Cognitive-Behavioral Treatment for Insomnia Comorbid with Psychiatric Disorders

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The International Classification of Sleep Disorders (ICSD-2) [1] defines insomnia as a difficulty in initiating/maintaining sleep or non-restorative sleep accompanied by decreased daytime functioning persisting for a period of at least 4 weeks. Insomnia afflicts about 10% of the population in western industrialized countries. Primary Insomnia (PI) occurs in about 2 to 4% of the adult population [2]. More frequently, however, insomnia occurs as a co-morbid condition together with other disorders.

Recently, Alison Harvey advanced the hypothesis that sleep disturbances, and particularly those associated with insomnia, may play a role in the causation and the maintenance of psychiatric disorders [3]. The theory is based on findings showing on the one hand that most psychiatric conditions are associated with alterations of sleep or a complaint of insomnia and on the other hand that the biological circuits regulating sleep overlap with those regulating affective and cognitive functioning [4]. Consistent with this hypothesis, at least with respect to depression, insomnia has been found to be a clinical predictor in a large sample [5]. Thus, in addition to these data coming from sleep research, innovative approaches in psychiatry [6] focus on the importance of comorbidity between clinical symptoms promoting a network approach to mental disorders instead of the current categorical approach followed in the major psychiatry diagnostic manual (DSM-IV: Diagnostic and Statistical Manual of Mental Disorders) [7]. Specifically, comorbidity would arise as a consequence of symptoms shared by different disorders, called ‘bridge symptoms’. In this framework, insomnia seems to be a relevant ‘bridge symptoms’, thus, it shares the highest percentage of connected symptoms. In other words, insomnia and psychiatric conditions should be treated not as symptoms of different disorders, but as symptoms of the patient's overall condition.

Cognitive-Behavioral Treatment for Insomnia (CBT-I) has been found to be efficacious both at short- and long-term for patients with PI [8-10]. Whether or not CBT-I is efficacious for Comorbid Insomnia (CI), instead, is still not fully understood. Consistent findings have been reviewed in 2005 by Smith et al. [11]. Although the authors concluded that the reviewed studies suggested an encouraging role of CBT-I in the treatment of CI, they also pointed out the scarcity of controlled studies or rigorous designs. Since 2005, new data have been published.

Some studies focused on CI, without specifically considering one specific comorbid condition. Edinger et al. [12] for example, published a Randomized Controlled Trial (RCT) in which people with PI (N=46) and CI with mixed psychiatric disorders (N=41) were assigned to CBT-I or sleep hygiene interventions. Results showed that CBT-I was equally efficacious for PI and CI and was superior to sleep hygiene education. In addition, an interesting study focused on a population of 19 psychiatric inpatients with different clinical symptoms as affective disorders, Post-Traumatic Stress Disorder (PTSD), substance dependence, and psychotic disorders [13]. Results showed that the attendance to one session of group behavioral therapy for insomnia was associated with improvement in insomnia symptoms as measured by self-report questionnaires. Finally, CBT-I for CI was found to be effective also when delivered as self-help booklet, although the improvement was higher in a group in which structured weekly therapist support by telephone was added [14].

Other studies considered the effect of CBT-I on insomnia comorbid with a specific psychiatric disorder. Specifically, in a RCT conducted by Manber et al. [15], including 30 patients with major depression (MDD) and insomnia, the authors could show that the addition of CBT-I to pharmacological antidepressant treatment produced better outcomes as compared with the combination of the antidepressant treatment with a quasi-desensitization control intervention. Outcome included both measures of insomnia (sleep diary, actigraphy and questionnaires) and of depression (questionnaires). The same group has shown that CBT-I improved sleep, perceived energy, self-esteem, and other aspects of well-being similarly in a group with and in a group without elevations in depressive symptoms [16]. However, this second study did not include a control intervention. A recent meta-analysis [17] has summarized the results about the efficacy of CBT-I in improving symptoms of anxiety, especially related to the conditions of Generalized Anxiety Disorder (GAD), Panic Disorder (PD), and PTSD. By reviewing a total of 50 studies, the authors concluded that CBT-I for individuals with either PI or CI has a positive, although moderate, effect on symptoms of anxiety. Thus, although beneficial aspects on anxiety were found, the authors underline that residual anxiety symptoms should always be carefully assessed after the conclusion of CBT-I. In addition, the studies included in this meta-analysis were very heterogeneous with respect to the construct of anxiety measured, suggesting that future research should focus on evaluating the efficacy of CBT-I for specific symptoms of anxiety. One recent study focused on the effect of CBT-I in the treatment of 15 patients with persistent persecutory delusions and insomnia [18]. Patients were administered 4 individual CBT-I sessions including behavioral and cognitive strategies. Results have shown impressive benefit of CBT-I not only in reducing insomnia but also the paranoia symptoms (both measured through self-report questionnaires). Although this was a pilot study including a small sample and without a control group, the impressive findings suggest that much more research is needed in understanding the role of the symptoms of insomnia in psychotic disorders and whether CBT-I could be an efficacious component of the treatment of these conditions.

In conclusion, the literature mentioned here suggests that the
inclusion of CBT-I in standard protocols for different psychiatric conditions could be beneficial for many patients. However, data are still preliminary, and more research is needed through the conduct of rigorous controlled designs on large samples. The data presented in these studies arise, moreover, additional questions which future research should assess. For example, insomnia being a main ‘bridge symptom’ in psychopathology, it could be possible that its treatment in a group of patient with only 1 comorbid condition (e.g. anxiety disorder) could prevent the development of a second comorbid condition (e.g. depression). Additionally, the promotion of CBT-I in the general population could, in part, reduce the risk of developing psychiatric symptoms. Finally, CBT-I for PI could benefit from the inclusion of strategies directed to assess personality aspects often evidenced in patients with PI as well as in patients with other psychiatric conditions, as perfectionism or emotion dysregulation. Consistently, these could be other ‘bridge symptoms’ in psychopathology, and thus their treatment in patients with PI could, at least partially, reduce the risk of developing clinical symptoms of other psychiatric conditions.

References