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Original article

Insomnia and depression

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Abstract

It is clear that insomnia and depression are intimately related, which may suggest an overlapping neurobiology. Although much progress has been made toward understanding how these disorders relate to each other, the exact neurobiological mechanisms that link them remain elusive. Sleep changes in depression may be associated with abnormal neurotransmission, genetic polymorphisms, HPA overactivity, impaired plasticity, or most likely a combination of factors. It is therefore crucial that sleep assessments go beyond traditional polysomnography to include a more expanded set of objective measures in the hope that these will uncover the common neurobiology that is thought to underlie insomnia and depression.
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Keywords: Insomnia; Depression; Neurobiology; Electroencephalogram; Slow wave activity

1. Introduction

Depression was once classically viewed as a cause of insomnia, but a growing body of epidemiological studies has suggested that insomnia may itself be a risk factor for depression [1–5]. As the relationship between sleep and depression has been explored over the years it has become apparent that each affects the other in a bi-directional manner; yet the question remains as to whether insomnia is a precursor to depression, a premorbid trait, or an independent risk factor [6,7]. Before this association can be fully defined, there is still much to learn about the shared neurobiology of sleep and mood.

2. Sleep disturbance and depression

Sleep disturbances (both insomnia and hypersomnolence) are so frequently observed in patients experiencing acute episodes of mood disorders that they form part of the diagnostic and statistical manual (DSM) criteria for these disorders. In particular, patients with depression often complain of difficulty getting to sleep, frequent awakenings during the night, early morning awakening, or nonrestorative sleep [8–10]. Epidemiological studies have shown that patients with mood disorders exhibit higher rates of sleep disturbance than the general population, and sleep disturbance can continue even during periods of remission [11]. On the other hand, patients with insomnia are up to 10 times more likely to have depression than normal sleepers [12–14], and individuals with persistent insomnia have a significantly higher risk of developing new-onset depression than those who have no sleep complaints [1,3,4]. Ford and Kamerow (1989) [1] found that 14% of patients with persistent insomnia had concurrent depression whereas depression occurred in less than 1% of patients who had no sleep complaints ($P < 0.001$; Figure 1). In fact, the occurrence of several psychiatric disorders was significantly higher in patients with insomnia than in individuals with no sleep complaints (Figure 1). Because of its prospective design, the study was also able to show that patients with persistent insomnia had a substantially higher risk of developing a new major depression compared with those whose insomnia resolved (odds ratios of 39.8 and 1.6, respectively, in comparison to those with no insomnia).

The temporal relationship between insomnia and psychiatric disorders may vary – for some patients, insomnia and psychiatric problems develop simultaneously, while for others they occur sequentially (Figure 2). Insomnia tends to precede or co-occur with mood disorders, whereas it tends to present at the same time or following onset of an anxiety disorder [15]. The more severe and chronic the insomnia, the greater the likelihood the individual will have a prior history of a mental disorder. For example, Ohayon and Roth reported that among a general population sample of almost 15,000 Europeans, those with severe insomnia were 6 times more likely to report a psychiatric condition in their past than subjects who did not experience insomnia. In contrast, those with moderate insomnia were only 1.4 times

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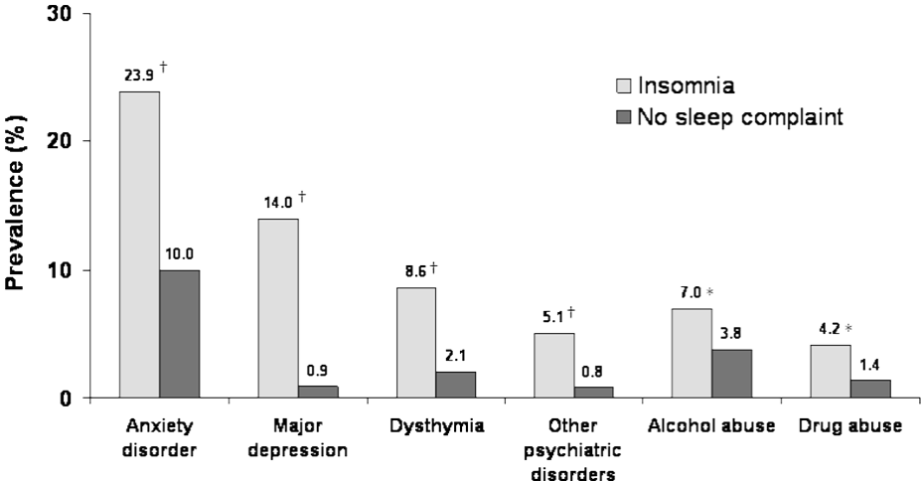

Fig. 1. Prevalence of comorbid psychiatric disorders in 811 individuals with insomnia [1]. †$P < 0.001$ vs. no sleep complaint; *$P = 0.05$ vs. no sleep complaint.

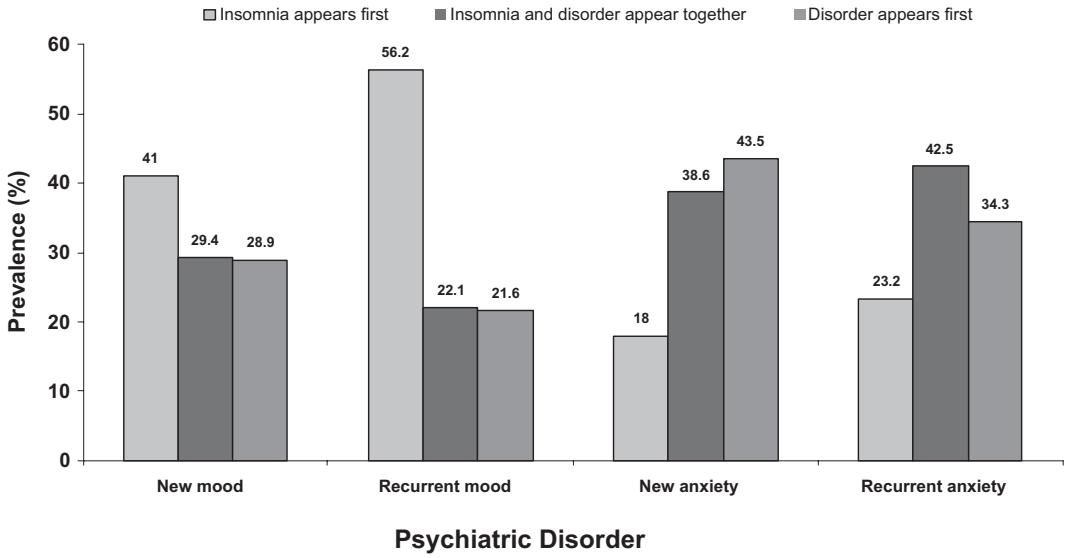

Fig. 2. Timing of insomnia related to onset of psychiatric illness (n = 14,915) [15].

more likely to report a psychiatric condition compared with those without insomnia [15]. Notably, some people with a current mental disorder reported a duration of insomnia of more than 5 years in addition to a past history of mental disorder, which suggests that insomnia may persist even when a mental illness is in remission [15]. Others regard insomnia as the most common refractory symptom of mood disorders, such as depression [16]. Furthermore, persistence of insomnia has been shown to be predictive of increased severity and recurrence of mood disorders [17,18].

3. Biological mechanisms for sleep changes in depression

The close correlation between depression and insomnia suggests there are common elements with regard to underlying neurobiology. There are a number of biological mechanisms that may explain altered sleep patterns in depressed patients, including (but not limited to) deficits in monoaminergic neurotransmission, abnormalities in circadian genes, overactivity of the hypothalamic–pituitary–adrenal (HPA) axis, and impaired functioning of plasticity-related gene cascades.

During normal sleep, electroencephalograph (EEG) activity typically reveals progressive transitions from light sleep (stage N1) to an "intermediate" level of sleep (stage N2), and then to "deep sleep" (slow wave sleep [SWS], characterised by slow delta waves; stage N3; Figure 3). The sleep stages N1–N3 comprise non-rapid eye movement (NREM) sleep which alternates across the night with episodes of REM sleep (Figure 3). REM sleep is initiated when the serotonergic and noradrenergic activity decreases and cholinergic activity increases, and ceases when the activity of the monoamines increases and cholinergic activity is reduced [20]. In patients with depression, REM sleep propensity is increased, leading to reduced REM latency, increased proportion of REM sleep,

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