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INSOMNIA: Conceptual Issues in the Development, Persistence, and Treatment of Sleep Disorder in Adults

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■ **Abstract** This paper critically reviews the evidence base for previously reported conceptual models of the development and persistence of insomnia. Although a number of perspectives have some empirical support, no one approach emerges as preeminent. Importantly, the efficacy of any particular psychological intervention cannot be taken as confirmation of presumed, underlying mechanisms. An integrated psychobiological inhibition model of insomnia is developed that accounts for the research data. The model views insomnia as arising from inhibition of de-arousal processes associated with normal sleep. It is proposed that sleep homeostatic and circadian factors are compromised by impairment of the automaticity and plasticity associated with good sleep, and that cognitive/affective processes activate the clinical complaint of insomnia. Common pathways for the action of cognitive-behavioral interventions are identified, and a research agenda is set for further conceptual and clinical study.

CONTENTS

AN INTRODUCTION TO INSOMNIA

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) defines primary insomnia as a complaint lasting for at least 1 month of difficulty initiating and/or maintaining sleep or of nonrestorative sleep (Am. Psychiatr. Assoc. 1994). The International Classification of Sleep Disorders-Revised (ICSD-R) uses the term "psychophysiologic insomnia" for such a complaint and associated decreased functioning during wakefulness. (Am. Sleep Disorders Assoc. 1997). ICSD-R regards insomnia of 6-month duration as chronic. Both systems differentiate insomnia from circadian rhythm disorders, in which timing of the major sleep period is out of alignment with the local clock; from parasomnias, in which behavioral events occur in association with sleep (e.g., sleepwalking, night terrors); and from secondary insomnias, in which psychiatric, neurologic, or medical problems present. Disorders such as sleep apnea, with associated respiratory impairment, and disorders of excessive sleepiness (e.g., narcolepsy) are also classified separately.

Conservative estimates for chronic insomnia range from 9–12% in adulthood and up to 20% in later life, and women present about two times more than men (Bixler et al. 1979, Mellinger et al. 1985, Ford & Kamerow 1989, Gallup Organisation 1991, Foley et al. 1995, Hoch et al. 1997). Sleep disturbance is a common complaint in general practice (Shocat et al. 1999) and once established may persist over many years (Mendelson 1995). Insomnia therefore constitutes a considerable public health problem. The direct costs of assessing and treating insomnia were approximated as $14 billion in the United States and FF10 billion in France in 1995 (Walsh & Engelhardt 1999, Leger et al. 1999).

Polysomnographic assessment (PSG) comprises monitoring of the electroencephalogram (EEG) along with muscle activity, eye movement, respiration, and blood oxygen saturation levels. However, PSG is not required unless clinical presentation raises the possibility of disorders such as sleep apnea (Gillin & Byerley 1990, Douglas et al. 1992, Reite et al. 1995, Am. Sleep Disorders Assoc. 1995a). Actigraphic assessment is helpful to identify disorders of circadian function. The wrist actigraph provides data on body movement over extended periods (typically 1 minute epochs for several weeks) and is a reliable index of sleep parameters (Sadeh et al. 1995, Am. Sleep Disorders Assoc. 1995b).

In practice, structured interview is recommended (Buysse et al. 1989, Morin 1993, Spielman & Anderson 1999, Espie 2000), supplemented by a sleep diary completed upon waking, comprising information on sleep-onset latency (SOL),

wake time after sleep-onset, and total sleep time (Espie 1991). Time in bed (TIB) is calculated by subtracting rising time from bedtime, and the sleep efficiency index is computed as the ratio of total sleep time to TIB expressed as a percentage. A sleep efficiency index of 85% is the upper limit for poor sleep (Frankel et al. 1976, Coates et al. 1982), and a minimum SOL or wake time after sleep-onset of 30 minutes per night is a threshold for clinical significance (Espie et al. 1989a, 2001a). Baseline values in outcome studies, however, are typically twice this level (Morin et al. 1999b). With medication, alcohol, and ratings of sleep quality commonly included, the sleep diary quantifies sleep experience and permits study of night-to-night variability, which is important because unpredictability of sleep is a feature of insomnia (Coates et al. 1982, Roth et al. 1976).

The American Academy of Sleep Medicine (AASM) has published practice parameters for the assessment (Chesson et al. 2000) and nonpharmacological treatment of insomnia (Chesson et al. 1999). The latter were derived from systematic review (Morin et al. 1999b) following meta-analyses demonstrating the efficacy of cognitive-behavioral treatment (CBT) for adults (Morin et al. 1994, Murtagh & Greenwood 1995). Another review came to similar conclusions (Edinger & Wohlgemuth 1999). Furthermore, clinical effectiveness studies have produced comparable results (Espie et al. 2001a,b), and CBT with or without pharmacotherapy compares favorably with pharmacotherapy alone for older adults (Morin et al. 1999a). CBT, therefore, may be the treatment of first choice for chronic insomnia (Espie 1999). The role of hypnotic medication has been debated (Kripke 2000, Kramer 2000, Buysse 2000), there being little evidence of long-term efficacy for any drug. Recent textbooks also provide useful discussion: Lichstein & Morin (2000) focus on late-life insomnia, and Pressman & Orr (1997) provide an overview of insomnia in medical patients. Finally, an AASM working group on research diagnostic criteria will report in 2002 (Edinger et al. 2002). Thus, there is a sizeable, systematic, and practitioner-oriented literature on insomnia. The conceptual foundations of insomnia, however, have been more neglected, and it seems timely to review models of the development, persistence, and treatment of insomnia, to evaluate explanatory mechanisms, and to propose an evidence-based integrated model.

THE CONCEPTUAL BASIS FOR THE DEVELOPMENT AND MAINTENANCE OF INSOMNIA

A wide range of factors may play some part in insomnia. It may be helpful to consider each of these before presenting a proposed integrated model.

Normal Sleep in Human Development

Wakefulness is not pathological. On the contrary, it might be considered the preferred state because a primary function of sleep is to ensure wakeful cortical

function (Horne 1988). Prolonged wakefulness reliably induces sleep, and failure to obtain at least a core amount (sleep deprivation) leads to impaired function. Two processes interact in normal sleep (Borbely 1994). The sleep homeostat "drives" the sleep-wake schedule toward a balanced requirement because prolonged wakefulness accrues "sleep debt" (Carskadon & Dement 1981) and sleep pays off the debt; the circadian timer regulates the biological clock in approximation to the 24-hour clock (Borbely 1994, Moore-Ede et al. 1982). These processes also regulate type of sleep (Dement 1960). Thus, young children have longer sleep periods than adults and have higher proportions of rapid eye movement (REM) and "deep sleep" (non-REM stages 3 and 4). Similarly, insufficient sleep induces recovery sleep comprising proportionately more REM and deep sleep. The sleep of older adults is more fragmented and lighter (Bliwise 1993, Carskadon et al. 1982), and homeostatic drive declines with age (Buysse et al. 1993), when disturbances of the sleep-wake schedule are less well tolerated (Webb 1981, Monk et al. 1992). Thus, increasing age represents a vulnerability factor to sleep disturbance.

Quality of Sleep

Investigations of PSG and self-report generally reveal modest positive correlation. Poor sleepers overestimate sleep disturbance relative to objective criteria (see Espie 1991, pp. 17–18). However, insomniacs have reported being awake when roused from light sleep (Borkovec et al. 1981), and modified EEG criteria are associated with greater accuracy (Coates et al. 1982). There is debate over whether insomniacs are sleepy in the daytime (Seidel et al. 1984, Mendelson et al. 1984, Chambers & Keller 1993, Lichstein et al. 1994). However, sleep should not be considered only in terms of chronobiological "fitness for purpose." The quality of the sleep experience is important. Indeed, ratings of sleep quality do not necessarily correlate highly even with subjective reports of sleep pattern. Unpublished analyses from our recent clinical cohort (Espie et al. 2001b; $n = 139$) reveal modest inverse association of SOL and wake time after sleep-onset with ratings of "sleep enjoyment" ($r = -0.30$ and -0.31) and "restedness after sleep" (-0.22 and -0.19).

Predisposing, Precipitating, and Perpetuating Factors

How then might insomnia develop? A useful conceptualization comprises predisposing, precipitating, and perpetuating components (Spielman & Glovinsky 1991). Both DSM-IV and ICSD-R report familial association with light, disrupted sleep, and ICSD-R reports anxious over-concern with health as predisposing. Indeed, insomniacs appear prone to introspection and worry (e.g., Kales et al. 1984, Edinger et al. 1988, Lundh et al. 1995, Schramm et al. 1995). Research suggesting elevated autonomic and metabolic rates also implies a vulnerability factor (Bonnet & Arand 1995, 1997a). However, predisposing factors alone are unlikely to create imbalance in sleep homeostasis or circadian timing, although they might impair sleep quality and, potentially, lead to sleep state misperception (see also Bonnet &

Arand 1997b). Reduced "plasticity" (see below) might also be a predispositional factor.

Transient sleep disorder is a likely context in which to identify precipitating factors. ICSD-R defines adjustment sleep disorder associated temporally with acute stress, conflict, or environmental change, and shiftwork schedule disorder as a transient phenomenon relating to work schedules. Because the homeostat and timer regulate natural variation, it can be hypothesized that there is generally sufficient plasticity to absorb the impact of such events, and to survive more prolonged change. The severity and impact of events may need to be greater to precipitate sleep disturbance in the absence of predisposing factors. Nevertheless, studies investigating the onset of chronic insomnia have commonly found that stress or life change was a factor at the time (Healey et al. 1981, Kales & Vgontzas 1992, Morgan & Clarke 1997). Presumably, some other mechanism accounts for the persistence of a sleep problem. Why only some disturbances develop into chronic insomnia when others spontaneously remit, and why good sleep can persist during chronic stress are important questions requiring further study.

To summarize, predisposition may interact with precipitating factors to create temporary sleep disruption, but in the absence of perpetuating factors, the plasticity of the sleep-wake schedule would drive toward homeostasis and reestablish good sleep. The literature has focused primarily on presumed factors maintaining insomnia.

Mental Disorder

A common misconception is that insomnia is a symptom not meriting treatment in its own right. However, insomnia can be either a symptom (e.g., a complaint of difficulty falling asleep) or a disorder (i.e., complaint plus significant distress and functional impairment) (Harvey 2001a, Lichstein 2000). The misconception is particularly evident in the mental health field but is unsupported by the literature.

Ford & Kamerow (1989) reported on 8000 respondents, revealing that the risk of developing depression was much higher in those with preexisting insomnia. Similarly, Eaton et al. (1995) reported that having a sleep problem was the highest precursor in terms of attributable risk, identifying 47% of new cases of depression the following year. Other longitudinal studies have confirmed such findings. One report on 262 older adults suggested that frequency of depressed affect related to poor sleep, even when age, sex, and health status were accounted for (Rodin et al. 1988). Breslau et al.'s (1996) work on 1007 21–30-year-olds found that gender-adjusted relative risk for major depression in people with a history of insomnia was 4.0 (95% CI 2.2–7.0). Weissman et al.'s (1997) epidemiological survey reported an odds ratio of 5.4 (95% CI 2.6–11.1) for first-onset depression in 414 people with insomnia and no psychiatric history. Insomnia was also an independent risk factor for panic disorder and obsessive compulsive disorder. Finally, the Johns Hopkins Precursors Study found that insomnia in young men was indicative of greater risk for depression and psychiatric distress that persists for 30 years (Chang et al. 1997).

Thus, insomnia cannot be accounted for simply as presumed mental disorder. Furthermore, sleep disturbance often fails to resolve upon recovery from depression. However, the possibility that insomnia shares some psychobiological diathesis with anxiety and mood disorder is worthy of further investigation.

Faulty Conditioning

Since first proposed by Bootzin (1972), an understanding of insomnia as the product of maladaptive sleep habits has had considerable appeal. Good sleep is seen as coming under the stimulus control of the bedroom environment, which acts as a discriminative stimulus for sleep (Bootzin et al. 1991). Difficulty falling asleep may result either from failure to establish discriminative stimuli for sleep or the presence of stimuli incompatible with sleep. Poor stimulus control, therefore, might compete with sleep drive by strengthening conditioned arousal, and with circadian timing by doing so at normal bedtime. The insomniac may also nap in an armchair and so strengthen associations between sleep and nonsleeping environments. Stimulus control treatment instructions comprise lying down to sleep only when sleepy, avoiding using the bed for activities other than sleep (sexual activity excepted), getting up if unable to sleep quickly (within 15–20 minutes), repeating rising from bed as necessary throughout the night, getting up the same time every day, and avoiding napping (Bootzin 1972, Bootzin & Epstein 2000).

Only a few studies have investigated conditioning in insomnia. Haynes et al. (1982) compared student insomniacs and noninsomniacs on 12 sleep-incompatible behaviors, but found only one that differentiated the groups. Furthermore, duration of engagement in sleep-incompatible activity was unrelated to SOL. They had previously reported that the number of sleep-incompatible behaviors was not related to sleep difficulty (Haynes et al. 1974). Although over half the chronic insomniacs in Espie et al.'s (1989a) outcome study reported reading or watching TV in bed, there was no comparison group of good sleepers. Tokarz & Lawrence (1974) separated situational (reestablishing bedroom cues for sleep) from temporal components (regularizing sleep routines) and found both reduced SOL in their student sample. Zwart & Lisman (1979) conducted a study of 47 undergraduates assigned to stimulus control (all instructions), temporal control (lie down only when sleepy, rise at same time each day, do not nap), noncontingent control (a fixed number of risings within 20 minutes of retiring), countercontrol (sit up in bed and read, watch TV etc. if unable to sleep), or no treatment. They found countercontrol as effective as stimulus control, suggesting that it may ensure contingent disruption of bed and bedtime as cues for mental arousal. Davies et al. (1986) used countercontrol with older adults and found it moderately effective, although their 30% reduction in wake time after sleep-onset is less than typically reported in this population (Morin et al. 1999c).

In spite of equivocal evidence for the mechanism of effect (i.e., is it a stimulus control paradigm?), there has been little recent work on the conceptual basis of stimulus control. Harvey (2000a) reported that primary insomniacs did not differ

from good sleepers on daytime napping, variable sleep scheduling, whether they stayed in bed or got up when unable to sleep, or on engagement in sleep-incompatible activities. Nevertheless, significantly lower sleep efficiency is typical of insomniacs, and this may evidence the need for improved stimulus control. Interestingly, Bootzin has reported that stimulus control reduces sleep anticipatory anxiety as well as improving sleep (Bootzin et al. 1999). More research is required because stimulus control interventions have consistently been found to be efficacious in meta-analyses (Morin et al. 1994, 1999b; Murtagh & Greenwood 1995). Indeed, they are the only procedures recommended by AASM as comprising "standard" nonpharmacological treatment for insomnia (Chesson et al. 1999).

Poor Chronobiological Timing

In sleep phase disorders people sleep relatively normally, but during the "wrong" hours. In Delayed Sleep Phase Syndrome (DSPS), for example, in which the sleep period is phase delayed, subjects experience sleep-onset insomnia if they attempt to sleep before they are ready to sleep, and in advanced sleep phase syndrome sleep-onset is early and the phase advance results in early waking. In primary insomnia there may be an element of chronobiological dysfunction. For example, some insomniacs go to bed early and spend excessive time in bed either habitually as in the case of older adults, or as a response to having slept poorly on previous nights (Morgan 2000, Morin 1993). This contributes to poor sleep efficiency, which is affected both by TIB and total sleep time (see above), and can be improved by spending less time in bed, sleeping longer, or by a combination of the two. Similarly, napping reduces nighttime sleep drive in adults. The literature may have failed to discriminate adequately between circadian disorders and primary insomnia (Morris et al. 1990, Lack & Wright 1993). In particular, DSPS may not have been identified in studies involving younger populations. Research diagnostic criteria require clarification (Edinger et al. 2002).

It is suggested that, in the absence of competing factors, good sleep is associated with optimal, stable, and accurate circadian timing. Stimulus control instructions contain elements of temporal adjustment (see Lacks 1987, pp. 51–53; Espie 1991, pp. 51–55) and may provide *zeitgebers* (Aschoff 1951) for healthy sleep. Sleep restriction is another technique that may act both as a circadian harmonic and a reinforcer of homeostatic drive. Patients are encouraged to reduce bedtime hours (by staying up late and/or rising earlier) to approximate TIB closer to actual sleep duration (Spielman et al. 1987). Sleep restriction compresses sleep toward greater continuity, reduces wakefulness in bed, and increases sleep efficiency. Once the sleep pattern is improved, TIB may be extended, at a rate of 15 minutes per night per week, until the patient no longer gains further sleep or sleep efficiency is at risk of being reduced. Wohlegemuth & Edinger (2000) reviewed empirical findings on the efficacy of sleep restriction, and the AASM support sleep restriction as a "guideline" intervention for insomnia (Chesson et al. 1999). The synergy between stimulus control and sleep restriction is evident, and they are often presented

together. We use the term "sleep scheduling" for the combination (Espie et al. 1998, 2001b).

Recent interest in using appropriately timed bright light exposure to entrain circadian timing also focuses attention on possible chronobiological explanations of sleep disorder. Early evening bright light shifts the circadian rhythm of core body temperature and improves PSG-defined sleep in sleep-maintenance insomnia (Campbell et al. 1993), and morning light reduces SOL (Lack et al. 1995). Light exposure may also have a role in managing sleep disturbance in dementia (e.g., Satlin et al. 1992).

Physiological Hyper-Arousal

In 1967 Monroe conducted an influential study comparing 16 good sleepers with 16 poor sleepers, suggesting that poor sleepers exhibit heightened autonomic arousal (higher rectal temperature, vasoconstrictions per minute, perspiration rate, skin conductunce, body movements per hour) both prior to and during sleep. This work has been partly replicated (Stepanski et al. 1989), but other studies have failed to demonstrate arousal differentials. Higher levels of hormones indicative of adrenocortical activity have been both supported (Johns et al. 1971, Adam et al. 1986) and denied (Frankel et al. 1973). Other early work reported less rapid decline in heart rate associated with sleep in insomniacs, but others found no significant relationship between sleep-onset and heart rate or frontalis electromyography (EMG) (Haynes et al. 1974, Good 1975, Browman & Tepas 1976). Now that heart rate variability in the progression through sleep is better understood in normal subjects (e.g., Baharav et al. 1995, Bonnet & Arand 1997a), further comparative study seems warranted. Freedman & Sattler (1982) found that, prior to sleep-onset, insomniacs had higher frontalis and chin EMG than good sleepers, and the literature on relaxation and/or biofeedback treatments (e.g., Borkovec & Weerts 1976, Borkovec & Sides 1979, Freedman & Papsdorf 1976) might appear to support muscle tension as a problem in insomnia. However, there is limited evidence of tension reduction as the active mechanism, and posttreatment changes in EMG, heart rate, or respiration have proven elusive (see Espie 1991, pp. 43–45; Bootzin & Rider 1997, pp. 322–26 for review).

Interest in physiological arousal has been rekindled by evidence that insomniacs display measurable neurobiological differences from normal sleepers. Bonnet & Arand (1995) compared 10 objectively defined insomniacs with age-, sex-, and weight-matched controls and found they had significantly increased oxygen use both day and night. They suggested that increased 24-hour metabolic rate could be magnified by stress or viewed as a "higher arousal set point." In a complementary controlled study, 9 subjects with sleep state misperception (SSM) were also found to have increased metabolic rate, but less so than the primary insomniacs (Bonnet & Arand 1997b), raising the possibility that SSM is a mild version of, or precursor to, psychophysiological insomnia. Hyper-arousal has also been investigated in PSG studies. Early reports suggested insomniacs had more beta and fewer

alpha frequencies in their EEG (Freedman 1987, Freedman & Sattler 1982). Recently, Merica et al. (1998) compared spectral characteristics of 20 insomniacs and 19 controls. For all frequencies below beta, insomniacs had slower rise rates and reached lower levels, whereas beta power was increased. In REM, insomniacs showed lower levels in delta and theta bands, whereas power in faster bands was increased. These findings are consistent with slow wave deficiency in insomnia accompanied by hyper-arousal of the CNS, suggesting that insomnia may result from increased cortical activation. They noted, however, that homeostatic control of slow wave activity appeared to be intact in the patient population. A study by Loewy & Bootzin (1998) also investigated hyper-arousal and found that event-related EEG activity, as measured by auditory evoked potentials, provided evidence of information processing during sleep.

Importantly, however, not all objective poor sleepers complain of insomnia, and not all subjective insomniacs have poor sleep (Edinger et al. 2000), suggesting that physiological arousal alone is an insufficient explanation.

Cognitive Hyper-Arousal

It was first reported 25 years ago that poor sleepers complain of mental alertness more than physiological arousal (Evans 1977). Other studies have consistently associated cognitive arousal more strongly with sleep disruption, and "having an overactive mind" has been the attribution rated most highly, both by insomniacs and noninsomniacs (Lichstein & Rosenthal 1980, Nicassio et al. 1985, Broman & Hetta 1994). Espie et al. (1989a) reported that the cognitive items of the Sleep Disturbance Questionnaire (e.g., "my mind keeps turning things over," "I am unable to empty my mind") were the most highly rated; Harvey (2000b) recently replicated these findings. The Sleep Disturbance Questionnaire has been found to have modest internal consistency ($\alpha = 0.67$) (Espie et al. 2000). Although there is no gold standard measure of cognitive activity, the Pre-Sleep Arousal Scale (Nicassio et al. 1985) is widely used and has satisfactory internal consistency for its somatic and cognitive subscales ($\alpha = 0.81$ and $\alpha = 0.76$, respectively). These constructs have some degree of independence (74% unshared variance). The cognitive subscale of the Pre-Sleep Arousal Scale has also demonstrated modest ($r = 0.35$) validity compared with voice-activated recordings of presleep thoughts as a criterion measure (Wicklow & Espie 2000).

Population survey confirms that people dissatisfied with sleep report mental activity near bedtime (Ohayon et al. 1997). However, there are conflicting conclusions from studies of the relationship between cognitive activity and sleep latency. Van Egeren et al. (1983) found that audiotape-recorded cognitions were significantly correlated with subjective sleep latency, but not with polysomnographic (PSG) assessment sleep, whereas Borkovec et al. (1979) and Kuisk et al. (1989) reported more frequent cognitive activity in insomnia confirmed by PSG. Sanavio (1988) reported a low correlation ($r = 0.09$) between presleep intrusion and self-reported sleep latency, and furthermore, found no advantage of a tailored

cognitively focused program in the treatment of sleep-onset insomnia. The possibility, therefore, remains that cognitive arousal is an epiphenomenon of nighttime wakefulness (Freedman & Sattler 1982, Morin 1993).

Support for a cognitive model has also come from studies involving experimental manipulation of presleep cognitive intrusions (e.g., Gross & Borkovec 1982, Hall et al. 1996). However, Haynes et al. (1981) exposed insomniacs and noninsomniacs to brief stressors and found a decrease in subjective and objective SOL among insomniacs on stress nights. They concluded that a mental processing task that disrupts sleep-related cognitive events may decrease SOL, implying that the nature of the intrusions may be critical to the effect upon sleep. Therefore, failure to differentiate thought content could result in limited comparability between studies.

Dysfunctional Thinking

The importance of emotional arousal has been stressed, because affect-laden cognitions are more likely to interfere with sleep (Espie 1991, Morin 1993, Haynes et al. 1981, Coyle & Watts 1991). Beliefs about the negative experiences and consequences of insomnia may foster the clinical complaint. This parallels research on "worry," posited as a generic trait (Barlow 1988, Meyer et al. 1990), and studies on unwanted intrusive thoughts (Rachman & De Silva 1978, Reynolds & Salkovskis 1992). Negative and distressing cognitions are likely to contribute to the development of obsessions; however, it is not the thoughts per se that are untypical or pathological, but the meaning and concern attributed to them (Clark & Purdon 1993, 1995). The conceptual relatedness of nighttime and daytime intrusions, therefore, appears considerable.

Insomniacs have more negative thoughts than good sleepers at bedtime (Nicassio et al. 1985, Van Egeren et al. 1983, Kuisk et al. 1989), and such thinking is reported even when wakened from light sleep (Borkovec et al. 1981). The thoughts of insomniacs may be dependent on emotional state. Investigating the relationship between worry and insomnia, Watts et al. (1994) found that much of the presleep mental activity of "worried insomniacs" revolved around work and general mental activity. In contrast, thoughts of "nonworried insomniacs" focused on the sleep process itself. Insomniacs may also feel less in control of their thinking (Watts et al. 1995). Gendron et al. (1998) reported that insomniacs with comorbid generalized anxiety disorder had greater cognitive activity at bedtime than insomniacs without generalized anxiety disorder, evaluated their thoughts as more intrusive and worrisome, and attempted cognitive avoidance strategies more frequently.

Morin (1993) has argued that beliefs and attitudes play a critical role. He devised a 30-item questionnaire to identify irrational, affect-laden thoughts that intrude prior to sleep-onset (dysfunctional beliefs and attitudes about sleep scale) (Morin 1993, Morin et al. 1993). This scale comprises misconceptions about the causes of insomnia, misattributions or amplifications of the consequences of insomnia,

unrealistic sleep expectations, diminished perceptions of control, and faulty beliefs about sleep-promoting practices. It has satisfactory psychometric properties and sensitivity to change after cognitive-behavioral treatment (CBT) (Espie et al. 2000, Morin et al. 2001), and may help identify subgroups of the insomniac population (Edinger et al. 1998). Furthermore, those with subjective insomnia report more dysfunctional sleep-related cognitions than do those with objective insomnia (Edinger et al. 2000), consistent with the view that cognition/affect may influence sleep report and perhaps mediate insomniac complaint.

Formal analysis of sleep-interfering cognitions has been reported in several studies. Coyle & Watts (1991) used an extended version of the Sleep Disturbance Questionnaire (Espie et al. 1989a) and reported two distinct factors: "sleep attitudes," reflecting anxiety about the sleep process, and "mental activity," reflecting nonspecific cognitive activity. Six factors of nighttime intrusive thoughts, i.e., trivial topics, thoughts about sleep, family and long-term concerns, positive plans and concerns, somatic preoccupations, and work and recent concerns, were identified in a study of young adults (Watts et al. 1994). Extending these findings by using a good-sleeper comparison group, Harvey (2000b) reported that presleep cognitive activity of insomniacs could be distinguished by being more focused upon worry about not getting to sleep, general worries, solving problems, the time, and noises in the house, and less focused upon "nothing in particular."

A study by Fichten et al. (1998) of the thoughts of older adults during wakeful periods yielded a 3-factor solution of generalized positive thinking, generalized negative thinking, and thoughts related to sleep. They suggested that insomniacs use positive thinking as a buffer to combat negative intrusions. Also recently, Wicklow & Espie (2000) obtained voice-activated audiotape recordings of spontaneous thoughts and sleep actigraphic data from 21 poor sleepers over 3 consecutive nights. Content analysis yielded 8 categories of presleep intrusion, and a regression model indicated that thinking about sleep and the anticipated consequences of poor sleep, along with general problem-solving were the strongest predictors of objective SOL. Intrusions were subsumed under one of 3 factors: active problem-solving (e.g., rehearsing/planning events), present state monitoring (e.g., thinking about sleep/not sleeping) and environmental reactivity (e.g., attending to external noises).

Paradox and Ironic Control

This model of insomnia proposes that anxiety responses may be conditioned not only to external, situational cues but also to the individual's behavior (Ascher & Turner 1979, Espie & Lindsay 1985, Espie 1991). Fear of performance failure (insomnia) and of anticipated negative consequences of that failure is described as performance anxiety. In paradoxical treatment counterproductive attempts to fall asleep are replaced by the intention of remaining passively awake or by giving up any direct effort to fall asleep (Ascher & Turner 1980, Fogle & Dyal 1983). This rationale is supportable in that good sleepers do not use any strategies to

fall asleep. Paradoxical intention has demonstrated efficacy in controlled trials (Turner & Ascher 1979, Espie et al. 1989b) and is an intervention that reflects a "moderate degree of clinical certainty" according to the AASM (Chesson et al. 1999). Paradox continues to be used within multicomponent CBT (e.g., Espie et al. 2001b) and may be particularly useful with patients who are resistant and reactive to therapeutic suggestions (Shoham et al. 1995).

Further evidence for this type of mechanism was provided by Ansfield et al. (1996), who explored the effects of different sleep-onset instructions in good sleepers under high or low mental load. Paradoxical wakefulness was found amongst those attempting to sleep while listening to marching music. This was interpreted in terms of Wegner's (1994) theory of a self-loading system that suggests that under certain conditions the thwarted attempt to control a particular mental state can yield the opposite of what is desired. They hypothesized that failure to fall asleep on a few occasions could occur when sleep is attempted under transitory mental loads, such as at times of stress. Eventually, a person's thoughts about being unable to sleep could constitute a debilitating mental load, which when combined with the continuing frustrated desire to fall asleep, could lead to chronic insomnia.

Harvey (2001b) has explored the effects of suppressing presleep cognitive activity on sleep-onset latency. A cohort of insomniacs and good sleepers were allocated to either a suppression condition ("suppress the thought most likely to dominate your thinking as you get into bed") or nonsuppression condition ("think about anything as you get into bed, including the thought you would most likely think about as you go to sleep"). Suppress participants reported longer sleep latencies and poorer sleep quality, regardless of whether they were insomniac or not. Harvey concluded that thought suppression appeared to have the opposite effect in that it prevented sleep-onset, in a manner consistent with Wegner's theory of ironic mental control.

There are parallels between Wegner's theory and the performance anxiety model that gave rise to the adaptation of paradoxical intention from the work of Frankl (1960). Indeed, Ansfield et al. (1996) propose that their results are consistent with theories of cyclic escalation of anxiety disorders (Ascher 1981) and worry about sleep (Borkovec 1982).

AN INTEGRATED PSYCHOBIOLOGICAL MODEL OF NORMAL SLEEP

There are, thus, differing explanations of insomnia, each having some empirical support. The rest of this paper attempts to integrate this evidence into a conceptual framework. A model of the normal sleep process is presented first, because a perspective on the pathway to sleeping well is likely to inform understanding of the development, maintenance, and treatment of insomnia.

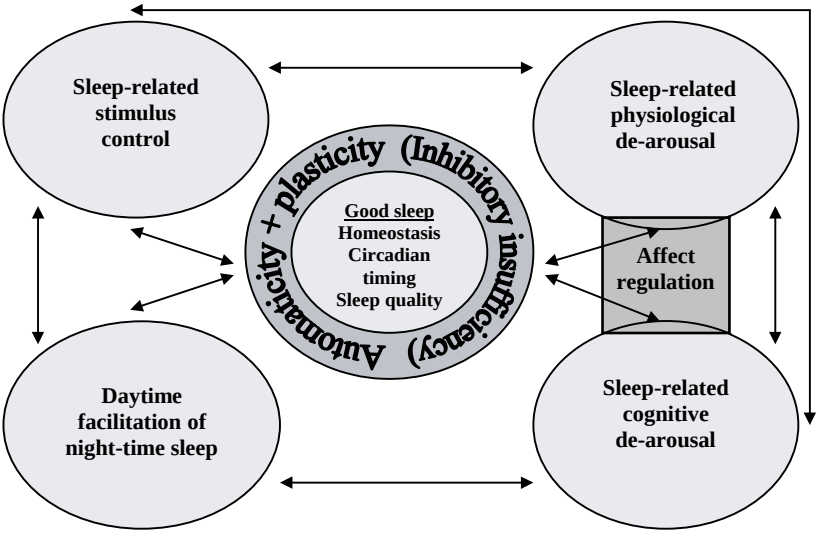

Figure 1 A psychobiological model of good sleep. Insomnia is proposed as resulting from chronic inhibition of one or more of the component processes.

Figure 1 summarizes hypothesized processes and interactions in normal sleep. The model proposes good sleep as the natural state of the human organism—its default state. That is, homeostatic and circadian processes, under normal circumstances, default to good sleep, not to insomnia. The core of the model is involuntary, harmonious interaction between homeostat and timer, which is associated with the self-perception of good quality sleep. Like all neurobehavioral systems, good sleep is assumed to have both functional plasticity and automaticity. These are presented as protective properties, "defending" the core.

Plasticity refers to the "absorb and readjust" capability of the sleep-wake system to accommodate living in the real world where situational and personal factors provoke variance and challenge normal, stable functioning. Night-to-night variability is tolerated but minimized by the good sleeper, for whom the sleep homeostat drives sleep-related behaviors effectively. In acute insomnia the norm would be recovery of sleep pattern, again reflecting the system's plasticity in function and default regression towards good sleep. Automaticity refers to the involuntary nature of the well-adjusted schedule, to the habitual, conditioned associations that are part of its stimulus control paradigm, and to the implicit expectations and assumptions the good sleeper has about sleep continuity and sleep quality. It is tempting to think of the model as the good sleeper's list of ingredients for good sleep, but that would be misleading. The good sleeper is not thought of as following a recipe to produce the perfect sleep. Rather, the good sleeper is regarded as essentially passive because internal and external cues act as automated setting conditions for sleep. Endogenous

cues to sleep such as physical and mental fatigue interact reciprocally with exogenous cues in the home environment. The good sleeper sleeps just as he walks, or talks—without thinking about it. This, presumably, is the insomniac's ambition.

The defensive properties of the good sleep paradigm, therefore, are seen as maintained by four interacting subsystems: sleep-stimulus control, physiological de-arousal, cognitive de-arousal, and daytime facilitation. In a reciprocal way, sleep homeostasis, circadian timing, and sleep quality serve to reinforce and maintain these behavioral, cognitive, and biological processes. That is, good sleep begets good psychobiological preparation for sleep, which begets good sleep.

The model predicts that the good sleeper accurately interprets physiological and mental signs of sleep readiness and, once in the bedroom, the stimulus environment there further reinforces de-arousal. Regular sleep habits for retiring and rising constitute a good predictive framework that exhibits both sensitivity and specificity. In terms of the former, a high percentage of nights that follow this pattern is associated with efficient sleep. In terms of the latter, sleep is highly specific to the bedroom and wakefulness to other environments; i.e., the good sleeper is less likely to lie awake in bed or need to sleep in the day or evening. Effective stimulus control reinforces not only circadian timing and sleep homeostasis but also contributes to cognitive de-arousal because high sleep efficiency precludes cognitive intrusion in bed. Both physiological and cognitive de-arousal are presumed to occur in parallel. Active information-processing recedes as the wake system disengages and the sleep system engages. The good sleeper is less likely to use time in bed for problem-solving or anxious thinking, nor have time to do so because sleep-onset is rapid, and nervous system adaptations proceed unhindered because the good sleeper has few inaccurate expectations or worries about sleep or wakefulness.

Crucially, the good sleeper experiences little affect associated with sleep. Affect regulation, which represents an interaction between cognitive and physiological processes, is proposed as functional when affect is essentially neutral. Dysregulation would occur with strong (negative or positive) emotions, both of which are arousing. Similarly, the good sleeper is seen as having few conscious expectations of sleep and puts no direct effort into the sleep process. Daytime attitudes and behaviors may also facilitate sleep. The good sleeper may make fewer attributions of daytime mood and performance to the preceding night's sleep. For example, when irritable or fatigued, he may be more likely to associate these with proximal events (e.g., pressure of work) than view them as contingencies of sleep pattern or sleep quality. Also, the good sleeper may be more effective at managing state/trait symptoms and/or may put less pressure on sleep to compensate for any excessive daytime routines.

Thus, the common pathway in the maintenance of sleep homeostasis, circadian timing, and sleep quality is proposed as an involuntary process of psychobiological de-arousal within which there is insufficient mental, behavioral, or physiological inhibition to impair the automaticity and plasticity of sleep. Implicit in the model is the expectation of regression towards normal sleep because of its fundamental importance both developmentally and functionally.

UNDERSTANDING INSOMNIA WITHIN THE PSYCHOBIOLOGICAL INHIBITION MODEL

At the simplest level, insomnia may be seen as a failure of automated sleep activation and maintenance. More specifically, insomnia may arise from acute inhibition of one or more of the processes normally contributing to good sleep (precipitating stage), perhaps in predisposed individuals, which may become persistent when chronic inhibition develops, preventing the natural recovery of good sleep (perpetuating stage). This conceptualization seems consistent with the lack of pathology or fundamental disorder of sleep-wake function in primary insomnia. Insomnia is proposed, therefore, as persistent loss of expression of normal sleep. The conceptualization is also consistent with evidence that chronic insomnia is amenable to cognitive-behavioral intervention (Morin et al. 1994, 1999b; Murtagh & Greenwood 1995). CBT may act to overcome inhibitory mechanisms and reestablish adaptive mental and situational "setting conditions" for the restoration of normal sleep.

Automaticity and plasticity would be weakened by inhibitory feedback from one or more of the attendant psychobiological processes (Figure 1 and Table 1). For example, poor stimulus control represented by sleep-incompatible behavior, diminished sleep-wake sensitivity and specificity, and irregular sleep habits could inhibit sleep and maintain insomnia. Similarly, affect-laden thinking would undermine cognitive de-arousal. The notion of "inhibitory sufficiency" is introduced because good sleepers do not necessarily observe all the rules of sleep preparation; e.g., good sleepers may read, drink caffeinated beverages, spend time anxiously reflecting on the day while in bed, be poor daytime copers, etc., without ever becoming insomniac. It helps to account for those individuals who are under chronic stress but who apparently still sleep well. Inhibitory sufficiency, therefore, represents the critical mass of inhibition required to outweigh the stability of an individual's default sleep pattern. Theoretically, it would need to be higher where there is strong sleep homeostasis and/or circadian timing, and less where there is predisposition to insomnia, strong precipitating factors or limited protection afforded by plasticity and automaticity.

Table 1 presents detail of the elements of the model and how they may become inhibited. Poor sleep-related stimulus control would be observed where the bedroom becomes a conditioned focus for waking activity and where the sensitivity/specificity of the sleep-wake schedule is compromised, primarily by remaining awake in bed, but also by sleeping in the daytime. Attempting to sleep longer on recovery nights and spending too long in bed could also inhibit the continuity and automaticity of sleep by reducing sleep efficiency. Stimulus control and sleep restriction treatments may be effective because they counteract the process of inhibition (stay up until sleepy, compress time in bed, etc.) and reinstate default sleep. The instruction to get up if not asleep within 15 minutes may also attenuate the inhibition associated with effort to sleep, as it can be likened to a "give up trying" instruction.

TABLE 1 Insomnia within the psychobiological inhibition model.

Factors contributing to good sleep	Insomnia: factors inhibitory to sleep homeostasis and circadian timing, and to the protection of good sleep afforded by automaticity and plasticity
Sleep stimulus control	
Sleep-compatible conditioning	Conditioned association of sleep-incompatible, waking activities (e.g., reading, watching TV, eating, talking, problem-solving) with bed and bedroom environment; keeping the light on
Sleep-wake sensitivity/specificity	Environmental latitude in sleep and wake behaviors: lying awake in bed either presleep or upon wakening, sleeping in the day, sleeping elsewhere than in bed
Regular sleep habits	Variable and/or reactive patterns: changing times for retiring and rising, extending time in bed to catch up on sleep, sleeping in at weekends, spending longer in bed than current sleep requirement—reduced sleep efficiency
Physiological de-arousal	
Sleep system engagement	Not feeling tired at bedtime, in bed too early, keeping the light on, sleep-incompatible activities, anxiety, trying too hard to sleep, tension, heart rate variability
Wake system disengagement	As above; active thinking and problem-solving, self-monitoring of internal (bodily and mental) cues, hypervigilance, poor sleep hygiene
Good sleep hygiene	Stimulants (e.g., caffeine, nicotine) in excess/near bedtime; alcohol withdrawal symptoms during the night; active exercise late evening; bedroom stuffy, hot, or cold; bed uncomfortable
Cognitive de-arousal	
Minimal cognitive drive	Rehearsing/planning/problem-solving thoughts in bed, thinking about events the previous or next day, preoccupation with sleep/sleeplessness, "stimulus hungry" mind, mind racing, unable to "switch off"
Accurate sleep-wake attribution	Dysfunctional beliefs and attitudes about sleep and consequences of not sleeping, not expecting to sleep, catastrophic thoughts, concern about next day well-being and coping
Affect regulation	
Minimal affect	Worry, anxiety, frustration, negativity or excitement, intensity in emotional tone associated with above cognitive or physiological processes
Minimal effort to sleep	Sleeplessness preoccupying: trying to control sleep/overcome insomnia, attempts to suppress thoughts/suppress affect, self-monitoring of alert/sleepiness state, performance effort to fall asleep, performance anxiety
Daytime facilitation of night sleep	
Accurate wake-sleep attribution	Attribution of impaired daytime mood, attention, performance to quality of sleep; expectation that sleep should compensate; blaming problems on insomnia; fatigue seen as pathognomic of insomnia; perception of self as insomniac
Effective coping skills	Experiencing time pressure; problems relaxing; worry, frustration, low mood; active late into evening; poor wind down

Some insomniacs report not being tired at bedtime, which undermines physiological de-arousal, and evidence of wakefulness when aroused from light sleep suggests slower engagement of the sleep system. Studies revealing elevated autonomic symptoms in poor sleepers suggest that inhibition of normal arousal is problematic. Sleep hygiene is traditionally regarded as a behavioral strategy. However, its components are primarily physiological in terms of the model of sleep inhibition. Excessive use of caffeine and strenuous exercise delay sleep onset, presumably through heightened arousal. Indeed, in one experimental study 400 mg of caffeine three times per day for one week produced increased arousal on metabolic measures and reports typical of insomniac complaint (Bonnet & Arand 1992). Similarly, environmental factors (temperature, humidity, light) could inhibit sleep physiologically. Use of alcohol may lead to dehydration or provoked awakenings because of ethanol metabolism. Observing good sleep hygiene, therefore, would remove some potential inhibitors of sleep. Similarly, relaxation-based treatments may either reciprocally inhibit autonomic activity (Wolpe 1958) and thus counteract the maintenance of physiological arousal or, perhaps more likely, facilitate mental (and physiological) de-arousal.

Problems with cognitive de-arousal appear central to insomnia. Insomniacs report intrusive thinking; some of this is reflective, but much is also worrisome. The active mind is likely to inhibit de-arousal, particularly where accompanied by negatively toned affect. The insomniac typically becomes concerned about sleeplessness and its immediate and longer-term negative consequences. This gives rise to emotional upset, ironic urgency, and performance anxiety associated with failed efforts to regain perceived loss of control over sleep. Indeed, the development of attentional bias for sleep- or insomnia-related thoughts may help to explain the maintenance of sleep disorders even after proximal sources of transient sleeplessness (e.g., stress) have passed. The fact that attempts to suppress thinking produce sleep disturbance even in normal sleepers is further evidence of insomnia arising from cognitively mediated sleep inhibition. Insomniacs often try too hard, thereby obviating passive acceptance of sleep, which is a hallmark of the good sleeper. The success of cognitive strategies for insomnia may depend, therefore, upon the extent to which they disable sleep-interfering mentation and affect. The central paradox is that deliberate efforts to do so fail to emulate the automatic nature of sleep-onset in good sleepers and may exacerbate preexisting sleep inhibition. The less direct mechanisms of behavioral interventions such as stimulus control upon cognitive processes therefore merit further attention, as do techniques using paradoxical instructions.

It is specious, of course, to set up physiological and cognitive arousal in direct competition. The psychobiological inhibition model presumes interaction between physiological, cognitive, affective, and behavioral subsystems, and indeed between daytime and nighttime variables. These relationships are represented by arrows in Figure 1. Their interaction contributes positively to the maintenance of good sleep. The corollary, however, is that interaction would also compound inhibitory effects. Indeed, Born et al. (1999) have demonstrated that normal sleepers, told that they

would be woken at 6 A.M., had significant increases in adrenocorticotropin during the hour preceding waking. This suggests that some form of cognitive priming may be linked to sleep-related biological responses.

Less attention has been paid to how daytime factors could contribute to insomnia. Good sleep may be inhibited by daytime preoccupation with impairments perceived to result from poor sleep. Just as attributional (sleep-wake) error can contribute to sleeplessness during the night, so it may form dysfunctional sleep-related schema during the day (wake-sleep). Self-perception of being insomniac may influence interpretation of everyday experiences, such as feeling fatigued or irritable, in a manner comparable to obsessional disorders in which special meaning is attributed to common thoughts and behaviors. Just as some people differ from controls only in respect of their interpretation of events, some insomniacs have normal or close-to-normal sleep, but perceive it to be abnormal. Insomniacs have been characterized as prone to worry and internalizing of anxiety. Such traits may be reinforced in the day, not only during the night. Furthermore, failure to manage daytime concerns effectively may put pressure on sleep when, in the absence of other stimulation or distraction, the insomniac spends time thinking, rehearsing, planning, and worrying. The model, therefore, includes poor coping skills and disturbed daytime affect as factors that may undermine preparation for sleep and may contribute to behavioral and cognitive sleep inhibition.

It may seem counterintuitive or even arbitrary to talk in terms of inhibition of de-arousal rather than excitation (hyper-arousal). However, the definition of insomnia as failure of expression of an essentially normal underlying sleep process is defensible. To remain awake the insomniac does not need to be hyper-aroused any more than one needs to be hyper-aroused at any other time to remain awake. Hyper-arousal is a sufficient but not necessary precondition to wakefulness. Furthermore, even if hyper-arousal were to occur initially to delay sleep it would not follow that remaining hyper-aroused would be necessary to stay awake thereafter. By comparison, if an individual is becoming sleepy (de-aroused) but subsequently becomes wakeful, it only seems necessary to infer that there has been attenuation of the de-arousal, and that explanation also seems sufficient. It is not being argued that hyper-arousal never occurs, but rather that inhibition of de-arousal does always occur. Inhibition is the lowest common denominator that can be reliably deduced. It is suggested that insomnia as a disorder of initiating or maintaining sleep is at its core a disorder of sleep engagement, not a disorder of excessive arousal.

Nevertheless, the model does account for hyper-arousal in insomnia. Hyper-arousal clearly would inhibit sleep, but it is hypothesized that there is within-group variability in insomnia (i.e., not all insomniacs are hyper-aroused). Variability between populations of insomniacs studied might explain apparently discrepant results reporting both physiological and mental measurement. Physiological hyper-arousal may be a distinct subtype of insomnia. De-arousal, however, is suggested as the reliable correlate of good quality sleep; anything else at or above a level sufficient to inhibit sleep may be associated with insomnia.

Why then has cognitive arousal been more strongly associated with insomnia than physiological arousal? It is suggested that cognition is invariably symptomatic of insomniacs' subjective nighttime experience, whereas physiological arousal is less invariably so. Accordingly, when asked to rate or attribute sleeplessness, insomniacs are likely to stress mentation and/or affect rather than physiological symptoms. There may be interpretation bias, and the possibility that cognitive activity is simply an epiphenomenon of sustained wakefulness or a by-product of the measurement process cannot yet be excluded. However, the cognitive/affective component of primary insomnia does seem to be a sine qua non in clinical practice. Like physiological arousal, cognitive arousal may represent a continuous dimension from de-arousal to hyper-arousal, exhibiting individual variability, but once again, de-arousal appears to be the correlate of good sleep. This helps to explain the common finding that insomniacs consistently appear hyper-aroused, relative to good sleepers, on measures such as the cognitive subscale of the Pre-Sleep Arousal Scale. It cannot be stated with conviction that they are hyper-aroused in any absolute sense, but it may be reasonably inferred that they are not de-arousing.

It should be noted that the model can also be applied to sleep-maintenance insomnia. This subtype is more commonly associated with mid- to late-life, when a propensity to lighter, more broken sleep develops and automaticity and plasticity may be compromised. It is suggested that in sleep-maintenance insomnia there is inhibition of the rapid, automated de-arousal response to the brief wakeful experiences typically found in older good sleepers. This results in more conscious processing of arousals, more frequent arousals, and times of extended wakefulness with difficulty returning to sleep. One way in which inhibition might occur would be through attentional bias for arousal and threat-related cues. The insomniac may selectively attend to brief arousals in the night and interpret these by means of established schema as evidence of inability to sleep. This could then set up the pernicious cycle of inaccurate attribution, affect, effort to sleep, physiological arousal and engagement of the wake system.

It is proposed, therefore, that the common pathway in insomnia is inhibition of the expression of normal sleep. It is further suggested that, whereas any of the subsystems may contribute to inhibition, it is cognitive/affective factors that serve as the "activating agent" for the phenomenology of insomnia and presentation of clinical complaint. This is evidenced by the consistent association of intrusive, affect-laden cognitions with primary insomnia and by the fact that some self-reported good sleepers sleep as poorly as insomniacs, but without experiencing concerns about sleep (Edinger et al. 2000). In the psychobiological inhibition model such cognitions are presumed to pervade nighttime and/or daytime thinking. A corollary to the suggestion of activation is that, if CBT intervention is to be effective, it must somehow close this cognitive/affective gate. This again is consistent with the proposition that stimulus control may achieve its effects by fostering cognitive de-arousal. Similarly, sleep restriction might be regarded as a paradigm involving behavioral experiments to evaluate assumptions about sleep requirements and their consequences. The circadian readjustments that form part

of both stimulus control and sleep restriction may, of course, take place without synchronous cognitive conceptual shift, but if that is the case it is suggested that insomnia complaint is likely to persist.

Relationship of the Psychobiological Inhibition Model to Other Models of Insomnia

Espie (1991, pp. 39–56) proposed a framework in which insomnia was conceptualized as mediating from activation of nervous system (central, autonomic), psychological (cognitive, emotional), or environmental (situational, temporal) arousal. These elements can all be accommodated in the current model, although the emphasis is now upon inhibition, impairing the automaticity of normal sleep. Daytime factors are also factored into the new model, and further detail of the cognitive dimension (from Espie 1992, Espie & Wicklow 2001) is presented in the cognitive de-arousal component.

Morin (1993, pp. 46–60) also presented an integrative conceptualization of insomnia. He suggested that hyper-arousal (emotional, cognitive, physiologic) is the central mediating feature of insomnia, which interacts with dysfunctional cognitions, maladaptive habits, and perceived consequences of insomnia. Although the psychobiological inhibition model makes no requirement of hyper-arousal, Morin's emphasis on cognitive factors parallels the cognitive/affective activation agent presented here. The role of dysfunctional thoughts and beliefs in the psychobiological inhibition model is consistent with Edinger et al.'s (2000) interpretation that these influence self-perception of insomnia and insomniac report. Morin also stressed the bi-directional influence of the components, such that consequences often become causes and vice versa, similar to the proposed reciprocal interaction of the elements of the psychobiological inhibition model.

Perlis et al. (1997) discussed discrepancies between PSG and subjective appraisal of sleep. They suggested that inconsistencies may be explained by the presence of high frequency EEG activity in insomnia around sleep-onset, which interferes with the development of mesograde amnesia, and results in insomniacs having blurred phenomenological distinction between sleep and wakefulness. The psychobiological inhibition model can accommodate such an association but would suggest that cortical arousal represents a failure to de-arouse, with hyper-arousal presenting only in some insomniacs. The reciprocal interaction between maintained physiological and psychological processes would inhibit both the transition to slower EEG activity and the experience of sleep-onset, but the model would suggest that it is cognitive/affective inhibition that is the more likely activating factor.

It is also noteworthy that the psychobiological inhibition model is consistent with Spielman's (1991) model of insomnia acquisition (presented above) and with Edgar's (1996) "opponent process" model. The latter proposes that circadian timing promotes wakefulness and opposes sleep drive, and that prolonged wakefulness leads to compensatory sleep responses. The psychobiological inhibition model assumes such an interaction between homeostat and timer, but also describes how

such an automated interaction may be inhibited by thoughts, emotions, and behavioral changes.

Some Implications of the Cognitively Activated Psychobiological Inhibition Model

This model raises testable hypotheses and suggests mechanisms for treatment effects. It is only possible to touch upon some of these. The central premise is that in insomnia the expression of normal sleep is chronically inhibited. Therefore, further study of sleep-related de-arousal in good sleepers is required. The model predicts phenomenology comprising attitudinal passivity towards sleep, with neutral expectations, compared with the negativity and worry of the insomniac. In terms of automaticity, it is suggested that the good sleeper is like the experienced car driver who executes a complex series of operations with minimal attentional load. In comparison, the insomniac is like the anxious learner driver: vigilant, deliberate, and errorful. The concept of automaticity in human learning has long been discussed as part of information-processing theory (Shiffrin & Schneider 1977). Its application to sleep and insomnia could be fruitful. The ability to automate is, presumably hard-wired in order to overcome problems associated with an otherwise limited-capacity information-processing system. Theories of paradox and ironic mental control are interesting because they emphasize allowing events to take their natural course, and de-emphasize the need for control and success. Effort to sleep and suppression of wakefulness, and its associated mental activity, may disengage automaticity.

Study of within-subject as well as within-group variability may lead to greater appreciation of how similarly or differently good sleepers and insomniacs respond to good and bad nights, in terms of their expectations, affect, and behavior. Furthermore, because cognitive/affective activation is seen as central, insomniacs may be differentiated not only from good sleepers, but also from noncomplaining poor sleepers. It seems important to identify this latter group for formal study and to consider degree/severity of complaint as a correlate of mental arousal/concern. The latter may be an inverse correlate of automaticity. Good sleepers and noncomplaining poor sleepers may make more benign attributions after a bad night, leaving automaticity intact. It is further suggested that sleep state misperception (SSM) may represent cognitive activation and perceived severity equivalent to that found in primary insomnia, thus differentiating SSM from good sleepers and noncomplaining poor sleepers, but not from insomniacs. Longitudinal study of SSM is required to investigate whether or not objective sleep disturbance develops over time. There is a need, however, to develop better measures of sleep quality, with sound psychometric properties, and to investigate the interrelationships between sleep quality, cognitive arousal, dysfunctional beliefs, attributional error, attentional bias, and affect, both in SSM and insomnia.

Comparison of the relapse patterns of various subgroups after bad nights or periods of poorer sleep would be interesting from another perspective. The interaction

of sleep homeostasis and circadian timing is thought to demonstrate impaired plasticity in insomniacs, who present sleep pattern variability. Their sleep-wake rhythm may be more brittle, and the repayment of sleep debt may not function as efficiently. Unfortunately, research data are usually analyzed as weekly mean values, thus concealing within-subject and within-group variability in sleep. Individual raw score variance has been reported only occasionally in insomnia, but further work using such data would be valuable. Theoretically, stability in sleep pattern would be displayed by small raw score standard deviations, and plasticity would be demonstrated by regression from higher values (e.g., at times of acute insomnia) to lower values. The time taken to accommodate and adjust from above to below some threshold value might be a useful measure of plasticity.

The "setting conditions" for insomnia are potentially wide-ranging, and further study employing experimental manipulation of situational, autonomic, mental, and affective variables would be valuable. The model would predict that only manipulations with a cognitive/affective activating component would lead to subjective sleep concern, which can vary independently of sleep disruption. The concept of inhibitory sufficiency requires investigation, and methods need to be developed to titrate experimental "doses" so that outcomes can be related to inputs. For example, if an experiment was conducted on the countercontrol procedure, it would be helpful to use standard stimuli (e.g., text to read, TV program to watch) validated for emotional valence, intrusiveness, imagery potential, etc. It should be borne in mind, of course, that disturbance of a single night is not insomnia, but only a bad night. Care must be taken, therefore, also to consider inhibitory sufficiency from the clinical perspective of generalized and enduring sleep disturbance where, presumably, retrospective experience influences response to prospective experimental factors.

The proposed model suggests that maintained arousal is the necessary and sufficient precondition for insomnia. Clearly, this hypothesis requires validation, and a between-group repeated-measures model utilizing daytime, pre-bedtime, presleep, and early-sleep measures of physiological and cognitive arousal in insomniacs and good sleepers would seem appropriate. This would also identify cases of hyper-arousal, proposed as a subpopulation of insomnia. In terms of intervention, the model suggests that cognitive behavioral strategies may act via the common pathway of disengaging mechanisms inhibitory to de-arousal, and facilitating reestablishment of automated normal sleep. Given the proposed importance of cognitive/affective de-activation, it would be particularly useful to investigate stimulus control and sleep restriction as de-activating in this context e.g., attitudinal shift, relinquishing control, precluding worry from bed. Their contribution to circadian timing and homeostatic drive, along with such de-activation, may explain the positive clinical outcomes achieved using these procedures. Specific cognitive interventions need to be explored in terms of direct versus indirect action upon sleep-related variables. Automaticity would predict that indirect (e.g., paradoxical) methods would be more efficacious than those that focus attention and intervention directly upon sleep or upon sleep-related thoughts.

CONCLUSION

Although there are a number of perspectives on the etiology and maintenance of insomnia, each of which has some empirical support, the conceptual basis lags some way behind treatment methodology and the evaluation of efficacy and effectiveness. An integrated psychobiological model has been proposed that differentiates insomnia from normal sleep in terms of inhibitory mechanisms and processes. It is hoped that the hypotheses raised here will stimulate interest in further experimental and clinical study, and that the resultant research process will improve both understanding and management of this common condition.

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