Insomnia Identity

Kenneth L. Lichstein – University of Alabama

Citation of published version:
Lichstein, K. (2017): Insomnia Identity. *Behavior Research and Therapy,* vol. 97.
DOI: https://doi.org/10.1016/j.brat.2017.08.005

Behaviour Research and Therapy, in press

Insomnia Identity

Kenneth L. Lichstein

University of Alabama

Correspondence concerning this article should be sent to Kenneth Lichstein, Department of Psychology, The University of Alabama, Tuscaloosa, AL 35487-0348. phone: 205 348-4962, fax: 205 348-8648, email: lichstein@ua.edu.

Abstract

Insomnia identity refers to the conviction that one has insomnia, and this sleep complaint can be measured independently of sleep. Conventional wisdom predicts that sleep complaints are synchronous with poor sleep, but crossing the presence or absence of poor sleep with the presence or absence of insomnia identity reveals incongruity with expected patterns. This review of existing research on insomnia identity processes and influence finds that about one-fourth of the population are uncoupled sleepers, meaning there is an uncoupling of sleep and sleep appraisal, and daytime impairment accrues more strongly to those who endorse an insomnia identity. Research supports the conclusion that there is a cost to pathologizing sleep. Individuals claiming an insomnia identity, regardless of sleep status, are at greater risk for a range of sequelae including self-stigma, depression, suicidal ideation, anxiety, hypertension, and fatigue. A broad research agenda is proposed with hypotheses about the sources, clinical mechanisms, and clinical management of insomnia identity.

key words: insomnia identity, cognitive factors, insomnia, uncoupled sleeper

Abbreviations

CBTi	Cognitive Behavior Therapy for Insomnia
CG	complaining good sleeper
CP	complaining poor sleeper
CTi	Cognitive Therapy for Insomnia
NG	noncomplaining good sleeper
NP	noncomplaining poor sleeper
NRS	Nonrestorative sleep
PSG	polysomnography
SE	sleep efficiency
SOL	sleep onset latency
TST	total sleep time
TWT	total wake time
WASO	wake time after sleep onset

People with insomnia construe particular significance from nighttime wakefulness. In the dark, in the quiet, in the lonely stillness, the aggrieved struggle to rescue sleep from vigilance. But insomnia impact isn't confined to sleep. Nighttime hardship taints daytime experience, inciting anticipatory worry and self-defeating compensatory behaviors (Harvey & Spielman, 2011). Insomnia is a 24-hour disorder characterized by dreary quality of life (Riedel & Lichstein, 2000). Though just as informative, some impute less insidious meaning from blemished somnolence (noncomplaining poor sleepers) and others ascribe sleep pathology when none is apparent (complaining good sleepers).

Insomnia is a sleep disorder, but it also may be a cognitive appraisal disorder. Two salient questions arise: how does one arrive at the conclusion that one is an insomniac and what are the consequences of this realization? This paper investigates a neglected cognitive aspect of insomnia in the hope of narrowing the gap between insomnia treatment goals and outcome. The premise of this paper is that the self-attribution of the insomnia label, termed insomnia identity, instigates a cognitive process that is predictive of the disorder and degrades quality of life. Further, it is hypothesized that insomnia identity is a drag on sleep treatment progress, beckons relapse, and commands intervention attention.

Interest in insomnia identity originated with the unexpected report that not all people with poor sleep report sleep distress (Fichten et al., 1995). Daytime impairment was likely to occur only when poor sleep was accompanied by sleep dissatisfaction. Daytime impairment was a function of one's attitude about sleep, not sleep. We have adopted the inclusive term insomnia identity to capture processes and effects associated with sleep dissatisfaction. It is now clear that the basis for adopting an insomnia identity is ironically often disengaged from sleep pattern.

Research has given credence to the terms complaining good sleepers, people whose sleep does not satisfy insomnia conventional benchmarks but who insist they have insomnia, and noncomplaining poor sleepers, people whose sleep satisfies most insomnia conventional benchmarks but who are content with their sleep. Pathologizing sleep, i.e., embracing an insomnia identity, is only partly determined by one's sleep pattern. This article is a review of past research exploring the characteristics and implications of insomnia identity.

Insomnia Identity Defined

Labeling oneself an insomniac creates an insomnia identity. Global complaints of poor sleep and general statements of sleep dissatisfaction are equally taken as evidence of insomnia identity. Determining the origin of insomnia identity is elusive, but plausible candidates can be eliminated. Data given below bolster the unlikely conclusion that insomnia identity is indifferent to the presence of good sleep.

For purposes of isolating insomnia identity from poor sleep, it is critical to evaluate sleep and sleep complaint independently. For example, if an individual reports difficulties falling asleep, that conflates poor sleep with a sleep complaint. This approach confounds sleep assessment with a valenced view of sleep. Present purposes would require that we ask the subject 'how long does it take you to fall asleep,' a nonjudgmental assessment. We would then ask "are you dissatisfied with your sleep?" The studies reported herein succeeded in this dual approach to assessing sleep and sleep complaint. Typically, sleep was evaluated by PSG or sleep diaries and sleep complaint by inquiring about sleep dissatisfaction.

We have used two assessment devices to establish the presence of an insomnia identity. Originally, we asked respondents to list any sleep disorders they might have (Lichstein, Durrence, Riedel, Taylor, & Bush, 2004). If they claimed insomnia, we presumed an insomnia

identity. Others have used similar approaches to determine the presence of an insomnia self-concept (Edinger et al., 2000; McCrae et al., 2005).

Our current research assesses insomnia identity more formally and directly with a Likert scale: I am an insomniac (with choices): Strongly disagree, Disagree, Undecided, Agree, Strongly agree. We are also exploring an alternative stem: I have insomnia, hoping to clarify the best way to capture this characteristic. The Likert approach recognizes that insomnia identity strength is graded.

We chose the term insomniac in the stem because we believed it would be more difficult to endorse than 'insomnia' and would betray a stronger insomnia identity presence, even though calling a person by their disorder is rightfully viewed as offensive by many. However, we are not calling people 'insomniac.' We are asking them if they call themselves that. The label insomniac beckons a durable, trait conceptualization compared to a more pliable state of having insomnia. We have not compared the two stems and do not know which is more revealing. The adequate assessment of insomnia identity remains an open question.

Indeed, the adequate assessment of what constitutes good and poor sleep for an individual is also elusive. Determining idiographic sleep status by applying nomothetic standards does not ensure a good fit. I return to this topic in the Discussion.

Individuals may or may not have poor sleep and may or may not complain of poor sleep. Crossing sleep status with complaint status yields four distinct groups, but there are not standardized terms for these groups and inconsistent language obscures clarity of communication. For example, individuals who exhibit poor sleep but do not view themselves as having a sleep problem have been called low distress poor sleepers (Fichten et al., 1995), subjective normal sleepers (Edinger et al., 2000), and noncomplaining poor sleepers (Lichstein,

Durrence, Taylor, Bush, & Riedel, 2003).

Figure 1 portrays the four types of match and mismatch that might occur when considering the presence or absence of poor sleep and sleep complaints. Isomorphic quadrants 1 (CP, complaining poor sleeper) and 4 (NG, noncomplaining good sleeper) capture the common understanding of insomnia and normal sleep, respectively. The highlighted off diagonal quadrants 2 (NP, noncomplaining poor sleeper) and 3 (CG, complaining good sleeper) depict sleep/complaint incongruity, the unlinking of sleep and sleep complaint. I refer to these individuals as uncoupled sleepers. This paper will adopt these terms, rather than repeatedly having to describe common characteristics of disparate terms across studies.

The Stigma of Insomnia Identity

Labeling

The stigma associated with mental illness diagnoses is longstanding, widespread, and well documented (Hinshaw, 2007). There are many reasons why stigma occurs. Typically, it originates with observation or anecdotes of unconventional behavior, but is compounded by the observer inferring unfounded character flaws, including poor self-control and responsibility avoidance.

Self-Labeling

Self-stigma occurs when negative stereotypes are internalized attendant to a professional diagnosis or when the diagnosis is self-conferred. Self-labeling creates stigma-induced distress similar to labeling from others, including low self-esteem, shame, restricted constructive activity, and bridled help-seeking (Corrigan, 2004; Corrigan & Watson, 2002; Pattyn, Verhaeghe, Sercu, & Bracke, 2014). Milder psychiatric conditions, including insomnia, are concealable (Hinshaw, 2007). Individuals can elect not to disclose its presence to others to elude stigma, but then may

endure the anxiety of secretive identity, deceit, and fear of discovery.

Self-labeling and insomnia. As documented below, insomnia presents a distinctive self-labeling environment because the majority of people with disturbed sleep do not seek professional help, and when they do, the diagnosis of insomnia is almost always initiated by the patient. Thus, people who adopt an insomnia identity account for virtually the entirety of the treated and untreated population of people with insomnia.

Four studies investigated stigma in insomnia using four methodologies: focus groups (Carey, Moul, Pilkonis, Germain, & Buysse, 2005; Kyle, Espie, & Morgan, 2010), audio diary (Kyle et al., 2010), individual interviews (Henry, Rosenthal, Dedrick, & Taylor, 2013), and questionnaires (Stinson, Tang, & Harvey, 2006). The most common stigma related reports were feeling misunderstood by peers and healthcare providers, their complaints were trivialized by others, and their sleep problem fostered social isolation (Carey et al., 2005; Henry et al., 2013; Kyle et al., 2010). People with insomnia reported shame when talking about their sleep problem (Henry et al., 2013; Kyle et al., 2010), and reported that anticipated stigma discouraged them from seeking treatment (Henry et al., 2013; Stinson et al., 2006).

Constricted treatment seeking in insomnia. Treatment exposure for the majority of people with insomnia is limited to over-the-counter hypnotics, alcohol, or self-help media (Johnson, Roehrs, Roth, & Breslau, 1998; Morin, LeBlanc, Daley, Gregoire, & Merette, 2006). Survey estimates reveal 52-84% of people with insomnia do not seek professional help (Aikens & Rouse, 2005; Bartlett, Marshall, Williams, & Grunstein, 2008; Estivill, 2002; Leger & Poursain, 2005; Morin, LeBlanc, et al., 2006; Ohayon & Smirne, 2002), and stigma avoidance is contributory.

Based primarily on studies of people with depression and anxiety disorders, and replicated

within insomnia (Morin, Gaulier, Barry, & Kowatch, 1992), 75% of people prefer psychological to pharmacological treatment (McHugh, Whitton, Peckham, Welge, & Otto, 2013). Psychological preference was even stronger among women and non-treatment seeking individuals, conforming to the characteristics of the insomnia population. Yet when people with insomnia receive treatment from a physician, nearly 90% of the time it is pharmacotherapy (Leger & Poursain, 2005). Explanations of low treatment seeking among people with insomnia must include disinterest in the expected treatment (Stinson et al., 2006).

When people with insomnia do receive professional care, it is almost always initiated by the patient, indicating the self-diagnosis has already been determined. It is unusual for a health care provider to inquire about possible insomnia without first being prompted by the patient (Benca, 2005).

Whether corroborated by a health care provider or not, the insomnia identity label is almost always self-assigned, and this process is not without cost. Based on the stigma and self-stigma literature, it is likely that some increment in psychological burden accrues to the individual when they assume an insomnia identity. Concordant with theoretical models of an anxiety-insomnia reciprocal exacerbation cycle (Harvey, 2002; Morin, Savard, & Blais, 2000; Perlis, Shaw, Cano, & Espie, 2011), adopting an insomnia identity likely aggravates the insomnia.

Sleep Pattern, Insomnia Identity, and Daytime Correlates

Germinal Studies

Fichten and her colleagues (1995) were the first to observe the curious disengagement of sleep complaint from sleep pattern. Anticipating that naturally occurring changes in sleep would instigate sleep complaints in a large proportion of older adults, this research group initially inquired about distress level among poor sleepers to investigate why some older adults

complained of insomnia and others did not (C. S. Fichten & E. Libman, personal communication, February 5, 2016).

Fichten et al. (1995) studied 396 community volunteers in the age range 55-89 years. Individuals labeled poor sleepers satisfied four criteria derived from a questionnaire: they reported at least 30 minutes of undesired wake time, at least 3 times a week, for at least 6 months, and rated subjective sleep difficulty high. They then rated distress about their sleep on a 10-point scale. Participants were assigned to three categories: good sleepers (NG, $n = 249$), high distress poor sleepers (CP, $n = 63$), and low distress poor sleepers (NP, $n = 84$). Poor sleep did not provoke distress in the majority of symptomatic individuals (63 vs. 84). Further analyses revealed daytime impairment, as measured by subjective sleepiness and fatigue, was significantly greater in high distress versus low distress poor sleepers. Most surprisingly, NP were not significantly more impaired than NG. Similarly, three out of four measures of anxiety and depression showed CP were significantly more elevated than NG, but NP were not. This was the first study to show that daytime impairment corresponds more closely with distress about sleep than with disturbed sleep.

A few years later, this same group (Alapin et al., 2000) replicated their results. They derived the same three groups from an older adult sample: NG, NP, CP. This study also included a college student cohort but the *N* of this group was too small to do low distress vs. high distress analyses. Significant differences between NP and CP did not occur on sleep parameters, but did occur on daytime functioning. CP scored significantly higher than NP on measures of sleepiness, concentration difficulty, nocturnal tension, and depression, but not fatigue. There was no significant difference between NP and NG on sleepiness, nocturnal tension, and depression. Again, daytime impairment more closely corresponds to sleep dissatisfaction than

sleep pattern. Taken together, these two studies challenged the conventional wisdom that dysfunction associated with insomnia was due to disturbed sleep and could best be alleviated by improving sleep patterns. Distress about sleep, contrasted with absence of distress about poor sleep, can be conceptualized as an insomnia identity effect.

McCrae et al. (2003) drew from a larger epidemiology study (Lichstein et al., 2004) to investigate the same three groups as the Fichten lab, but also separately analyzed young old (age 60-74 years) and old old groups (age 75-98 years). The following results occurred in both age groups. Comparing NG, CP, and NP, two health measures (count of medical disorders and medication use) and two mood measures (depression and anxiety) were significantly higher in the CP compared to the other two. NG and NP did not differ on any of these measures. McCrae et al. (2003) replicated prior findings (Alapin et al., 2000; Fichten et al., 1995), but could not rule out that differences in mood and sleep pattern were accounted for by health inequality.

Emergent Literature

Five studies closely related to Fichten et al. (1995) have appeared since the pioneering articles cited above. This group of studies further clarifies the correspondence between insomnia identity and sleep pattern and the relation between these two factors to daytime impairment. Another seven studies were published by the Penn State Cohort, and these are considered separately below because of their distinctive methodology.

These studies vary across a range of subject characteristics, methodologies, and measures. What they have in common is they all replicate and extend Fichten et al.'s (1995) findings. To preview the results of this group of studies, research has shown that polysomnography (PSG) verified poor sleep may not produce a sleep complaint (Edinger et al., 2000) and the same with sleep documented with sleep diaries (Lichstein et al., 2003; McCrae et al., 2005), the presence of

sleep complaints are often accompanied by PSG verified good sleep (Edinger et al., 2000) and the same with sleep documented with sleep diaries (Lichstein et al., 2003; McCrae et al., 2005), CG exhibit comparable or higher levels of daytime impairment as people with insomnia (i.e., both poor sleep and a sleep complaint) (Edinger et al., 2000; McCrae et al., 2005; Ustinov et al., 2010; Woosley, Lichstein, Taylor, Riedel, & Bush, 2016), and NP exhibit comparable levels of daytime functioning as normal sleepers, NG (Edinger et al., 2000; McCrae et al., 2005).

Epidemiology studies. Our lab conducted a random survey of people's sleep and daytime functioning experience (Lichstein et al., 2004) and have published three articles related to insomnia identity from these data (Lichstein et al., 2003; Ustinov et al., 2010; Woosley et al., 2016).

We (Lichstein et al., 2003) collected 2 weeks of sleep diaries and numerous demographic and daytime functioning questionnaires from a random sample of 735 community dwelling individuals, age range 20-98 years. Poor sleep was defined according to empirically derived, quantitative criteria (sleep onset latency [SOL] or wake time after sleep onset [WASO] ≥ 31 minutes, ≥ 3× a week, for ≥ 6 months). We observed the uncoupling of poor sleep and insomnia complaint. Results salient to the present discussion are presented in Figure 2. Of the 257 people with poor sleep (top row), 120 did not complain of insomnia (47%, NP). Of the 214 people who complained of insomnia (left column), 77 did not have poor sleep (36%, CG). Overall, 137 people in our sample qualified for an insomnia diagnosis, having both poor sleep and a sleep complaint, but nearly 1.5 times as many people (197) experienced one but not the other. Concordant poor sleep and sleep dissatisfaction is the exception.

Ustinov et al. (2010) applied a hierarchical blocks multiple regression model with an *N* of 734 to study insomnia identity. Control variables were entered on three steps: a set of

demographic variables, a set of health variables, and a set of sleep variables from 2 weeks of sleep diaries: SOL, WASO, terminal wake time, and total sleep time (TST). A single dichotomous variable, presence or absence of insomnia complaint (i.e., insomnia identity) was entered on step 4. This model was run five times to predict questionnaire data on depression, anxiety, fatigue, sleepiness, and insomnia impact.

After a total of 17 control variables, insomnia identity significantly predicted depression and had the largest beta weight compared to all 17 variables. Of the four sleep variables, only WASO was significantly related to depression. Near identical results were obtained in predicting anxiety. For this analysis, only age had a higher beta weight and WASO was again the only sleep variable related to outcome. With minor variation, the same results prevailed for sleepiness, fatigue, and insomnia impact. Insomnia identity was significant in all three, and it had the largest beta weight in two of these, sleepiness and fatigue. Among the sleep parameters, only WASO was significant for sleepiness, SOL and TST were significant for fatigue, and SOL, WASO, and TST were significant for insomnia impact. No sleep variable had a higher beta weight than insomnia identity in the five regression runs.

Woosley et al. (2016) studied suicidal ideation as measured by item 9 on the Beck Depression Inventory, $N = 768$. Binary logistic regression predicting presence or absence of suicidal ideation tested main effects for presence or absence of sleep complaint and presence or absence of poor sleep. Sleep complaint proved significant, but poor sleep did not. Subjects with a sleep complaint were nearly twice as likely as subjects with no sleep complaint to endorse suicidal ideation, odds ratio = 1.92. Subjects who exhibited both insomnia complaint and poor sleep (CP) were no more likely to report suicidal ideation than CG. These data found no role for poor sleep in predicting suicidal ideation above insomnia identity.

The epidemiology series from the Penn State Cohort. The Penn State Cohort (as they refer to themselves) published seven studies (Fernandez-Mendoza et al., 2011; Fernandez-Mendoza et al., 2010; Fernandez-Mendoza et al., 2015; Fernandez-Mendoza et al., 2012; Vgontzas, Liao, Bixler, Chrousos, & Vela-Bueno, 2009; Vgontzas et al., 2010; Vgontzas, Liao, Pejovic, et al., 2009) based on a large community sample to evaluate the interaction between total sleep time and insomnia complaint. The core of this series comprised 1,741 subjects who completed one PSG night and numerous psychological and physiological measures, although the *N* was smaller in some of these studies due to missing data and variation in screening criteria. The design crossed sleep status (normal > 6 hours TST or short < 6 hours TST) with presence or absence of an insomnia complaint. Given the assumption that good and poor sleep can be distinguished solely by a 6 hour TST boundary on one PSG night, the resulting grid permits evaluation of insomnia identity.

In the most cited study in this series, Vgontzas and his colleagues (Vgontzas, Liao, Bixler, et al., 2009) found that neither short sleep nor insomnia complaint independently conferred significant risk of hypertension, after controlling for a wide range of factors including age, gender, body mass index, diabetes, smoking, and alcohol. But when short sleep was accompanied by a sleep complaint, their interaction yielded a 350% - 500% increased likelihood of hypertension depending on the degree of short sleep. The association between combined PSG short sleep and insomnia complaint with hypertension has been partially replicated (Bathgate, Edinger, Wyatt, & Krystal, 2016), but this study did not include a short sleep without insomnia complaint comparison group.

The Penn State Cohort reported additional significant interactions that featured the influence of insomnia identity. The joint presence of short sleep and insomnia complaint was associated

with heightened neuropsychological deficits including psychomotor processing speed, visual conceptual tracking, set switching, and short-term visual memory (Fernandez-Mendoza et al., 2010) and increased risk for type 2 diabetes (Vgontzas, Liao, Pejovic, et al., 2009). But the joint presence of normal sleep and insomnia complaint was associated with increased risk for sleep state misperception, also known as paradoxical insomnia, and elevated MMPI anxiety and depression (Fernandez-Mendoza et al., 2011).

Long-term follow-up studies on this sample ranging between 7 and 14 years also reported interactions between sleep and sleep complaint. Men, but not women, who complained of insomnia and had short sleep duration were at heightened risk for all-cause mortality (Vgontzas et al., 2010). The combination of short sleep and insomnia complaint presented the highest risk for incident depression (Fernandez-Mendoza et al., 2015) and incident hypertension (Fernandez-Mendoza et al., 2012).

Determination of sleep status by polysomnography in an experimental study. One study used PSG to classify good and poor sleepers (Edinger et al., 2000). This study recruited subjects from several sources: VA patients, sleep center patients, and volunteers at a research center. Using DSM-IV interview criteria, they identified 57 people diagnosed with insomnia and 51 gender and age-matched normal sleepers.

Independent of insomnia status classification, subjects were judged to have good or poor sleep based on a mix of TST and sleep efficiency (SE) criteria derived from 6 PSG nights. Normal sleepers had a mean PSG TST across 6 nights > 6.5 hours and poor sleepers had a mean TST < 6 hours. Subjects whose TST averaged between 6 and 6.5 hours were assigned to the good sleeping group if their mean SE > 85% or, in the case of subjects 60 years of age or older, if their mean SE ≥ 80%. Subjects in the TST band between 6 and 6.5 hours not assigned to the

good sleeping group were classified as poor sleepers. Thus, by crossing presence or absence of insomnia report with presence or absence of poor PSG sleep, this study produced the same 2 × 2 matrix described in Figure 1.

As shown in Figure 3, 41% of this sample were uncoupled sleepers, meaning their sleep pattern and sleep appraisal were discordant. Based on the top row, 39% (22 of 57) of those who have poor sleep did not assert an insomnia identity, NP. Based on the left column, 39% (22 of 57) of those who claim an insomnia identity were good sleepers, CG. It should be noted that because this was not a random sample, in that about 50% of subjects were recruited from an insomnia clinic and 50% from the community, these data are not generalizable to the population.

Edinger et al. (2000) measured daytime functioning in depression, anxiety, and dysfunctional insomnia related cognitions (five subscales). They found multivariate, but not univariate, statistically significant differences between the quadrants. The pattern across these seven measures was consistent. I will summarize data just for depression and anxiety. Figure 4 reveals an identical pattern for these two outcomes. The two noncomplaining groups reported the least disturbance and the two complaining groups the most. Indeed, contrary to conventional wisdom attributing impairment to poor sleep, sleep complaint absent poor sleep (CG) had the highest levels of both depression and anxiety and poor sleepers absent complaint (NP) the lowest. Elevated depression/anxiety tracked insomnia identity (i.e., insomnia complaint), not poor sleep.

Older adults. McCrae et al. (2005) relied on 2 weeks of sleep diaries to classify sleep in 103 older adult volunteers recruited from the community. They used the same quantitative sleep criteria as Lichstein et al. (2003) to differentiate good and poor sleepers (SOL or WASO ≥ 31 minutes, ≥ 3× a week, for ≥ 6 months), and they determined insomnia identity by the presence or absence of a sleep complaint.

As shown in Figure 5, uncoupled sleepers represented a third of the sample. The majority (56%) of poor sleepers did not complain about their sleep (NP, 24 of 43, top row). A third (37%) of individuals who endorsed an insomnia identity were rated as good sleepers (CG, 11 of 30, left column).

Objective sleep, assessed by 2 weeks of actigraphy, confirmed sleep status assignment. Several measures (SOL, SE, total wake time [TWT]) documented worse sleep in CP compared to NG, and one measure (TWT) documented worse sleep in NP compared to NG. It is noteworthy that actigraphy found no significant differences between complaining vs noncomplaining subjects within the good or poor sleep groups, ruling out a sleep confound.

Questionnaires were used to assess common demographic characteristics including count of medications and medical disorders, depression, anxiety, sleepiness, and fatigue. There was a significant main effect for sleep complaint on medical disorders: those who had a sleep complaint regardless of good or poor sleep had more medical disorders. Of the remaining daytime functioning variables, only fatigue produced significant findings: greater fatigue in CG compared to NP.

These improbable results are displayed in Figure 6. The ascending sequence of fatigue impairment with the two noncomplaining groups low and the two complaining groups high and with NP lowest and CG highest exactly replicates the sequence reported by Edinger et al. (2000) for depression and anxiety (see Figure 4).

Uncoupled Sleepers: An Integrative Summary

Table 1 extracts data from Figures 2 and 5 to summarize category counts for CP, NP, CG, and NG from the two studies that reported such data. Edinger et al. (2000) derived these same four categories, but was not a random sample, and therefore does not suit the purposes of this

analysis.

The percent distributions for these two studies closely replicate, particularly for CP and CG. CP ranged from 18 to 19%, NP from 16 to 23%, CG from 10 to 11%, and NG from 48 to 55%. Combined, a total *N* of 838 reveals 19% of subjects are CP, 17% NP, 11% CG, and 54% NG. Joining categories CP and NG, the sleep/complaint congruous groups, 73% of individuals conform to conventional expectations of sleep and sleep appraisal. Joining categories NP and CG, the sleep/complaint incongruous groups, 28% of individuals are uncoupled sleepers.

Table 2 presents the Penn State Cohort data parallel to Table 1. Three of the studies in this series broke down the total N to support categorical percentages (Fernandez-Mendoza et al., 2011; Fernandez-Mendoza et al., 2010; Vgontzas et al., 2010). The same subjects participated in each of these studies but the Ns varied due to factors such as missing data for specific measures. But the percentages are consistent from one study to the next, and I will focus on the data averaged across the three studies. Because the Penn State Cohort did what amounted to a median split on TST, the proportion of their sample of "poor sleep" groups (CP and NP) was larger than that reported in Table 1, and their normal sleeper groups (CG and NG) smaller. Nevertheless, they did replicate the uncoupled sleep phenomenon, albeit at a lower level. They found 13% of their sample was uncoupled.

The reigning diagnostic systems require both reported poor sleep and a sleep complaint to render an insomnia diagnosis (American Academy of Sleep Medicine, 2014; American Psychiatric Association, 2013), and the present data (Table 1) illustrate the wisdom of this tenet. Consider the two poor sleep categories, CP (19%) and NP (17%). Jointly, they represent 36% of the integrated sample, but 17/36 (47%) of these individuals are not dissatisfied with their sleep. These data suggest that when an insomnia diagnosis is rendered solely on the basis of poor sleep,

it will be wrong 47% of the time according to formal diagnostic criteria. Similarly, consider the two complaint categories, CP (19%) and CG (11%). Jointly, they represent 30% of the sample, but 11/30 (37%) of these individuals do not have poor sleep by conventional standards. These data suggest that when an insomnia diagnosis is rendered solely on the basis of sleep complaint, it will be wrong 37% of the time, and this conclusion is particularly disturbing in two respects. First, it is the sleep complaint that motivates individuals to seek treatment. These are the people most likely to be seen in doctors' offices for insomnia, and for a variety of practical reasons, no more than a token sleep assessment is usually performed. Second, the most common treatment given for insomnia is sleep medication. Based on these data, 37% of the time, hypnotics are prescribed to correct a sleep problem that does not exist. These conclusions are tempered by acknowledging health risk may accrue to NP and to CG even though these individuals do not satisfy formal diagnostic criteria. Idiographic factors will sometimes rightfully outweigh consensus standards.

It is important to note that these data do not come from treatment-seeking individuals, and the current data may not be representative of clinical samples. But it is not known if individuals seeking insomnia treatment are more or less likely to be CG. In the only clinical data available, Edinger et al. (2000) reported on 57 complaining individuals in an insomnia clinic, and 22/57 (Figure 3, 39%) were CG, a rate consistent with that derived from Table 1.

Cognitive Behavior Therapy for Insomnia (CBTi)

CBTi, the application of cognitive/behavioral science to treat insomnia, was introduced nearly half a century ago. As attested to by numerous reviews and meta-analyses, CBTi claims high efficacy (e.g., Lichstein, Vander Wal, & Dillon, 2012; Morin, Bootzin, et al., 2006; Okajima, Komada, & Inoue, 2011). Within an insomnia identity framework, it may be

worthwhile to reexamine CBTi.

A Closer Look at CBTi Efficacy

CBTi efficacy is not without limitations. Harvey and Tang (2003) pointed out that CBTi has done well but there is substantial room for improvement. Effect sizes, a common indicator of treatment potency, are generally in the medium to large window ranging up to 1.0. In contrast, CBT for other disorders such as depression and anxiety disorders often achieve effect sizes in the 1-2.5 range.

Graded CBTi success depends on the rigor of criteria (Espie, Inglis, & Harvey, 2001; Harvey & Tang, 2003; Morin et al., 1999; Morin, & Wooten, 1996). About 70% of treated subjects have significantly better sleep outcomes than control subjects, 50% of treated subjects exhibit clinically meaningful change, and 30% of people administered CBTi are converted to normal sleepers, but about 20% of people with insomnia are unresponsive to CBTi. By inference, there are neglected aspects of insomnia and there are untested, novel intervention strategies yet to be discovered.

Cognitive Processes in CBTi

Many causes of insomnia have been identified, including perverse conditioning, hyperarousal, biopsychosocial stressors, comorbidity, and genetic vulnerability (Perlis et al., 2011). Arguably more than any other factor, cognitive arousal has marshaled strong evidence in support of its causal role in insomnia.

From the early days of empirical investigations of disturbed sleep, cognitive processes in insomnia commanded much interest (Bootzin, Herman, & Nicassio, 1976; Borkovec, & Fowles, 1973; Hauri, 1969). Years later, the potency of sleep related cognitions was invoked to explain

sleep versus sleep dissatisfaction asynchrony as a function of sleep-interpreting processes (Lundh & Broman, 2000), a mechanism that may be conceptual kin to insomnia identity.

In more recent times, inventive experimental and descriptive studies from Harvey's lab comparing people with insomnia to normal sleepers more clearly illuminated cognitive mechanisms in insomnia. Compared to normal sleepers, people with insomnia are more prone to complain of elevated cognitive activity at bedtime (Harvey, 2001), worry about sleep at bedtime (Harvey, 2000; Ree & Harvey, 2006; Semler & Harvey, 2004), experience unpleasant images (Nelson & Harvey, 2003a, 2003b), overestimate sleep disturbance (Tang & Harvey, 2004), and indulge sleep-related negativistic thinking (Harvey & Greenall, 2003; Ree, Pollitt, & Harvey, 2006).

This same research group also engaged the alternative methodological strategy of providing differential challenges to two or more groups of people with insomnia. By this approach, Harvey and her collaborators demonstrated that induced sleep perceptions alter daytime functioning (Semler & Harvey, 2005), clock watching exacerbates insomnia (Tang, Schmidt, & Harvey, 2007), and self-monitoring fuels insomnia concerns (Semler & Harvey, 2006, 2007).

Dozens of experimental and descriptive studies from other labs spanning a diverse range of insomnia phenotypes have corroborated Harvey's findings on the role of cognitive arousal. As examples, cognitive arousal fosters disturbed sleep (Carney, Harris, Falco, & Edinger, 2013; Fichten et al., 2001; Morin, Rodrigue, & Ivers, 2003; Wicklow & Espie, 2000), people with insomnia report cognitive arousal has primacy over physiological arousal (Espie, Brooks, & Lindsay, 1989; Lichstein & Rosenthal, 1980; Nicassio, Mendlowitz, Fussell, & Petras, 1985), pre-sleep cognitive arousal was more strongly related to sleep disturbance than pain complaints among chronic pain patients (Byers, Lichstein, & Thorn, 2016; Smith, Perlis, Smith, Giles, &

Carmody, 2000), people with insomnia have an attentional bias toward the bedroom environment (Jones, Macphee, Broomfield, Jones, & Espie, 2005; MacMahon, Broomfield, & Espie, 2006), and in treatment studies, change in cognitive arousal predicts sleep outcomes (Morin, Blais, & Savard, 2002; Sunnhed & Jansson-Frojmark, 2015).

The evidence affirming the critical role of cognitive arousal in insomnia is overwhelming. But two aspects of cognitive arousal have been neglected in the insomnia literature: catastrophizing and insomnia identity. Catastrophizing has assumed a central position in understanding pain experience (Sullivan et al., 2001), and might apply in equal measure to insomnia. A few studies have explored catastrophizing among people with insomnia (Barclay & Gregory, 2010; Harvey & Greenall, 2003; MacDonald, Linton, & Jansson-Fröjmark, 2008; Winsper & Tang, 2014), but for the most part, this process has remained on the margin of insomnia theory. The second aspect, insomnia identity, shall be the focus of recommendations for future research.

A Broad Research Agenda

There is ample research to substantiate the existence of insomnia identity. But there is a scarcity of research to fully explore the many dimensions of this concept. This section identifies three important areas for future research: sources, clinical mechanisms, and clinical management of insomnia identity. Each part is structured with research hypotheses that may contribute to a more robust understanding of insomnia identity and salient therapeutic processes. It is intended that such plausible speculation will stimulate clarifying research.

Hypothesized Sources of Insomnia Identity

This section speculates on the origins of insomnia identity or immunity to it. Uncoupled sleepers, NP and CG, embody the paradox of insomnia identity and represent two competing

processes fostering either increased risk for or protection from insomnia identity. The challenge is to understand the origins of how people become disposed toward or resistant to this belief system.

Demography. There are well established risk factors for insomnia, including older age, female gender, a history of transient insomnia episodes, low socio-economic status, and medical/psychiatric comorbidity (Lichstein, Taylor, McCrae, & Petrov, 2017). Might there be demographic, comorbidity, and insomnia profile predictors of endorsing or resisting insomnia identity? As plausible examples, perhaps the emergence of insomnia identity is mediated by exposure to nonsleep chronic illness, or perhaps multiple moderators such as gender, marital status, and advanced or delayed sleep phase combine to instigate or insulate sleep effects on insomnia identity.

Paradoxical insomnia, skewed sleep expectations, or preconception? Individuals who falsely perceive sleep disturbance have been labeled subjective insomnia, sleep state misperception, and more recently, paradoxical insomnia (Edinger & Krystal, 2003). These individuals self-report severe sleep pattern disturbance, but this is disconfirmed by PSG, and daytime impairment proportional to their complaint is absent. Though this is a sleep/sleep complaint uncoupling, it is of a different sort than is described herein in two respects. First, misperceived sleep does not have a role in insomnia identity uncoupling, as these individuals might self-report poor sleep without a sleep complaint (NP) or report what appears to be good sleep with a sleep complaint (CG). Second, reversing the pattern of paradoxical insomnia, daytime impairment among individuals described as uncoupled in the present context is proportional to their sleep complaint, but not to their sleep.

Insomnia identity uncoupling is more likely a problem of unrealistic sleep standards. Subjects convinced that normal sleepers fall asleep in less than 10 minutes may conclude that their own SOL of 15 minutes, containing a hefty 50% wake appendage, evinces serious insomnia. The widening gap between one's sleep and perceived normal sleep may fortify convictions of sleep pathology (Mulla et al., in press). This is a testable hypothesis. I predict more stringent sleep expectations among CG than others.

Alternatively, sleep standards may be reasonable but idiosyncratic. Clinicians' diagnostic decisions regarding mental disorders are more strongly driven by theory than symptom (Kim & Ahn, 2002a). Because individuals' favored theories vary in interpreting a common set of symptoms, this is a biasing mechanism that undermines diagnostic reliability. Laypersons exhibit a similar process in judging mental illness (Kim & Ahn, 2002b). Individuals may have preconceptions of what constitutes insomnia that causes them to endorse an insomnia identity based on a symptom profile that would be disconfirming to others. With regard to CG, it is unknown what weighted combination of symptom features drives their conviction.

Nonrestorative sleep (NRS). Much is not known about people who awaken in the morning complaining about the quality of their sleep and not feeling well-rested, unaccompanied by subjective or objective evidence of sleep disturbance (Stone, Taylor, McCrae, Kalsekar, & Lichstein, 2008). But it is known that NRS is not unusual and carries with it impaired daytime functioning.

The recently developed, well-validated Restorative Sleep Questionnaire (Drake et al., 2014) provides an important research tool to investigate the relation between insomnia identity and NRS. I hypothesize that heightened levels of NRS occur among good sleepers who profess an insomnia identity (CG).

Personality bias. There are personality types who are bent on complaining though there is weak underpinning (CG) or who defy complaining even when confronted by adversity (NP). Unexplained or unfounded health complaints are not uncommon (Nezu, Nezu, & Lombardo, 2001), and may reflect characterological bias. If this were true, then we would expect CG would also be more prone to exhibit or complain about other medical/psychiatric disorders. But this may not be a fair test because of the high comorbidity found in insomnia. A more revealing investigation would be to determine if CG are more inclined to complain about general life circumstances unrelated to health.

If we consider CG exhibit elements of hypochondriasis, there is no comparable diagnosis for the opposite of hypochondriasis, NP. But there are personality traits that dispose individuals to minimize health complaints. Happy (Lyubomirsky, King, & Diener, 2005), sociable (Berscheid, 2003) people are less likely to report physical symptoms and more likely to claim higher health satisfaction. To extrapolate, it may be worthwhile to investigate if positive affect and social support congregate more strongly with NP. Conversely, might grumpy, lonely people be prone to CG?

Hypothesized Clinical Mechanisms of Insomnia Identity

This section speculates on insomnia identity clinical processes and effects. Insomnia identity is likely not dichotomous. It is graded and the strength of insomnia identity exerts a proportional influence on clinical mechanisms. Based on studies of cognitive processes in insomnia, discussed above, I assume that nocturnal vigilance, negative attentional bias toward sleep imperfections, and worry about sleep processes and effects flourish when an individual affirms an insomnia identity. I hypothesize that insomnia identity is an organizing structure that mobilizes alerting responses. If this is true, then the following clinical mechanisms are

predictable.

Therapist outcome expectations. It has long been known that therapists who expect positive outcomes are more likely to obtain them (Frank, 1968). There are no data on therapists' views of how amenable people with insomnia are or if patients' insomnia identity is infectious. Therapy may suffer when therapists' outcome expectations are tainted by patients' fractious insomnia identity.

Insomnia identity fuels chronicity. Perhaps insomnia identity fits the role of predisposition in Spielman's 3P Model (Spielman, Conroy, & Glovinsky, 2003). It primes the individual's vulnerability to psychological/physical stress and sensitizes the individual to sleep irritants, thereby also fueling the second stage of the model, precipitants. Indeed, insomnia identity could emerge at any point along this hypothesized path, including the third phase, perpetuation, and help sustain insomnia. Spielman's Model traces the evolution of insomnia from an acute, transient disturbance to a chronic disorder, and insomnia identity may be one of the foundational factors motivating this path.

Insomnia identity is a drag on treatment. Insomnia treatment response should be inversely proportional to the strength of one's insomnia identity, assuming insomnia identity is not one of the treatment targets. Treatment is pushing against conviction. I anticipate two mechanisms by which insomnia identity may operate as a moderator variable. A strong insomnia identity may take a toll on patient motivation by discounting sleep gains and fostering discouragement. And a strong insomnia identity may sabotage therapy progress by curtailing adherence, given the aforesaid demotivation. In either case, this would appear as psychotherapeutic resistance motivated by cognitive-behavioral processes (Jahn & Lichstein, 1980).

Insomnia identity provokes recurrence. The depression literature has drawn on

psychological scar theory to partly explain recurrence (Burcusa & Iacono, 2007; Elgersma, Glashouwer, Bockting, Penninx, & de Jong, 2013). This concept postulates that a depressive episode corrupts the individual's sensitization to stress, social interactions, cognitions, or personality, rendering the person more vulnerable to future episodes.

An analogous process may be instigated by insomnia identity in individuals who have experienced sleep disturbance. An insomnia episode will not necessarily instill a strong insomnia identity in all individuals. I predict that individuals who emerge from a bout of insomnia with a strong insomnia identity are more likely to engage in worry, vigilance, and negative attentional bias with regard to insomnia triggers or even mild-moderate symptoms of disturbed sleep. These individuals are sleep scarred and are thereby more likely to have recurrent insomnia.

Hypothesized Clinical Management of Insomnia Identity

This section speculates on interventions to mitigate insomnia identity. I identify established treatments or variations on treatments that could be fruitful in altering insomnia identity. If insomnia identity is a construct worth intervention, then this goal is served by the independent assessment of sleep and sleep complaint. The insomnia treatment literature does not routinely conduct such dual assessment, but this may prove valuable in illuminating mechanisms in the insomnia syndrome.

Mindfulness is an effective intervention for insomnia (Black, O'Reilly, Olmstead, Breen, & Irwin, 2015; Ong et al., 2014), and perhaps its calming influence would make it well-suited for defusing insomnia identity, but this mechanism has not been evaluated. Plausibly, persons with the strongest insomnia identity would benefit the most from mindfulness based treatments, including acceptance and commitment therapy (Hayes, Strosahl, & Wilson, 2011). The concept

of metacognition, thoughts about thoughts, has gained traction as an exacerbating influence in many domains and is likely relevant to insomnia identity (Ong, Ulmer, & Manber, 2012; Palagini et al., 2016). Mindfulness/acceptance therapies hold the promise of metacognitive deactivation.

A perspective that is easily distinguished from mindfulness therapies is rational emotive behavior therapy (Walen, DiGiuseppe, & Dryden, 1992). This approach attempts to modify maladaptive cognitions and sometimes takes the form of a Socratic method. Following are examples of questions that can be posed to clients to initiate a discussion designed to challenge and reform core beliefs of an insomnia identity.

- What are your standards for normal sleep?
- How did you derive your standards for normal sleep?
- How close do you have to be to these standards to consider yourself a normal sleeper?
- Can you be a normal sleeper if you are not a perfect sleeper?
- Does having troubled sleep change the way you think about yourself?
- What do you think the impact of seeing yourself as an insomniac is?
- If your sleep improved, how would that change the way you think about yourself?

This line of questioning is similar to that in cognitive therapy for insomnia (Morin et al., 2000), but possesses a greater emphasis on the person's self-concept compared to attitudes about sleep and sleep consequences.

Discussion

When sleep self-appraisal is disengaged from sleep, perception carries more weight than actuality. Perceived sleep disturbance poses a health risk, even when accompanied by good sleep. Perceived satisfactory sleep confers no health risk, even in the presence of disturbed

sleep. These findings have been replicated across labs, age groups, methods of sleep assessment, and measures of impaired health.

Although the presence of causal influence, characterization of causal mechanisms, and the direction of causality between reported poor sleep, sleep complaint, and impaired daytime functioning are poorly understood, these are correlated attributes. Specifically, none of the studies reported in this article address causality associated with insomnia identity. The intimate linkage between these three processes has long been formally recognized in the diagnostic systems and continues in both the DSM-5 (American Psychiatric Association, 2013) and ICSD-3 (American Academy of Sleep Medicine, 2014). By definition, if you do not complain about your sleep, you may still experience daytime dysfunction, but you do not have insomnia. However, recognition of the linkage between sleep complaint and daytime impairment in the absence of poor sleep, i.e., CG, constitutes a paradigm shift in understanding insomnia mechanisms that commands corresponding changes in assessment and treatment of insomnia. In this conceptualization, sleep self-appraisal takes precedence over sleep, a position previously advocated by Ohayon, Caulet, Priest, and Guilleminault (1997). If the preponderance of insomnia health risk is conveyed by insomnia identity, not sleep, then insomnia identity should be among the primary insomnia outcome measures by which treatment is judged.

The seven studies from the Penn State Cohort (Fernandez-Mendoza et al., 2011; Fernandez-Mendoza et al., 2010; Fernandez-Mendoza et al., 2015; Fernandez-Mendoza et al., 2012; Vgontzas, Liao, Bixler, et al., 2009; Vgontzas et al., 2010; Vgontzas, Liao, Pejovic, et al., 2009) took a different approach from the other studies testing this phenomenon in studying the interaction between sleep and complaint. They were primarily interested in short sleep effects and essentially did a median split on TST from one PSG night to identify short and normal

sleepers separated by a 6-hour boundary. Their group assignment method departs from others in this review and may be testing a different process than good and poor sleep. Yet nevertheless, they consistently found the joint operation of insomnia identity and short sleep conferred elevated health risk, as did other labs. However, unlike several other studies (Edinger et al., 2000; McCrae et al., 2005; Ustinov et al., 2010; Woosley et al., 2016), but one of their studies found that the co-occurrence of insomnia identity and good sleep (CG) also conferred elevated health risk (Fernandez-Mendoza et al., 2011).

In order to identify uncoupling, a good versus poor sleep determination is made, and this assessment is inexact. All of the studies in this literature relied on a variety of quantitative sleep parameters. Consensual agreement on these standards is lacking, the boundaries are fuzzy, and individual sleep needs vary. Perhaps, some uncoupled individuals are responding to qualitative as opposed to quantitative dimensions of sleep. No study used sleep stages or EEG anomalies to discriminate good and poor sleepers. For example, beta EEG intrusion may sway sleep appraisal (Perlis, Smith, Andrews, Orff, & Giles, 2001). And no study used measures outside of routine sleep assessment, such as brain glucose metabolism, which varies between people with insomnia and normal sleepers (Kay et al., 2016). Patients may be drawing on a wider range of information to judge their sleep than are sleep researchers.

The proportion of people with insomnia that have a strong insomnia identity is unknown. But for those that do, it follows that interventions focusing on sleep behavior, such as stimulus control and sleep restriction, are likely to secure smaller benefits for daytime functioning than interventions aimed at easing feverish insomnia identity cognitions. People seek insomnia treatment because they are unhappy with their sleep. According to Table 1, I documented 244 unhappy sleepers (CP = 156 + CG = 88), but CG accounted for 36% of these 244 people (88 ÷

244). These figures are based on population sampling and may not generalize to clinical populations. Edinger et al. (2000) studied treatment seeking individuals and found that 39% of them were CG (Figure 3), closely replicating Table 1. If 36-39% of the clinical population are CG, then interventions like stimulus control and sleep restriction are attempting to modify good sleep behavior in these individuals. Even more disturbing, perhaps 36-39% of people treated for insomnia have been prescribed hypnotics and exposed to hypnotic side effects, when sleep was already good.

According to the most recent recommendations by the American Academy of Sleep Medicine (Littner et al., 2003), PSG is not recommended in the routine assessment of insomnia, other forms of sleep monitoring such as actigraphy and sleep diaries are not required, and the most common clinical practice is to assess sleep by interview. These practice guidelines may not work well for uncoupled sleepers because the evaluation of sleep and sleep complaint are not well differentiated by interview.

NP do not show up at our clinics or volunteer for clinical research. They are a clinically invisible population. But NP is an interesting group and could perhaps inform us about healthy traits, such as resilience, coping, and stubborn optimism. The vast literature on psychological factors in illness, has focused on personality predictors of disease, but has also revealed the hypo-reactivity of Type B personality, a harbinger of health (Smith & Gallo, 2001). It would be helpful to learn the source of NP and processes by which it is acquired.

A critical role of review papers is to plan a course of future research (Torraco, 2005), and the usual importance of this aspect is elevated further in reviews of nascent research areas. The research agenda described earlier in this paper identifies three main areas of inquiry: sources, clinical mechanisms, and clinical management of insomnia identity. I suggest plausible avenues

of investigation for each topic to strategically expand knowledge of this concept, illuminate its import, and curb its impact. These lines of research hold the promise of securing incremental therapeutic gains for people with insomnia.

When dread intermingles with sleep, the bedroom is a neutral or welcoming environment by day, a dystopia by night. But for some, though the night problem exists only in their mind, it still degrades experience during both night and day. Findings that sleep and sleep complaints are not always synchronous are not new. Investigators have long observed discrepancies between sleep and sleep complaints (Adam, Tomeny, & Oswald, 1986; Carskadon et al., 1976; Ohayon et al., 1997; Rosa & Bonnet, 2000), but the research reported in this paper engaged in systematic inquiry to more clearly reveal salient attributes and established the foundation for a science of uncoupled sleepers.

Acknowledgements

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

This paper benefitted from comments on an earlier draft by graduate students, Sarah Emert and Hylton Molzof, and colleagues, Daniel Buysse and Christina McCrae.

References

Adam, K., Tomeny, M., & Oswald, I. (1986). Physiological and psychological differences between good and poor sleepers. *Journal of Psychiatric Research, 20*, 301-316.

Aikens, J. E., & Rouse, M. E. (2005). Help-seeking for insomnia among adult patients in primary care. *Journal of the American Board of Family Practice, 18*, 257-261.

Alapin, I., Fichten, C. S., Libman, W., Creti, L., Bailes, S., & Wright, J. (2000). How is good and poor sleep in older adults and college students related to daytime sleepiness, fatigue, and ability to concentrate? *Journal of Psychosomatic Research*, *49*, 381-390.

American Academy of Sleep Medicine. (2014). *International classification of sleep disorders, third edition: Diagnostic and coding manual*. Darien, IL: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders, fifth edition, DSM-5*. Arlington, VA: Author.

Barclay, N. L., & Gregory, A. M. (2010). The presence of a perseverative iterative style in poor vs. good sleepers. *Journal of Behavior Therapy and Experimental Psychiatry, 41*, 18-23.

Bartlett, D. J., Marshall, N. S., Williams, A., & Grunstein, R. R. (2008). Predictors of primary medical care consultation for sleep disorders. *Sleep Medicine, 9*, 857–864.

Bathgate, C. J., Edinger, J. D., Wyatt, J. K., & Krystal, A. D. (2016). Objective but not subjective short sleep duration associated with increased risk for hypertension in individuals with insomnia. *Sleep, 39*, 1037–1045.

Benca, R. M. (2005). Diagnosis and treatment of chronic insomnia: A review. *Psychiatric Services, 56*, 332–343.

Berscheid, E. (2003). The human's greatest strength: Other humans. In L. G. Aspinwall & U. M. Staudinger (Eds.), *A psychology of human strengths: Fundamental questions and future*

directions for a positive psychology (pp. 37–47). Washington, DC: American Psychological Association.

Black, D. S., O'Reilly, G. A., Olmstead, R., Breen, E. C., & Irwin, M. R. (2015). Mindfulness meditation and improvement in sleep quality and daytime impairment among older adults with sleep disturbances: A randomized clinical trial. *JAMA Internal Medicine, 175*, 494-501.

Bootzin, R. R., Herman, C. P., & Nicassio, P. (1976). The power of suggestion: Another examination of misattribution and insomnia. *Journal of Personality and Social Psychology, 34*, 673-679.

Borkovec, T. D., & Fowles, D. C. (1973). Controlled investigation of the effects of progressive and hypnotic relaxation on insomnia. *Journal of Abnormal Psychology, 82*, 153-158.

Burcusa, S. L., & Iacono, W. G. (2007). Risk for recurrence in depression. *Clinical Psychology Review, 27*, 959-985.

Byers, H. D., Lichstein, K. L., & Thorn, B. E. (2016). Cognitive processes in comorbid poor sleep and chronic pain. *Journal of Behavioral Medicine, 39*, 233-240.

Carey, T. J., Moul, D. E., Pilkonis, P., Germain, A., & Buysse, D. J. (2005). Focusing on the experience of insomnia. *Behavioral Sleep Medicine, 3*, 73-86.

Carney, C. E., Harris, A. L., Falco, A., & Edinger, J. D. (2013). The relation between insomnia symptoms, mood, and rumination about insomnia symptoms. *Journal of Clinical Sleep Medicine, 9*, 567-575.

Carskadon, M. A., Dement, W. C., Mitler, M. M., Guilleminault, C., Zarcone, V. P., & Spiegel, R. (1976). Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *American Journal of Psychiatry, 133*, 1382-1388.

Corrigan, P. (2004). How stigma interferes with mental health care. *American Psychologist, 59*,

614–625.

Corrigan, P. W., & Watson, A. C. (2002). The paradox of self-stigma and mental illness. *Clinical Psychology: Science and Practice, 9*, 35–53.

Drake, C. L., Hays, R. D., Morlock, R., Wang, F., Shikiar, R., Frank, L., . . . Roth, T. (2014). Development and evaluation of a measure to assess restorative sleep. *Journal of Clinical Sleep Medicine, 10*, 733-741.

Edinger, J. D., Fins, A. I., Glenn, D. M., Sullivan, R. J., Jr., Bastian, L. A., Marsh, G. R., . . . Vasilas, D. (2000). Insomnia and the eye of the beholder: Are there clinical markers of objective sleep disturbances among adults with and without insomnia complaints? *Journal of Consulting and Clinical Psychology, 68*, 586-593.

Edinger, J. D., & Krystal, A. D. (2003). Subtyping primary insomnia: is sleep state misperception a distinct clinical entity? *Sleep Medicine Reviews, 7*, 203-214.

Elgersma, H. J., Glashouwer, K. A., Bockting, C. L. H., Penninx, B. W. J. H., & de Jong, P. J. (2013). Hidden scars in depression? Implicit and explicit self-associations following recurrent depressive episodes. *Journal of Abnormal Psychology, 122*, 951-960.

Espie, C. A., Brooks, D. N., & Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 20*, 143-153.

Espie, C. A., Inglis, S. J., & Harvey, L. (2001). Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: Analyses of outcome data at the 12 months posttreatment. *Journal of Consulting and Clinical Psychology, 69*, 58–66.

Estivill, E. (2002). Behaviour of insomniacs and implication for their management. *Sleep*

Medicine Reviews, 6(Suppl. 1), S3-S6.

Fernandez-Mendoza, J., Calhoun, S. L., Bixler, E. O., Karataraki, M., Liao, D., Vela-Bueno, A., … Vgontzas, A. N. (2011). Sleep misperception and chronic insomnia in the general population: Role of objective sleep duration and psychological profiles. *Psychosomatic Medicine, 73*, 88–97.

Fernandez-Mendoza, J., Calhoun, S., Bixler, E. O., Pejovic, S., Karataraki, M., Liao, D., … Vgontzas, A. N. (2010). Insomnia with objective short sleep duration is associated with deficits in neuropsychological performance: a general population study. *Sleep, 33*, 459-465.

Fernandez-Mendoza, J., Shea, S., Vgontzas, A. N., Calhoun, S. L., Liao, D., & Bixler, E. O. (2015). Insomnia and incident depression: role of objective sleep duration and natural history. *Journal of Sleep Research, 24*, 390–398.

Fernandez-Mendoza, J., Vgontzas, A. N., Liao, D., Shaffer, M. L., Vela-Bueno, A., Basta, M., & Bixler, E. O. (2012). Insomnia with objective short sleep duration and incident hypertension: The Penn State Cohort. *Hypertension, 60*, 929-935.

Fichten, C. S., Creti, L., Amsel, R., Brender, W., Weinstein, N., & Libman, E. (1995). Poor sleepers who do not complain of insomnia: Myths and realities about psychological and lifestyle characteristics of older good and poor sleepers. *Journal of Behavioral Medicine, 18*, 189-223.

Fichten, C. S., Libman, E., Creti, L., Amsel, R., Sabourin, S., Brender, W., & Bailes, S. (2001). Role of thoughts during nocturnal awake times in the insomnia experience of older adults. *Cognitive Therapy and Research, 25*, 665-692.

Frank, J. D. (1968). The influence of patients' and therapists' expectations on the outcome of psychotherapy. *British Journal of Medical Psychology, 41*, 349-356.

Harvey, A. G. (2000). Pre-sleep cognitive activity: A comparison of sleep-onset insomniacs and good sleepers. *British Journal of Clinical Psychology, 39*, 275-286.

Harvey, A. G. (2001). I can't sleep, my mind is racing! An investigation of strategies of thought control in insomnia. *Behavioural and Cognitive Psychotherapy, 29*, 3-11.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869-893.

Harvey, A. G., & Greenall, E. (2003). Catastrophic worry in primary insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 34*, 11-23.

Harvey, A. G., & Spielman, A. J. (2011). Insomnia: Diagnosis, assessment, and outcomes. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 838-849). St. Louis: Saunders.

Harvey, A. G., & Tang, N. K. Y. (2003). Cognitive behaviour therapy for primary insomnia: Can we rest yet? *Sleep Medicine Reviews, 7*, 237-262.

Hauri, P. (1969). The influence of evening activity on the onset of sleep. *Psychophysiology, 5*, 426-430.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2011). *Acceptance and commitment therapy: The Process and practice of mindful change* (2nd ed.). New York: Guilford.

Henry, D., Rosenthal, L., Dedrick, D., & Taylor, D. (2013). Understanding patient responses to insomnia. *Behavioral Sleep Medicine, 11*, 40-55.

Hinshaw, S. P. (2007). *The mark of shame: Stigma of mental illness and an agenda for change*. New York: Oxford University Press.

Jahn, D. L., & Lichstein, K. L. (1980). The resistive client: A neglected phenomenon in behavior therapy. *Behavior Modification, 4*, 303-320.

Johnson, E. O., Roehrs, T., Roth, T., & Breslau, N. (1998). Epidemiology of alcohol and medication as aids to sleep in early adulthood. *Sleep, 21*, 178-186.

Jones, B. T., Macphee, L. M., Broomfield, N. M., Jones, B. C., & Espie, C. A. (2005). Sleep-related attentional bias in good, moderate, and poor (primary insomnia) sleepers. *Journal of Abnormal Psychology, 114*, 249-258.

Kay, D. B., Karim, H. T., Soehner, A. M., Hasler, B. P., Wilckens, K. A., James, J. A., . . . Buysse, D. J. (2016). Sleep-wake differences in relative regional cerebral metabolic rate for glucose among patients with insomnia compared with good sleepers. *Sleep, 39*, 1779-1794.

Kim, N. S., & Ahn, W. (2002a). Clinical psychologists' theory-based representations of mental disorders predict their diagnostic reasoning and memory. *Journal of Experimental Psychology: General, 131*, 451-476.

Kim, N. S., & Ahn, W. (2002b). The influence of naive causal theories on lay concepts of mental illness. *American Journal of Psychology, 115*, 33-65.

Kyle, S. D., Espie, C. A., & Morgan, K. (2010). ": : : Not just a minor thing, it is something major, which stops you from functioning daily": Quality of life and daytime functioning in insomnia. *Behavioral Sleep Medicine, 8*, 123-140.

Leger, D., & Poursain, B. (2005). An international survey of insomnia: under-recognition and under-treatment of a polysymptomatic condition. *Current Medical Research and Opinion, 21*, 1785-1792.

Lichstein, K. L., & Rosenthal, T. L. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *Journal of Abnormal Psychology, 89*, 105-107.

Lichstein, K. L., Durrence, H. H., Riedel, B. W., Taylor, D. J., & Bush, A. J. (2004). *Epidemiology of sleep: Age, gender, and ethnicity*. Mahwah, NJ: Erlbaum.

Lichstein, K. L., Durrence, H. H., Taylor, D. J., Bush, A. J., & Riedel, B. W. (2003). Quantitative criteria for insomnia. *Behaviour Research and Therapy, 41*, 427-445.

Lichstein, K. L., Taylor, D. J., McCrae, C. S., & Petrov, M. E. (2017). Insomnia: Epidemiology and risk factors. In M. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (6th ed., pp. 761-768). Philadelphia: Elsevier.

Lichstein, K. L., Vander Wal, G. S., & Dillon, H. R. (2012). Insomnia III: Therapeutic approaches. In C. M. Morin & C. A. Espie (Eds.), *The Oxford handbook of sleep and sleep disorders* (pp. 453-470). New York: Oxford University Press.

Littner, M., Hirshkowitz, M., Kramer, M., Kapen, S., Anderson, W. M., Bailey, D., . . . & Woodson, B. T. (2003). Practice parameters for using polysomnography to evaluate insomnia: An update. *Sleep, 26*, 754-760.

Lundh, L. G., & Broman, J. E. (2000). Insomnia as an interaction between sleep-interfering and sleep-interpreting processes. *Journal of Psychosomatic Research, 49*, 299-310.

Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: Does happiness lead to success? *Psychological Bulletin, 131*, 803–855.

MacDonald, S., Linton, S. J., & Jansson-Fröjmark, M. (2008). Avoidant safety behaviors and catastrophizing: Shared cognitive-behavioral processes and consequences in co-morbid pain and sleep disorders. *International Journal of Behavioral Medicine, 15*, 201-210.

MacMahon, K. M. A., Broomfield, N. M., & Espie, C. A. (2006). Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome using the dot-probe task. *Sleep, 29*, 1420-1427.

McCrae, C. S., Rowe, M. A., Tierney, C. G., Dautovich, N. D., DeFinis, A. L., & McNamara, J. P. H. (2005). Sleep complaints, subjective and objective sleep patterns, health, psychological

adjustment, and daytime functioning in community-dwelling older adults. *Journal of Gerontology: Psychological Sciences, 60B*, P182-P189.

McCrae, C. S., Wilson, N. W., Lichstein, K. L., Durrence, H. H., Taylor, D. J., Bush, A. J., & Riedel, B. W. (2003). 'Young old' and 'old old' poor sleepers with and without insomnia complaints. *Journal of Psychosomatic Research, 54*, 11-19.

McHugh, R. K., Whitton, S. W., Peckham, A. D., Welge, J. A., & Otto, M. W. (2013). Patient preference for psychological vs pharmacologic treatment of psychiatric disorders: A meta-analytic review. *Journal of Clinical Psychiatry, 74*, 595-602.

Morin, C. M., Blais, F., & Savard, J. (2002). Are changes in beliefs and attitudes about sleep related to sleep improvements in the treatment of insomnia? *Behaviour Research and Therapy, 40*, 741-752.

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998-2004). *Sleep, 29*, 1398-1414.

Morin, C. M., Gaulier, B., Barry, T., & Kowatch, R. A. (1992). Patients' acceptance of psychological and pharmacological therapies for insomnia. *Sleep, 15*, 302-305.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A. J., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia. *Sleep, 22*, 1134-1156.

Morin, C. M., LeBlanc, M., Daley, M., Gregoire, J. P., & Merette, C. (2006). Epidemiology of insomnia: Prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. *Sleep Medicine, 7*, 123-130.

Morin, C. M., Rodrigue, S., & Ivers, H. (2003). Role of stress, arousal, and coping skills in primary insomnia. *Psychosomatic Medicine, 65*, 259-267.

Morin, C. M., Savard, J., & Blais, F. C. (2000). Cognitive therapy. In K. L. Lichstein & C. M. Morin (Eds.), *Treatment of late-life insomnia* (pp. 207-230). Thousand Oaks, CA: Sage.

Morin, C. M., & Wooten, V. (1996). Psychological and pharmacological approaches to treating insomnia: Critical issues in assessing their separate and combined effects. *Clinical Psychology Review, 16*, 521-542.

Mulla, M. M., Lewis, J. A., Hamilton, J. C., Tutek, J., Emert, S. E., Witte, T. H., & Lichstein, K. L. (in press). *The role of perceived sleep norms in subjective sleep appraisals and sleep-related illness behavior*. *Journal of Behavioral Medicine*.

Nelson, J., & Harvey, A. G. (2003a). An exploration of pre-sleep cognitive activity in insomnia: Imagery and verbal thought. *British Journal of Clinical Psychology, 42*, 271-288.

Nelson, J., & Harvey, A. G. (2003b). Pre-sleep imagery under the microscope: a comparison of patients with insomnia and good sleepers. *Behaviour Research and Therapy, 41*, 273-284.

Nezu, A. M., Nezu, C. M., & Lombardo, E. R. (2001). Cognitive-behavior therapy for medically unexplained symptoms: A critical review of the treatment literature. *Behavior Therapy, 32*, 537-583.

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the pre-sleep state: The development of the pre-sleep arousal scale. *Behaviour Research and Therapy, 23*, 263-271.

Ohayon, M. M., Caulet, M., Priest, R. G., & Guilleminault, C. (1997). DSM-IV and ICSD-90 insomnia symptoms and sleep dissatisfaction. *British Journal of Psychiatry, 171*, 382–388.

Ohayon, M. M., & Smirne, S. (2002). Prevalence and consequences of insomnia disorders in the general population of Italy. *Sleep Medicine, 3*, 115-120.

Okajima, I., Komada, Y., & Inoue, Y. (2011). A meta-analysis on the treatment effectiveness of

cognitive behavioral therapy for primary insomnia. *Sleep and Biological Rhythms, 9*, 24-34.

Ong, J. C., Manber, R., Segal, Z., Xia, Y., Shapiro, S., & Wyatt, J. K. (2014). A randomized controlled trial of mindfulness meditation for chronic insomnia. *Sleep, 37*, 1553-1563.

Ong, J. C., Ulmer, C. S., & Manber, R. (2012). Improving sleep with mindfulness and acceptance: A metacognitive model of insomnia. *Behaviour Research and Therapy, 50*, 651-660.

Palagini, L., Bruno, R. M., Paolo, T., Caccavale, L., Gronchi, A., Mauri, M., . . . Drake, C. L. (2016). Association between stress-related sleep reactivity and metacognitive beliefs about sleep in insomnia disorder: Preliminary results. *Behavioral Sleep Medicine*, 14, 636-649.

Pattyn, E., Verhaeghe, M., Sercu, C., & Bracke, P. (2014). Public stigma and self-stigma: Differential association with attitudes toward formal and informal help seeking. *Psychiatric Services, 65*, 232-238.

Perlis, M., Shaw, P. J., Cano, G., & Espie, C. A. (2011). Models of insomnia. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 850-865). St. Louis: Saunders.

Perlis, M. L., Smith, M. T., Andrews, P. J., Orff, H., & Giles, D. E. (2001). Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep, 24*, 110-117.

Ree, M. J., & Harvey, A. G. (2006). Interpretive biases in chronic insomnia: An investigation using a priming paradigm. *Behavior Therapy, 37*, 248-258.

Ree, M. J., Pollitt, A., & Harvey, A. G. (2006). An investigation of interpretive bias in insomnia: An analog study comparing normal and poor sleepers. *Sleep, 29*, 1359-1362.

Riedel, B. W., & Lichstein, K. L. (2000). Insomnia and daytime functioning. *Sleep Medicine*

Reviews, 4, 277-298.

Rosa, R. R., & Bonnet, M. H. (2000). Reported chronic insomnia is independent of poor sleep as measured by electroencephalography. *Psychosomatic Medicine, 62*, 474-482.

Semler, C. N., & Harvey, A. G. (2004). An investigation of monitoring for sleep-related threat in primary insomnia. *Behaviour Research and Therapy, 42*, 1403-1420.

Semler, C. N., & Harvey, A. G. (2005). Misperception of sleep can adversely affect daytime functioning in insomnia. *Behaviour Research and Therapy, 43*, 843-856.

Semler, C. N., & Harvey, A. G. (2006). Daytime functioning in primary insomnia: Does attentional focus contribute to real or perceived impairment? *Behavioral Sleep Medicine, 4*, 85-103.

Semler, C. N., & Harvey, A. G. (2007). An experimental investigation of daytime monitoring for sleep-related threat in primary insomnia. *Cognition and Emotion, 21*, 146-161.

Smith, M. T., Perlis, M. L., Smith, M. S., Giles, D. E. & Carmody, T. P. (2000). Sleep quality and presleep arousal in chronic pain. *Journal of Behavioral Medicine, 23*, 1-13.

Smith, T. W., & Gallo, L. C. (2001). Personality traits as risk factors for physical illness. In A. Baum, T. A. Revenson, & J. E Singer (Eds.), *Handbook of health psychology* (pp. 139-173). Mahwah, NJ: Erlbaum.

Spielman, A. J., Conroy, D., & Glovinsky, P. B. (2003). Evaluation of insomnia. In M. L. Perlis & K. L. Lichstein (Eds.), *Treating sleep disorders: Principles and practice of behavioral sleep medicine* (pp. 190-213). New York: Wiley.

Stinson, K., Tang, N. K. Y., & Harvey, A. G. (2006). Barriers to treatment seeking in primary insomnia in the United Kingdom: A cross-sectional perspective. *Sleep, 29*, 1643-1646.

Stone, K. C., Taylor, D. J., McCrae, C. S., Kalsekar, A., & Lichstein, K. L. (2008).

Nonrestorative sleep. *Sleep Medicine Reviews, 12*, 275-288.

Sullivan, M. J. L., Thorn, B., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., & Lefebvre, J. C. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *Clinical Journal of Pain, 17*, 52-64.

Sunnhed, R., & Jansson-Frojmark, M. (2015). Cognitive arousal, unhelpful beliefs and maladaptive sleep behaviors as mediators in cognitive behavior therapy for insomnia: A quasi-experimental study. *Cognitive Therapy and Research, 39*, 841-852.

Tang, N. K. Y., & Harvey, A. G. (2004). Correcting distorted perception of sleep in insomnia: a novel behavioural experiment? *Behaviour Research and Therapy, 42*, 27-39.

Tang, N. K. Y., Schmidt, D. A., & Harvey, A. G. (2007). Sleeping with the enemy: Clock monitoring in the maintenance of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 38*, 40-55.

Torraco, R. J. (2005). Writing integrative literature reviews: Guidelines and examples. *Human Resource Development Review, 4*, 356-367.

Ustinov, Y., Lichstein, K. L., Vander Wal, G. S., Taylor, D. J., Riedel, B. W., & Bush, A. J. (2010). Association between report of insomnia and daytime functioning. *Sleep Medicine, 11*, 65-68.

Vgontzas, A. N., Liao, D., Bixler, E. O., Chrousos, G. P., & Vela-Bueno, A. (2009). Insomnia with objective short sleep duration is associated with a high risk for hypertension. *Sleep, 32*, 491-497.

Vgontzas, A. N., Liao, D., Pejovic, S., Calhoun, S., Karataraki, M., Basta, M., … Bixler, E. O. (2010). Insomnia with short sleep duration and mortality: The Penn State cohort. *Sleep, 33*, 1159-1164.

Vgontzas, A. N., Liao, D., Pejovic, S., Calhoun, S. L., Karataraki, M., & Bixler, E. O. (2009). Insomnia with objective short sleep duration is associated with Type 2 diabetes. *Diabetes Care, 32*, 1980-1985.

Walen, S. R., DiGiuseppe, R., & Dryden, W. (1992). *A practitioner's guide to rational-emotive therapy* (2nd ed.). New York: Oxford University Press.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy, 38*, 679-693.

Winsper, C., & Tang, N. K. Y. (2014). Linkages between insomnia and suicidality: Prospective associations, high-risk subgroups and possible psychological mechanisms. *International Review of Psychiatry, 26*, 189-204.

Woosley, J. A., Lichstein, K. L., Taylor, D. J., Riedel, B. W., & Bush, A. J. (2016). Insomnia complaint versus sleep diary parameters: Predictions of suicidal ideation. *Suicide and Life-Threatening Behavior, 46*, 88-95.

Table 1

Distribution of noncomplaining poor sleepers (NP), noncomplaining good sleepers (NG), complaining poor sleepers (CP), and complaining good sleepers (CG) across two studies.

	CP (Insomnia)	NP	CG	NG (Normal Sleep)	Total
	N (%)	N (%)	N (%)	N (%)	N
Lichstein et al., 2003	137 (19)	120 (16)	77 (10)	401 (55)	735
McCrae et al., 2005	19 (18)	24 (23)	11 (11)	49 (48)	103
Combined	156 (19)	144 (17)	88 (11)	450 (54)	838

Table 2

Distribution of noncomplaining poor sleepers (NP), noncomplaining good sleepers (NG), complaining poor sleepers (CP), and complaining good sleepers (CG) across three studies from the Penn State Cohort.

	CP (Insomnia)	NP	CG	NG (Normal Sleep)	Total
	N (%)	N (%)	N (%)	N (%)	N
Fernandez-Mendoza et al., 2011	82 (9)	365 (42)	60 (7)	359 (41)	866
Fernandez-Mendoza et al., 2010	51 (8)	219 (32)	65 (10)	343 (51)	678
Vgontzas et al., 2010	33 (4)	344 (46)	22 (3)	342 (46)	741
Average	55 (7)	309 (41)	49 (6)	348 (46)	761

		Insomnia Complaint	
		Present	Absent
Poor Sleep	Present	① complaining poor sleeper (CP) [insomnia]	② noncomplaining poor sleeper (NP)
	Absent	③ complaining good sleeper (CG)	④ noncomplaining good sleeper (NG) [normal sleep]

Figure 1. The figure presents a conceptualization of sleep/complaint categories by crossing presence or absence of a sleep complaint with presence or absence of poor sleep. The principal diagonal (quadrants 1 and 4) conveys common characterizations wherein poor sleep and sleep complaints are either both present or both absent. The off diagonal categories (quadrants 2 and 3, highlighted) convey mismatches wherein poor sleep occurs without sleep complaint or good sleep is accompanied by a sleep complaint. Sleep and sleep complaint are asynchronous in quadrants 2 and 3. Individuals characterized by these two quadrants may be termed uncoupled sleepers.

		Insomnia Complaint	
		Present	Absent
Poor Sleep	Present	137	120
	Absent	77	401

Figure 2. Distribution of sleep types from Figure 2b in Lichstein et al. (2003). The off diagonal (highlighted) indicates 27% of subjects (197 of 735) are uncoupled sleepers.

		Insomnia Complaint	
		Present	Absent
Poor Sleep	Present	35	22
	Absent	22	29

Figure 3. Distribution of sleep types from Edinger et al. (2000). The off diagonal (highlighted) indicates 41% of subjects (44 of 108) are uncoupled sleepers.

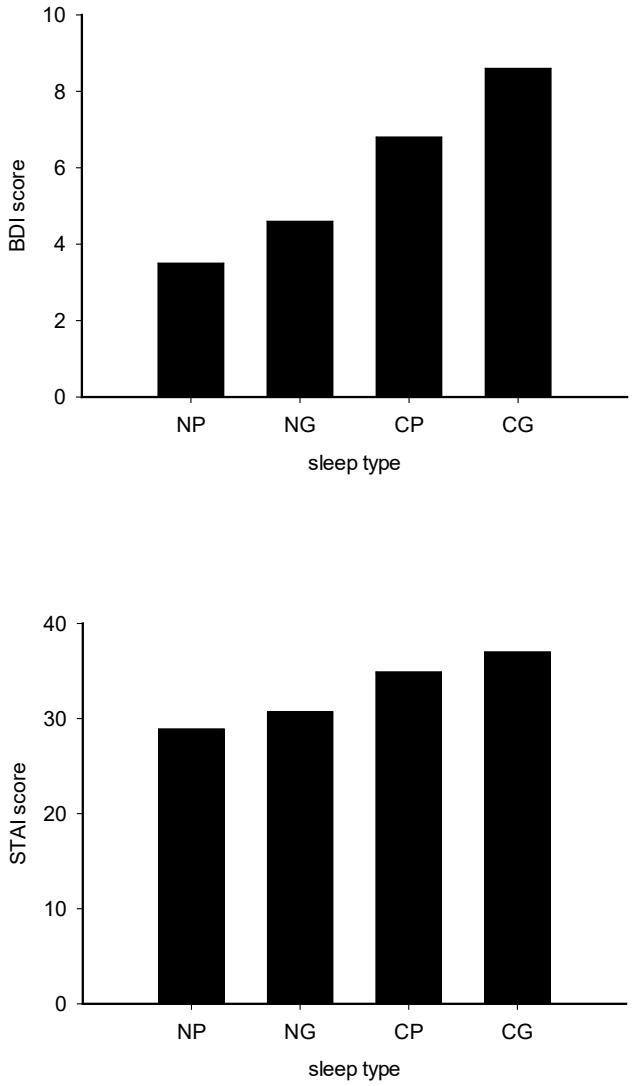

Figure 4. Edinger et al. (2000) data on depression (Beck Depression Inventory, top panel) and anxiety (State-Trait Anxiety Inventory, bottom panel) comparing noncomplaining poor sleepers (NP), noncomplaining good sleepers (NG), complaining poor sleepers (CP), and complaining good sleepers (CG).

		Insomnia Complaint	
		Present	Absent
Poor Sleep	Present	19	24
	Absent	11	49

Figure 5. Distribution of sleep types from McCrae et al. (2005). The off diagonal (highlighted) indicates 34% of subjects (35 of 103) are uncoupled sleepers.

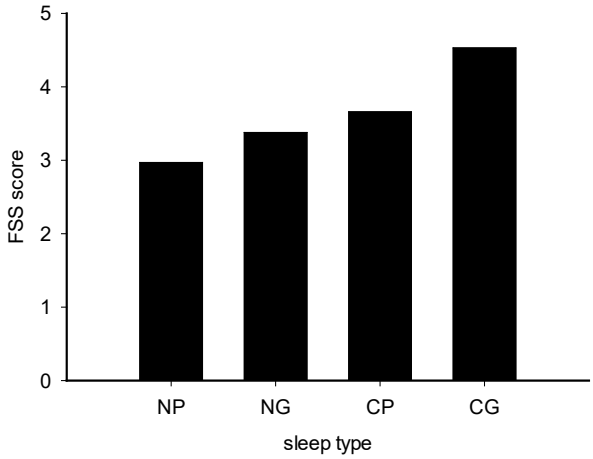

Figure 6. McCrae et al. (2005) data on fatigue (Fatigue Severity Scale) comparing noncomplaining poor sleepers (NP), noncomplaining good sleepers (NG), complaining poor sleepers (CP), and complaining good sleepers (CG).