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CLINICAL REVIEW

Acute insomnia: Current conceptualizations and future directions

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SUMMARY

Despite significant contributions made in the area of persistent/chronic insomnia, especially with regard to the underlying mechanisms driving its maintenance, the area of acute insomnia has received comparatively little attention. The aim of this paper is to review the literature with regard to understanding the situational and personaological circumstances that surround the development of acute insomnia. The review begins by examining how the existing diagnostic systems conceptualise acute insomnia. Theoretical models that explain, or inferentially explain, the transition between normal sleep and acute insomnia are then explored and evaluated. The review then examines the current evidence base in terms of the pathogenesis of acute insomnia from naturalistic and experimental studies. Overall, the findings from the review confirm the paucity of evidence available but perhaps more importantly highlight the need for a structured diagnosis of acute insomnia as the first step in a research and treatment strategy. To this end a diagnostic system, drawing on the existing literature on stress and the systems used to diagnose depression, is forwarded and justified and a research agenda advanced.

Introduction

In June 2005, the National Institute of Health (NIH)[1] published a 'State of the Science' statement on the manifestations and management of insomnia. Amongst its conclusions was the suggestion that the natural history of insomnia has not been adequately profiled and that targeted research in this domain is required. Very little is known about the pathogenesis and aetiology of insomnia and particularly the transition from normal sleep to acute insomnia, and from acute to chronic insomnia. This influential document brought the issue of the natural history of insomnia to the fore but perhaps not for the first time, as several prior calls for such research already existed in the literature.[2–4] Delineating the factors that moderate and mediate the transitions between normal sleep and acute insomnia and acute insomnia and chronic insomnia would allow for a better understanding of the development of the disorder whilst simultaneously providing a platform for preventative work.[5]

The aim of this review is to move the agenda on the natural history of insomnia forward, especially in the area of acute insomnia. The aim is accomplished by first reviewing how the various nosological systems define acute insomnia. This is followed by a comprehensive summary of how the existing models of insomnia conceptualise the acute form of the disorder and the potential transitions from normal sleep to acute insomnia. Following this review, the existing evidence for the conceptualisation and definition of acute insomnia is summarised in terms of predisposition and precipitation and critically evaluated. The review concludes with suggestions for future research starting with a set of proposed criteria for the diagnosis of acute insomnia and a speculative look at treatment options. Finally, it should be noted that the literature on acute insomnia is limited and thus much of the discussion must rely upon inferences that can be drawn from existing research on chronic insomnia.

The diagnosis of acute insomnia

When looking for 'caseness' (i.e., the occurrence or non occurrence of an illness, disease, or disorder), three central concepts are crucial in determining whether the individual has reached or exceeded a threshold and can then be classified as a 'case'.[6] These concepts all focus around the presenting symptoms and associated features and include the duration, frequency, and severity of symptoms. Additionally, when conceptualising the onset of illness, such as acute insomnia, one must also take into account the circumstances surrounding its initiation (i.e., triggering factors). There are three diagnostic systems used to define 'caseness' in sleep

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research and practise: the international classification of diseases (ICD-10),[7] the diagnostic and statistical manual of mental disorders - fifth edition (DSM-V),[8] and the international classification of sleep disorders – second edition (ICSD-2).[9] Two of these three diagnostic manuals use a categorical multiaxial system (DSM and ICD) and each outline different criteria to define insomnia types and subtypes.

International classification of diseases (ICD)

Although there is no specific diagnosis of acute insomnia (also known as adjustment insomnia, stress-related insomnia, transient psychophysiological insomnia, transient insomnia, adjustment disorder, or short term insomnia[e]) under the ICD-10, inferences can be made from how the diagnosis of nonorganic insomnia (NI) (F51.0) is defined. To be classified as NI the disorder must be present for at least one month, triggered by a stressor and must result in both distress and interference to psychosocial or occupational functioning. In terms of frequency, the individuals' sleep should be disturbed for at least three nights per week. In terms of severity, no criteria are provided on qualitative (e.g., mild, moderate, or severe) or quantitative (e.g., equal to or greater than 30 min of sleep onset latency (SOL) or wakefulness after sleep onset (WASO)) terms. Given these considerations, acute insomnia may be construed as a form of Nonorganic Insomnia that occurs for less than one month. With respect to the trigger it follows that since both acute and chronic insomnia are depicted as nonorganic disorders, both would require a non-biological antecedent. In terms of daytime sequelae, while the ICD stipulates that there be an attention to or awareness of daytime consequences there is no reason to suppose that during the acute form of the disorder the individual is or is not focused more on the occurrence of the insomnia versus the precipitant. Similarly, there is no reason to assume differences between the acute and chronic phases in terms of frequency. This said it is at least plausible that an acute episode may represent a more intense form of the disorder with an increased frequency of sleep continuity disturbances. Finally as there are no criteria for severity in the chronic phase there is no basis on which to make an inference about the acute phase. As with frequency it is also plausible that during the acute episode the sleep continuity disturbance is more severe rather than less.

Diagnostic and statistical manual of mental disorders (DSM)

The DSM-V is due to be published in 2013. Under the proposed framework the predominate change in the area of insomnia is the sublimation of insomnia due to general medical conditions, mental disorders, or other sleep disorders under the title of primary insomnia (PI) (307.42). Within the context of primary insomnia, the diagnosis insomnia disorder has been chosen to reflect this change from primary vs. secondary insomnia. Moreover, there are six main criteria for a differential diagnosis (A–F) with A = A subjective complaint of dissatisfying sleep, and B = A reported problem in initiating sleep, maintaining sleep, having early morning awakenings and/or non-restorative sleep. Additionally there must be reports of distress or impairment (C), for a minimum of three nights per week (D), for at least three months (E) despite adequate opportunities for sleep (F).

Similar to the ICD, the DSM-V[f] has no specific diagnosis of acute insomnia but inferences can be made from this diagnosis of Primary Insomnia. This is especially true because PI is described in terms of disease progression and includes, as stages of the chronic disorder, both an acute and subacute phase. Acute and subacute are only described in terms of duration where the former is less than one month and the latter is between one month and three months. Whilst the DSM-V does not provide information about triggering factors it is interesting to note that the previous DSM-IV-TR did. In that version it was suggested that PI develops from a biopsychosocial stressor but could also develop without an identifiable antecedent. Given that the identification of a biopsychosocial stressor is not 'necessary' for the diagnosis of PI in DSM-IV-TR this may explain why it was removed from the DSM-V. With respect to daytime effects it is stated that 'The sleep complaint is accompanied by significant distress or impairment in social, occupational, or other important areas of functioning'.[8] With respect to severity there are no quantitative criteria but qualitative criteria have recently been adopted (although these may change). Qualitative criteria are based on scores from retrospective assessments accomplished with instruments such as the Insomnia Severity Index (ISI).[10] This approach allows for the assessment of sub and super thresholds of symptomatology and the assignment of a numeric value for severity. The latter, while essentially quantitative, is still not based on a naturally-occurring ratio metric that is centred on time and universally used by patients (e.g., time to fall asleep and time awake at night).

Given these considerations, acute insomnia may be construed as a form of PI as the duration criteria suggests acute insomnia is a form of the disorder that lasts for up to one month. As indicated in the DSM-V there is no criterion on triggering factors and thus no inference is possible about the role of this in acute insomnia. In terms of daytime effects, like the ICD, there is the requirement that sleep loss has an impact on daytime functioning but once again it is unclear whether this is relevant for the definition of acute insomnia. In terms of frequency and severity, like the definition of NI, there is no sound basis to suggest differences between the acute and chronic phases of PI. That is, as noted above, frequency and severity in acute insomnia may or may not represent more intense forms of this disorder.

International classification of sleep disorders

The ICSD-2 is the only nosology to provide a direct conceptualisation of acute insomnia which is referred to as adjustment insomnia (AI). AI is defined as occurring for a period of up to three months (up to one week is defined as acute and between one week and three months is subacute). With regard to triggering factors the ICSD stipulates that an identifiable 'stressor' be present. Further, it is explicitly stated that there should be no learned associations or conditioning to the bedroom environment or routine. Presumably this specification is made because these considerations represent perpetuating factors and as such are relevant to chronic insomnia. With respect to daytime consequences it is specified that the focus be on the effects of the stressor as opposed to the sleep problem. With respect to frequency and severity there are no criteria. It is interesting to note that the qualitative dimensions of mild, moderate, and severe were present under its earlier definition of adjustment sleep disorder (ASD) (307.41-0).

Comparing the ICD, DSM, and ICSD

Only one nosological system explicitly defines acute insomnia with a clear trigger, duration, and course provided. This said given the inferences made above one can compare and contrast the three given formulations. This is presented in Table 1. With respect to duration, the DSM and ICSD agree that a period of up to three months is the overall timeframe for acute insomnia whereas the ICD places this within a month. When considering the subcategories of acute and subacute a different picture emerges, with the ICD and DSM converging on the shortest period of sleep disturbance being up to

[e] This is the list provided under the heading of Adjustment Insomnia in the ICSD-2.

[f] This is based on the next to final draught of the DSM-V where it is possible that further revisions may be made before the close of the cannon.

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Table 1
The existing diagnostic systems approach to acute insomnia.

	DSM-V	ICSD-2	ICD-10
	PI	AI	NI
Trigger	x	Any identifiable stressor	Only a non organic stressor
Minimum frequency	3 nights per week	x	3 nights per week
Duration	0–3 months	0–3 months	0–1 month
Course	Acute 0–1 month Subacute 1–3 months	Acute 0–7 days Subacute 1 week - 3 months	x
Qualitative severity	Retrospective self reports	x	x
Quantitative Severity	x	x	x

AI – acute insomnia.
DSM-V – diagnostic and statistical manual of mental disorders - Fifth Edition.
ICD-10 – international classification of diseases.
ICSD-2 – international classification of sleep disorders - Second Edition.
NI – nonorganic insomnia.
PI – primary insomnia.

one month. With respect to trigger, both the ICD and ICSD require that a precipitating factor be identified whereas the DSM does not. The other difference is in the case of the ICD where the precipitator is presumably limited to a psychosocial stressor whereas in the ICSD this may also include biological or organic factors. With respect to the frequency dimension, both the DSM and ICD provide a 'three nights per week' minimum where the ICSD has no criteria. Finally, with respect to severity none provide true (ratio) quantitative criteria, although the DSM-V has a relative qualitative dimension, based upon cut-off scores from self-report scales.

Theories and perspectives on the aetiology of acute insomnia

There are currently four human models of insomnia that specifically outline the circumstances under which an acute period of sleep disturbance could occur and each will be discussed in turn. It should also be acknowledged that many of the other models of insomnia allude to characteristics that may predict the likelihood of acute insomnia occurring in response to a stress or the circumstances under which a stressor of threat may directly result in a period of acute sleep disturbance. In each of these cases, the specific contribution from that model will be reviewed, albeit briefly.

The '3P' model

In 1986/1987, Spielman and colleagues[11,12] proposed a cumulative model of the progression from normal sleep to chronic insomnia (the 3P model: predisposing, precipitating, and perpetuating). For the purposes of this review only the two (predisposing and precipitating) factors relevant to acute insomnia are discussed. These two aspects of the model advance a stress-diathesis conceptualisation of insomnia. One of the model's central tenets is that predisposing factors make some more vulnerable to insomnia than others. These predisposing factors are then compounded by a precipitating event, resulting in the threshold of insomnia being surpassed and a period of sleep continuity disruption beginning. In terms of the specific predisposing and precipitating factors that can lead to acute insomnia, in the original conceptualisation, which has continued to be fleshed out over time and across successive articles and chapters,[13–15] it is suggested that predisposing factors are within the biopsychosocial sphere and precipitating factors are conceptualised in terms of threats or stressful life events (e.g., a promotion, an exam, medical complaint). The main aspect of the model that is appealing is that it has high face validity (in the context of chronic insomnia it is supported by the high efficacy of therapies that derive from the theory). The main drawback is that no specific characteristics or circumstances are postulated at each progressive stage, as such, making it difficult to test.

The high risk model of threat perception (HRMTP)

In 1998, Perlstrom and Wickramasekera[16] proposed a model of insomnia derived from their earlier work on somatoform disorders. The central tenet of the HRMTP is that a perceived threat creates high levels of physiological arousal inhibiting the normal sleep process. Additionally, these levels of arousal are influenced through interplay between four predisposing characteristics; high susceptibility to hypnosis, high neuroticism, repression, and a tendency to catastrophize. These four characteristics work in two ways a) by increasing the sensitivity towards a perceived threat and/or b) by magnifying the sympathetic response to the threat. The main advantage of this model is that it details a very specific set of circumstances that account for the initiation of acute insomnia. The main drawback is the same as its main advantage in that by specifying the precise circumstances, other pertinent factors are easily overlooked.

Lundh and Broman's interactive model

In 2000, Lundh and Broman[17] developed a model of insomnia that put forward a proposed course from normal sleep to chronic insomnia. The central tenet of the model is that the pathogenesis of chronic insomnia is a two-process phenomenon. The first process (sleep interfering) is similar to the position proffered by most insomnia models: insomnia occurs as a hyperarousal phenomenon. Lundh and Broman further specifie that the hyperarousal response be subject to the moderating influences of the individuals' predisposition (i.e., a low arousal threshold via emotional sensitivity or a slowed habituation following a stressful life event). The second process (sleep interpreting), which is Lundh's and Broman most unique contribution, is that the pathogenesis of insomnia also requires a subjective appraisal component. That is, the individual must perceive the sleep disturbance or the consequences of the sleep disturbance to be problematic. Finally, the personality characteristic of perfectionism is identified as important as it feeds into the appraisal process. The main advantage of this model is that it highlights and provides insight into the factors that allow for the switch from normal sleep to acute insomnia. Additionally, it offers an explanation for intra-individual differences in the stress response. The main drawback to this model is that it does not discuss how the two processes interact but appears to construe them as orthogonal.

The psychobiological inhibition model

In 2002 Espie[18] proposed a model of the development and maintenance of insomnia. The Psychobiological Inhibition Model's central tenet is unique in that unlike the other models of insomnia,

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the emphasis is not on a form of 'hyperarousal' inhibiting sleep but insomnia being a failure to inhibit wakefulness. The model states that normal sleep, as a biological function, has two inherent features; 'plasticity' and 'automaticity'. Plasticity works by absorbing the effects of psychosocial or environmental stressors that could disrupt sleep. Automaticity refers to the involuntary nature of how sleep is regulated under normal circumstances. Further, automaticity implies that coping efforts at best intrude upon, and at worst deregulate, the normal function of sleep. These two processes not only respond to external triggers and events but also endogenous factors arising from mental and biological processes. Within this model acute insomnia is conceptualised as a natural consequence of stress (perceived or actual). After what is presumably part of the flight-or-fight response the individual should then default back to normal sleep aided by automaticity and plasticity. This recovery however may not occur provided that several factors become operational. Espie and colleagues frame these factors in terms of attention, intention, and sleep-related effort.[19] The model therefore allows for the distinction between acute or chronic insomnia by the presence or absence of these compensatory practises. Espie does allude to the presence and influence of predispositional factors that can affect the efficacy of the two processes but, like Spielman et al., does not state what these may be. The main advantage of this model is that it specifically focuses on the failure to inhibit wakefulness that opens the research agenda to the examination of insomnia as i) a 24 h disorder and ii) a neurobiological phenomenon not of hyperarousal but persistent wakefulness. Additionally, (for the first time) this frames acute insomnia as part of a normal biological process and that it is only the chronic form of the disorder that is pathological. The main drawback is that there is no discussion on the timing or circumstances that determine whether one recovers or becomes subject to the pathogenic factors of attention, intention or effort.

Models for which inferences about acute insomnia are possible

As noted above, some of the models directly address acute insomnia whilst many do not. With respect to the latter, a subset of these models, despite being dedicated to the aetiology of chronic insomnia nonetheless may allow for inferences about the nature of the acute form of the disorder. At first glance Bootzin's[21] Stimulus Control model does not appear to offer insight regarding the initiation or development of acute insomnia. This model focuses on how behavioural responses to insomnia exacerbate the problem and therefore appears to be singularly focused on chronic insomnia. On closer inspection it could be inferred that these behaviours (extensive time spent in bed whilst awake and engaging in behaviours other than sleep and sex in the bedroom) may already be in place when a precipitant occurs thus lowering the threshold for acute insomnia. Morin's Microanalytic Model[22] presents a vicious cycle of insomnia that is fed and feeds on arousal, dysfunctional cognitions, maladaptive habits, and consequences. It could be inferred that the factors that contribute to chronic insomnia as a vicious cycle also predispose one to bouts of acute insomnia. For example, unrealistic expectations of sleep may reduce the individual's tolerance for sleep disruption and make them more inclined to view it as acute insomnia and such worries may exacerbate the severity of this acute episode. The central tenet of the neurocognitive model[23] is that conditioned cortical arousal is permissive of processes that directly and indirectly perpetuate insomnia including augmented sensory and information processing and long and short term memory functioning. Inherent in this model is that these alterations occur as a final end product and therefore only occur in chronic insomnia. The inference that can be made here is that the flight-or-flight response itself initiates the augmentation and that cortical arousal serves only to perpetuate this phenomenon after the stress has abated. It could be further inferred that these effects vary with the individuals' predisposition to stress and level of reactivity. Harvey's cognitive model[24] offers a perspective on the acute phase of insomnia in terms of predispositional and precipitating factors. There is the suggestion of a pre-existing vulnerability to focus on sleep loss and the consequences of sleep loss during the initial stress response. Here, it could be inferred that the individual responds in a characteristic (predispositional) manner by employing maladaptive cognitions (i.e., negative cognitive style, monitoring) and behaviours throughout the day and night. Taking each of these models, or inferring from them, provides a seemingly endless series of possibilities in terms of predisposing and precipitating factors and their interrelationships.

Empirical work related to the phenomenon of acute insomnia

Cano-Saper's rodent model of acute insomnia

As the only model to directly map the stress/threat component of acute insomnia, the central aim of this analogue model is to better understand the neurobiology of acute insomnia.[20] In this instance a male rat was removed from his cage at the peak of the sleep period to a cage that had been occupied by another male rat, evoking the animals territorial nature, and promoting the flight-or-fight response. Under these conditions there was simultaneous activation in regions associated with both the maintenance of wakefulness and the maintenance of sleep. Corresponding to this neurobiological finding was an increase in EEG frequencies typically associated with wakefulness that also appear peri-sleep onset and during nonREM sleep in patients with chronic insomnia. Additionally, sleep continuity disruption was also observed. The major strength of this model is that it shows the effects of a psychosocial stressor, as opposed to a physical stressor, on the neurobiological markers and substrates of sleep, lending credence to the oft-alluded link between stress (perceived or actual threat) and sleep continuity disturbance. The main drawback is that as this is an animal model its capacity to translate to humans is limited and no inferences can be drawn about the perceptual dimension and its relevance for acute insomnia.

Prospective epidemiologic studies

Naturally, a great deal of the presumptions, assumptions, and inferences about acute insomnia rest upon the idea that there is a linear relationship between normal sleep, first onset of acute insomnia, multiple episodes of acute insomnia, and chronic insomnia. Evidence for this is scarce but there are indications that this may be the case. For example, one study has examined the course of insomnia following hospitalisation.[25] They showed that the rate of chronic insomnia (using the cut-off for chronic insomnia from the Sleep Impairment Index) had increased from 10% to 19% within three months following discharge. Furthermore, the main predictors of this transition were levels of sleep disturbance, duration of insomnia, and levels of sleep-related dysfunctional beliefs. Where the duration of insomnia was measured (up to a week - transient, between a week and a month - unclassified, between a month and three months - short-term, and three months or longer - chronic), between groups analyses were not undertaken, limiting an understanding of the course of insomnia.

Another study by Morin et al.[26] also provides an indication of the course of insomnia. They showed that although those with subsyndromal insomnia at baseline were 3 times more likely to remit, as opposed to develop the full syndrome, the most frequent course over the three years was from subsyndromal to syndromal.

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More importantly, they separately analysed the good sleepers (assessing at six months and one year) to determine the incidence of subsyndromal and syndromal insomnia.[27] They report that 14.43% developed subsyndromal insomnia by six months and 2.37% had developed syndromal insomnia. In terms of first episode onset, 5.77% had their first subsyndromal bout within six months and 1.57% had their first syndromal bout within six months. Furthermore, the number of life events experienced, number of negative events, and the intensity of the event (positive and negative) within the last six months, differentiated between good sleepers and both insomnia groups.

An additional longitudinal study of the natural history of insomnia, which should also be acknowledged, is the Zurich Study.[28,29] Within that study three insomnia subtypes were defined by duration; Continued Insomnia (CI) (lasting at least two weeks), Repeated Brief Insomnia (RBI) (at least monthly but lasting less than two weeks), Occasional Insomnia (OI) (less frequent). The findings showed that between the three sampling-periods (1979, 1981, and 1986) there was an overall increase of 27% between reporting no insomnia or OI at baseline and reporting RBI or CI at either follow-up period. Furthermore, the authors report that of those with OI at baseline ($n = 77$), 15.6% remained within this category over the course of the follow-ups whereas 57.1% were reclassified as RBI or CI, and 27.2% no longer reported insomnia (i.e., remission).

The latter follow-up reclassified the groups into 2–3 weeks of insomnia (equivalent to CI in the first analysis), recurrent brief insomnia (equivalent to RBI), and occasional brief insomnia (equivalent to OI) and added a fourth group, one month insomnia, which set a minimum duration.[29] The cumulative weighted prevalence for each subtype was as follows: 19.8% (one month insomnia), 9.7% (2–3 week insomnia), 20.6% (recurrent brief insomnia), and 17.5% (occasional brief insomnia). Additionally, over the total sampling period (20 years) 41% developed a more enduring form of insomnia whereas 32% developed a briefer form.

Whilst these data do provide an indication of the prevalence and natural course of 'briefer' forms of insomnia, methodological issues limit the generalisability of these findings to acute insomnia. At each sampling period participants were asked to retrospectively report sleep problems over the last twelve months, thus current sleep problems were not examined or followed prospectively. Moreover, the definitions of insomnia subtypes did not exclude other sleep disorders, which may have been masked as insomnia and as such skewed the results, and there were no minimal criteria on the frequency of occurrence.

Finally, a longitudinal study from Sweden examined the course of insomnia over a year.[30] The sampling period at baseline and follow-up spanned three months retrospectively and defined insomnia based upon a) a reported problem over the previous three months, b) for at least three nights per week, c) daytime decrements in performance or functioning, and d) a sleep onset of $\geq$ 30 min, sleep maintenance difficulties after $\geq$ 30 min, or early awakenings of $\geq$ 30 min. The reported cumulative incidence of insomnia over the sampling period was 2.8%. This said, although the study's definition of insomnia meets the time-sampling duration, severity, and frequency criteria for the present definition of acute insomnia, the definition of insomnia precluded any separation between acute and chronic insomnia.

In order to gain a definitive insight into the development of insomnia, in terms of its natural course and its incidence and prevalence, a prospective longitudinal study, beginning with a sample of good sleepers, with sampling rate of at least weekly (although ideally daily), and to have a sampling period long enough to capture i) acute episodes of insomnia ii) the resolution of those episodes or transition to chronic insomnia would be needed.

Naturalistic and cross-sectional studies

Although naturalistic studies of insomnia are rare, there are a few notable exceptions that evaluate the link between stress and 'poor sleep'. These studies have successfully shown life-event stress to correlate with poor sleep in the recently bereaved[31,32] and those recently exposed to a significant stressor (e.g., natural disasters/industrial accidents/war).[33–36] In these instances reduced sleep continuity is evident and changes in sleep architecture have also been documented.[37] Understandably, most of these studies do not fit the context of acute insomnia, as these groups are usually recruited months or years after the initiation of the antecedent. Two prospective studies that merit particular attention are by Torsvall et al. on ship engineers during on-call periods.[38,39] In both studies on-call nights were rated poorer in terms of subjective sleep quality even when no alarms occurred, presumably due to the anxiety caused by uncertainty. Moreover, in the first study they all showed reduced SWS and reduced power density during the first sleep cycle (when no alarms sounded). The stress/sleep relationship is further buttressed by the more temporally relevant work of Morin and colleagues.[40] They demonstrated an ongoing, albeit small, relationship between daily hassles and poor sleep quality (measured through sleep diary) over a 21-day period in good sleepers. Interestingly, they also found pre-sleep arousal (cognitive and somatic) and maladaptive coping styles mediated the relationship between daily stress and poorer perceived sleep in the good sleeper group.

There also exists one cross-sectional study that directly compares normal sleepers, people with acute insomnia, and people with chronic insomnia on cognitive coping styles.[41] Acute insomnia was defined as having the symptoms of chronic insomnia for less than six months (consistent with the DSM-IV-TR's definition of acute insomnia). Distraction (focussing away from the stressor) was related to not reporting a 'case' of acute insomnia. Additionally, comparing those with chronic insomnia and those with acute insomnia, the latter were more likely to employ a self-punishment coping style. Predictably both people with acute and chronic insomnia worried more than normal sleepers.

Probably the most compelling data that exist on acute insomnia comes from the Espie group.[42] They examined attentional biases for sleep-related cues in two groups of people with sleep onset insomnia (acute and persistent) following the diagnosis of cancer. Acute insomnia was defined as the development of insomnia symptoms between 0 and 12 weeks since diagnosis whilst persistent insomnia was defined as the development of symptoms within 12 weeks since diagnosis but lasting for at least 12 months. Further, sleep onset insomnia was defined as a longer than 30 min SOL for at least four nights per week. They found that people with acute insomnia reported significantly higher levels of psychological distress than those with persistent insomnia. Further, where there was no difference between the groups on reaction times to cancer words, those with persistent insomnia showed an additional attentional bias towards sleep from the emotional stroop task whereas those with acute insomnia did not.

A final development that warrants attention is the seminal work of Drake et al. on 'sleep reactivity'.[43] The guiding premise for this programme of research is that individual differences exist in how people respond to stress, with some more predisposed to respond with increased sympathetic nervous system activation, resulting in a period of acute sleep disruption. The Ford Insomnia Response to Stress Test (FIRST) was developed as a self-report measure of this pre-existing vulnerability. The FIRST has been shown to predict poor sleep, measured polysomnographically (i.e., low sleep efficiency and increased SOL), in response to acute sleep disruptors (i.e., acute administration of caffeine, first-night effect of PSG).[44,45]

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Moreover, higher scores on the FIRST have also shown to be a significant risk factor for the development of insomnia in normal sleepers over 13-month period[46] A recent study has also demonstrated that dysfunctional coping strategies, arousability (cognitive and emotional), and neuroticism predict scores on the FIRST.[47]

Inferences about predisposing factors from cross-sectional studies

Making inferences about acute insomnia from cross-sectional studies is difficult because factors may be perpetuating and not predisposing. Characteristics such as perfectionism,[48] a vulnerability to worry,[49] ambitiousness,[50] neuroticism,[51] low extraversion,[52] vulnerability to depression,[53] and susceptibility to hypnosis[54] have differentiated normal sleepers from people with chronic insomnia. Whether these traits are a cause or consequence of acute insomnia is unclear. Bonnet and Arand[55,56] addressed, and provided, evidence against several of these traits (using the Minnesota Multiphasic Personality Inventory) being a consequence of chronic insomnia in two studies. In the first they induced 'insomnia-like' sleep parameters in normal sleepers. In the second, by restricting the sleep of a group of people with chronic insomnia further than what would be their 'normal sleep'. Neither study resulted in state-wise exacerbation, suggesting the personality characteristics that contribute to insomnia are not likely the consequence of acute sleep disturbance, although naturally-occurring insomnia or insomnia of longer duration may produce these outcomes.

Experimentally induced stress studies

There may be real merit to analogue studies of acute insomnia that inhibit the actual physical process of sleep (i.e., partial or total sleep deprivation, mismatches in sleep scheduling, administration of pharmacological substances which inhibit sleep, or other physical stressors) but these are not considered here for several reasons. First, these stressors do not naturally fit with the concept of a trigger outlined in the diagnostic systems, in that, both the ICD and ICSD point to psychosocial stressors. Second, the models of insomnia, and the evidence from Morin's et al. study,[40] emphasise appraisal and coping as crucial in mediating the relationship between the stress response and sleep. Finally, those studies which use sleep disruption as an analogue for the stressor (i.e., first-night effect) are self-confounding as they cannot tease apart the stress from its impact on the sleep experience.

Only a few studies have utilised acute psychosocial stress paradigms to examine the effects on sleep continuity. In most cases performance anxiety has been created (participants have to deliver a speech the next morning) or cognitive load is significantly increased (music playing during the sleep onset period). Most research in the area comes from studies conducted in the early 80's and the findings are generally consistent in that sleep disturbance occurs following a stress induction.[57–59] One prominent study used the performance anxiety paradigm to examine the effects of stress on rapid eye movement (REM) sleep in normal sleepers.[60] Neuroticism and coping did not moderate the relationship between acute stress exposure and REM latency but the interactions between social support seeking and stress exposure and avoidance coping and stress exposure did. More interestingly was the finding that the stress exposure had no effect on any subjective or objective sleep variables (e.g., SOL and WASO) except in terms of reduced REM counts during the last REM period. This study was expanded upon by Hall et al.[61] and showed that this anticipatory performance anxiety led to increased sympathetic activity (as indexed by heart rate variability) throughout the night.[62] A further study, using a similar performance anxiety paradigm, also demonstrated the effects on sleep disruption (reduced sleep efficiency index (SEI), increased SOL, and increased Stage 1).

There are several potentially confounding factors in these studies that should be taken into account. Stress inducement studies are time limited, allowing the participant to estimate, and potentially 'bank', additional resources needed to cope. Additionally, knowing when a stressor will end is not usual in the real world and may attenuate the observed stress response.[63] There is one quasi experiment that has addressed some of these issues. This prospective study aimed to examine the link between sleep preoccupation, stress, and sleep over the course of a natural stressor (exams).[64] As levels of poor sleep efficiency increased (closer to the exam), levels of perceived stress and sleep preoccupation also increased proportionately. This study lends support to the idea that stress induction can model acute insomnia but only provided ecologically valid and/or definitively provocative stressors are utilised.

Conceptualising acute insomnia

At this juncture there is a temptation to conclude that more research is needed before one can establish the diagnosis of acute insomnia. This could be ideally accomplished by a series of longitudinal studies that have a sufficient temporal resolution to identify the factors that govern the relevant transitions. In the absence of such studies forward progress could be accomplished by tentatively crafting diagnostic criteria based upon the information summarised above. Accordingly, we propose a series of criteria for 'caseness' for acute insomnia (Table 2) and a justification for each of the central concepts.

Before looking at each of the defining criteria it is important to note that any examination of insomnia, be it acute or chronic, should begin with the complaint and a comprehensive sleep history assessment. This assessment ideally should determine the actual nature of the complaint in addition to covering the criteria for caseness. For example, the reported subtype (initial, middle, late, or mixed) should be included, as is traditionally the case with chronic insomnia (and proposed within the DSM-V). The rationale for including this is that interventions can be linked to treatment gains in relation to the initial presenting complaint. It is not a defining criterion as there is scarce information on these subtypes, in all forms of insomnia, which limit their predictive capability regarding the course of the disorder or specifically indicate one treatment option over another.

Trigger

1) Any life event or train of life events that results in a significant reduction in quality of life from the individuals' ideal; 2) Distress at current situation. In this formulation, the primary precipitant is

Table 2
The proposed diagnostic for acute insomnia.

	Acute Insomnia
Trigger	1) Any life event or train of life events which results in a significant reduction in QoL from the individuals ideal 2) Distress at current situation
Minimum frequency	3 or more nights per week
Duration	3 days −3 months
Course	3–14 days: acute 2–4: weeks Transient 1–3: months subchronic
Qualitative severity	mild/moderate/severe as defined by the patient
Quantitative severity	(+30 min SOL; +30 min WASO)

QOL − quality of life.
SOL − sleep onset latency.
WASO − wakefulness after sleep onset.

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acknowledged as a significant life event whilst also accounting for the potential of a stress response threshold being exceeded through cumulative losses or a chronic stressor (Fig. 1). Moreover this formulation allows for the potential of predispositional factors increasing the stress response beyond the threshold for acute insomnia or decreasing the threshold at which the stress response threshold results in acute insomnia (Fig. 2). The rationale behind the use of quality of life as the measure of the trigger comes from Hobfoll's characterisation of stress.[65–67] Previous models generally define stress in terms of its outcome (e.g., physiological effects, cognitive appraisals of the effects), which limits the ability to prospectively examine its causes.[65] Hobfoll's viewpoint is that all stress is caused by a perceived (a threat to) or actual loss of resources. Resources cover the full biopsychosocial spectrum and can be internal (e.g., self-esteem) or external (e.g., time, money). As such, a single significant loss, a series of cumulative losses, or an inability to acquire sufficient resources to protect against a potential loss causes stress. Additionally, as Hobfoll conceptualises stress as a neutral construct, both positive and negative life events could equally impact on sleep as it is the threat of, loss of, or failure to retain, resources that evokes the stress response. For example, although a wedding is usually constructed as a positive event, the money, time, and effort involved in its planning could sufficiently tax an individuals' resources, resulting in an acute stress response. Distress is specified as a criterion to add clarity to Hobfoll's resource loss theory as it is possible that one or more stressors can substantially impact on quality of life whilst not being perceived as particularly distressing.[68] Moreover, the distress component affords 'normalizing' or stress buffering processes, such as coping style and social support, to be considered, as suggested by several of the models of insomnia described earlier.

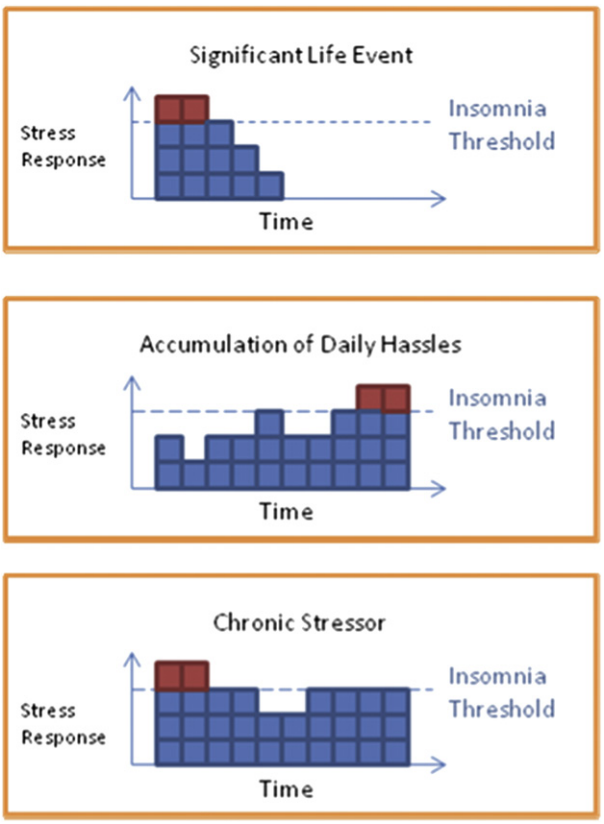

Fig. 1. The influence of different stressors on acute insomnia.

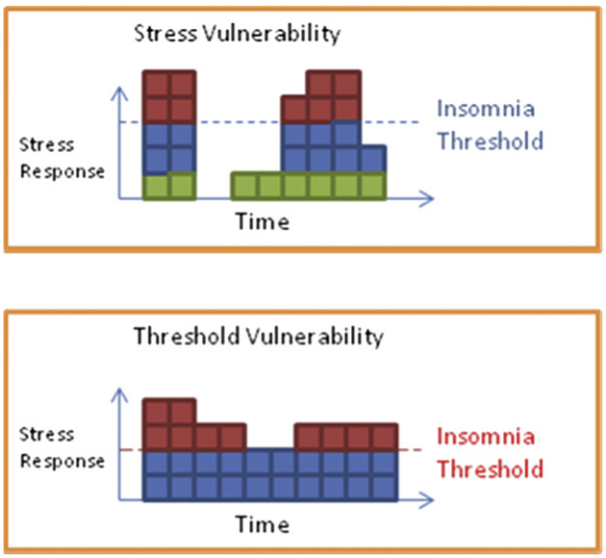

Fig. 2. The influence of predispositional factors on acute insomnia.

Frequency - 3 or more nights per week

Given that there is some range in what constitutes normal 'night to night' variability and given that the intent in framing this classification is the identification of poor sleep when it reaches the threshold of being a disorder it follows that the same minimum criteria applied to chronic insomnia be applied to acute insomnia.[69] For that reason a three night minimum disruption is recommended.

Duration and course - 3 days-3 months

The identification of 3 days to 3 months represents the entirety of the acute interval. The minimum stipulation, as noted above, of three nights allows for normal variation and the framing of such occurrences as not insomnia per se but simply a few bad nights. The maximum is in keeping with ICSD and DSM-V classification of the cut off for chronic insomnia being three months or more. Whilst this category could be left as a unitary construct or construed dimensionally, sub categorisation is not only in keeping with the current classifications systems but also allows for the distinction between what appear to be relevant time frames within the literature. Accordingly, 3–14 days is identified as the shortest form of the disorder (pre-transient). The upper limit of two weeks derives its precedent from its use throughout the classification of mental disorders. For example, in order to be diagnosed with depression the symptoms must be present for two weeks.[8] The interval between two weeks and one month may be best identified as transient insomnia. The cut off of one month is based on the precedent that many research studies adopt this duration as sufficiently prolonged as to meet criteria for chronic insomnia.[41] A further potential advantage is that it allows for this timeframe to be contrasted with the preceding timeframe for the determination of when subjects begin to engage in compensatory behaviours. Finally, the sub-chronic phase (one to three months) is the period under which a switch between stress-related sleep loss and sleep loss-related stress would most likely occur.

A natural addition to these criteria would be to distinguish first episode of acute insomnia from recurrent episodes. In the absence of precedent it is recommended that the criteria used for major depressive disorder be used here. That is an episode of acute insomnia is discriminated from a second episode of acute insomnia

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given a two-month period of being subsyndromal or symptom free. If this condition is not met under apparently recurrent episodes then a separate diagnosis of idiopathic insomnia should be considered.

Severity

A qualitative severity criterion allows for a reasonable metric of subjective distress whilst not imposing arbitrary cut points. In keeping with the classification system set forth by the ICSD-2, it may be useful to use the descriptors of mild, moderate, or severe to denote subjective severity. In the absence of established quantitative criteria for severity (in all three of the nosological systems), one is left to the precedent that is used throughout the literature and substantiated by a single empirical study to set the threshold for caseness. The majority of research studies adopt a greater than or equal to 30 min threshold for the identification of SOL and WASO.[70] In this instance there is no reason to suppose that acute insomnia differs from chronic insomnia in terms of the minimum length of time for sleep onset or awakening.

Concluding remarks

While the major strength of this review is the operationalisation of the concept of acute insomnia, perhaps its greater value resides in the conceptualisation of this phenomenon as part of a larger, and non pathological, biopsychosocial process. That is, acute insomnia is part of the stress response and as such is likely to be adaptive and not pathological[g] or as Spielman and Glovinsky state "No matter how important sleep may be, it was adaptively deferred when the mountain lion entered the cave."[13] (p.3) Conceiving of acute insomnia in this manner has at least two important implications. First, it requires that we come to a better understanding of the factors that determine whether one resumes normal sleep as opposed to develops the pathological condition of chronic insomnia. Second, when the factors that differentiate acute and chronic insomnia are explicated this will provide new directions in prevention and treatment.

Potential implications for treatment

Previous research on the treatment of acute insomnia

Where pharmacological intervention studies during the 'pre-transient' phase of acute insomnia do exit, the context of these studies can be seen to limit their relevance to the full duration of acute insomnia. These studies rest largely on the assumption that the central feature of pre-transient insomnia is a rapid change in sleep timing in response to a stressor. To that end, paradigms such as caffeine use, the first-night effect, and phase shifting have been utilised. Essentially, these studies have examined, and in many cases successfully demonstrated, the ability of pharmacological agents to 'normalize' sleep. That said, the main drawback is the timing involved. It is unlikely that a few nights of sleep disruption is a sufficient model of acute insomnia, especially if this is planned and known by the subject to be a time-limited disruption. As such, the uncertainty seen in acute insomnia is not likely to be mirrored by the experimentally induced stressor and not result in a significant threat or sustained loss to the individuals' resources. Even if the stressor has sufficient valence to elicit a form of 'pre-transient insomnia' it is not known whether the agent is addressing the insomnia or fixing/masking a potentially normal reaction to stress. It is still not known how people across the full range of acute insomnia sleep and how their sleep compares to normal sleep, this form of acute stress-induced 'pre-transient insomnia', or chronic insomnia.

Within the area of non-pharmacological treatment, precedence for treatment within the range of acute insomnia also exists. There have been several trials of cognitive behaviour therapy for Insomnia (CBT-I) that set the minimum duration criteria for insomnia at one month. The difficulty is there is usually no maximum duration. As such, the treatment effects are diluted across the full course of insomnia making it difficult to assess efficacy during the 'sub-chronic' phase of acute insomnia.

Taking these points into account, when addressing potential treatment implications, based on this conceptualisation of acute insomnia, three questions emerge:

1) Is it feasible that acute insomnia can be identified and/or responded to in a timely manner?
2) Is it possible that an intervention for acute insomnia has the potential to derail the occurrence of chronic insomnia?
3) What would the optimal treatment approach be?

With reference to the first question, providing defining criteria to acute insomnia is a crucial first step if it is to be identified and addressed in both research and clinical practice. Where the pre-transient phase has been experimentally manipulated and people in a pre-chronic phase have been recruited for studies, there is no information on those in the transient phase (two weeks to one month). Considering the time needed to account for night-to-night variability and normal reactions to stress, treatment would be unlikely to be sought or initiated within the pre-transient phase. These considerations also underscore the need for the trigger, and its impact, to be taken into account alongside the duration criteria during assessment. This information may provide an indication of the switch point between a normal, albeit disrupted, sleep pattern and a sleep disorder which may in turn help identify the point at which treatment is warranted. More importantly, the acute insomnia has to be recognised and acted upon by the individual. The timeline of these two events is something that is unknown at present and will likely guide the answer to this question.

The question as to whether the prevention of chronic insomnia is attainable if addressed during an acute phase is curious. Whilst an important goal, the answer as to whether acute insomnia naturally transits to chronic insomnia or the exact rates of transition/remission are as yet unknown. These questions would need answering before an attempt towards prevention would be possible. Although there are indications that the natural pathway is from acute to chronic (as discussed earlier) it is also known that there are a significant number of individuals who remit and as yet it remains a mystery as to why some people transit, in one direction or the other, whilst others do not.

This final question is likely to be of significant interest provided that the first two questions are answered. Based on the previous research summarised earlier (pharmacological and non-pharmacological trials), improvements in sleep are attainable during both the pre-transient (three days to two weeks) and sub-chronic (one to three months) phases and so it stands to reason that improvements are also possible during the transient phase (two weeks to one month). However, at what point is 'normalizing' sleep enough and when should dysfunctional thinking, stimulus control and other components be introduced. As such, this question should be reframed to determine at which point pharmacotherapy is warranted and at which point do non-pharmacological treatments become the first-line treatment option, or is acute insomnia best treated through a combined protocol?

[g] In keeping with the above statement is the expression 'we live with insomnia today because at some point in our evolutionary history, insomnia allowed us to live' (Dean Handley, Sepracor, Circa 2005).

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In essence, the answer to all three questions cannot be addressed without a stronger evidence base, most notably examining the switch point between a sleep disruption and a sleep disorder. Moreover, there needs to be a significant change in the zeitgeist. Getting an individual to recognise and act upon the problem at an early stage, providing a treatment pathway that recognises acute insomnia as a problem, and having a health-care provider with the knowledge and expertise to assess and treat the problem. These are essential goals in attempting to deal with acute insomnia.

Practice points

1) Acute Insomnia is a normal biopsychosocial response to a perceived or actual stressor.
2) Identifying acute insomnia will allow for the development of preventative interventions at both individual clinical levels and at the societal level.

Research agenda

1) Given the proposed definition of acute insomnia, the first order of business will be to determine the incidence and prevalence rates of this phenomenon.
2) The second order of business will be to identify the factors that discriminate between acute and chronic insomnia.
3) Multiple longitudinal studies are needed to define, based on evidentiary grounds, the duration and severity of acute insomnia and determine the point at which attention shifts from the precipitant to sleep and compensatory behaviours are engaged.
4) More stress induction studies, looking at sleep continuity disturbance, and the mechanisms behind this, are required.
5) Behavioural and genetic studies examining predispositional and precipitating factors would help delineate cause and consequence.
6) Studies need to be conducted with an eye towards defining how hyperarousal is manifested across the clinical trajectory and discriminative from the construct of the failure to inhibit wakefulness.
7) Additional analogue studies looking at what psychosocial stressors do and do not induce acute insomnia (i.e., intense hedonic experiences that induce the same level of physiological arousal but do not create insomnia).
8) Additional studies looking at predispositional characteristics and how they interact with the flight or fight response (i.e., rat strains that vary in respect to basal anxiety levels).
9) While it is still a matter of debate whether chronic insomnia is episodic in nature, acute is episodic by definition. Therefore, it may be useful to test the clinical course conceptualisation for its relevance (e.g., age of first episode, age of onset, number of episodes, onset and duration of index episode, relapse and recurrence).

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