sm}. In step 2, time functions (linear) will be

added as a covariate to the null model (Table 5-3, Row 2), producing a latent growth curve model. As such, the model will specify that $WASO_{sm}$ for person *j* on day *i* is a function of: average $WASO_{sm}$, linear time, a between-persons random error term, and a within-person random residual component. Next, SOL_{sm} will be added to the model. In step 3 (Table 5-3, Row 3) the estimates of the fixed and random intercepts and fixed linear slopes for each sleep variable will be added. Thus, the daily $WASO_{sm}$ for each person will be predicted by: average level of $WASO_{sm}$, linear time, the between-person effects of mean-level SOL_{sm}, the within-person effects of SOL_{sm}, a between-person random error term, and a within-person random residual component.

In the second model, Step 1 (Tables 5-5, Row 1), the null (baseline) model, will estimate only a fixed and random intercept for SOL_{sm} (Bryk & Raudenbush, 1992). This model will specify that SOL_{sm} for person *j* is a function of the overall group-average SOL_{sm}, a between-person random error term, and a within-person random residual component. This step provides a comparison for the step 3 model, which includes $WASO_{sm}$. In step 2, time functions (linear) will be added as a covariate to the null model (Table 5-5, Row 2), producing a latent growth curve model. As such, the model will specify that SOL_{sm} for person *j* on day *i* is a function of: average SOL_{sm}, linear time, a between-persons random error term, and a within-person random residual component. Next, $WASO_{sm}$ will be added to the model. In step 3 (Table 5-5, Row 3) the estimates of the fixed and random intercepts and fixed linear slopes for each sleep variable will be added. Thus, the daily SOL_{sm} for each person will be predicted by: average level of SOL_{sm}, linear time, the between-person effects of mean-level $WASO_{sm}$, the within-person effects of $WASO_{sm}$, a between-person random error term, and a within-person random residual component.

All models will be estimated using the Maximum Likelihood (ML) method. The ability of the first model to predict $WASO_{sm}$ and the second model to predict SOL_{sm} better than the

baseline model (i.e., Deviance) will be used as an index of Goodness of Fit. Improvements in predictability will be determined by the amount of reduction of within- and between-person residual variances compared to the baseline model (Bryk & Raudenbush, 1992). Decreases in residual and intercept variances represent a proportional reduction of the prediction error, which is analogous to R^2, and will be used as an estimate of within- and between-person effect sizes. The amount of agreement between model predicted values and actual values will be calculated as an estimate of an overall effect size.

Specific Aim 4

To determine the intraindividual daily relationship between SOL_{sm} and $WASO_{sm}$ to TST_o.

Data analysis. A multilevel modeling (MLM) approach was used to predict within-person and between-person relationships of SOL_{sm} and $WASO_{sm}$ to TST_o. $WASO_e$ and SOL_{sm} will be used to predict TST_o using a four step MLM approach. Step 1 (Table 5-7, Row 1), the null (baseline) model, will estimate only a fixed and random intercept for SOL_{sm} (Bryk & Raudenbush, 1992). This model will specify that TST_o for person *j* is a function of the overall group-average TST_o, a between-person random error term, and a within-person random residual component. This step provides a comparison for the step two models which includes time. In step 2, time functions (linear) will be added as a covariate to the null model (Table 5-7, Row 2), producing a latent growth curve model. As such, the model will specify that TST_o for person *j* on day *i* is a function of: average TST_o, linear time, a between-persons random error term, and a within-person random residual component. In step 3 (Table 5-7, Row 3) the estimates of the fixed and random intercepts and fixed linear slopes for each sleep variable will be added. Thus, the daily TST_o for each person will be predicted by: average level of $WASO_{sm}$, linear time, the between-person effects of mean-level $WASO_{sm}$, the within-person effects of $WASO_{sm}$, a between-person random error term, and a within-person random residual component. In step 4

(Table 5-7, Row 4) the estimates of the fixed and random intercepts and fixed linear slopes for SOL_{sm} will be added. Thus, the TST_o for each person will be predicted by: average level of TST_o, linear time, the between-person effects of mean-level SOL_{sm} aned $WASO_{sm}$, the within-person effects of daily-centered SOL_{sm} and $WASO_{sm}$, a between-person random error term, and a within-person random residual component.

All models will be estimated using the Maximum Likelihood (ML) method. The ability of a model to predict TST_o better than the baseline model (i.e., Deviance) will be used as an index of Goodness of Fit. Improvements in predictability will be determined by the amount of reduction of within- and between-person residual variances compared to the baseline model (Bryk & Raudenbush, 1992). Decreases in residual and intercept variances represent a proportional reduction of the prediction error, which is analogous to R^2, and will be used as an estimates of within and between-person effect sizes. The amount of agreement between model predicted values and actual values will be calculated as an estimate of an overall effect size.

CHAPTER 5
RESULTS

The main objective of the study was to better understand sleep misperception by investigating SOL_{sm} and $WASO_{sm}$ in a sample of community dwelling older adults. This objective was achieved by examining daily values and biweekly averages of SOL_{sm} and $WASO_{sm}$ in relation to1) the amount of within- to between-person variability, 2) the amount of within- to between-person variability by sleep complaint status, 3) each other, and 4) objective total sleep time (TST_o). The main objective was achieved through these four aforementioned specific aims. Results will be discussed separately for each aim.

Specific Aim 1

The first specific aim was to investigate SOL_{sm} and $WASO_{sm}$ across two weeks to determine the amount of within- to between-person variability in SOL_{sm} and $WASO_{sm}$. Intraindividual variability (IIV) analyses were used to determine the relative amount of within- to between-person variability in SOL_{sm} and $WASO_{sm}$. Dissection of SOL_{sm} and $WASO_{sm}$ variables revealed a considerable amount of within- to between-person variability suggesting that individuals are highly variable in sleep misperception across two weeks. There was greater within-person variance in participant's SOL_{sm} than between-person variance, and almost as much within-person variance in participant's $WASO_{sm}$ as between-persons. Specifically, SOL_{sm} was found to vary within-persons over 150% as much as it varies between-persons and within-persons $WASO_{sm}$ displayed 95% of the amount of between-person variability. For a listing of the amount of within-person variability compared to between-person variability, see Table 5-1. For a graphical depiction of the relative amount of within-person to between-person variability, see Figure 5-1.

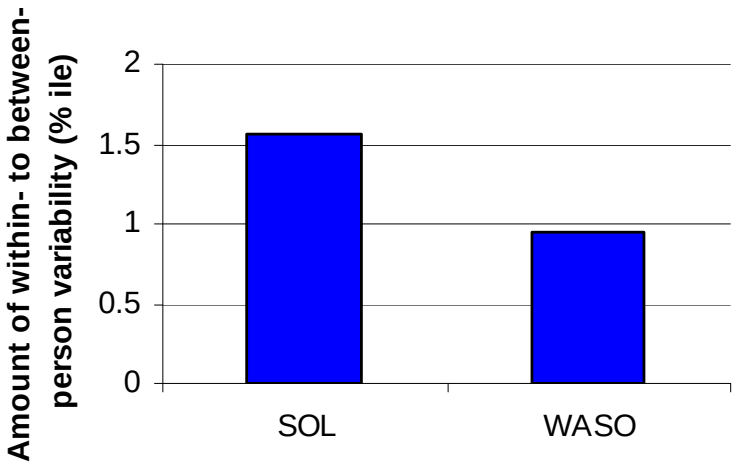

Figure 5-1. Relative amount of within-person variability compared to between-person variability after controlling for any linear or quadratic effects of time.

Specific Aim 2

The second specific aim was to determine the amount of within- to between-person variability in SOL_{sm} and $WASO_{sm}$ by sleep complaint status. Intraindividual variability analyses were used to determine the relative amount if within- to between-person variability in SOL_{sm} and $WASO_{sm}$ within each of the sleep groups (i.e., sleep complaining and sleep non-complaining). As compared to other individuals with the same complaint status, participants were nearly as, and generally more variable within-persons as compared to between-persons in SOL_{sm} and $WASO_{sm}$. Both the sleep non-complaining and complaining groups had large amounts of within-person variability in their sleep estimates as compared to between-person variance in sleep estimates. However, there was great variability in the complainers in both SM variables (Figure 5-2).

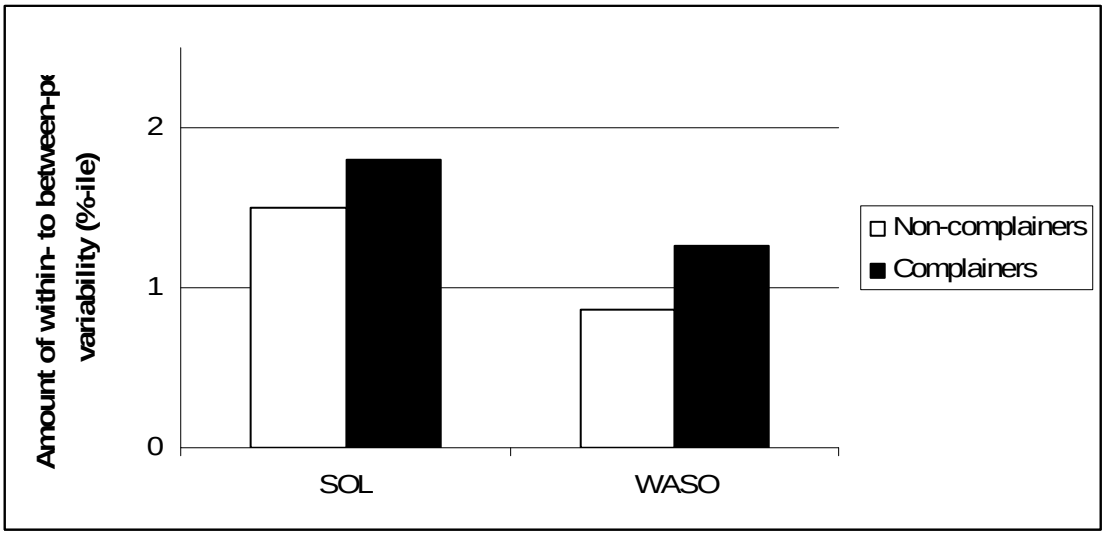

Figure 5-2. Relative amount of within-person variability compared to between-person variability after controlling for any linear or quadratic effects of time.

Specific Aim 3

Aim 3 was to investigate the relationship between day-to-day fluctuations in SOL_{sm} and $WASO_{sm}$. A multilevel modeling (MLM) approach was used to predict within-person and between-person relationships between SOL_{sm} and $WASO_{sm}$. Because the self-reported perception of SOL and WASO were recorded by the participants at the same time each day, and because the experience of either could impact the perception of the other while reporting, determining the relationship of SOLsm and WASOsm requires that two models be built: one using SOLsm to predict WASOsm and the other using WASOsm to predict SOLsm.

Multilevel Model for $WASO_{sm}$

The intraclass correlation coefficient (ICC), which serves as an index of the amount of within- and between-person variability to be explained (Bryk & Raudenbush, 1992), was 0.41. This indicates that, 41% of the overall variability in $WASO_{sm}$ was within-person and 59% was between-person. For a complete listing of model parameters and estimates obtained at each step of the model building process, see Table 5-3.

In the final MLM for $WASO_{sm}$, SOL_{sm} was a significant between-person, level 2, predictor, $\beta = 0.62$, $t(101.43) = 3.29$, $p < .01$. At the within-person level, level 1, the predictor Day, $\beta = -0.03$, $t(129.50) = -3.11$, $p < .05$, was significant. There was a nonsignificant trend at the within-person level, level 1, for the predictor SOL_{sm}, $\beta = -0.10$, $t(91.09) = -1.78$, $p = .08$. The model also contained a significant random effect of SOL_{sm}, $\beta = 0.13$, *Wald's Z* = 3.83, $p < .01$. This model explained approximately 10% of the within-person variance and 20% of the between-person variance. See Table 5-4 for a total listing of predictor estimates and significance levels.

Multilevel Model for SOL_{sm}

The intraclass correlation coefficient (ICC), which serves as an index of the amount of within- and between-person variability to be explained (Bryk & Raudenbush, 1992), was 0.17. This indicates that, 83% of the overall variability in SOL_{sm} was within-person and 17% was between-person. For a complete listing of model parameters and estimates obtained at each step of the model building process, see Table 5-5.

In the final MLM for SOL_{sm}, $WASO_{sm}$ was significant between-person, level 2, predictor, $\beta = 0.21$, $t(98.09) = 3.98$, $p = .01$. At the within-person level, level 1, the predictors Day, $\beta = -0.74$, $t(204.24) = -3.47$, $p < .01$, and $WASO_{sm}$, $\beta = -0.1$, $t(87.59) = -2.01$, $p < .05$, were significant. The model also contained a significant random effect of $WASO_{sm}$, $\beta = 0.13$, *Wald's Z* = 4.02, $p = .01$. This model explained approximately 8% of the within-person variance and 17% of the between-person variance. See Table 5-6 for a total listing of predictor estimates and significance levels.

Specific Aim 4

Aim 4 was to determine the intraindividual daily relationship between SOLsm and WASOsm to TSTo. Multilevel modeling (MLM) was used to predict within-person and between-person relationships of SOLsm and WASOsm to TSTo.

Multicollinearity

Prior to running the multilevel models to predict TST_o within-person and between-person correlations were run between $WASO_{sm}$ and SOL_{sm} variables to determine the extent to which they shared variances. Formal multicollinearity diagnostic procedures are not available for multilevel modeling. However, aim three of this study showed that $WASO_{sm}$ and SOL_{sm} are collinear. Therefore, to control for their collinear relationship this diagnostic procedure will extract the shared variance from of SOL_{sm} from $WASO_{sm}$ so that when they are individually added to the MLM the significant level will represent the unique contribution of each variable to the model.

Within-Person Multicollinearity

The within-person correlational analysis revealed significant collinearity between SOL_{sm} and $WASO_{sm}$ ($r = 0.-14$, $p < .05$).

Between-Person Multicollinearity

The between-person correlational analysis revealed significant collinearity between SOL_{sm} and $WASO_{sm}$ ($r = 0.44$, $p < .01$).

Total Sleep Time (TST_o)

The intraclass correlation coefficient (ICC) for TST_o is 0.51. This indicates that, 49% of the overall variability in TST_o was within-person and 51% was between-person. For a complete listing of model parameters and estimates obtained at each step of the model building process see Table 5-7.

In the final MLM for TST_o, Day, β = -0.74, $t(111.74) = -1.99$, $p < .05$, $WASO_{sm}$,β = -0.15, $t(49.77) = -2.47$, $p = .02$, and SOL_{sm}, β = 0.32, $t(67.74) = 5.02$, $p < .001$, were significant within-person, level 1, predictors. At the between-person level, level 2, SOL_{sm}, β = 0.67, $t(98.18) = 2.19$, $p = .03$, was the only significant predictor of average TST_o. The model also contained a significant random effect of Day, β = 2502.51, *Wald's Z* = 6.67, $p < .001$, $WASO_{sm}$, β = 0.11, *Wald's Z* = 2.13, $p = .03$, and SOL_{sm}, β = 0.10, *Wald's Z* = 2.41, $p = .02$.

This model explained approximately 11% of the within-person variance and 16% of the between-person variance. See Table 5-8 for a total listing of predictor estimates and significance levels.

Table 5-1. Amount of within- and between-person variability

Variable	Sample standard deviation (SD)	Individual standard deviation (ISD)	ISD / SD
SOL_{sm}	16.48	25.74	1.56
$WASO_{sm}$	29.89	28.42	0.95

Table 5-2. Amount of within- and between-person variability by complaint status.

Complaint Status	Variable	Sample Standard Deviation (SD)	Individual Standard Deviation (ISD)	ISD / SD
No	SOL_{sm}	23.76	35.51	1.49
	$WASO_{sm}$	26.27	22.8	0.87
Yes	SOL_{sm}	12.17	21.90	1.80
	$WASO_{sm}$	34.29	43.28	1.26

Table 5-3. Steps taken in building the $WASO_{sm}$ multilevel model

	$WASO_{sm}$									
Models	AIC	BIC	-2LL	Δ-2LL	*df*	Δ*df*	s^2_b	s^2_w	r^2_b	r^2_w
(1) Null	4954.14	4969.80	4948.14	--	101.42	--	1.31	1.83	--	--
(2) Time added	4938.83	4964.93	4928.83	19.31**	136.68	35.26	1.29	1.75	.02	.04
(3) SOL_{sm} added	4811.11	4868.87	4811.11	117.7***	102.48	-32.20	1.15	1.52	.11	-.13

Notes: AIC = Akaike's Information Criterion; BIC = Schwarz's Bayesian Criterion; -2LL = -2 log likelihood; Δ-2LL = change in –2LL relative to preceding model; s^2_b = unexplained intercept-related (between subjects) variance; s^2_w = unexplained residual-related (within subjects) variance; r^2_b = between-subjects pseudo R-squared, an estimate of the amount of between subjects variance (estimated from null model) explained by fixed and random predictors; r^2_w = within-subjects pseudo R-squared, an estimate of the amount of within subjects variance (estimated from null model) explained by fixed and random predictors. *** Deviance is significant at the 0.001 level. ** Deviance is significant at the 0.01 level. * Deviance is significant at the 0.05 level.

Table 5-4. The relationship between SOL_{sm} and $WASO_{sm}$

Fixed effects					
Predictor variable	*B*	*SE*	*df*	*t*	*p*
Within-person					
Day	-0.03	0.01	129.50	-3.11	<.01
SOL_{sm}	-0.10	0.06	91.09	-1.78	.08
Between-person					
SOL_{sm}	0.62	0.19	101.43	3.29	<.01
Random effects					
Covariance parameter estimate	*B*	*SE*		*Z*	*p*
Within-person					
Day	<0.01	<0.01		2.42	<.05
SOL_{sm}	0.13	0.03		3.83	<.01
				Within pseudo R^2	-.13
				Between pseudo R^2	.11

Notes: Variables with a subscript ‘sm‘ indicate they were calculated by subtracting the subjective Seep Diary measure from the respective variable measured by objective Actigraphy.

Table 5-5. Steps taken in building the $WASO_{sm}$ multilevel model

	$WASO_{sm}$									
Models	AIC	BIC	-2LL	Δ-2LL	*df*	Δ*df*	s^2_b	s^2_w	r^2_b	r^2_w
(1) Null	14062.79	14084.86	14062.79	--	103.00	--	198.82	1007.27	--	--
(2) Time added	14061.61	14087.93	14051.61	11.18	227.01	124.01	173.36	981.60	.13	-.03
(3) SOL_{sm} added	13342.36	13384.12	13326.36	725.25***	172.55	54.46	182.45	839.37	.17	-.08

Notes: AIC = Akaike's Information Criterion; BIC = Schwarz's Bayesian Criterion; -2LL = -2 log likelihood; Δ-2LL = change in –2LL relative to preceding model; s^2_b = unexplained intercept-related (between subjects) variance; s^2_w = unexplained residual-related (within subjects) variance; r^2_b = between-subjects pseudo R-squared, an estimate of the amount of between subjects variance (estimated from null model) explained by fixed and random predictors; r^2_w = within-subjects pseudo R-squared, an estimate of the amount of within subjects variance (estimated from null model) explained by fixed and random predictors. *** Deviance is significant at the 0.001 level. ** Deviance is significant at the 0.01 level. * Deviance is significant at the 0.05 level.

Table 5-6. The relationship between SOL_{sm} and $WASO_{sm}$

Fixed effects					
Predictor variable	*B*	*SE*	*df*	*t*	*p*
Within-person					
Day	-0.74	0.21	204.24	-3.47	<.01
SOL_{sm}	-0.10	0.05	87.59	-2.01	<.05
Between-person					
SOL_{sm}	0.21	0.05	98.09	3.98	<.01
Random effects					
Covariance parameter estimate	*B*	*SE*		*Z*	*p*
Within-person					
Day	0.47	0.45		1.06	.29
SOL_{sm}	0.13	0.03		4.02	<.01
				Within pseudo R^2	-.08
				Between pseudo R^2	.17

Notes: Variables with a subscript ‘sm‘ indicate they were calculated by subtracting the subjective Seep Diary measure from the respective variable measured by objective Actigraphy.

Table 5-7. Steps taken in building the TST_o multilevel model

					TST_o					
Models	AIC	BIC	-2LL	Δ-2LL	*df*	Δ*df*	s^2_b	s^2_w	r^2_b	r^2_w
(1) Null	15315.60	15331.37	15309.60	--	102.03	--	2401.47	2522.99	--	--
(2) Time added	15301.92	15328.20	15291.92	17.68	124.40	22.37	2317.90	2316.36	.08	.03
(3) $WASO_{sm}$ added	14763.58	14805.34	14747.58	544.34***	110.82	13.58	2203.65	2370.94	.06	.08
(4) SOL_{sm} added	14680.95	14738.36	14658.95	88.63	110.16	0.66	2021.14	2252.68	.11	.16

Notes: AIC = Akaike's Information Criterion; BIC = Schwarz's Bayesian Criterion; -2LL = -2 log likelihood; Δ-2LL = change in –2LL relative to preceding model; s^2_b = unexplained intercept-related (between subjects) variance; s^2_w = unexplained residual-related (within subjects) variance; r^2_b = between-subjects pseudo R-squared, an estimate of the amount of between subjects variance (estimated from null model) explained by fixed and random predictors; r^2_w = within-subjects pseudo R-squared, an estimate of the amount of within subjects variance (estimated from null model) explained by fixed and random predictors. *** Deviance is significant at the 0.001 level. ** Deviance is significant at the 0.01 level. * Deviance is significant at the 0.05 level.

Table 5-8. Sleep misperception variables predicting TST_o

Fixed effects					
Predictor variable	*B*	*SE*	*df*	*t*	*p*
Within-person					
Day	-0.74	0.37	111.74	-1.99	<.05
$WASO_{sm}$	-0.15	0.06	49.77	-2.47	.02
SOL_{sm}	0.32	0.06	67.74	5.02	<.01
Between-person					
$WASO_{sm}$	0.09	0.18	98.58	0.51	.61
SOL_{sm}	0.67	0.31	98.18	2.19	.03
Random effects					
Covariance parameter estimate	*B*	*SE*		*Z*	*p*
Within-person					
Day	2502.51	374.96		6.67	.01
$WASO_{sm}$	0.11	0.05		2.13	.03
SOL_{sm}	0.10	0.04		2.41	.02
			Within pseudoR^2		.11
			Between pseudoR^2		.16

Notes: Variables with a subscript ‘sm‘ indicate they were calculated by subtracting the subjective Seep Diary measure from the respective variable measured by objective Actigraphy. The Subscript 'o' indicates the variable was measured by objective Actigraphy.

CHAPTER 6
DISCUSSION

Review of Findings

Aim 1 and 2

Sleep misperception is common but highly variable among older adults. The finding that older adults are almost as variable in $WASO_{sm}$ (ISD/SD = .95) and more variable in SOL_{sm} (ISD/SD = 1.56) within-persons as between-persons is noteworthy, because it conflicts with the traditional view that sleep misperception is a consistent trait like behavior isolated to patients with insomnia. Indeed, an ISD/SD value of 1.56 means that 85% of the total variance of the variable SOL_{sm} across two weeks is found within-persons, and an ISD/SD value of .95 means that nearly 50% of the total variance in the variable $WASO_{sm}$ was within-persons. While, standard statistical techniques largely disregard within-person variability as error, herein it is argued that large amounts of within-person variability relative to between-person variability (i.e., ISD/SD > .50) may be meaningful and predictable.

Results from Aim 2 provide further evidence that sleep misperception is not best explained by a between-persons phenomenon in older adults. Some researchers argue that sleep misperception found among insomnia patients is limited to a subgroup making up about 25% of individuals with insomnia. From this perspective, that sleep misperception is limited to a subgroup of insomnia patients, we would expect to find larger amounts of between-persons variability than within-persons in a sample of individuals with sleep complaint. However, this is not what was found in this study. In fact, older adults with sleep compliant had between 65% and 75% more within-persons variability than between-persons variability in the two sleep misperception variables tested in this study (i.e., SOL_{sm} and $WASO_{sm}$, respectively). These findings supplement previous findings from our lab in which we found that the number of days

overestimated SOL and WASO only occurred on average about 7 and 4 days respectively among older adult sleep complainers (Kay & McCrae, 2008). In addition, these results help explain the findings of a previous single night study which found that insomnia patients where just as likely to over- as under-estimate SOL (Vanable et al., 2000). The results of the present study suggest that the probability of older adults with insomnia over- or under-estimating may be better predicted by within-person variability than by between-persons variability. Because on any given day individuals with sleep complaint are as likely to over-estimate SOL as under-estimate it, investigating the temporal or day-to-day factors that fluctuate with sleep misperception may yield more valuable information about the cause of this phenomenon than investigating the more tonic factors such as personality or diagnostic categorization. Ultimately, studies that rely on a single night and averaging methods may not appropriately represent the construct of sleep misperception and certainly are not adequate to describe the longitudinal patterns of sleep misperception. The validity of sleep state misperception as a categorical diagnostic categorization may have limited validity among older adults.

Aim 3

Though not previously validated, the convention of combining average SOL_{sm} and $WASO_{sm}$ seems to be supported by this study, because SOL_{sm} and $WASO_{sm}$ co-occured on average. Since there was no interaction between complaint status and the between-persons relationship between SOL_{sm} and $WASO_{sm}$, we can conclude that regardless of whether there is a complaint or not, individual sleep misperceptions are related on average. However, SOL_{sm} and $WASO_{sm}$ do not necessarily co-occur on the same night. This finding, that the SOL_{sm} and $WASO_{sm}$ shared a negative relationship on the daily level, adds to the apparent discrepancy in findings between Mercer et al, (2002) who found that sleep misperceptions occurred during researcher probe throughout the night and Coates et al. (1987) who found that sleep

misperception occurred only in the early part of the night and during spontaneous but not researcher induced awakenings later in the night. The major difference between these two studies is the location and the induction of arousal that may be associated with the laboratory as compared to the home environment. The former occurred in the laboratory while the later occurred in the home environment. Those individuals who where probed in the laboratory may have been more aroused throughout the night than those in the home environment.

Because SOL_{sm} and $WASO_{sm}$ did not co-occur daily, the suggestion made by Perlis et al. (2001) that sleep misperception cannot be reduced to a general pessimistic bias imposed consistently on all aspects of sleep by individuals with sleep complaint is strengthened. Sleep misperception does not occur every day among sleep complainers, and SOL_{sm} and $WASO_{sm}$ do not fluctuate in the same direction night-to-night. In an additional analysis, sleep complaint was added as an additional predictor variable and the relationship between SOL_{sm} and $WASO_{sm}$ was not found to be different by complaint status. Thus, sleep misperception may not be fundamentally different among sleep complainers and non-complainers beyond the frequency/severity.

Aim 4

This analysis revealed a complex relationship between sleep misperception and TST_o. Specifically, SOL_{sm} was significantly related to longer TST_o, while $WASO_{sm}$ was significantly related to shorter TST_o. One explanation for the directional difference in SOL_{sm} and $WASO_{sm}$ on TST_o is that $WASO_{sm}$ is related to a more pervasive problem of arousal leading to earlier wake times and/or reduced sleep during the night while SOL_{sm} may be related to a more phasic arousal problem occurring primarily during the sleep onset period. Vanable et al. (2000) argued that poorer sleep quality may motivate individuals to exaggerate their sleep problems, thus explaining sleep misperception. Contrary to their theory, this analysis clearly suggests that when

individual's TST_o is greater than usual, they are more likely to overestimate SOL_{sm} and when their sleep is shorter than usual, they are more likely to overestimate $WASO_{sm}$, regardless of complaint status. We argue that SOL_{sm} may represent a form of localized sleep deprivation that reduces the likelihood of $WASO_{sm}$ and extends the length of sleep, similar to the effects of global sleep deprivation.

Summary of Results

These analyses suggest that though sleep misperception is common among older adults, it is not limited to individuals with sleep complaint. Identification of sleep perceiver subtypes of insomnia may be the result of limited observations or averaging techniques that overlook individual's highly variable sleep behavior. The common view that sleep misperception represents an exaggeration bias does not have support. This study suggests that older adults with sleep misperception follow patterns more similar to sleep deprivation than exaggeration. The validity of sleep misperception as a distinct sleep disorder that is quantitatively different in patterns among insomnia patients and normal sleepers should be reviewed. Additional studies are needed to quantify the intraindividual variability of sleep misperceptions in diagnosed insomnia and sleep state misperception patients.

Study Limitations

This secondary analysis study was based on a convenience sample and not a scientific experimental study. Limitations of the present study include restricted generalizability of results. The results of this study may not generalize beyond community dwelling older adults. There are other methodological concerns as well. The use of a convenience sample restricted the diversity of the sample. The participants were primarily European Caucasian, and college educated. The homogeneity of the sample prevents reliable generalization to younger or diverse populations. Additionally, individuals were excluded from the study if they presented with sleep disorders

other than insomnia (e.g. sleep apnea, periodic leg movements). Approximately one-half of the elderly population experience one or both of these conditions (Ancoli-Israel, Kripke, Mason, & Kaplan, 1985).

A New Model of Sleep Misperception

One prominent theory of sleep argues that there are two major sleep systems, or processes (Borbely, 1982). The circadian or C-process is an endogenous process imposing sleep on the brain based on species specific biological rhythms while the S-process or homeostatic process progressively imposes sleep on the brain proportional to the amount of previous wakefulness. These systems function on both global and local levels of brain functioning. However, the body/mind and sleep/wake false dichotomies complicate the discussion of sleep misperception. First, the idea that the mind is orthogonal from what occurs in the brain is scientifically invalid. All mental events correspond to brain events. However, to reduce the mind to the sum of the parts of the brain is equally, though philosophically, invalid. From the perspective of the brain, for example, there is no meaning in the perception of sleep as wakefulness. This false dichotomy is apparent in delineations of cognitive arousal as a function of mental arousal while physiological arousal is supposed to be a function of body arousal. However, cognitive arousal corresponds to activation of neurons in executive centers of the brain while physiological arousal corresponds to activation of the descending sympathetic nervous system in the brain. Both are physiological and both occur in distant, though interconnected, parts of the brain (discussed in detail below). All that matters in relationship to sleep misperception is whether the source of arousal activates consciousness centers of the brain while global sleep has set in.

Second, sleep and wake are not dichotomous global state of brain, physiology, or consciousness. The sleeping brain is dynamic and never in a ubiquitous pattern of neuroactivity in all areas of the brain at any time (Roth, Achermann, & Borbely, 1999; Werth, Achermann, &

Borbely, 1997). Though the use of EEG has become the 'gold standard' for determining sleep onset, early sleep researchers warned against the sole use of EEG as a measure of sleep onset (Kleitman, 1963). Physiologically, transitions between sleep and wake occur on a continuum in space and time with no clear either/or. While arbitrary cutoffs have been entrenched in sleep research, these delineations are arbitrary by all accounts (Tryon, 2004). In healthy young populations, self-report measures of sleep onset tend to occur later than EEG defined sleep and EEG defined sleep tends to occur later than behavioral indices of sleep (Tryon, 2004) and currently there is no evidence that this pattern holds in diverse patient populations. Based on the localized model of sleep, it is entirely possible for this typical pattern to occur in any order. Moreover, even though EEG definitions of sleep were created based on behavioral correlates, EEG definitions of sleep and behavioral indices often disagree. One study found that dogs given atropine resulted in EEG defined sleep while the dog was clearly awake and active (Wikler, 1952). Certain brain damage in humans has been shown to induce EEG defined sleep while the individuals are observably awake (Hamoen, 1954 cited in (Hauri & Olmstead, 1983). Actigraphy may be a valid indication that the afferent motor centers of the brain have gone to sleep, while EEG may indicate that the neocortex has fallen asleep, and self-report may indicate the point in time that the consciences centers of the brain have fallen asleep. People with sleep misperception may have a longer transition period with a unique brain dynamic unlike healthy sleep onset such that behavioral and EEG based indices reflect a sleep state while the persons cognitive centers of the brain remain awake and the person remains aware of self and time.

The idea that consciousness "switches off" at sleep onset is as untenable as the idea, almost ubiquitously held (Saper, Chou, & Scammell, 2001), that there is a "sleep switch" inducing the dichotomy between sleep and wake. Nonetheless, assumptions, highlighted in the sleep switch

theory remain highly accepted and popular among sleep experts. Levels of conscious awareness, attention to and processing of the environment, similar to process observed in wakefulness, are on a continuum which corresponds to the level and depth of sleep of areas of the brain involved (Czisch et al., 2002). For example, during sleep some level of attention is attuned to external stimuli and depending on the context of stimuli sleep will continue or terminate. Specifically, the participants' name induced awakenings more successfully than a neutral stimulus (Perrin, Garcia-Larrea, Mauguiere, & Bastuji, 1999). Explicit to sleep misperception, between 27 and 50% of healthy sleepers reported not having slept minutes after sleep spindles emerged and between 10 and 5% of these individuals reported wakefulness during stage 2 and stages 3-4 sleep, respectively (reviewed in Bonnet & Moore, 1982). The hypothalamic sleep switch theory must be reconsidered and may more appropriately be called the hypothalamic sleep promoting theory that interacts with other sleep promoting and arousal systems in the brain. Even early sleep researchers acknowledged that cogitation occurs during sleep (Mullin, 1938). Unfortunately, contemporary sleep experts seem unable to embrace the idea that the incongruence between a participant's self-reported experience and EEG defined states is the product of limited recording resolution. Evidence suggests that EEG is unable to measure the physiological state of the brain areas involved in consciousness. As Sewitch (1984) argued,

> When a sleeper is awakened at some point during the night and asked to evaluate his or her subjective state prior to the awakening, he or she is confronted with a decision between two alternatives, sleep, or wakefulness. Consequently, it seems appropriate to consider the issue of perceiving sleep and wakefulness in the context of a decision problem. There are three elements essential to any decision problem. [One of these essential elements is that] there must be two 'states of the world' and in the case of deciding about one's subjective state during the sleep period, those two states are wakefulness versus sleep. (p. 244-245)

In fact, sleep and wake are not dichotomous; therefore, the decision need not be wake vs. sleep. Although teaching patients how to judge what the EEG defines as sleep is effective in treating sleep misperception (Downey & Bonnet, 1992), teaching patients to accurately report

their experience in terms of EEG cutoffs does not necessarily inform why they needed this training in the first place, nor does it necessarily mean that their experience has substantively changed. Ignoring the patient self-reported state may prevent the best treatments for sleep misperception from being formed. This technique may indirectly improve insomnia by teaching patients that most of the brain is obtaining restful sleep, thus reducing anxiety. However, it probably does not directly mean that the whole brain, including the perceptual areas of the brain, is receiving healthy sleep.

Implications for Sleep Research, Diagnoses, and Treatments

It is argued that sleep misperceptions may result in two ways. First, simultaneously high activation in the sleep promoting and arousal systems of the brain would allow self-awareness to occur during lighter stages of sleep. Second, low localized sleep drive in consciousness centers of the brain in the presence of high global sleep drive may allow isolated areas to remain active during EEG defined sleep. These two possible pathways to sleep misperception while having a similar symptoms (perception of EEG defined sleep as wakefulness), may be different in their consequences. The former would induce maintained activation in only isolated areas of the brain, representing a form of localized sleep deprivation in only those affected areas of the brain, while the later would be related to a more pervasive flattening of the S-process in localized areas of the brain. The treatment of sleep misperception resulting from hyperarousal may employ relaxation techniques. Conversely, to treat sleep misperception related to a localized S-process deficiency may involve enhancing daytime activation of affected brain areas. This would force sleep dept to accumulate in those areas, thus, enhancing sleep in those areas of the brain and consolidating their sleep processes with the whole brain's sleep/wake pattern. In kind, the mechanisms of insomnia onset may come through either of these forms, suggesting that at least two subtypes of insomnia seem logical: the first resulting from hyperarousal, the other from a S-process

deficiency. It is time that researchers begin to focus more on the mechanisms of insomnia to determine its subtypes, rather than exclusively studying diagnostic categories developed from clinically observed symptom constellations.

Sleep misperception has been at the center of the sleep disorder debate. The validity of all insomnia studied and the diagnosis of insomnia itself was at one time considered to be threatened by the discovery of sleep misperception (Borkovec et al., 1979). Indeed, one may wonder, "What if there are no global sleep problems among insomnia patients?" or "What will become of all the studies that include a large subgroup of participants, as many as half all insomnia patients, who do not have global sleep deficiencies?" Since that time, sleep state misperception has been accepted by most researchers and clinicians. Moreover, its systematic validation has become a fixed pursuit of the field. Regrettably, these fears seem to have led the field to embrace a system of diagnostic categories that require retrospective validation at the expense of scientific exploration and prospective progress in understanding insomnia and its relationship to sleep misperception. One explanation offered by Borkovec (1981) that should be reconsidered is "that insomnia patients may….base their evaluation of sleep on a different set of internal and/or external cues relative to good sleepers" (p.609). A slight modification to this statement may be more accurate: "that [hyperaroused individuals with or without a sleep complaint] may base their evaluation of sleep on a different set of internal and/or external cues relative to [individuals that are not hyperaroused]." As sleep experts, and society as a whole, continue to define sleep as an either/or state, individuals who experience sleep misperception may have difficulty determining which category their subjective experience belongs (Lamarche & Ogilvie, 1997) since it may not fit in either. The validity of sleep state misperception as a distinct disorder has little support. However, identification of sleep misperception in a patient should receive treatment even if

objective measures are absent, because sleep misperception may be effectively treated. Unlike Borkovek (1981) who once argued that the presence of localized arousal events may require "a revision of the EEG criteria for sleep, at least for some clinical populations" (p608), we conclude that altering the criteria of a global measure based on a localized event may not adequately capture the localized event. That is, this approach may either over- or under-approximate the impact of localized activity on a global scale. The results of this study shows that there is much knowledge that can be gained by determining the amount of discrepancy between the perception of sleep onset and the EEG global assessment on sleep onset on both the daily level and the within-persons level.

Treatment implications

Sleep medicine has made dramatic headway in the diagnosis and treatment of sleep problems. Yet, the development of a universally accepted definition of insomnia remains illusive. Moreover, many insomnia treatments employ a "shotgun" approach, that is, giving multiple interventions and hoping one works. Often this approach is effective; however, in some cases it is not as efficient or effective as desired. Sleep medicine is at the point where understanding the pathophysiology of insomnia, and sleep in disorders highly related to insomnia more generally, is required so that future treatments can be developed (Dang-Vu et al., 2007; Mahowald & Schenck, 2005). Most importantly, sleep research and medicine must accept that sleep is not a whole brain event (Krueger & Obal, 2003). In addition, regional abnormalities in sleep architecture need to be better understood through research, and specifically targeted in treatment. More importantly, localized sleep abnormalities, in the form of extended activation during sleep, can be couched in the framework of localized sleep deprivation. Like global sleep deprivation, localized sleep deprivation may predispose individuals to a host of mental and physical health problems. Given the hypothesis that isolated areas of the brain remain active, or

are prevented from sleeping deeply with the rest of the brain during sleep misperception, these areas may be subjected to a form of localized sleep deprivation. Like global sleep deprivation (Durmer & Dinges, 2005), localized sleep deprivation may predispose individuals to problems related to the areas of the brain that are affected in sleep misperception. Moreover, sleep misperception may explain the high comorbidity rates of hyperarousal related health conditions and insomnia. For example, between 50-70% of individuals with chronic pain commonly report sleep problems (Pilowsky, Crettenden, & Townley, 1985). The high co-morbidity between these health problems hints at a common pathophysiology, but the relationship between these conditions remains undetermined.

There may be three potential factors related to sleep misperception. If the combination of these factors is better understood new treatments may be developed to improve sleep locally and, thus, prevent or improve potential problems. For example, if there is a localized S-process deficiency in only the prefrontal cortex, a treatment such as biofeedback may be developed to enhance the sleep need of that area. In another situation, if there is increased arousal in the anterior cingulated, new treatments may be able to reduce localized arousal behaviorally, allowing the individual to sleep more deeply at night with the rest of the brain. Thus, both potentially eliminating sleep misperception and localized sleep deprivation.

Future Directions

This study highlights the needed and utility of IIV and MLM analyses in sleep research. Sleep researchers need to look beyond single night and averaging research methods in sleep research broadly and sleep misperception research specifically. Sleep misperception, as with insomnia, appears to be periodic rather than consistent among sleep complainers, not only from night-to-night but also throughout the night. Moreover, it is interesting that over two weeks average SOL_{sm} correlated with average $WASO_{sm}$, yet, this was not true on a nightly basis.

Additional studies are needed to replicate these findings in younger populations and in diagnostic groups. More importantly, neuroimaging studies that combine functional/structural imaging, PSG measures, and self-report in concert are needed to uncover the biopsychophysiological mechanism of sleep misperceptions.

Regional sleep theories argue that sleep is not a whole brain event, but that each region of the brain has its own sleep patterns and needs (Kattler, Dijk, & Borbely, 1994; Krueger & Obal, 1993, 2003; Krueger, Obal, & Fang, 1999; Lavie, 1993; Mahowald & Schenck, 1992; Mukhametov, Supin, & Poliakova, 1984; Vyazovskiy, Borbely, & Tobler, 2000; Vyazovskiy, Borbely, & Tobler, 2002; Yasuda, Yasuda, Brown, & Krueger, 2005; Yoshida et al., 2004). Sleep and wakefulness are not dichotomous global states. Although there is ample evidence of this, few have embraced this idea and integrated it into how they think about and do sleep research. Indeed, this idea has been limited to basic animal research and theory (Krueger & Obal, 2003; Mahowald & Schenck, 1992), few healthy human studies (Czisch et al., 2004; Kattler et al., 1994; Kaufmann et al., 2006; Maquet, 2000; Wehrle et al., 2005), and recently, a handful of neuroimaging studies of selected sleep disorders. Several marine mammals and bird species provide evidence that sleep and its functions can occur in isolated areas of the brain simultaneously while other areas continue to perform wake-like brainwaves and functions, such as attending to the environment (Mukhametov, Supin, & Poliakova, 1984). Even in humans, isolated events of activity indicative of wakefulness occur during sleep (Dement & Kleitman, 1957) and isolated events of slow wave activity indicative of sleep occur during wakefulness (Durmer & Dinges, 2005). Both of these scenarios are far more common than realized. When one considers that the brain is a complex organ and is never in a ubiquitous state of identical activity in all areas of the brain during wakefulness, it seems odd that sleep is treated as a whole

brain event. For example, most EEG sleep research relies on as few as one EEG electrode that provides summations of brain wave activity, particularly activity occurring on the surface of the cortex. While few would dispute that daytime brain activity is localized during wakefulness, even fewer have considered that such localization can occur during sleep. Indeed, EEG research with multiple leads (Roth et al., 1999) and neuroimaging studies support this hypothesis (Kaufmann et al., 2006). It is clear that there is a continuum of cumulative global states between sleep/wake stages (Kaufmann et al., 2006). Furthermore, given that sleep is not a whole brain event, there is little support for the idea that sleep can only occur in the complete absence of consciousness (Mercer et al., 2002). Consciousness during sleep would result if areas of the brain responsible for consciousness remain active while other areas deactivate into slow wave activity. The prefrontal cortex, and the anterior cingulate cortex (Bush, Luu, & Posner, 2000) have been posited as the seat of consciousness. Given that sleep misperception is the subjective perception of wakefulness during sleep, undetectable to most EEG procedures (i.e., sleep misperception), may represent extended microarousal in these consciousness centers during nonrapid eye movement sleep (NREMs). Sleep misperception may represent a form of localized sleep deprivation resulting in the dysregulation and dysfunction in these isolated and related areas of the brain. This may be the source of sleep disorders and mental and physical health problems. Specifically, we posit that sleep misperception is the result of concurrent activation of both the sleep system (forcing EEG defined sleep to occur) and the ascending arousal system preventing selected areas from sleeping with the rest of the brain. While a microstructure indication of localized activation in sleep misperception has been identified employing EEG (Mercer et al., 2002) the location of this sleep problem has not been studied to our knowledge. Cumulative global states are simply the amalgamation of diverse brain functions and physiological activity.

The goal of research in the future should be aimed at defining the mechanisms and factors involved that predict the days when sleep misperception will occur or not. These studies may help determine whether arousal directly or indirectly is related to sleep misperception and how a local of global S-process deficiency may be involved. This line of research may finally be able to confirm or refute the uncertainty of Borkovec (1981) when he said, "The experience of wakefulness during sleep is indeed a remarkable phenomenon. Although the effects may be ultimately determined to be due to artifact or gross EEG definitions of sleep, its elucidation should contribute to an understanding of some of the contributors to the experience of insomnia" (p 609). The findings from the present study contribute to decades of research aimed at understanding sleep misperceptions role in insomnia and may be the beginning steps in a new avenue of insight to this problem.

APPENDIX A
SLEEP DIARY

SLEEP DIARY

ID# _____ Week 1 ___ Week 2___

Please answer the following questionnaire **WHEN YOU AWAKE IN THE MORNING**. Enter yesterday's day and date and provide the information to describe your sleep the night before. Definitions explaining each line of the questionnaire are given below.

EXAMPLE

yesterday's day yesterday's date	TUES 10/14/97	day 1	day 2	day 3	day 4	day 5	day 6	day 7
1. NAP (yesterday)	70							
2. BEDTIME (last night)	10:55							
3. TIME TO FALL ASLEEP	65							
4. # AWAKENINGS	4							
5. WAKE TIME (middle of night)	110							
6. FINAL WAKE-UP	6:05							
7. OUT OF BED	7:10							
8. QUALITY RATING*	2							
9. BEDTIME MEDICATION (include amount & time)	Halcion 0.25 mg 10:40 pm							

*Pick one number below to indicate your overall QUALITY RATING or satisfaction with your sleep.
1. very poor, **2.** poor, **3.** fair, **4.** good, **5.** excellent

APPENDIX B
HEALTH SURVEY

HEALTH SURVEY

Please **PRINT** and Supply **ALL** Information

ID# _____ Height _____ Weight _____

Race _____

1. Do you have a sleep problem? yes or no
   If yes, describe (e.g., trouble falling asleep, long or frequent awakenings, sleep apnea):

   _____

   If yes, on average, how many nights per week do you have this problem? _____

   How long have you had this sleep problem? _____years _____months

2. Please indicate whether you or your bed partner have noticed any of the following:
   Are you a heavy snorer? yes no
   Do you have difficulty breathing or gasp for breath during sleep? yes no
   Do your legs jerk frequently during sleep or do they feel restless before sleep onset? yes no
   Do you have sleep attacks during the day or paralysis at sleep onset? yes no

If yes to any of the questions under #2, please explain and indicate how often symptoms occur:

_____

_____

3. Indicate with a check mark if you have the following health problems, and put the number of years you've had each problem:

Yes ***Years***

___ _____ **Heart disease**
___ _____ Cancer
___ _____ AIDS
___ _____ High blood pressure
___ _____ Neurological disease (ex: seizures, Parkinson's)
___ _____ Breathing Problems (ex: asthma, emphysema)
___ _____ Urinary problems (ex: kidney disease, prostate problems)
___ _____ Diabetes
___ _____ Chronic Pain (ex: arthritis, back pain, migraines)
___ _____ Gastrointestinal (ex: stomach, irritable bowels, ulcers)

4. Please list any mental health disorders you have and the number of years you've had the disorder(s)

_____

_____

(OVER)

5. List any other health problems you have (and the number of years you've had the problem).

_____

6. Medical and mental health disorders may disrupt sleep. Medication may also disturb sleep. Please list any disorder or medication that affects your sleep and describe how it affects sleep.

_____

_____

7. List ALL **medications** taken within the past month, the frequency with which they are taken (e.g., daily, 3 times a day, weekly), time of day, and the purpose of the medication.

	Medicine	Frequency	Time of Day	Purpose
a.				
b.				
c.				
d.				
e.				
f.				
g.				

8. List ALL **vitamins** taken within the past month, the frequency with which they are taken (e.g., daily, 3 times a day, weekly), time of day, and the purpose of the medication

	Vitamin	Frequency	Time of Day	Purpose
a.				
b.				
c.				
d.				

9. On average, how many alcoholic drinks do you drink per week? _____
10. On average, how many cigarettes do you smoke per day? _____
11. On average, how many caffeinated drinks do you have per day? _____
12. What is your highest level of education? _____
13. If you have a spouse, what is his or her highest level of education? _____

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BIOGRAPHICAL SKETCH

Mr. Kay is a graduate student in the Department of Clinical and Health Psychology at the University of Florida studying to be a sleep specialist as a clinical psychologist. Mr. Kay has been involved in sleep research for 5 year. His training began in the animal sleep research lab of Dr. James Krueger at Washington State University. He continued his research at Brigham Young University studying the biological and behavioral correlates of infant's diurnal sleep in Rhesus Macaques and received the American Academy of Sleep Medicine Young Investigator Honorable Mention Award in 2008 for his work in this area. He is currently studying sleep misperception in older adult in Dr. Christina McCrae's sleep research lab. He won the College of Public Health and Health Profession (PHHP) Young Research Award and the Department of Clinical and Health Psychology Best Fall Symposium Presentation Award in 2008 on his work in the area of sleep misperception. Specific research interests include the functions of sleep, localization of brain functions during sleep, sleep in development, and the sleeping brain in mental and physical illnesses. Mr. Kay is committed to helping make cognitive-behavioral treatments for sleep problems more integrative, accessible, and effective in clinical practice.