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Summary

Cognitive processes play an important role in the maintenance, and treatment of sleep difficulties, including insomnia. In 2002, a comprehensive model was proposed by Harvey. Since its inception the model has received >300 citations, and provided researchers and clinicians with a framework for understanding and treating insomnia. The aim of this review is two-fold. First, we review the current literature investigating each factor proposed in Harvey’s cognitive model of insomnia. Second, we summarise the psychometric properties of key measures used to assess the model’s factors and mechanisms. From these aims, we demonstrate both strengths and limitations of the current knowledge of appropriate measurements associated with the model. This review aims to stimulate and guide future research in this area; and provide an understanding of the resources available to measure, target, and resolve cognitive factors that may maintain chronic insomnia.

Keywords: sleep; insomnia; cognitive model; measurement; review
List of Abbreviations

ACT  
Actigraphy

CBT-I  
Cognitive behaviour therapy for insomnia

CI  
Catastrophising Interview

DBAS  
Dysfunctional beliefs and attitudes about sleep

DISRS  
Daytime insomnia symptom response scale

DBT  
Dot-probe task

FFT  
Flicker fusion task

GCTI  
Glasgow content of thoughts inventory

GSES  
Glasgow sleep efficiency scale

PSAS  
Pre-sleep arousal scale

PSG  
Polysomnography

REM  
Rapid Eye Movement sleep

SAAQ  
Sleep anticipatory anxiety questionnaire

SCT  
Stroop colour task

SRBQ  
Sleep related behaviour questionnaire
Introduction

Sleep is a necessary part of human existence (1,2), and people who sleep poorly may be subject to poor social, occupational and educational functioning (2,3). Insomnia is the most common sleep difficulty (4,5), defined as difficulty initiating and/or maintaining sleep or waking up too early, which disrupts functioning (6,7). Between 25-37% of adults frequently experience at least 1 insomnia symptom, with 5-10% diagnosed with an insomnia disorder (5,8,9). Comorbidity is common, including substance abuse, anxiety, and depression (10,11), which can be a consequence, risk, or even contributing factor (12). Understanding factors that may contribute to, and maintain insomnia is of high clinical relevance.

It is widely acknowledged that cognitive processes play a central role in the maintenance of insomnia. Targeting cognitions is thus important for the effective treatment of the disorder (13). Several key models of insomnia have highlighted the role of various cognitive processes (e.g., the psychobiological inhibition model [14]; the sleep interfering-interpreting process model [15]; and the microanalytic model [16]). The most widely cited of these models is the cognitive model of insomnia (17), which will be the focus of this review. This model of insomnia has been largely accepted by both researchers and clinicians working in the field of sleep disorders. Most notable is the models focus on what maintains, rather than what may cause, insomnia (17,18). That is, the model is said to focus on those features of insomnia likely to be active when a person seeks help for the disorder, and thus the features that would be important to target in a psychological interventions (see Ref 18). The focus on factors maintaining insomnia has particular clinical relevance given evidence that people with insomnia are unlikely to seek professional help until their insomnia has become chronic (19)

1 Based on citation rates presented on Scopus database, September 2014.
When first proposed in 2002, Harvey’s model was based on an extensive review of the insomnia, sleep and anxiety fields (e.g., 16,20,21), providing an accessible and cohesive understanding of the role of cognitions in the maintenance of insomnia. The model proposed several mechanisms, said to perpetuate insomnia, and placed importance both on nighttime and daytime processes (17). While excessive negatively toned cognitive activity (e.g., worry, rumination) lies at the centre of the model, other components either exacerbate or lead to other factors, that together result in a perceived sleep deficit. Other components include (i) safety behaviours, (ii) dysfunctional beliefs, (iii) arousal and distress, (iv) selective attention and monitoring, and (v) distorted perception of deficit (see Figure 1).

The role of cognitions in the maintenance of insomnia is particularly highlighted through the acknowledgement of the efficacy of cognitive behavior therapy for insomnia (CBT-I). CBT-I is considered an efficacious treatment for insomnia, with long term benefits and few side effects (22). However, when dismantling CBT-I, the evidence for cognitive therapy alone is not yet strong (23). Consequently, it is important to fully investigate each factor in the cognitive model, and pathways between them, in the hope that current cognitive therapy will be more potent. This can be made easier if clinicians and researchers are made aware of what measures target cognitive factors. To assist in stimulating further research in this field, the aims of this review were two fold. First, we have provided an update on the past 5 years of research, since recommendations were made to improve knowledge of the cognitive processes related to insomnia in a 2009 review (24). While we acknowledge other factors, outside of those proposed in this model that may impact on insomnia (most notably depression), this review specifically focuses on providing an update on evidence of those factors highlighted in Harvey’s model of insomnia. Second, we have also provided an overview of the key measures for each component of Harvey’s cognitive model for the maintenance of insomnia (17). The intention is to make clinicians cognizant of current
evidence-based measures to use during CBT-I, and to highlight important areas for future research on Harvey’s model.

**Components of the Cognitive Model of Insomnia: The Current State of Evidence**

**Excessive negatively toned cognitive activity.** This factor lies at the centre of Harvey's model, with all components being related to it (see Figure 1; 17). Several correlational and experimental studies support an association between negative cognitions and sleep disturbance (e.g., 24-28). Two cognitive styles which have received attention in the sleep and insomnia field are rumination and worry. While both involve repetitive negatively-valenced thinking, their content varies. Rumination is where one makes attributions for their disturbed mood or symptoms (e.g., "because I did not sleep last night I cannot concentrate today"); 29,30). Worry involves repetitive thinking about the future and consequences (e.g., "because I feel anxious I will not be able to sleep tonight"); 31). While there is robust evidence of the impact of worry, and particularly worry about sleep, on exacerbating sleep difficulties in poor sleepers and in insomnia (31,32), rumination has received less attention, with little focus on comparing the processes. Investigations of rumination in analog samples, showed higher levels of rumination in poor, compared to good, sleepers, as well as a general association between high levels of rumination and poorer sleep (28,33). A 2010 study was the first to explore, and directly compare, worry and rumination in a clinical sample of over 200 adults with primary insomnia (31). Interestingly, while both processes uniquely contributed to poor sleep, it was high levels of rumination, but not worry, that was associated with poorer sleep quality, sleep efficiency and more time awake after sleep onset, in insomnia. Indeed, rumination impacted on insomnia above any impact of depression (31). While the lack of association between worry and sleep was surprising, the authors acknowledged that this may
have resulted from the measure used (Penn State Worry Questionnaire; 34) being too broad and thus not related to sleep disturbance (31). Consequently, future research comparing these two processes remains pertinent.

Catastrophising is a third cognitive process to receive more attention in recent years, and is defined by compounding catastrophic thoughts (e.g., “because I cannot sleep I will not function and never get a job”) (25). Although catastrophising was not explicitly highlighted in Harvey’s original model (13), Espie’s Psychobiological Inhibition Model identifies catastrophising as a meaningful contributor (14). Compared to good sleepers, those diagnosed with an insomnia disorder report more catastrophic thinking about the consequences of sleep (e.g., “If I don’t get enough sleep my job will be on the line”; 35), and experience increased anxiety and discomfort (34), which also provided support for cognitive activity leading to arousal and distress (discussed below; see Figure 1; 17). While Harvey’s model does not explicitly distinguish between these various cognitive styles, based on evidence of each contributing to sleep, there is clearly a need for research to focus on extending our knowledge of how these processes uniquely impact on sleep. Further developing our understanding of these processes would be particularly beneficial for improving the potency of CBT-I.

**Measures of cognitive activity.** Table 1 presents a summary of the psychometrics and clinical utility of measures assessing key components of Harvey’s cognitive model of insomnia. Reviewed measures include the Glasgow content of thoughts inventory (GCTI; 36), the sleep anticipatory anxiety questionnaire (SAAQ; 37), the cognitive subscale of the pre-sleep arousal scale (PSAS; 38), the Glasgow sleep effort scale (GSES; 39), and the daytime insomnia symptom response scale (DISRS; 29,31,40,41). To date, many do not provide clinical cut-offs (e.g., SAAQ, PSAS), significantly reducing their clinical utility, and some would benefit from replication to confirm their validity and reliability (e.g., the GSES
and the DISRS). Further, it would be beneficial to explore whether clinical cut-offs could differentiate factors that maintain insomnia versus, for example, other sleep or mental health disorders (31).

Moreover, there is some debate about what is actually measured by some of these tools (i.e., worry vs. rumination vs. catastrophising) (31). Many questionnaires provide a general measure of negative cognitive activity, rather than individual cognitive processes. Currently, the only measure to focus on insomnia-specific rumination is the DISRS (29). This measure was created based on the symptom-focused rumination subscale of the rumination styles questionnaire, which focusses on rumination and depression (31,40). Additional items were then added based on current knowledge of insomnia (see Ref 29). Thus far, the DISRS has demonstrated good psychometric properties and the ability to distinguish good and poor sleepers, however replication is needed (see Table 1). The most commonly implemented measurement of a catastrophising thinking style is the aptly named catastrophising interview (25,35,42), which has also been used in paediatric samples (<18 yrs old; e.g., Ref 25,35). Scores are based on the number of catastrophising steps generated during the interview, and ceases when the individual can no longer generate responses (e.g., Ref 35). However, little validity and reliability of this technique exists.

**Dysfunctional Beliefs and Safety behaviours.** Harvey proposed underlying beliefs about sleep and use of safety behaviours exacerbate negatively toned cognitive activity (see Figure 1). There is ample evidence supporting the link between beliefs and sleep, including reductions in unhelpful beliefs following CBT-I (43). Perhaps the most extensively used insomnia cognitive measure is the dysfunctional beliefs and attitudes about sleep scale (DBAS; 44). The DBAS has adequate reliability, and is responsive to treatment of samples with primary insomnia (43). It comes in several forms, with the 16-item and original 30-item form showing the best reliability (see Table 1; α =.72-.88). The original DBAS included 5
subscales: (i) misconceptions of the causes of insomnia; (ii) misattributions or amplifications of the consequences of insomnia; (iii) unrealistic sleep expectations; (iv) diminished perceptions of control and predictability of sleep; and (v) faulty beliefs about sleep-promoting practices (44), yet the internal consistencies of some were unacceptable (e.g., ‘diminished perceptions’ [α=.41] and ‘faulty beliefs’ [α=.34]), questioning their use. The DBAS focuses on broad underlying beliefs or schemas, rather than specific automatic thoughts (45). Clinicians may identify themes that the insomnia patient endorses, which can be used to develop behavioural experiments to challenge and alter dysfunctional beliefs.

Safety behaviours are proposed to be associated with beliefs about sleep and exacerbate negatively toned cognitions (17). Broadly, safety behaviours assist the individual in preventing, or avoiding, the feared outcome (i.e., drink alcohol to avoid sleeplessness; consume caffeine to avoid daytime dysfunction). Safety behaviours are maladaptive because they both prevent disconfirming unhelpful beliefs, and increase the likelihood that the feared outcome will occur (e.g., that they will not be able to initiate sleep). There is good evidence for the association between safety behaviours and underlying beliefs (e.g., r = .49) (46,47). However, recent research suggests safety behaviours may be indirectly associated with insomnia severity via maladaptive beliefs about sleep (46). That is, rather than a direct association between safety behaviours and poor sleep, engaging in safety behaviours may exacerbate underlying beliefs, which in turn negatively impacts on sleep. Further still, what seems to be important to insomnia patients is not necessarily the frequency of safety behaviours, but their perceived importance for adequate sleep, especially when predicting the severity of the insomnia experience (47).

The sleep related behaviours questionnaire (SRBQ; 47,48), assesses the frequency of 32 safety behaviours, during the day or night, on a scale from 1 (almost never applies to me) to 5 (applies to me almost always). Preliminary psychometric evidence of the SRBQ is
presented in Table 1. A limitation is that it only provides a measure of frequency, which may not be necessarily related to insomnia severity (47). Hood and colleagues added 2 items to each of the SRBQ’s 32 items (47). Using a 0 to 10 scale, one item assessed the strength of which the person believes the particular behaviour is necessary for sleep, and the second item measured the anticipated distress if the safety behaviour is not performed. A ‘utility’ score is created based on the average ratings across the additional items. While the frequency and utility scores were highly correlated ($r = .80$), only utility was related to insomnia severity. It will be important for future research to continue testing the SBRQ’s reliability and validity, and relationships with insomnia. Moreover, the creation of a shorter scale to reduce time burden is likely to enhance its use in a clinical setting.

**Arousal and Distress.** Negatively toned cognitive activity leads to physiological arousal and distress (17). Both cognitive- and somatic-arousal are associated with susceptibility to stress-related insomnia (49). Typical markers of elevated physiological arousal in adults with insomnia include elevated heart rates, increased temperature, activated sympathetic nervous system during sleep, and abnormal hormone secretion (50,51). Insomnia patients can experience heightened arousal both during the night and day (51,52). Experimental studies manipulating both physiological and psychological arousal show negative effects on sleep (53). This includes people with insomnia reporting increased subjective hyperarousal when presented with negative sleep stimuli (e.g., picture of a person lying awake in bed), and increased physiological arousal (referred to as a craving response) when presented with positive sleep stimuli (e.g., picture of a person asleep in bed)(54). While most studies focus on the role of arousal in effecting sleep onset latency, there is also evidence that psychological distress is associated with increased physiological arousal during the non-rapid eye movement sleep period of individuals with insomnia. This suggests pre-
sleep psychological distress also contributes to nighttime physiological arousal, and thus wakings after sleep onset (55).

**Measures of arousal and distress.** Two measures outlined in Table 1 target self-reported arousal during the pre-sleep or daytime period. The first 5 items of the previously discussed SAAQ (37) ask the individual to rate their pre-sleep somatic complaints (e.g., heart beating when attempting sleep). Similarly, the previously discussed PSAS (38) contains eight (out of 16) items that target somatic complaints (e.g., shortness of breath or labored breathing). These somatic subscales have shown pleasing reliability and validity (see Table 1), yet, more work is needed to validate these self-report measures against objective measures (i.e., EEG, heart rate, metabolic rate).

**Selective monitoring and attention.** The next step in Harvey’s model is that arousal and distress leads to selective attention and monitoring (17). Heightened scanning of the one's self and their environment can lead to a negative feedback loop by increasing awareness of cognitions and behaviours (Figure 1). Selective monitoring may include monitoring physiological signs (e.g., heart rate, temperature) and/or external stimuli (e.g., noises outside, the time) that may inhibit sleep. The majority of studies support this aspect of the model (56-60), including evidence of sleep-related attentional biases differentiating good sleepers, moderately poor sleepers, and those with insomnia (56). Moreover, the role of selective attention has been demonstrated through experimental studies that have shown manipulating a person's selective attention to a stimulus adversely affects sleep (24). Recent innovative claims are that the selective attention mechanism may be an inability to shift from internal/external stimuli (60), and that threat stimuli may trigger an anxious vigilance-avoidance response (arousal and distress) (61). These findings have important clinical implications, and as such, warrant replication and further exploration.
Measures of selective monitoring and attention. The majority of research on sleep-related attentional biases have either used modified versions of the dot-probe task (DPT; 61), the Stroop colour task (SCT; 63,64), or the flicker fusion task (FFT; 65). Usually these tasks are computerised (i.e., stand-alone laptop), and since the recent introduction of tablets, apps are now available (66), making them accessible by clinicians to use with their patients in a clinical setting. As these tasks produce measurements in milliseconds, their use over web applications is not recommended due to variability in internet speed (i.e., inflating measurement error). For techno-adverse clinicians, the SCT may be administered in pen-and-paper format.

In brief, the DPT presents either sleep-related images (e.g., pillow) or words (e.g., night) along with neutral images/words. Images/words are removed and a single 'probe' (e.g., arrow pointing right) replaces one of them. One is required to press the appropriate arrow button on the keyboard (e.g., right arrow) as quickly as possible. The hypothesis is that an insomnia patient's attention is drawn to the sleep-related stimulus and thus responds more quickly to the probe they were already looking at. The SCT uses coloured neutral and sleep-related words. The task is to quickly name the colour of the word without reading it. In this case, the hypothesis is that insomnia patient's attention is additionally drawn to the sleep-related words, and that this extra cognitive load takes more milliseconds to complete. Finally, the FFT quickly interchanges an image of neutral images (e.g., chair, fruit, shovel, etc.) with additional sleep-related images (e.g., slippers, pillow, teddy bear, etc.). A button is pressed as soon as they perceive a change. The hypothesis being an insomnia patients' attention is drawn more rapidly to sleep-related stimuli and thus their reaction times are quicker. Although engaging, there is limited psychometric data on these tasks. To be more useful in a clinical
setting, normative data of good sleepers and patients with insomnia would help clinicians ascertain the severity of their patient’s selective attention and monitoring.

**Misperception of deficit.** The final component of the model is that individuals with insomnia possess a distorted perception, commonly applied to their own sleep (e.g., that they report less sleep than they actually get). Misperception may be present despite good objective sleep (26) or an exaggeration of actual sleep deficit (67). When insomnia patients receive negative feedback (versus positive), regarding their sleep (e.g., that the sleep obtained was bad quality), they report more negative thoughts, monitor for sleep related threats, and engage in safety behaviours (68). A recent comprehensive paper reviewed 13 possible contributions to misperception, including: exaggerating the sleep complaint, psychological distress causing the misperception, worry and selective attention towards sleep-related threats, two insomnia subtypes (with/without misperception), and sleep being misperceived as wake (for full list see ref 69). Out of the 13, three had the best support. These were misperceiving sleep as being awake, worry and selective attention towards threats, and brief awakenings. Brief awakenings are likely to occur from light stages of sleep (e.g., stage 2) and rapid eye movement (REM) sleep, so it is not surprising individuals with insomnia are more likely to report being awake for longer than good sleepers. Reasons proposed for this phenomenon include a greater amount of mentation during sleep (i.e., mentation that more closely resembles waking mentation), and a selective bias due to previous experiences of nocturnal wakefulness (67). Although polysomnography (PSG) is not a standard measurement for insomnia, there is precedence for its use (70,71). If the clinician is part of a multi-disciplinary sleep disorders clinic, PSG would allow for the opportunity to probe insomnia patients’ perception of sleep when waking from light sleep, by asking the question over an intercom “Do you think you have been awake or sleep”, followed by “How long do
you think you have been awake/sleep?" (67). In most cases, PSG is a time and financial expense not afforded in most clinics. The standard is therefore the simultaneous use of wrist actigraphy and sleep diary, which can be readily compared for discrepancies, and fed back to the insomnia patient (68).

Finally, misperception can be applied to daytime activities (e.g., performance; 17). Although subjective daytime complaints are common among those suffering insomnia (63, 69), and constitute a core diagnostic component of the disorder (5), the majority of studies demonstrate a lack of impairment in the objective performance of insomnia patients when compared to good sleepers (72-75). For these relatively short-duration performance tasks, hyperarousal may act as a compensatory mechanism allowing insomnia patients to rally cognitive resources to achieve a comparable level of performance to that of good sleepers (49). In less demanding everyday situations, they may not rally such cognitive resources. This issue remains unclear and warrants further investigation to guide clinicians regarding the emphasis cognitive therapy should place on addressing the apparent discrepancy between subjective and objective daytime functioning.

Summary

In the 12 years since the publication of Harvey’s cognitive model of insomnia (13) evidence has accumulated to show that each component and pathway exists for insomnia patients, and in some cases poor sleepers (compared to good sleepers). Based on evidence presented in this review on the consensus of cognitive factors impacting sleep in insomnia, the research diagnostic criteria may benefit from being updated to increase the focus on the importance of these processes. In this review we have also provided an overview of the key measures used to assess each cognitive process in Harvey’s model that may maintain insomnia. We have primarily focused on measures that can be used in clinical settings. The purpose was not only to provide researchers with an up-to-date psychometric review, but also
to provide clinicians with an overview of their clinical utility. The literature has demonstrated pleasing reliability and validity for many measures (Table 1). However, we also highlight some important cautions (e.g., reliabilities of subscales; differentiating specific thought processes [worry, rumination, catastrophising]). Developing clinical cut-offs will also enhance these measures’ clinical utility. Moreover, while it was not the focus of this review, there remains a need for research to focus on how different cognitive factors may impact insomnia differently in different populations (e.g., older adult, different co-morbidities). We hope this review serves as a stimulus for replication and novel exploration of the tools to measure cognitive processes associated with the maintenance of insomnia.

**Research Agenda**

- Clinical activity of the tools used will be enhanced with the development of clinical cut-off scores.
- More research is needed to test distinctions between worry, rumination, and catastrophising in relation to the insomnia experience.
- New studies as well as replication studies are needed to validate the measurement of safety behaviours, and self-reported arousal and distress.
- A greater focus on the assessment of the misperception of performance deficits is needed given daytime impairment is a common complaint of insomnia patients.

**Practice Points**

- Several measures with good psychometric properties are available to test excessive negatively toned cognitive activity.
- Currently there is only one measure to assess dysfunctional beliefs (DBAS) and safety-behaviours, yet there is excellent support for the DBAS.
- Two subscales from measures (PSAS, SAAQ) may be used to assess arousal and distress.
- Novel assessment of attentional bias may be performed with new apps.
- If the resources exist, misperception of sleep may be assessed with PSG at a multi-disciplinary sleep clinic, or wrist actigraphy, both of which should be compared to self-report (e.g., sleep diary).
References


Table 1

Summary of Measures of Sleep Cognitions, their Psychometric Properties and Clinical Utility

<table>
<thead>
<tr>
<th>Measure</th>
<th>Number of Items</th>
<th>Psychometric Properties</th>
<th>Tested Aspect of Harvey's Model</th>
<th>Summary of Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catastrophising interview</td>
<td>1-10 items (ends when patient cannot generate more thoughts)</td>
<td>Unknown</td>
<td>Excessive negatively-toned cognitive activity</td>
<td>Catastrophising has been linked to sleep onset. No information on validity or reliability of measure</td>
</tr>
<tr>
<td>Daytime insomnia symptom response scale (DISRS; 29)</td>
<td>20 item scale (8 items come from previously validated Ruminion Style Questionnaire; 31,40,41)</td>
<td>Cronbach’s alpha = .80 - .88</td>
<td>Excessive negatively-toned cognitive activity</td>
<td>Acceptable psychometric properties but replication necessary. Distinguish good sleepers and those with insomnia. Preliminary evidence to support importance of rumination as distinct issue. No information on clinical cut-off scores</td>
</tr>
<tr>
<td>Dysfunctional beliefs and attitudes about sleep scale</td>
<td>Original scale: 30 items</td>
<td>Cronbach’s alpha = .69 - .88</td>
<td>Beliefs</td>
<td>Widely used and validated. Better to use total score rather than subscale scores</td>
</tr>
<tr>
<td>Glasgow content of thoughts inventory (GCTI; 36)</td>
<td>25 items</td>
<td>Cronbach’s alpha = .87</td>
<td>Excessive negatively-toned cognitive activity</td>
<td>Only 1 study focused on psychometric properties</td>
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<tr>
<td></td>
<td></td>
<td>Distinguishes good sleepers/people with insomnia</td>
<td></td>
<td>Includes items on rehearsal and planning cognitions, based on evidence for common pre-sleep thought content (70)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Relationship with DBAS</td>
<td></td>
<td>Targets specific pre-sleep automatic thoughts: these may be more accessible than the DBAS items for use in clinical work</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Score of ≥ 42 (out of 100) identified 100% insomnia (sensitivity) and 86% good sleepers (specificity)</td>
<td></td>
<td>Clinical cut-offs provided, although</td>
</tr>
<tr>
<td>{DBAS; 44}</td>
<td>Short forms: range from 10 to 28 items</td>
<td>for the 16-item version ((\alpha = .80 - .88)) and 30-item version ((\alpha = .72 - .80))</td>
<td>Adequate psychometric properties with people of different ages, ethnicities and with comorbid conditions</td>
<td>Recommended: either 16-item or original 30-item version</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distinguishes good sleepers/people with insomnia</td>
<td></td>
<td>Better measure of broader beliefs than of negative automatic thoughts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sensitive to treatment changes (CBT)</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Clinical cut-off score is provided for 16-item DBAS (&gt;3.8 indicates clinical level of unhelpful beliefs)</td>
<td></td>
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</tr>
<tr>
<td>Glasgow sleep effort scale</td>
<td>7 items</td>
<td>Cronbach’s alpha = .70 - .77</td>
<td>Beliefs</td>
<td>Very early stages of use – more replication required</td>
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<tr>
<td>(GSES; 39)</td>
<td></td>
<td>Showed evidence of divergent validity</td>
<td>Excessive negatively-toned cognitive activity</td>
<td>Measures a very specific area of cognitions (sleep effort).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Content validity uncertain – may not cover cognitive and behavioural aspects of sleep effort</td>
<td></td>
<td>May be more useful to use a more general measure of sleep cognitions/beliefs first</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinical cut-off score provided (total score &gt;2 identified 93% of people with insomnia &amp; 87% of good sleepers)</td>
<td></td>
<td>Then could use GSES if sleep effort is implicated as a problem for individuals</td>
</tr>
</tbody>
</table>

| Pre-sleep arousal scale    | 16 items (8 somatic, 8 cognitive) | Cronbach’s alpha = .67 - .88 | Excessive negatively-toned cognitive activity | More replication needed |
| (PSAS; 38)                | 13 item scale (8 somatic, 5 cognitive) | Distinguishes good sleepers/people with insomnia | Arousal and distress | Measures cognitions and somatic arousal |
|                           |                                   | Particularly useful with sleep-onset difficulties | Adequate psychometric properties | Acceptable psychometric properties |
|                           |                                   | Adequate psychometric properties | No clinical cut-off scores provided |
with children in one study

Related to measures of anxiety, depression, somatic anxiety symptoms and cognitive anxiety symptoms

<table>
<thead>
<tr>
<th>Sleep related behavior questionnaire (SBRQ; 47)</th>
<th>32 item scale</th>
<th>Chronbach’s alpha = .83-.92</th>
<th>Safety Behaviours</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Distinguishes good and poor sleepers</td>
<td>Covers a wide range of safety behaviours</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sensitive to treatment changes</td>
<td>No information on clinical cut-offs</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sleep anticipatory anxiety questionnaire (SAAQ; 37)</th>
<th>10 items (5 somatic, 5 cognitive)</th>
<th>Cronbach's alpha = .83 - .84</th>
<th>Excessive negatively-toned cognitive activity</th>
<th>Measures cognitions and somatic arousal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Distinguished adults with sleep-onset insomnia from general student population</td>
<td>Arousal and distress</td>
<td>Trend towards significant change in scores pre- and post-treatment (Intensive Sleep Retraining; 71). Significant differences were found when only the cognitive scale was considered (and not the somatic scale).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Related to measure of pre-sleep arousal and somatic symptoms</td>
<td></td>
<td>Initial reports show good psychometric properties</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Related to measures of sleep self-efficacy, anxiety sensitivity and worry.</td>
<td></td>
<td>No clinical cut-off scores provided</td>
</tr>
</tbody>
</table>
Figure 1. Cognitive model of insomnia (Harvey, 2002), with acronyms for discussed measures, listed under the component they are proposed to measure. Published with permission from [17].