# Insomnia and Sleep Disorders

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## Abstract

Sleep is common to all animals and yet there are so many mysteries surrounding its function. Insomnia, the most common sleep disorder, is prevalent and debilitating and has been shown to play a role in the onset and maintenance of other mental health disorders, such as depression, anxiety, and bipolar disorder. This chapter outlines a basic overview of sleep, followed by a summary of cutting-edge research investigating insomnia as well as its relationship to other psychiatric disorders. It is suggested that a bidirectional relationship exists between sleep and emotion and research supporting this framework is addressed briefly. We describe the treatment of choice for insomnia, cognitive behavioral therapy, as well as outline other exciting developments in the treatment of insomnia, including bright light therapy, intensive sleep retraining therapy, and Internet-based treatments. Finally, we end on a discussion of areas that are ripe for future investigation, including biological mechanisms, sleep medications, and other sleep disorders such as hypersomnia.

## INTRODUCTION

All animals need to sleep, from ants to dolphins to humans. As the knowledge base builds, scientific consensus on why we need sleep is growing. What is very clear is that if we do not get enough sleep there are serious consequences to both our physical and emotional health. Insomnia, which involves difficulty falling asleep, staying asleep, or waking earlier than desired in the morning, is one of the most common health problems. On its own, it is associated with considerable functional impairment but beyond that, insomnia is very frequently comorbid with, and predicts the development of, several psychological and medical conditions.

Over the past decade, as an understanding of the adverse consequences of insufficient sleep has emerged, research on insomnia and other sleep disorders has exploded. The study of insomnia is incredibly interesting from a scientific point of view as it provides insights into emotion processing and regulation as well as learning and memory, which are key fields studied in psychology. Furthermore, it is fascinating from a clinical perspective as it

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seems likely that effective interventions for insomnia may assist in the prevention of other mental health disorders and physical disorders, too. There appear to be bidirectional relationships between sleep disturbance and emotional dysregulation in that both impact on one another. Research into these relationships is providing important clues about mechanisms involved in insomnia and are detailed later. Other sleep disorders include hypersomnia, delayed sleep phase disorder (DSPD), sleep apnea, narcolepsy, night terrors, parasomnias, and restless leg syndrome. This chapter focuses primarily on insomnia, and touches upon hypersomnia and DSPD.

## FOUNDATIONAL RESEARCH

### Circadian Rhythms and Sleep

Many biological processes, including sleep, occur in a daily rhythm, roughly around a 24-h clock. Our internal body clock is influenced by several factors in the environment, termed *zeitgebers*. The most powerful zeitgeber is light/dark. Other cues that help us entrain our rhythms include temperature, exercise, and meal times. The internal pacemaker lies in the suprachiasmatic nucleus (SCN) of the brain, which receives light signals directly from the retina of the eye.

### Sleep Stages

Human sleep is divided into REM (rapid eye movement) and non-REM sleep, which is further divided in to four stages (stages 1, 2, 3, and 4). Stages 1 and 2 are "light" stages of sleep and stages 3 and 4 are "deep," more restorative stages of sleep in which slow-wave sleep occurs. Non-REM sleep appears to be important for conservation of energy and restoration (Kryger, Roth, & Dement, 2005) as well as the consolidation of certain types of memories (Diekelmann & Born, 2010). REM sleep is also important in learning and memory and is particularly critical in emotion processing and regulation (Karni, Tanne, Rubenstien, Askenasy, & Sagi, 1994; Walker & Stickgold, 2006; Yoo, Gujar, Hu, Jolesz, & Walker, 2007).

### Two-Process Model of Sleep

The two-process model is the prevailing theory of sleep regulation, which proposes that sleep and wakefulness are dependent on two processes, a circadian process and a homeostatic process (Borbely, 1982). The circadian rhythm is an internal biological clock that runs approximately on a 24-h basis. Variations in melatonin, temperature, and levels of arousal operate according to

our circadian rhythms (Lack & Bootzin, 2003). Through the homeostatic process, "sleep pressure" (or the need to sleep) increases with time spent awake. When a person is sleep deprived, the tendency to sleep increases. Conversely, when one has a long nap, the tendency to sleep is decreased. These two processes operate in tandem in that the propensity to sleep is more likely when levels of alertness (the circadian process) are relatively low and sleep pressure (the homeostatic process) is high. As a corollary, following an afternoon nap, one may find it difficult to fall asleep at one's regular "circadian bedtime" because homeostatic sleep pressure is low. Similarly, circadian arousal may prevent sleep onset from occurring if one tries to go to sleep early following a poor night of sleep, even though sleep pressure is high.

## CUTTING-EDGE RESEARCH

Exciting developments, outlined later, are being made in the treatment of insomnia and in the understanding of the mechanisms underpinning the relationship between insufficient sleep and psychiatric disorders.

### Sleep and Psychiatric Disorders

Evidence for the importance of sleep in psychiatric disorders has grown enormously in recent years. Certainly, we have all had the experience of poor sleep affecting our mood and the research is beginning to catch up by explaining this interaction at the behavioral, cognitive, and biological levels. This research is extremely important in helping to identify possible therapeutic targets for both insomnia and other psychiatric disorders.

Given that insomnia frequently predates and predicts psychiatric disorders, sleep disturbance is no longer considered an epiphenomenon in psychiatric disorders; rather, it is now viewed as an important and underrecognized mechanism in the cause and maintenance of psychiatric disorders. This change has recently been reflected in the latest edition of the Diagnostic and Statistical Manual (DSM-5), in which the diagnosis of primary insomnia has been renamed insomnia, to eliminate the distinction between primary and secondary insomnia.

### A Bidirectional Model Between Sleep and Emotion

Recently, we and others have proposed that the relationship between sleep disturbance and processes of emotional dysregulation in psychiatric disorders is likely to be bidirectional (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010; Harvey, Murray, Chandler, & Soehner, 2011). This simple model proposes that mood, anxiety, and other emotional symptoms interfere with

sleep, and that, conversely, the effects of sleep deprivation contribute to mood dysregulation and emotional disturbance. It is clear anecdotally and through experimental manipulations that worry and rumination, key features of anxiety and depressive disorders, affect our sleep. Equally, exciting research from Dr. Matthew Walker's group at the University of California, Berkeley has shown that just a single night of sleep deprivation causes significant increases in emotional reactivity to negative stimuli and corresponding increases in activity in the amygdala, an area of the brain that plays a key role in emotion (Yoo *et al*., 2007). Not only has increased emotional activity to negative stimuli been observed but it has also been shown that sleep deprivation leads to amplified neural reactivity in reward networks in response to pleasant stimuli (Gujar, Yoo, Hu, & Walker, 2011). Indeed, in an article entitled "Overnight therapy? The role of sleep in emotional brain processing," Walker and his colleague emphasize the role of REM sleep in processing emotional memories and note that there has been a "renaissance" of research with a focus on sleep and neurocognitive processes involved in emotion regulation. Certainly, this is a fascinating area which is sure to grow in the future. We suggest that this bidirectional model between sleep and emotion is valuable as an initial organizing structure for the existing and future research. The study of sleep across psychiatric disorders is an exciting domain for interdisciplinary research across behavioral, social, cognitive, and neurobiological levels of explanation.

### Cognitive Behavioral Therapy for Insomnia (CBT-I)

Cognitive Behavioral Therapy for Insomnia (CBT-I) is the most effective psychological treatment for insomnia. Unlike pharmacological treatments, its effects persist after a course of treatment has been terminated (Morin, 2006). The aim of CBT-I is to foster sleep-conducive behaviors and to change unhelpful and unrealistic expectations about sleep and daytime functioning. CBT-I has been shown to increase total sleep time and improve sleep quality, while reducing the time it takes to fall asleep and the number of awakenings in the night time. It is a multicomponent therapy which involves cognitive therapy, stimulus control, sleep restriction, behavioral experiments, and psychoeducation about good sleep habits. One of the more paradoxical components of CBT-I is sleep restriction. The aim of this is to match time in bed as closely as possible with time asleep by limiting the amount of time the patient sleeps in the initial stages of therapy. This helps the patient to consolidate sleep, associate the bed with sleep, and to shorten sleep onset times as she/he accumulates a sleep debt through mild sleep deprivation. Stimulus control, which aims to create a positive association between the bed/bedroom and sleep by encouraging the patient to get out of bed when

not tired and only use the bed for sleep and sex, while restricting other activities in bed (e.g., watching television, checking e-mails), has also been shown to be an important contributor to treatment gains. CBT-I is generally conducted over six to eight sessions, and similar to CBT treatments for other disorders, it involves in-session activities as well as homework to be completed between sessions.

### Treatment Development

Progress toward establishing treatments has been good. However, given that effect sizes are small to moderate and many patients derive no benefit, there is certainly room for improvement (Harvey & Tang, 2003). Advances in treatment of insomnia are likely to have valuable widespread ramifications because it seems probable that treating insomnia will translate into reductions in other mental health disorders such as anxiety and depression, as insomnia is known to be predictive of the onset of these disorders. This section outlines a few key areas of treatment development currently under research.

The role of light and dark in sleep is well understood and there is growing evidence that light and dark therapy is likely to be important in the treatment of mood disorders (Harvey, 2011). The use of *bright light* is another key development in the treatment of sleep disorders. Morning bright light will bring forward (phase advance) the circadian rhythm and is therefore an effective therapeutic intervention for "owls," whose preference for staying up late and sleeping in late interferes with their daily duties. On the other hand, evening bright light delays the circadian rhythm and is therefore effective for "larks," those who wake up earlier that they would like to because of an abnormally advanced sleep phase (Lack & Wright, 2007). Although the use of light boxes have been shown to be useful in the treatment of DSPD, adherence is not optimal and therefore, the use of "informal" light exposure (e.g., sunlight in the morning, reduced light from electronic devices in the evening) is an important part of treatment.

Light therapy may also be combined with *sleep phase chronotherapy*, which involves moving sleep time 3 h later each night, around the clock, until the desired sleep time is established. More commonly, however, light therapy is combined with shifting sleep and wake times more gradually (say, 30 min per week) forward or backward depending on the desired sleep/wake schedule. For example, a patient who wishes to move their bedtime from 12 a.m. to 10 p.m. would set a regular wake time and endeavor to get morning light as soon as possible after waking. Simultaneously, they would shift their bedtime backwards half an hour each week until a regular pattern is established.

*Intensive Sleep Retraining Therapy.* Intensive sleep retraining therapy, which involves a one-off 25-h treatment period of sleep deprivation in which patients are only allowed to sleep for a few minutes at a time, has shown promising results for the treatment of insomnia, with reductions observed in time taken to fall asleep and total sleep time (Harris, Lack, Kemp, Wright, & Bootzin, 2012). During this treatment, participants are allowed a series of 50 half-hourly sleep opportunities. If they do not sleep within 20 min, the patient is asked to move out of the bed on to a chair to read or watch DVDs until the next trial begins. If sleep is initiated, participants are only allowed to sleep for 3 min before being woken. On each trial where the participant does sleep, they are asked about their perception of whether or not they slept and provided with accurate feedback regarding whether sleep onset occurred or not. Even the most extreme insomnia patients are able to sleep in this protocol. Intensive sleep retraining therapy is thought to work in several ways, including (i) by helping people to experience what it is like to fall asleep quickly and to learn that they are capable of falling asleep fast; (ii) by providing exposure to sleep deprivation (which is often feared by patients with insomnia); and (iii) by helping to correct sleep misperception (sleep/wake discrimination). It has very rapid results, although an even larger treatment effect size has been observed when this treatment was combined with five sessions of stimulus control therapy.

*Internet-Based Treatments.* In recent years, Internet-based treatments for a number of mental disorders have grown enormously. Indeed, there are a few different groups of researchers working on Internet-based treatments for insomnia. For example, Colin Espie and colleagues in Scotland have developed "Sleepio," a CBT Internet program developed as a self-help treatment for insomnia. "SHUTi," developed in the United States, is another example of an online self-help program for insomnia and randomized controlled trials of these programs are currently under way. Internet-based treatments are particularly helpful in the dissemination of evidence-based treatments for insomnia because they are easily accessible and lessen the burden on the health system as specialist clinician time is reduced.

## Targeting Sleep in Adolescence

Adolescence has emerged as a crucial time point to intervene with sleep. This is due to two key factors—(i) Many mental health disorders such as anxiety and depression have their onset in adolescence and insomnia is a risk factor for the emergence of these disorders; and (ii) the natural biological delay in the circadian cycle that occurs in adolescence along with increased use of

technology and other changes in social pressures are now competing more than ever with the need to sleep.

Clinically, when one's sleep schedule is heavily delayed and this interferes with one's ability to conform to a required schedule (e.g., school or work start times), one may meet criteria for a diagnosis of DSPD. An obvious corollary of DSPD in adolescents, who are required to wake early to be at school, is chronic sleep deprivation. This can put such a teenager at risk for the development of psychiatric and physical disorders as well as have deleterious effects in the academic and social domains. Several complex interactions between biology, psychology, and social context make adolescence a particularly important phase for mechanistic and early intervention research. Indeed, our group is currently investigating whether a brief intervention to target DSPD in teenagers will help reduce risk across social, academic, physical, and psychological domains.

## KEY ISSUES FOR FUTURE RESEARCH

One of the great things about working on sleep is that there is so much more to learn. Despite the fact that sleep takes up about one-third of our life, very little is known about its function. Certainly, it is clear that insomnia is common and debilitating and there are a number of exciting areas for future research in this disorder and other sleep disorders.

### Hypersomnia

More recently, an understanding of the importance of hypersomnia has developed (Kaplan & Harvey, 2009). It is still unclear whether hypersomnia is a disorder of excessive sleep or excessive time in bed. That is, do people with this disorder actually sleep for excessively long periods or simply spend large amounts of time in bed? As has been pointed out by our group previously, the use of objective measures of such as actigraphy (which assesses an individual's movement as a fairly accurate proxy of actual sleep) will help shed light on this quandary (Kaplan & Harvey, 2009). It may also be the case that psychological factors such as avolition and daytime fatigue emerge as being more important to this disorder than objective sleep. What is clear, however, is that as with insomnia, hypersomnia is prevalent and persistent across mood disorders and there is certainly a need for greater research into this disorder and its treatment.

### Transdiagnostic Treatment

Given the high rates of co-occurrence between insomnia and psychiatric disorders as well as evidence suggesting that insomnia contributes to the onset,

maintenance, and relapse of a range of psychiatric disorders, we have suggested that insomnia may be a transdiagnostic process across psychiatric disorders (Harvey, 2008). Therefore, the development of a single treatment that can be applied to individuals with comorbid insomnia across disorders has the potential to provide great benefits to public health. However, research is needed to determine whether it is possible to develop one treatment protocol that will be effective across psychiatric disorders. Secondly, given the research showing that insomnia predicts the onset and maintenance of other psychiatric disorders, it appears to be a logical consequence that the treatment of insomnia will improve the functioning and symptomatology of individuals with psychiatric disorders and indeed even prevent the onset of some cases, yet this is still to be determined empirically.

### Sleep Versus Circadian Rhythm Disturbance?

Sleep disturbances and circadian rhythm abnormalities, although overlapping, are not identical processes. That is, although the circadian rhythm contributes to sleep disturbance, other factors such as stress also play a role. Similarly, the circadian rhythm not only controls sleep but also other biological processes. Both sleep and the circadian rhythm have been implicated in psychiatric disorders, although at this stage their individual contribution to psychiatric disorders is unknown. Therefore, future research on the involvement of sleep versus circadian influences in psychiatric disorders is needed.

### Biological Mechanisms

Work is already beginning to show that the sleep/circadian systems are closely linked to serotonergic and dopaminergic function, which points to one common mechanism potentially underlying sleep and mood/anxiety disorders (see Harvey *et al*., 2011 for review). The manipulation of specific stages of sleep may be a valuable approach to answer important questions about the contribution of REM and non-REM sleep to psychiatric disorders, and indeed it may be possible to selectively manipulate different stages of sleep for therapeutic benefit. For example, it is known that individuals with depression typically have shortened REM latency and higher REM density (Coble, Foster, & Kupfer, 1976). Although widely documented, the underlying mechanisms and reasons for this excess of REM sleep in depression are poorly understood. Interestingly, antidepressant medications suppress REM sleep (Mayers & Baldwin, 2005). Similarly, it has been suggested that individuals with posttraumatic stress disorder (PTSD) fail to show a suppression of brain adrenergic activity during REM sleep (Shad, Suris, & North, 2011; Taylor *et al*., 2008) and this suppression has been

proposed to be central to the emotional processing of negative memories (Walker & van Der Helm, 2009). Consistent with this, an adrenergic antagonist (Prazosin), which reduces adrenergic activity during REM sleep (Shad *et al*., 2011; van der Helm *et al*., 2011) has been shown to reduce symptoms of PTSD. Understanding the mechanisms underpinning insomnia and the relationship between sleep processes and psychiatric disorders is invaluable in developing better treatments for these disorders.

### Sleep Medications

Finally, it is worth noting that further research into sleep medications is warranted. Hypnotic medications such as Zolpidem (Ambien) and Temazepam (Restoril) are some of the most widely used medications in Western societies (Kripke, Langer, & Kline, 2012). Yet, it seems to be that the type of sleep one obtains when under the influence of medications such as Zolpidem is quite different to naturally occurring sleep. That is, naturally occurring sleep is known to be essential to learning and memory consolidation. In contrast, pharmacologically induced sleep appears to actually impair critical sleep-dependent brain processes. For example, in a study involving developing kittens, Seibt *et al*. (2008) found that Zolpidem reduced cortical plasticity by 50%. This suggests that these agents may be of concern to developing human children and adolescents, potentially as well as adults, who continue to need sleep for growth, repair, learning, plasticity, and optimal emotional functioning.

## REFERENCES

Baglioni, C., Spiegelhalder, K., Lombardo, C., & Riemann, D. (2010). Sleep and emotions: A focus on insomnia. *Sleep Medicine Reviews*, *14*, 227–238. doi:10.1016/j.smrv.2009.10.007

Borbely, A. (1982). A two process model of sleep regulation. *Human Neurobiology*, *1*, 195–204.

Coble, P., Foster, F. G., & Kupfer, D. J. (1976). Electroencephalographic sleep diagnosis of primary depression. *Archives of General Psychiatry*, *33*, 1124.

Diekelmann, S., & Born, J. (2010). The memory function of sleep. *Nature Reviews Neuroscience*, *11*, 114–126.

Gujar, N., Yoo, S.-S., Hu, P., & Walker, M. P. (2011). Sleep deprivation amplifies reactivity of brain reward networks, biasing the appraisal of positive emotional experiences. *The Journal of Neuroscience*, *31*, 4466–4474.

Harris, J., Lack, L., Kemp, K., Wright, H., & Bootzin, R. (2012). A randomized controlled trial of intensive sleep retraining (ISR): A brief conditioning treatment for chronic insomnia. *Sleep*, *35*, 49–60.

Harvey, A. G. (2008). Insomnia, psychiatric disorders, and the transdiagnostic perspective. *Current Directions in Psychological Science*, *17*, 299–303.

Harvey, A. G. (2011). Sleep and circadian functioning: Critical mechanisms in the mood disorders? *Annual Review of Clinical Psychology*, *7*, 297–319.

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review*, *31*, 225–235.

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

Kaplan, K. A., & Harvey, A. G. (2009). Hypersomnia across mood disorders: A review and synthesis. *Sleep Medicine Reviews*, *13*, 275–285.

Karni, A., Tanne, D., Rubenstien, B. S., Askenasy, J. J., & Sagi, D. (1994). Dependence on REM sleep of overnight improvement of a perceptual skill. *Science*, *265*, 679–682.

Kripke, D. F., Langer, R. D., & Kline, L. E. (2012). Hypnotics' association with mortality or cancer: A matched cohort study. *BMJ Open*, *2*. doi:10.1136/bmjopen-2012-000850

Kryger, M. H., Roth, T., & Dement, W. C. (2005). *Principles and practice of sleep medicine* (4th ed.). Philadelphia, PA: WB Saunders Co..

Lack, L. C., & Bootzin, R. B. (2003). Circadian rhythm factors in insomnia and their treatment. In M. Perlis & K. Lichstein (Eds.), *Treatment of sleep disorders: Principles and practice of behavioral sleep medicine*. New York, NY: John Wiley and Sons, Inc.

Lack, L. C., & Wright, H. R. (2007). Treating chronobiological components of chronic insomnia. *Sleep Medicine*, *8*, 637–644.

Mayers, A. G., & Baldwin, D. S. (2005). Antidepressants and their effect on sleep. *Human Psychopharmacology: Clinical and Experimental*, *20*, 533–559. doi:10.1002/hup.726

Morin, C. M. (2006). Cognitive-behavioral therapy of insomnia. *Sleep Medicine Clinics*, *1*, 375–386.

Seibt, J., Aton, S. J., Jha, S. K., Coleman, T., Dumoulin, M. C., & Frank, M. G. (2008). The non-benzodiazepine hypnotic zolpidem impairs sleep-dependent cortical plasticity. *Sleep*, *31*, 1381–1391.

Shad, M. U., Suris, A. M., & North, C. S. (2011). Novel combination strategy to optimize treatment for PTSD. *Human Psychopharmacology: Clinical and Experimental*, *26*, 4–11.

Taylor, F. B., Martin, P., Thompson, C., Williams, J., Mellman, T. A., Gross, C., … Raskind, M. A. (2008). Prazosin effects on objective sleep measures and clinical symptoms in civilian trauma posttraumatic stress disorder: A placebo-controlled study. *Biological Psychiatry, 63*, 629–632.

van der Helm, E., Yao, J., Dutt, S., Rao, V., Saletin, J. M., & Walker, M. P. (2011). REM sleep depotentiates amygdala activity to previous emotional experiences. *Current Biology*, *21*(23), 2029–2032.

Walker, M. P., & Stickgold, R. (2006). Sleep, memory, and plasticity. In *Annual review of psychology* (Vol. 57, pp. 139–166). Palo Alto, CA: Annual Reviews.

Walker, M. P., & van Der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological Bulletin*, *135*, 731.

Yoo, S.-S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep—a prefrontal amygdala disconnect. *Current Biology*, *17*, R877–R878.

## FURTHER READING

Harvey, A. G. (2011). Sleep and circadian functioning: Critical mechanisms in the mood disorders? *Annual Review of Clinical Psychology*, *7*, 297–319.

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review*, *31*, 225–235.

Morin, C. M. (2006). Cognitive-behavioral therapy of insomnia. *Sleep Medicine Clinics*, *1*, 375–386.

Walker, M. P., & van Der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological Bulletin*, *135*, 731.

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