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Treatment of insomnia – A preventive strategy for cardiovascular and mental disorders

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ABSTRACT

Insomnia is defined as a difficulty initiating or maintaining sleep with daytime impairments like increased fatigue, decreased attention, and increased mood irritability for at least three nights a week for a period of 3 months. DSM 5 has established a new overarching category of insomnia disorder, thus replacing the old primary vs. secondary dichotomy.

Insomnia has been traditionally viewed as a symptom or consequence of somatic or mental disorders. The last decade has seen a paradigm shift, focusing more on the question whether insomnia as an independent diagnostic entity may be predictive for other disorders/diseases, especially in the cardiovascular and mental area. There is now strong evidence that insomnia is a risk factor for cardiovascular disorders on the one hand and for mental disorders, especially depression, on the other hand. These data raise the question, whether early and adequate treatment of insomnia may be preventive for the somatic or mental sequelae of the disorder. The first-line treatment for insomnia is cognitive behavioral treatment (CBT-I) which comprises techniques like education about sleep, behavioral recommendations, relaxation training and cognitive restructuring. These techniques are usually delivered as a treatment package in one to eight sessions. CBT-I has been shown to be very effective for the treatment of insomnia. There is now also growing evidence that this type of treatment is effective in comorbid insomnia, i.e. insomnia coupled with mental or somatic disorders. First tentative data indicate that insomnia treatment may be effective as prevention for mental and somatic disorders.

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1. Introduction

A review article on the epidemiology of insomnia indicated that approximately 6% of the adult population in industrialized countries suffer from chronic insomnia according to DSM-IV criteria. These criteria encompass day- and nighttime symptoms; the night-time symptoms include difficulties initiating or maintaining sleep or early morning awakening, daytime symptoms include impairments like decreased attention and motivation, increased fatigue, tension headache, and increased mood irritability (Ohayon, 2002). Whereas insomnia as a transient phenomenon is ubiquitous and frequently remits spontaneously, data from a natural history investigation indicated that 74% of the individuals who fulfilled most of the criteria for at least 4 weeks, will still suffer from insomnia 12 months later (Morin et al., 2009). It has been shown for insomnia in its chronic form that it is associated with adverse health outcomes and a reduced quality of life (Kyle, Morgan, & Espie, 2010). Several prescriptive longitudinal studies have now documented that individuals suffering from insomnia have an increased risk for developing or dying from cardiovascular diseases (Sofi et al., 2014). Insomnia occurs with a frequency of 25–60% in the context of neurological disorders (Mayer, Jennum, Riemann, & Dauvilliers, 2011). Other authors (Jaffee, Winter, Jones, & Ling, 2015; Sexton, Storsve, Walhovd, Johansen-Berg, & Fjell, 2014) have described that insomnia may be involved in the development of cognitive impairments and that there is a relationship between poor sleep quality and cortical atrophy in elderly adults living in the community. Concerning insomnia and mental disorders, a recent meta-analysis (Baglioni et al., 2011) found that insomnia significantly increases the risk of developing depression in subsequent years. This relationship has also been demonstrated by meta-analytic studies for suicidal ideation/behavior and insomnia. Furthermore, growing evidence shows that insomnia is a risk factor for work disability, sick leave and reduced work performance (Kucharczyk, Morgan, & Hall, 2012). Thus it is not surprising that insomnia is associated with indirect costs for our health care systems and society (Léger & Bayon, 2010).

Summarizing, chronic insomnia as a common disorder, has a strong impact on somatic and mental health and is coupled with high costs for health care systems. The DSM 5 (American Psychiatric Association, 2013, S. 5) has acknowledged this impact by creating the new overarching diagnostic category insomnia disorder (ID) replacing traditional primary/secondary insomnia concepts.

2. Pathophysiological models of insomnia

2.1. 3 P model

The 3 P model of insomnia (Fig. 1) was proposed by Spielman, Caruso and Glovinsky (1987) and describes predisposing, precipitating and perpetuating factors of the disorder. Predisposing factors are characteristics ("traits") which render a person vulnerable for poor sleep. Importantly, these factors do not inevitably lead to insomnia – they rather increase the risk to react to stressors with sleep disturbance. Within the recent years, several studies have been published which argue in favor of a genetic predisposition for insomnia (Hublin, Partinen, Koskenvuo, & Kaprio, 2011; overview: Palagini, Biber, & Riemann, 2014). The heritability of the disorder has been estimated at 55% in men and 43% in women (Drake, Friedman, Wright, & Roth, 2011). Furthermore, psychological factors, as for example perfectionistic personality traits are a risk factor for insomnia (Azevedo et al., 2010; Jansson-Fröjmark & Linton, 2007). Perfectionism is a personality profile comprising doubt, extremely high standards and fear of making mistakes.

Precipitating factors are stressful events which immediately precede the onset of insomnia. Patients often describe stress at work, health concerns and family conflicts as triggers for the first onset of their insomnia (Bastien, Vallieres, & Morin, 2004). While in most people, sleep disturbances resolve when the stressful event is managed, the 'perpetuating' factors in the 3 P model describe why insomnia becomes chronic. A crucial perpetuating factor is inadequate sleep hygiene, i.e. irregular sleep-wake rhythms, prolonged bedtimes and daytime naps which decrease sleep pressure and thus make falling asleep difficult. Other perpetuating factors are worry and rumination over the sleeping problem as well as the intake of substances with a sleep disturbing effect (alcohol, nicotine, caffeine, etc.).

2.2. Cognitive model

In their cognitive model of insomnia (Fig. 2), Harvey et al. describe how cognitive and behavioral processes contribute to the maintenance of insomnia in the form of a vicious circle (Harvey, 2002; Harvey, Tang, & Browning, 2005). The patients notice a sleep disturbance which may initially be related to a stressful event. They then start worrying about their insomnia and its adverse consequences for their life and their health. Because they perceive their sleep disturbance as a threat, they closely monitor their sleep, which leads to an increased awareness of sleep disturbance and a potential overestimation of sleep deficits. The perceived sleep deficit then results in worried arousal about the consequences of insomnia. Increased arousal, in turn, aggravates the sleeping problem. Dysfunctional safety behaviors such as extended bedtimes and daytime naps further maintain insomnia.

2.3. Hyperarousal model

Increased levels of physiological arousal, mental activity and emotional reactivity are characteristic for patients with insomnia – both at night and during the day (see Fig. 3, Bonnet & Arand, 1997;

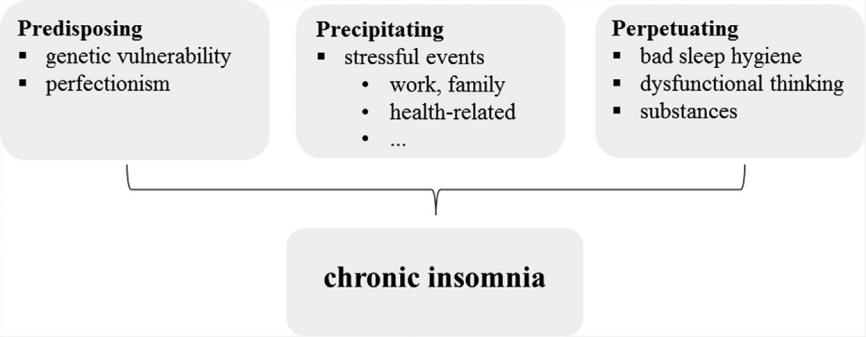

Fig. 1. The 3-P Model of Insomnia (compare Spielman et al., 1987).