CBT-I and acute insomnia: considerations and controversies

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INTRODUCTION

WHAT IS ACUTE INSOMNIA?

Before discussing the treatment of Acute Insomnia, within a CBT-I framework, it is appropriate to provide a working definition of the concept. When attempting to differentiate what makes as case of any specific disease or disorder, as well as what does not make a case, there are usually three perspectives to work from – nosology, theory and empirical work. In many instances, these three work in tandem, with new insights in each domain influencing the other two. However, that has been limited with respect to Acute Insomnia.

The Nosological Perspective

The term ‘Acute Insomnia’ was first listed in the International Classification of Diseases in 1977 (ICD). This was followed closely, in 1979, by the, as was then, American Sleep Disorders Association in 1979 and Diagnostic and Statistical Manual of Mental Disorders in 1987. As time progressed and as the definition of Insomnia Disordera changed, so did the definition of Acute Insomnia (otherwise known as short-term insomnia, transient insomnia, adjustment insomnia or stress-related insomniab). Currently, the ICD-10 identifies Acute Insomnia under the framework of meeting all the criteria for Non-Organic Insomnia, except duration (i.e. not being present for at least one month)1. Both the International Classification of Sleep Disorders – 3rd Edition2 and DSM-53 follow the same logic as the ICD-10, in that, Acute Insomnia is defined on the basis of not meeting the duration criterion for Insomnia Disorder (however, in these instances three months). In fact, the DSM-5 explicitly states that if all other criteria are met, bar duration, it is to be defined as Acute, or Short-term, Insomnia with a specific classification of – ‘another specified insomnia disorder’3. Therefore, within each framework, Acute Insomnia has largely been defined on the basis of not meeting criteria for Insomnia Disorder. This leads to the first consideration regarding its assessment and management. When does Acute Insomnia become Insomnia Disorder and when should it be managed? Working from the current nosologies, Acute Insomnia is defined on the basis of having Insomnia Disorder for less than 1 or 3 months. It is unclear, however, on what basis the current duration criteria for Insomnia Disorder originated. Previous iterations of the DSM, as well as the ICD and ICSD have outlined differing duration criteria with some going as far as six-months. Conversely, is insomnia experienced for a week, or two, still Acute Insomnia? In essence, what changes should we expect to see, if any, that signify the beginning, and end, of Acute Insomnia? Where this is not outlined explicitly in any nosology, and there is a very limited evidence base in this area, there is one suggestion embedded within many of the models of insomnia.

The Theoretical Perspective

Spielman’s model of insomnia is, arguably, the first model of insomnia that outlines the trajectory of insomnia, from its pre-morbid, to chronic state4-6. With reference to Acute Insomnia, Spielman suggests that the precipitant (i.e. a major life event), which may be biological or social in nature, is the main driver of insomnia prior to its transition to Insomnia Disorder. Although the idea that Acute Insomnia has to be driven by a major life event has been questioned with respect to the accumulation of daily hassles and/or chronic stress7-8 as triggers, the idea of the precipitant being the driver of Acute Insomnia has remained central to the models which followed Spielman (e.g. Perlis and colleagues9, Harvey10, Lund & Browman11, Perlstrom & Wickramasekera12, Buysse and colleagues13). The only model, which explicitly outlines the trajectory from normal sleep to Acute Insomnia, is from Espie14. Like Spielman, Espie suggests that Acute Insomnia originates from a stressor and during the acute phase it is the impact of the stressor that determines the resultant sleep disturbance. Further, Espie signifies the transition to Insomnia Disorder when the source of the stress changes to sleep as opposed to the stressor itself and manifests through increased attention to sleep as well as focused intention and increased effort to regain sleep. One of the challenges of the existing models is that although they may specify a specific change that signifies the transition from Acute Insomnia to Insomnia Disorder, such as Espie does, none provide a timeline for when this change would occur.

This stress-diathesis perspective outlined in each of the insomnia models also leads to a second consideration. If Acute Insomnia manifests in response to a precipitant event, and perpetuating factors are not altogether evident during this phase, why would a CBT-I framework be an appropriate treatment strategy? The central tenet of CBT-I is to identify and manage sleep-related dysfunctional thoughts, feelings and behaviors (i.e. perpetuating factors), with the precipitant usually ignored within the framework of CBT-I. Therefore, if it were the case that perpetuating factors are minimal, if evident at all, during the acute phase then it would stand to reason that a stress-reduction based treatment strategy would be more meaningful and effective candidate. That said, a careful examination of Spielman’s model demonstrates two stages where the insomnia threshold has been breached but is not at the point of Insomnia Disorder (see Figure 1). The latter stage of which suggests the introduction of perpetuating factors, albeit minimally.

INSERT FIGURE 1 HERE

The Empirical Perspective

As might be expected, without a consensus definition or a model that outlines a specific timeline within which to examine Acute Insomnia, its study has been limited15. The majority of research that has been undertaken has been in an attempt to understand the pathophysiology of Insomnia Disorder using a stressor as an analogue for Acute Insomnia. Various methods have been employed such as physiological (e.g. caffeine supplementation, rapid change in sleep timing), environmental (e.g. noise, light and temperature) and psychological (e.g. social stress test, complex cognitive tasks) challenge16-21. In each case the ‘stressor’ employed has been shown to disrupt both sleep continuity and sleep architecture22-23.

The validity of these studies, as an analogue for Acute Insomnia, or even Insomnia Disorder, however, is questionable. The majority of these studies do not employ a research protocol lasting a week with at least three nights of stress-related disruption. As such these studies do not meet the minimum frequency or, by design, duration criteria set out in any of the nosologies. It could also be argued that they are unlikely to meet criteria for daytime disorder or dysfunction as in most cases research participants are required, by ethics boards and committees, to have a period of recovery the day following the night of sleep disruption. Another consideration is with regard to the aspect of informed consent procedures. In most, if not all, cases the subject in the study would have been informed that they were likely to face a stressor either before bed or in the morning. This does not fit with the phenomenology of insomnia, at least in terms of Insomnia Disorder, with respect to its perceived unpredictable nature24-26.

On the basis of the existing nosologies, the theories advanced by Spielman, Espie and others, and the existing evidence Ellis and colleagues created a working definition of Acute Insomnia in 20127. This definition (see Table 1 for the full criteria) suggests that Acute Insomnia should be defined as a self-reported disruption in sleep continuity for between two-weeks and three months in duration.

INSERT TABLE 1 HERE

WHAT DO WE KNOW ABOUT ACUTE INSOMNIA?

On the basis of this definition, Acute Insomnia has a point prevalence of between 7.9% (UK) and 9.5% (USA) and for just over half of those reporting Acute Insomnia (51.2%) it will be reported as a first episode. Furthermore, the annual incidence of Acute Insomnia is in the region of 31-36%27.

The sleep of individuals with first onset Acute Insomnia differs from that of normal sleepers - subjectively in terms of increased time awake during the night (at both sleep onset and over the course of the night), a higher number of nocturnal awakenings and a lower sleep efficiency and objectively in terms of higher sleep fragmentation at night, via actigraphy28-29, and higher amounts of stage 2 (N2) sleep and lower amounts of Slow Wave Sleep (N3) via polysomnography29. Further, those with first onset Acute Insomnia tend to show, in addition to these differences in subjective and objective sleep, a slight circadian delay in addition to decrements in daytime energy expenditure, broadly resembling a 90-minute cycle – most pronounced during the morning30. Importantly, none of the objective differences observed in these studies (i.e. higher N2, lower N3) are generally seen in those with Insomnia Disorder31-32, suggesting that Acute Insomnia may be quantitatively different from full Insomnia Disorder. At this juncture, however, it is uncertain whether these differences are specific to Acute Insomnia, in general, or whether they only present during the first episode of any form of insomnia.

In terms of other differences between individuals with Acute Insomnia and those with Insomnia Disorder there is one cross-sectional study that compared groups on individual differences in coping styles33. The main finding from that study was that those with Acute Insomnia employed self-punishment, as a strategy to deal with unwanted intrusive thoughts, more so than those with Insomnia Disorder. That said, as the study employed a duration criteria of six months to differentiate the groups, it is unknown how this data fits with the current definition of Acute Insomnia.

WHAT DO WE KNOW ABOUT THE TRANSITION FROM ACUTE INSOMNIA TO INSOMNIA DISORDER?

Subjective sleep continuity data, during the first episode, does not differ between those with Acute Insomnia who go on to develop Insomnia Disorder and those who naturally remit29. However, those who transition to Insomnia Disorder demonstrate two differences in their sleep architecture compared to normal sleepers and those who would naturally remit29. Those who would go on to develop Insomnia Disorder showed a shorter REM onset latency (mean minutes 66.32) compared to normal sleepers (mean minutes 92.69) and those who would remit (mean minutes 97.08) at baseline. Furthermore, lower amounts of N3 (6.51% of total sleep) compared to normal sleepers (16.99% of total sleep) were observed. As the number of life events experienced over the previous year, stress levels over the previous month nor current levels of anxiety and depression did not differ between those who would later remit and those who would develop Insomnia Disorder it is unlikely that these results could be explained as an epiphenomenon to the precipitating factor(s) that triggered the insomnia. What these figures point to, especially the relatively early onset of REM, is a vulnerability for the onset of depression34-35. Again, however, it is unclear whether these are features of Acute Insomnia in general or specific to the first episode. Whilst this does not directly impact on the treatment choice for Acute Insomnia what this does point to is the wider issue of the need to address Acute Insomnia due to the potential for the onset of depression.

There is one small study that compared individuals with Acute Insomnia against those with Insomnia Disorder in terms of sleep-related cognitions and behaviors, sleep-associated monitoring, perceived stress and worry and both objective (actigraphy) and subjective (sleep Diary and Insomnia Severity Index) sleep36. Whereas differences between the groups were observed in terms of higher levels of stress being reported by those with Acute Insomnia, no differences between the groups in terms of sleep-incompatible cognitions and behaviors were evident. Albeit tentatively due to the sample size this finding suggests that perpetuating factors do have a role to play during Acute Insomnia (as defined on the basis of meeting criteria for DSM-5 Insomnia Disorder between two weeks and three months in duration). A more recent study adds support to this idea, as sleep-related dysfunctional beliefs were a significant predictor of those who would transition to Insomnia Disorder, within a three-month window, in a group of hospitalized individuals37. On the basis of these studies it would appear that a CBT-I framework might be a treatment candidate for this population.

TREATING ACUTE INSOMNIA WITH CBT-I

Working from the premise that i) perpetuating factors are a feature of Acute Insomnia but are likely to be in their infancy, as suggested by Spielman, and ii) brief therapies for insomnia, such as Abbreviated CBT-I (ACBT-I) and Brief Behavioral Therapy for Insomnia (BBTI), can effect change in individuals with Insomnia Disorder38-40, Ellis and colleagues developed a ‘one-shot’ CBT-I intervention specifically to prevent the transition from Acute Insomnia to Insomnia Disorder41. To date, this is the only intervention that has been applied to this population. The intervention comprised a pamphlet and a single 60-70 minute face-to-face session. The rationale for a single session came from Edinger42, who demonstrated that it is possible to ‘front load’ CBT-I into a single session with good results. The rationale for the amount of contact time came from an examination of the previous brief therapies which suggested approximately 60 minutes was sufficient to elicit change in individuals with Insomnia Disorder38,40,43.

The One-Shot Pamphlet

The pamphlet was framed as the 3D’s (**D**etect, **D**etach and **D**istract) and was broadly aligned to two of the principle components contained within traditional CBT-I (i.e. Stimulus Control and Cognitive Therapy). In addition, there was a brief description of Acute Insomnia (framed as stress-related sleep loss) and the suggestion that although it was unlikely that the bedroom environment caused the problem, if the bedroom were too hot or cold, too light or too noisy it could make the problem worse. The pamphlet contained four key points:

* How to record a sleep diary – The aim of the sleep diary was threefold; i) as a method of recording patterns in symptoms and symptom improvements, ii) as a method to determine the Sleep Restriction prescription in the treatment session and iii) something to be taken to a General Practitioner if the individual did not wish to or could not attend a single session but would still like support for their insomnia. The instructions were focused on recording the main symptomology of insomnia – Sleep Latency, Number of Awakenings, Wake After Sleep Onset, and the core elements needed to calculate Sleep Efficiency (Time in Bed and Total Sleep Time). The diary was to be recorded, every day, for a minimum of one continuous week.
* Stimulus Control instructions – Based upon Bootzin’s original concepts44, the aim was to recondition the relationship between the bedroom and sleep whilst simultaneously increasing the sleep drive. The instructions were to i) only use the bedroom for sleep and sex, ii) leave the bedroom if unable to sleep and iii) not to sleep in any other environment.
* Cognitive Control instructions – The aim of this element was to reduce the potential for nocturnal rumination and worry by giving the individual a sense of control over their previous, and next, days activities. The instructions were to put the day to bed before the individual went to bed by creating a period of reflection (kept in the form of lists) a few hours before bedtime. This period involved reflecting on what the individual had achieved that day, what they had to do the following day and what they had put into place in an attempt to deal with the demands of the following day.
* Distraction Techniques – As a form of articulatory suppression45 the aim was to engage the individual with Acute Insomnia to address nocturnal intrusive thoughts using one of three techniques – visual, numeric or alphabetical. These techniques were designed to be meaningless but mentally consuming with the intention of overloading executive function in the brain.

The pamphlet was designed to be provided a week prior to the treatment session, alongside a sleep diary, so that the initial sleep prescription could be discussed during the single session.

The One-Shot Treatment Session

The single session outlines two further elements taken from traditional CBT-I; Sleep-related Psychoeducation and Sleep Restriction. The psychoeducation component involves a brief discussion, using pictorial versions of Spielman’s 3 P Model4-6 and Bobely’s Two Process Model46 of sleep/wake regulation, on what insomnia is and why it occurs. The other aspect of this component involves a discussion on individual differences in sleep need throughout the lifespan. As with traditional Sleep Restriction, Total Sleep Time from the sleep diary is used to determine the prescribed sleep schedule over the following week (i.e. Total Sleep Time becomes Time in Bed). The individual, with help from the therapist, then agrees the morning anchor time and is then instructed to stick to the schedule for the next week. Following, the titration schedule for following weeks, based upon Sleep Efficiency, is discussed with the following rules – less than 85% the individual goes to bed 15 minutes later for the next week, between 85-89% the prescription stays the same for the next week and over 90% the individual can go to bed 15 minutes earlier than previously for the next week. It is also reinforced that the individual should not reduce their Time In Bed to less than 5 hours, irrespective of what the sleep diary calculations suggest. Safeguarding incase of excessive daytime sleepiness are also discussed at this point. Finally, the time remaining is devoted to discussing potential barriers and solutions to the treatment47.

The evidence for the One Shot

There are currently three studies that have examined the impact of the One Shot41, 48-49. The first contained a feasibility study on the pamphlet alone in terms of reductions in cognitive and somatic tension (significant reductions on both domains were evident one week post delivery) as well as a Randomized Controlled Trial of the full intervention41. The second study mirrored the first in that it was on a self-selecting community-based sample with the aim to determine whether the One Shot could be delivered in a group context and whether outcomes would differ based upon individualized vs. group treatment48. The third and final study looked at a particularly vulnerable population (prison inmates49) where rates of Insomnia Disorder are high (approximately 61%50) and the evidence suggests that the insomnia develops within that environment51.

The findings from each respective study have been promising in terms of remission rates and reductions in insomnia symptoms at one-month post treatment follow ups (see Table 2). In terms of adherence, using a rule of 15 minutes outside of prescribed sleep scheduled times during the first week of treatment being an indicator of non-adherence, 60% of those in the first study were adherent compared to 72.76% in the second study and 90% in the final study. As can be seen in Table 2, however, the effect sizes are quite variable between the first study and the second and third. There are several potential reasons for this including therapist factors, the inclusion of individuals with a range of chronic physical and/or psychological illnesses in the first study but not the other two, adherence rates and/or the small sample sizes involved. These issues should be examined closely in further studies of the One Shot. Interestingly, there were no significant differences between group treatment versus individualized treatment, suggesting it can be delivered in groups (although those in groups were less adherent – 53.85% vs. 91.67%). Further, where the final two studies examined changes in anxiety and depression scores, significant reductions were evident in both cases.

INSERT TABLE 2 HERE

THE FUTURE OF ACUTE INSOMNIA (ASSESSMENT AND MANAGEMENT)

Clearly there is still much work to be done in understanding both the phenomenology of Acute Insomnia and its place within the pathophysiology of Insomnia Disorder. Whilst a definition now exists, this needs further empirical testing, especially with regard to the timing of the definition. Further testing of the One-Shot, in addition to other interventions aimed at Acute Insomnia, are also warranted. This is especially true when considering the slight circadian delay observed in individuals with Acute Insomnia, which is not yet featured in current treatment protocols.

Recently, Edinger and colleagues showed poorer treatment outcomes, using pharmacotherapy or CBT-I, in those with Insomnia Disorder whose first episode of insomnia and depression occurred in childhood compared to those who developed both conditions, for the first time, in adulthood52. That said, it could not be determined whether the poorer treatment response was due to: i) the age of first onset, ii) the length of exposure to insomnia (first onset at 13.1 + 5.27 years old for childhood onset vs. 38.6 + 12.2 years old for adult onset) or iii) the number of episodes of insomnia experienced (2.5 + 3.05 vs. 1.7 + 2.38). This, albeit tentatively, points to another area of future research with respect to the assessment and management of Acute Insomnia. Are there differences between first onset Acute Insomnia and a recurrent episode of Acute Insomnia, which may require different treatment approaches? Finally, an examination of the relationship between ‘sleep reactivity’ and Acute Insomnia may be fruitful, especially considering recent research which demonstrates that an episode of insomnia may impact on subsequent episodes53-54.

SUMMARY

The term ‘Acute Insomnia’ has been part of the language of sleep medicine since the late 1970’s. Despite that, a comprehensive research agenda on the topic has only relatively recently been advanced. This has, at least until now, prevented a clinical viewpoint on the assessment and management of Acute Insomnia. Whilst there is a CBT-I focused intervention, designed to circumvent the transition from Acute Insomnia to Insomnia Disorder, the results from the trials undertaken have been quite variable and have been limited by quite small sample sizes. The findings from the review suggest there is still much work to be done with regard to the assessment, diagnosis and management of Acute Insomnia.

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