Sleep Medicine 53 (2019) 205

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Editorial

Do we need neuroimaging to treat insomnia effectively?

In recent years, the diagnosis of insomnia disorder has shown striking developments concerning its conceptualization, assessment and treatment. Notably, the main comprehensive models on insomnia have been behavioral or psychological [1]. These theoretical conceptualizations led to numerous intervention techniques, which continued to be used over the years [2,3]. Several studies such as randomized controlled trials (RCT) and meta-analyses have reinforced the efficacy and efficiency of cognitive-behavioral therapies for insomnia (CBT-I) [3]. It is also true that in the field of neuropsychiatric disorders (eg, depression, anxiety disorders), progress in neuroscience methods and technologies such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) has contributed to therapy improvement [4]. However, several abuses and misinterpretations have been documented regarding the role of neuroimaging methods in explaining human behavior [5]. The efficacy level of CBT-I in successful cases has enabled several patients to reduce symptoms and improve their quality of life with short treatment protocols. Nonetheless, CBT-I is not proven to treat all insomnia patients successfully (approximately 20% of insomnia patients do not respond to CBT-I [6]). Thus, it is pertinent to question whether, in the future, it will be possible to use expensive techniques such as fMRI or others to diagnose or even monitor treatment effects of interventions for insomnia. There is no doubt that neuroimaging is an important tool in insomnia research; however, it is unlikely that there is a revolution in insomnia treatment triggered solely by neuroscience, at least in psychophysiological insomnia or stress-related insomnia. Nonetheless, the contributions of neuroimaging techniques may be useful if we use them to complement and to improve our understanding of CBT-I effects [4,7].

We know from our recent knowledge about neuroplasticity that there are modifications in the brain structure and function of insomnia patients, after any intervention. A problem may arise when sleep specialists attempt to explain the insomnia experience as a brain disorder. We should utilize neuroimaging but not forget that insomnia is a multidimensional experience. Investment on expensive technologies with the expectation that we can explain insomnia because there is dysfunction in area X or Y or network Z is quite simplistic and dangerous. That is, brain dysfunctions in certain areas might represent correlates of insomnia disorder but not be the cause of it. In other words, we cannot confound correlation with causality at the explanatory level. These dysfunctions should direct our attention to biopsychosocial framework enriching our understanding of insomnia disorder. The data resulting from neuroimaging studies should provide us valuable clues in understanding insomnia and its treatment that make us reconsider steps to diagnose insomnia and will be fundamental for our enhancement of non-pharmacological therapies. The peril will not be in the use of these technologies but in the interpretations and inaccurate conclusions that can be extracted.

In the future, the role that we should expect from neuroimaging in insomnia treatment should be supplementary to behavioral paradigms and RCTs. Neuroimaging will be very important in our understanding of cerebral dynamics modifications after successful and unsuccessful CBT-I; it will further complement and advance the existing comprehensive models of insomnia [8]. By and large, insomnia researchers may and should use neuroimaging in their studies. However, we as researchers will have to be cautious in our conclusions.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2017.08.005.

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28 July 2017
Available online 24 August 2017

http://dx.doi.org/10.1016/j.sleep.2017.08.005