

Multiple Causality in the Impairment of Daytime Functioning Associated with Insomnia: Is it Time to Reconsider Insomnia Subtypes?

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Approximately 10-30% of individuals experience insomnia worldwide [1,2]. Chronic insomnia causes disturbances to nighttime sleep, including prolonged sleep latency, disturbance of sleep maintenance, and non-restorative sleep. It can also cause various Impairments of Daytime Functioning (IDFs) such as fatigue, mood disturbance, irritability, reduced motivation and interest, attention deficit, and memory impairment, with a consequent decline in occupational, social, or leisure quality [3]. A socioeconomic study has predicted that IDFs have a greater impact on the global economy rather than disturbances in night time sleep resulting from insomnia [4]. As a result, IDFs associated with insomnia have recently been a subject of particular interest. However, since the biological background and pathogeny of IDFs has not yet been clarified, they are not unconsidered in the differential diagnosis of insomnia subtypes in the latest version of the International Classification of Sleep Disorders (ICSD-2) [5], published in 2005. However, the criteria of "primary insomnia" in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) will be changed to "insomnia disorder" in the fifth edition (DSM-V) [6], which will be released in 2013 [7]. In line with this modification, IDFs will be clearly defined as specified sequelae based on specific biological vulnerabilities to illness, contrasting with the traditional, ambiguous definitions of IDFs.

It was suggested that the type and severity of subjective symptoms related to IDFs vary according to the insomnia subtype classification in ICSD-2 [8]. Insomnia associated to a mental disorder and idiopathic insomnia was associated with symptoms of mood disturbance and low sleepiness; and psychophysiological insomnia and inadequate sleep hygiene were related to symptoms of daytime tension and low fatigue. Whereas paradoxical insomnia showed subtle symptoms with mild severity of IDFs, and insomnia associated to a mental disorder showed the most severe IDFs.

Despite this relatively extensive understanding and categorization, the data on the severity of cognitive dysfunction related to IDFs arising from different insomnia subtypes is scarce. In particular, there is still controversy regarding whether primary insomnia patients exhibit cognitive dysfunctions as IDFs or not. A recent meta-analysis indicated a small to moderate magnitude of cognitive impairment in insomnia patients that involved episodic memory, problem solving, and some working memory skill domains [9]. However, a great heterogeneity in the magnitude of these effects on cognitive functions suggests that other factors may have produced variation among these studies. The heterogeneous subtype of insomnia may contribute to this variance in cognitive functions.

At least 2 overlapping biological vulnerabilities may trigger IDFs, one of which is vulnerability to sleep loss per se, and the other is stress vulnerability that might trigger IDFs in chronic insomnia via various cognitive dysfunctions. It is well established that attention is one of the cognitive processes most vulnerable to sleep loss [10], and yet it is essential to virtually every cognitive process. Thus, a wide range of cognitive abilities might be impaired by accumulated sleep loss. In

contrast, stress vulnerability, which includes vulnerabilities to illness or insanity, is not always associated with sleep loss, but is a common cause of insomnia and various other physical and mental ailments.

Recently, our own research indicated that a personality trait of neuroticism, which includes signs of stress vulnerability and often presents with a chief complaint of subjective sleep quality deterioration (sleep dissatisfaction), actually gives rise to improve daytime attention levels and higher cognitive performance via overcoming sleep loss [11]. Most paradoxical and psychophysiological insomniacs often share this personality trait of neuroticism, and therefore, they may show somewhat better daytime cognitive functions than healthy subjects with a less pronounced neuroticism trait. Their daytime dysfunctions may therefore be limited to complaints of subjective mental and physical problems, and the resulting social and occupational maladaptation.

Differences in IDFs between insomnia subtypes arise from the interaction between these 2 vulnerabilities, and thus, we cannot attribute IDFs to a specific common cause in all cases of insomnia. This idea helps us to understand the therapeutic dynamics of Cognitive Behavioral Therapy for Insomnia (CBT-I). Given therapeutic strategy and efficacy of CBT-I [12], it may reduce stress vulnerability rather than the vulnerability to sleep loss. Moreover, objective sleep deterioration (loss of sleep duration or sleep architecture impairments) may contribute to impairment in sleep-dependent neuroplasticity that in turn results in the deterioration of off-line memory consolidation [13,14]. Another vulnerability associated with the sleep-dependent neuroplasticity presumably contributes to the IDFs of social and occupational maladaptation.

Future diagnostic methods for insomnia subtypes using the next-generation classifications of sleep disorder should be developed with consideration of the biological backgrounds of IDFs, which may not only confirm the causality of primary insomnia but also contribute to the adoption of a strategy for therapeutic intervention based on insomnia subtypes. If we accept this concept, paradoxical insomnia and associated insomnia share the same pathological condition with anxiety or somatoform disorder defined in the DSM-IV, making it necessary to categorize them as general mental disorders rather than just specific sleep disorders.

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References

1. Soldatos CR, Allaert FA, Ohta T, Dikeos DG (2005) How do individuals sleep around the world? Results from a single-day survey in ten countries. *Sleep Med* 6: 5-13.
2. Ohayon MM, Reynolds CF 3rd (2009) Epidemiological and clinical relevance of insomnia diagnosis algorithms according to the DSM-IV and the International Classification of Sleep Disorders (ICSD). *Sleep Med* 10: 952-960.
3. Roth T, Ancoli-Israel S (1999) Daytime consequences and correlates of insomnia in the United States: results of the 1991 National Sleep Foundation Survey. II. *Sleep* 22 Suppl 2: S354-358.
4. Daley M, Morin CM, LeBlanc M, Grégoire JP, Savard J (2009) The economic burden of insomnia: direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep* 32: 55-64.
5. American Academy of Sleep Medicine (2005) *The International Classification of Sleep Disorders: Diagnostic & Coding Manual*. (2nd edn), American Academy of Sleep Medicine, Westchester, IL.
6. Espie CA, Kyle SD, Hames P, Cyhlarova E, Benzeval M (2012) The daytime impact of DSM-5 insomnia disorder: comparative analysis of insomnia subtypes from the Great British Sleep Survey. *J Clin Psychiatry* 73: e1478-1484.
7. Lawson AK, Wright CV, Fitzgerald LF (2013) The Evaluation of Sexual Harassment Litigants: Reducing Discrepancies in the Diagnosis of Posttraumatic Stress Disorder. *Law Hum Behav* .
8. Sánchez-Ortuño MM, Edinger JD, Wyatt JK (2011) Daytime symptom patterns in insomnia sufferers: is there evidence for subtyping insomnia? *J Sleep Res* 20: 425-433.
9. Fortier-Brochu E, Beaulieu-Bonneau S, Ivers H, Morin CM (2012) Insomnia and daytime cognitive performance: a meta-analysis. *Sleep Med Rev* 16: 83-94.
10. Killgore WD (2010) Effects of sleep deprivation on cognition. *Prog Brain Res* 185: 105-129.
11. Yoshiike T, Kuriyama K, Honma M, Kim Y, Nishikawa T (2013) Neuroticism simultaneously enhances daytime wakefulness and deterioration of subjective sleep quality via facilitating bilateral prefrontal efficiency: a preliminary study. Under submission.
12. Van Houdenhove L, Buyse B, Gabriëls L, Van den Bergh O (2011) Treating primary insomnia: clinical effectiveness and predictors of outcomes on sleep, daytime function and health-related quality of life. *J Clin Psychol Med Settings* 18: 312-321.
13. Cipolli C, Mazzetti M, Plazzi G (2013) Sleep-dependent memory consolidation in patients with sleep disorders. *Sleep Med Rev* 17: 91-103.
14. Kuriyama K, Mishima K, Suzuki H, Aritake S, Uchiyama M (2008) Sleep accelerates the improvement in working memory performance. *J Neurosci* 28: 10145-10150.