Pain, sleeping problems and their many relatives

Although it has been neglected for quite some time, the mutual relationship between pain and sleeping problems has recently become the focus of significant scientific and clinical interests [2,4]. The – at first glance – unusual perspective that disturbances of regular night sleep cause or exacerbate pain the next day is now widely acknowledged and even influences recent developments in pain management. For example, first attempts have been made to evaluate the effects of the Cognitive-Behavioral Therapy of insomnia on pain [7]. And not surprisingly, as soon as the association between the pain and sleep disturbance was established, the search for the neurobiological and psychosocial links began. For example, it has been shown that nocturnal episodes of pain lead to changes in sleep architecture and to phases of arousal, which may even trigger awakening [3]. The reciprocal is also true; sleep fragmentation weakens the endogenous pain inhibitory system [6]. As we have gained some new insights, so far, so good. But we have not been asked to give up old ones.

Fortunately, studies such as the one by Buenaver et al. [1] in this issue of Pain, together with its theoretical background have started to challenge some of our well beloved beliefs. Wouldn’t we all like to agree that cognitive processes such as pain catastrophizing have mainly a direct influence on pain processing and that theoretical and experimental modeling of this influence is sufficient for our understanding? In fact, the report of Buenaver et al. shows convincingly that catastrophizing, an apparently pain-specific form of worrying and rumination leads – as an indirect cause of pain – first and foremost to sleep disorders. These disorders are in turn the direct cause of pain in chronic conditions such as temporomandibular disorder.

Another but similar theoretical challenge is already awaiting us. Depression is associated with alterations in pain processing, including chronic pain, sleep disorders in the form or fragmentation and early awakening, as well as rumination and worrying. Which are the pathways of direct, indirect and modulated causation in the resulting network? The situation might be considered to be even more complicated because cognitive dysfunctions – including failures of problem solving – can also be sequelae of chronic pain, sleep disorders and depression, whereas worrying and rumination may correspond to the individuals’ awareness of cognitively insoluble problems.

The pain-fear avoidance model of Vlaeyen and Linton [8], which has received common acceptance for the understanding of chronic musculoskeletal pain, offers at least three areas where sleep disorders can interact with the model. (i) Pain experience may be directly exacerbated by sleep discontinuity; (ii) pain catastrophizing as a specific form of worrying and rumination may promote sleeping problems; early awakening may in turn offer the temporal platform for this type of inefficient cognitive problem solving; (iii) depression includes sleep disorders as a major symptom and constitutes a condition sensitive to all changes in sleep continuity. Such considerations, triggered by findings such as those reported by Buenaver et al., advocate strongly for the inclusion of sleep problems into the theoretical modeling of chronic pain, as both potential indirect and direct causes. Given the all-encompassing restorative functions of sleep for regular pain processing (especially pain inhibition), mood regulation, cognitive functioning (including efficient problem solving), and the like, validates the assumption an omnipresent relevance of sleeping problems.

The conclusion at this point has to be that sleep and its related disorders continue to be not sufficiently considered in theoretical models as to their contribution to an understanding of pain and nociception. Moreover, not only does a psychosocial account of pain impose this assumption, but also the neurobiology of pain and sleep appears closely related and has common links, for example, to the metabolism of serotonin and stress hormones, which may be interrelated in complex interactions between sleeping disorders and pain [5].

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References


Stefan Lautenbacher *  
Physiological Psychology, University of Bamberg, Germany  
* Tel.: +49 951 8631851; fax: +49 951 8631976.  
E-mail address: stefan.lautenbacher@uni-bamberg.de