

**Manuscript category:** Review Article

**Title:** Hyperarousal and failure to inhibit wakefulness in primary insomnia: “Birds of a feather”?

**Running Title:** Processes in insomnia

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**Word Count:** 5883

## **Abstract**

Primary insomnia (PI) is one of the most prevalent sleep disorders. For this reason, over the last decades, several comprehensive and etiological theories have been proposed. In this paper we review some of the main theoretical models of insomnia and discuss the two most studied processes for comprehension of insomnia: the hyperarousal and the failure to inhibit wakefulness or psychobiological inhibition hypotheses. Some clinical implications of the models are described. In the end, we propose that the two processes are complementary and both are relevant to the understanding of clinical insomnia.

*Key words:* primary insomnia, hyperarousal hypothesis; psychobiological inhibition hypothesis

## **Introduction**

Primary insomnia (PI) is a clinical condition characterized by a marked difficulty in initiating or maintaining sleep, waking up too early and cannot go back to sleep or experiencing non-restorative sleep that is not due to a comorbid medical or psychiatric disorder.<sup>1,2</sup> Briefly, it results on a pathological reduction of sleep time at night or experiencing a non-restorative sleep. Insomnia is a highly prevalent health complaint afflicting approximately 50% of the general population.<sup>3</sup> Prevalence of PI in general population ranges from 3 to 5%.<sup>4</sup> PI is one of the most common clinical entities within all sleep disorders.<sup>1,4,5</sup>

### *Models of insomnia and its relationship to the concept of hyperarousal*

The set of theories about the etiology and pathophysiology of PI has led researchers and clinicians to propose integrated approaches. From the mid-80s of the twentieth century the emphasis was put mainly on the psychological, emotional, and psychopathological variables. However, from the 90's and with the advent of neuroimaging techniques, theoretical approaches started to highlight the role of neurobiological variables. Following these developments, the concept of hyperarousal became popular as a predisposing factor for insomnia, which combined with other constructs such as personalistic traits or genetic components would make individuals more or less vulnerable to develop an insomnia disorder.<sup>4,3</sup> The comprehensive list of the models we present follows a hybrid orientation, although with a more behavioral focus. For a brief review of the main physiological models see other references.<sup>6,7</sup> For a review of the *Drosophila* model of insomnia and the cage exchange model see Perlis et al.<sup>8</sup> The list of models we present in the current paper (i.e., internalization of conflict's model; behavioral perspective of Spielman; stimulus control model; microanalytic model of Morin; hybrid cognitive-behavioral model of Lundh and

Broman; neurocognitive theory of insomnia; Espie's psychobiological inhibition model; the cognitive model of Harvey; and bottom-up model of Riemann) is entirely from our responsibility and tries whenever possible to follow chronological criteria. Similarly, it should be noted that some of these models are different in their explanation level. For example, some theoretical approaches are more interested in understanding of the maintenance factors in insomnia rather than on etiological ones.<sup>9</sup> According to the literature review we have performed, the models selected in this paper seemed to be the most comprehensive and appropriate ones for the purposes of the present work. For a brief explanation of each of the models see Table 1.

#### *Internalization of conflict's model*

The first attempt to relate empirically insomnia with psychological constructs comes with the internalization model of conflicts.<sup>10</sup> Based on the input from psychodynamic and psychosomatic medicine insights dominant at the time, the authors used a psychological assessment tool widely used for studying the personality, the MMPI (Minnesota Multiphasic Personality Inventory), and assessed hundreds of people. The results showed significantly higher levels of depression, conversion hysteria and psychasthenia in individuals with PI compared to control groups without sleep complaints. Following this, it was defined a "typical" personality type for the PI patient: a person with a more pronounced tendency to internalize problems and emotions (e.g., depression) than to externalizing (e.g., acting-out). This predisposition to internalize conflicts would lead to increased levels of emotional activation, which in turn would lead to physiological hyperarousal, transforming the person into someone less prone to sleep.<sup>11</sup> Another study, with 528 subjects (428 insomniacs and 100 controls) using the same psychological measure confirmed the initial pioneering study. The profile of individuals with insomnia was homogeneous between people from rural areas and

semi-urban areas being characterized by high levels of neurotic depression, rumination, anxiety, chronic inhibition of emotions, and an inability to express hostile feelings (e.g., anger).<sup>12</sup>

In terms of implications for treatment, these authors suggested that:

(...) [it] should primarily center around psychotherapeutic treatment to elicit emotional discharge during the day to minimize emotional arousal at night and to deal directly with the fear of sleeplessness itself by means of supportive psychotherapy or behavioral techniques. Adjunctive short-term use of hypnotic drugs may be utilized to diminish physiologic arousal in insomniacs, which is a result of their unexpressed and internalized negative emotionality (p. 354).

These data were supported by another study using the MMPI, which found a “psychosomatic profile” in patients with insomnia.<sup>13</sup> There are still few investigations on personality characteristics of patients with insomnia, and as such it is proposed by several authors as an issue to invest in the future.<sup>9</sup> Some recent studies that are trying to follow this direction are worth mentioning.

A research of Spiegelhalder et al. reinforced the idea that there is a personalistic pattern, containing obsessive traits, in patients with insomnia compared to patients with other sleep disorders. The authors assessed/operationalized this construct using an original construct: punctuality.<sup>14</sup> The recent studies about personality traits of individuals with insomnia have brought new data, specifically about the fact that patients with PI seem to differentiate themselves from other patients with other subtypes of insomnia. There are researches which emphasize there is no particular pathognomonic profile related to PI while others suggest that PI patients are more pessimistic, fearful, shy and reveal higher fatigue level compared to

general population samples.<sup>15</sup> Other studies point to a similar profile in insomnia patients and individuals with various psychosomatic diseases.<sup>16</sup> A recent research concluded that there is a negative correlation among the severity of insomnia and personality related variables such as looking for new sensations ("novelty seeking"), dependence for rewards ("reward dependence") and cooperation ("cooperativeness"). Moreover, the same research found a positive relationship among "harm avoidance", "self-transcendence" and "sleep-related cognitions" and insomnia severity.<sup>17</sup> Other studies have investigated also the association between sleep/insomnia and perfectionism.<sup>18,19</sup> In summary, the studies concerning the profile of individuals with insomnia are within what many authors consider to be emotional hyperarousal.<sup>20</sup>

#### *Behavioral perspective of Spielman*

The approach regarding PI by Arthur Spielman may be considered one of the most important ones, since it will influence subsequently further psychological (i.e., cognitive-behavioral models) and biological approaches.<sup>9</sup> This model suggests that there are three factors related to the etiology and maintenance of PI: a) *predisposing factors* - that refer to behavioral traits of vulnerability prior to insomnia, encompassing biopsychosocial spectrum variables such as biological (e.g., hyperarousal of different neurobiological systems), psychological (e.g., tendency to worry and ruminate excessively), and social (e.g., bed companion with inconsistent sleep schedule, social pressures to sleep without respecting the sufficient hours of sleep) variables; b) *precipitating factors* - which concerns life situations perceived and interpreted by the person as threatening and stressful, and as such, inducing insomnia symptoms (e.g., death in the family, sudden illness); and finally, c) the *maintenance factors* - which relate roughly with the maladaptive strategies that people develop to cope with insomnia (e.g., excessive time in bed, staying in bed although not sleeping).<sup>9,21</sup> This

model turns out to explain why people with PI sleep well in new environments, even in the first night on a sleep laboratory. This is known as "reverse first night effect".<sup>22</sup> The environment is new, has few visual and temporal cues for activation (arousal) and allows transitory improvement in the patient's complaints. This also explains the fact that many patients fall asleep, for example, in the living room watching TV, and consequently when they go to the room/bed, drowsiness disappears and a scenario of widespread arousal installs. It should be noted that alternative views have been developed over the years about the role of these different factors, in particular, regarding the role of the predisposing factors that seem not to be as static as previously thought.<sup>23</sup> This perspective is also known as the model of the 3 P's, since the designation of the three factors begins with the letter "P" - predisposing, precipitating and perpetuating.<sup>7</sup>

#### *Stimulus control model*

The stimulus control model proposed by Bootzin is based on the conditioning history of individuals.<sup>24</sup> Starting from the assumption that the same stimulus can generate varied responses, this author suggests that the PI develops when the sleep-related stimuli (e.g., bedtime, bed or room) failed to generate responses associated (only) to sleep, yielding alternatively responses inconsistent with the sleep behavior or sleep induction, such as reading, working or concerning about not being able to fall asleep. In this sense, because there is a learned association between stimuli and responses that is not conducive to sleep, the clinician can help patients to inhibit, strengthen or promote new learning conditions.<sup>25</sup>

#### *Microanalytic model of Morin*

The Canadian psychologist Charles Morin proposed in the early 90s of the last century a guiding multifactorial and integrative model to understand how the vicious cycle related with

PI installs and becomes autonomous and independent from the precipitating factor that might have originated it. This theory stresses psychosocial aspects, namely cognitive arousal, but also includes biophysiological variables, being the first model to suggest a more articulated concept of hyperarousal covering affective, behavioral and physiological domains. Thus, according to this author, there are four major categories of maintenance factors that, together, explain how a person cannot to get rid of insomnia: (1) *activation*, characterized by an abnormal and excessive activity in physiological (i.e., activity related to the central and peripheral nervous system that maintains arousal and is incompatible with, or interferes with the process of inducing sleep ), cognitive (i.e., related to an intense activity in terms of alertness, intrusive thoughts related to own sleep behavior, daily concerns, eager anticipations, ...), and emotional systems (i.e., related with stressors which induce a permanent state of activation, personality traits such as neuroticism and perfectionism, or psychopathology); (2) *dysfunctional cognitions* (i.e., which relate to cognitive arousal and that crystallize in a more profound way forming core beliefs or cognitive schemes that once activated or hypervalent they will guide the subsequent processing of information); (3) *maladaptive habits* (i.e., excessive time in bed, napping); (4) and the *pernicious consequences of the interaction of all these factors*.<sup>20</sup> It is due to Morins` works and his theoretical model that cognitive techniques (developed initially for depressive and anxiety disorders) targeted for therapeutic intervention become frequently used in clinical practice. This author, in addition to his many important contributions to the insomnia´s study, investigated two fundamental cognitive errors or distortions in patients with PI: catastrophizing and probability over estimation.<sup>26</sup> This theoretical model by Morin, together with behavioral models such as the Spielman´s theory, is one of the most cited in the literature, underlying most of contemporary clinical practice in the field of insomnia.



### *Hybrid cognitive-behavioral model of Lundh and Broman*

The Lundh and Broman's model is clearly an approach that gives more prominence to insomnia's maintenance processes.<sup>27</sup> The authors note that there are two types of psychological processes forming the core of the experience of insomnia: (1) *processes that interfere with sleep-related levels of cognitive, emotional and physiological arousal*, which may be moderated by personality characteristics such as neuroticism, and (2) *processes that make the individual prone to perceive or interpret their sleep patterns in a distorted or dysfunctional way* - usually linked to inaccurate beliefs that people hold about sleep in general, causal attributions associated with partial or total sleep deprivation, or attentional biases (among others). Despite this distinction, the authors suggest that in practice there is a bidirectional relationship between these two processes, influencing each other. High levels of arousal often leads to biased interpretations or cognitive distortions regarding own sleep difficulties; in turn, ruminations, worries, and negative beliefs and attitudes regarding sleep, often lead to exacerbation of arousal. In short, this is an approach with important clinical implications, which allows that treatment strategies will be selected depending on the identification of the process more compromised to each individual.<sup>28</sup> If the sleep-interfering processes are the most predominant, then the treatment may focus on strategies such as relaxation, stimulus control, restriction of time in bed, or techniques derived from cognitive-behavioral paradigms of third generation such as mindfulness and acceptance; if on the other hand the processes involved are essentially those that lead the individual to perceive or interpret their sleep patterns in a distorted or dysfunctional way, then cognitively oriented methodologies may be more relevant. Nonetheless, in most cases, both processes are compromised.

### *Neurocognitive theory of insomnia*

The neurocognitive theory represents an advance in conceptualization of insomnia, extending the behavioral or psychological classical models to put in evidence biophysiological variables, which have been understudied by previous models. This model highlights the concept of hyperarousal, and divides it into the three distinct components considered by previous models, namely: somatic, cognitive, and cortical. *Somatic hyperarousal* corresponds to high rates of metabolism; *cognitive hyperarousal* concerns psychological constructs such as rumination or worry; and *cortical hyperarousal* concerns to cortical high activity recorded by electroencephalography or event-related potentials (e.g., EEG and ERP, respectively) or other measures related to the activity of central nervous system (e.g. structural and functional neuroimaging – MRI and fMRI, respectively).<sup>29,30</sup> These authors call attention to the "paradoxes of insomnia" maintained by patients (i.e., perceiving hypnotic treatment as being more effective than it actually is; overestimating sleep latency and underestimating total sleep time; and perceiving sleep, as recorded by polysomnography, as wakefulness). Traditionally, somatic activation is seen as something physical/corporal - thus studied preferentially with physiological or biological methods, while the cognitive activation is regarded as a "matter of mind," being privileged for this purpose by traditional psychological records.<sup>29</sup> The same investigators stresses that the approach should not be orthogonal but integrated, and the cortical activity may be interpreted as a correlate of cognitive activity, for example.

### *Espie's psychobiological inhibition model*

Colin Espie, an experienced clinical psychologist and researcher in the domain of insomnia, proposes that the circadian and homeostatic processes are inhibited in chronic insomnia. He and his colleagues developed the AIE model (A-attention; I-intention; E-effort)

which consists of a cognitive model aiming to further understand the development and maintenance of chronic insomnia.<sup>31,3</sup> This model may even represent, according to its proponents, a subtype of PI that may be called AIE syndrome.<sup>32</sup> This perspective focuses on the cognitive mechanisms related to the general hyperarousal underlying PI, and it tries to explain insomnia symptomatology by the same mechanisms underlying normal or standard sleep. For sleep to occur, there must be a general dearousal (i.e., decreased activation / deactivation), at the cognitive and physiological levels, together with a set of automated actions concerning stimulus control or practices related to good sleep hygiene. In this sense, this model states that the three main components of cognitive dysfunction in PI are: *selective attention to sleep, explicit intention to sleep, and effort to fall asleep*. However, in contrast: 1) normal sleep is an automatic and involuntary process that can be inhibited by selectively directing attention to itself - according to Troxel, German and Buysse is the same as saying that "trying to sleep is not only frustrating, but biologically impossible: Sleep is not a volitional behavior, but the state that the brain switches into when it is ready to do so, based on homeostatic and circadian factors" (p. 273)<sup>33</sup>; 2) automaticity is committed to a self-implicit instruction to sleep, and 3) the explicit effort to try to sleep results in the development of maladaptive or dysfunctional behaviors, such as alcohol intake.<sup>31,34</sup> From a pragmatic way, it may be assumed that the selective attention, the explicit intention and the effort to sleep represent three distinct, albeit related, modes: the "scanning mode", the "planning mode" and the "performing mode", respectively. This model proposed by Espie et al. although more focused on cognitive processes, supports the probable mechanisms by which cognitive-behavioral strategies used in the treatment of insomnia are effective, that is, precisely because they would facilitate the disinhibition of neurobiological structures responsible for inducing sleep.<sup>8</sup> This model further explains why individuals with insomnia tend to obtain better sleep outside the usual sleeping place: because they do not have the

specific goal or explicit intent to sleep. Furthermore, it gives strength to the use of the paradoxical intention's technique. In sum, these authors elaborated an alternative explanation to the classical hyperarousal hypothesis, which despite its value needs more research.

### *The cognitive model of Harvey*

The cognitive model of Allison Harvey is a more faithful approach to the classic cognitive theories of psychopathology.<sup>35</sup> According to Harvey and her team, insomnia is maintained by a profusion of cognitive processes that operate either at night or during the day. These cognitive processes relate to (1) *worry*, (2) *selective attention and monitoring*, (3) *misperception of sleep and daytime dysfunction*, (4) *dysfunctional beliefs*, and (5) *dysfunctional safety behaviors*.<sup>36</sup> Briefly, this cognitive model emphasizes an individualized case formulation-driven treatment, and it considers that the worries (Harvey & Greenall, 2003), for instance, at bedtime, will lead the nervous system to rise sympathetic arousal and physiological distress.<sup>37</sup> This arousal state, similar to what occurs in threat, harm or challenge situations (i.e., stress in general), will decrease the attentional field turning the person more aware of possible clues or threatening stimuli (monitoring), and preventing her/him from relaxing. The attention and monitoring are typically directed to stimuli signaling potential threats related to sleep (or lack of it), such as internal stimuli, such as certain bodily sensations, or external environment stimuli, such as the clock displaying the hours.<sup>38</sup> Therefore, it will strengthen unrealistic or dysfunctional beliefs that maintain the vicious cycle of insomnia. To deal with the perceived threat, the person performs certain open or covert behaviors (i.e., safety behaviors) which aim to reduce anxiety and decrease sleep latency. Generally, these are dysfunctional behaviors (e.g., taking psychotropic drugs, alcohol abuse, trying hard to sleep – sleep effort) in the sense that they prevent the patient from having experiences that challenge their beliefs, thus making it more likely that the fears of

patients persist.<sup>39</sup> A very important component in this model in terms of therapeutic protocol design is the emphasis on behavioral experiments.<sup>37</sup> This cognitive model allows for a flexible treatment for many patients because the intervention options and sequence are adjusted depending on the analysis of the most relevant maintenance factors for each patient.

### *Bottom-up model of Riemann*

In an article published in 2010, Riemann and colleagues gathering input from various scientific fields, organized the literature so far supporting the hypothesis of hyperarousal in insomnia. The authors' initial thesis was that there is empirical evidence organized in the literature that converges to the hypothesis of hyperarousal in insomnia, gathering input from various scientific domains (e.g., neuroendocrine, neuroimaging, neuroimmunologic, and electrophysiological).<sup>3</sup> The bottom-up model proposes that PI results partially from a genetically determined dysfunction in sleep–wake regulating neural circuitries. However, this approach does not ignore the relevance of perpetuating factors or maintenance mechanisms, such as tendency to ruminating both during the night and during the day. In terms of comprehensive models of PI, it is suggested that there should be a balance between a top-down approach (which focus on the importance of cognitive processes on the physiological activation, such as Perlis et al. or Espie models)<sup>29,31</sup> and a bottom-up approach privileging an explanation based on a genetically determined dysfunction of the neural mechanisms that regulates the sleep-wake cycle, essentially located in the brainstem, hypothetically causing cognitive and emotional disturbances. The authors state that it is necessary to integrate data from neurobiological research (including genetic studies that have shown robust in understanding the hyperarousal of PI in order to complement (but not to neglect) psychological or behavioral models.<sup>3</sup> Although there are conflicting data and it is currently impossible to determine cause-effect relationships, Riemann et al. suggest that it is reasonable

to assume that only those individuals with a certain genetic vulnerability for insomnia are likely to develop chronic insomnia.<sup>5</sup> Still, we should recognize that "(...) there is no definitive evidence that supports any single theory of insomnia. Likewise, the biological or neurophysiological markers have adequate sensitivity and specificity demonstrated" (p. 256).<sup>40</sup>

### *The instability hypothesis of REM sleep*

A complementary view to the hypothesis of hyperarousal in insomnia establishes that what causes sleep complaints in these patients is the instability occurring during REM sleep. This hypothesis has been based on studies that point to the existence of common micro and macro-awakenings in these patients during REM sleep. This is the sleep stage where there is greater neuropsychophysiological activation. In this way, researchers suggest that this stage is where individuals with a more pronounced degree of activation are more sensitive to arousals and sleep fragmentation. Also according to this model, concerns about sleep difficulties and their consequences are common themes in the dreams of insomnia patients. Arousals occurring in REM sleep make dream cognitions more accessible to conscious perception, to memorizing and its retrieval upon waking in the morning, resulting in an experience of interrupted and not restorative sleep. This fragmentation in REM sleep seems to point to a dysfunction in the limbic and paralimbic areas of the brain that are especially active at this sleep stage.<sup>41</sup> REM sleep appears to be particularly vulnerable to pre-sleep concerns, which might lead therefore to a retrospective memory of this phase as waking time, and to perceive sleep quality as poor. In short, for these authors, the hyperarousal in PI is expressed primarily as a pronounced change in REM sleep.

### *Metacognitive model of insomnia*

The model of two levels of arousal related with sleep (metacognitive model of insomnia) by Ong et al. represents an attempt to develop a conceptual framework of insomnia within the third generation models of cognitive-behavioral therapies.<sup>26</sup> This theoretical proposal clearly emphasizes the role played by metacognitions (referred in this model as secondary activation). Put it simply, this model concerns to how one relates to thoughts about sleep and not directly to cognitive activity directly related to the inability to sleep. It denotes a marked influence of Lundh and Broman's model.<sup>27</sup> This perspective, despite the satisfactory results found/obtained until now, is still an exploratory one, as it lacks more systematic research. It is the first model that attempts to develop a theoretical explanation on the mechanisms that underlie the promising results that treatments based on mindfulness and acceptance have achieved.

**INSERT TABLE 1 HERE**

### **Discussion**

#### *The hyperarousal's hypothesis*

It should be noted that in this paper was chosen the term "hyperarousal hypothesis" like Bonnet and Arand suggest to refer to hyperactivation.<sup>42</sup> Other authors use alternative designations as "hyperactivation perspective", "hyperactivation theory" or "hyperarousal model".<sup>3,5</sup>

PI is one of the sleep disorders included in the International Classification of Sleep Disorders (AASM, 2005) and is currently regarded as a "psychobiological disorder".<sup>5</sup> Consequently, hypotheses about its etiology and pathophysiology have been generated trying to reconcile the traditional psychological models with the findings within the modern

neuroscience. In this line, the hyperarousal proposal emerges as representing an integrated perspective of insomnia, and corresponding to an increased level of overall stimulation in the organism, affecting physiological, cognitive, and emotional systems.<sup>43,42,44</sup> The most recent studies, as mentioned by Pejovic & Vgontzas, suggest that “insomnia is a disorder of hyperarousal present throughout the 24-hour sleep/wake cycle, rather than a disorder of sleep loss” (p. 65).<sup>45</sup>

Many insomnia understanding models have attempted, since their beginning, to incorporate the concept of hyperarousal. However, one aspect that can be criticized in some models that explain this construct is that there seems to be no consensus about what the boundaries are to this hypothetical hyperactivation. That is, there are authors who discuss disparate systems (e.g., cognitive, emotional, ...), subdivide other systems that might fit together into other subsystems such as the physiological activation and cortical activation, others suggest that hyperarousal concerns essentially to neurobiological activation, and there are also researchers who refer to hyperarousal as a concept that meets any kind of arousal, regardless of the system. Thus, we are witnessing a paradoxical situation in this field. If on the one hand the view that there is a hyperarousal in PI seems to be consensual, on the other, there is no consensus on what the term covers. Riemann et al. report that even after about 20 years from the initial conceptualization of PI by Hauri and Fischer (1986) there was little accumulation of studies on the physiological component (i.e., somatic tension) of insomnia.<sup>3,46</sup> In this study, for operationalization purposes, we assume the definition of hyperarousal as any significantly pronounced activity in neurobiological, cognitive and/or emotional systems, either self-reported or objectively observed by different methods, and which differs from the average of a group without major sleep problems or disorders. As a complement to the suggested models we should invest in the study of affective and personalistic variables.<sup>9,47</sup> For instance, it would be germane to realize to which extent



neuroticism predisposes to, or it results from PI. Contextual and environmental factors should likewise be further investigated. Regardless of the comprehensive model on the etiology and maintenance of insomnia, it should be noted that the psychophysiological stress plays a major role, similar to what occurs in other neuropsychiatric disorders.<sup>48</sup> Although the study of the hyperarousal in insomnia represents a field eminently useful and advantageous, it should be recognized that despite the extensive research to date, one cannot discern whether it is due to hyperarousal that individuals develop insomnia or vice versa, i.e., whether the activation of various biological systems is due to a clinical manifestation of insomnia *per se*. Still, there are authors who point out that probably the relationship is bidirectional.<sup>3</sup> Moreover, the evidence is still insufficient to discern whether the hyperarousal itself is constant/stable or variable/floating throughout the night (and day).<sup>31</sup> However, Riemann et al. state that "given the cyclic nature of the sleep process itself with well-known oscillations between slow-wave and REM sleep and other dynamic changes with even shorter period lengths during sleep, it seems reasonable to assume that the hypothetical hyperarousal also fluctuates, which might explain that neuroimaging methods with different sampling times may produce divergent results "(p. 26).<sup>3</sup>

In sum, in the scope of insomnia research, there are two possible processes involved: the hyperarousal and the failure to inhibit wakefulness. Note, however, that these mechanisms are not necessarily opposites, but rather complementary.<sup>8</sup> The hyperarousal hypothesis states that PI patients have significantly higher levels of general arousal than good sleepers. Although this hyperarousal is generalized, the cognitive component should be emphasized. Several PI patients refer that the presence of dysfunctional thoughts – overactivity of the cognitive system – is the most disturbing feature of insomnia.<sup>49,50</sup> Besides, one must note that this dysfunctional activity albeit very frequent at bedtime, it is also common during the daytime.<sup>3</sup> On the other hand, the failure to inhibit wakefulness process posit that as bedtime

approaches, PI patients have difficulty in their ability to relax; the typical arousal levels of the daytime wakefulness tend to remain constant, not allowing the action of neurobiological structures and neurobiochemical mediators responsible for inducing sleep.<sup>31</sup> Espie's model is clearly a perspective that fits in the process of failure in inhibiting wakefulness.

*Is there room for more theories of insomnia?*

As stated by Harvey, we also think that there are still many contributions to be made to theory and intervention in PI.<sup>37</sup> Although very satisfactory results are obtained with the techniques derived from the models generated so far, we believe that with refinement and improvement of brain imaging techniques, among others, the behavioral models will establish themselves in a more solid and consistent way. Secondly, and based on new paradigms of neuroscience, our group is very interested in studying the default mode network (DMN) in these patients. One of our tasks is to understand whether the DMN is relevant to insomnia and to enrich the existing empirically well-established insomnia models. With the help of neuroimaging techniques and developments in clinical psychology and psychiatry research, we believe that the two processes for insomnia addressed in this paper, will be both improved. We hope in the future to further understand how the simultaneous action of both processes may lead to better insomnia treatments.

#### **Acknowledgments/Disclosure:**

First author has a PhD Grant from the Portuguese Foundation for Science and Technology (FCT - Fundação para a Ciência e Tecnologia) with the following reference:

SFRH/BD/77557/2011.



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**Table 1.** An overview on insomnia models

Insomnia model	Description
Internalization of conflict's model <sup>10,11</sup>	<ul style="list-style-type: none"> <li>• A pioneer model that focused on the personality features of insomnia patients.</li> </ul>
Behavioral perspective of Spielman <sup>21</sup>	<ul style="list-style-type: none"> <li>• The behavioral theory of insomnia states that in insomnia there are three factors that one should understand: predisposing factors, precipitating factors and maintenance factors.</li> </ul>
Stimulus control model <sup>24</sup>	<ul style="list-style-type: none"> <li>• Initially conceptualized by Richard Bootzin. It assumes that there are a maladaptive conditioning between stimuli that causes arousal and stimuli that are sleep-inducing. It is the theoretical support of the most efficacious treatment for insomnia (i.e., stimulus control technique).</li> </ul>
Microanalytic model of Morin <sup>20</sup>	<ul style="list-style-type: none"> <li>• One of the most studied and well-known theories about insomnia. It originated several useful psychological assessment instruments. Although centered on maintenance factors of insomnia, this perspective suggest three levels of arousal: cognitive, emotional and physiological.</li> </ul>
Hybrid cognitive-behavioral model of Lundh and Broman <sup>27,28</sup>	<ul style="list-style-type: none"> <li>• This is a perspective which differentiates processes that interfere with sleep-related levels of cognitive, emotional and physiological arousal and processes that make the individual prone to perceive or interpret their sleep patterns in a distorted or dysfunctional way; besides, this perspective tries to include concepts derived from mindfulness approaches.</li> </ul>
Neurocognitive theory of insomnia <sup>29,30</sup>	<ul style="list-style-type: none"> <li>• The first behavioral-based model which emphasizes neurobiological variables in insomnia and operationalizes the cortical arousal concept.</li> </ul>
Espie's psychobiological inhibition model <sup>31</sup>	<ul style="list-style-type: none"> <li>• Psychobiological inhibition model posits that one of the most critical issues in insomnia is the difficulty regarding inhibit the arousal typical or normal from wakefulness period. Further, this conceptualization tries to understand the "insomnia experience" from the normal sleep parameters.</li> </ul>
The cognitive model of Harvey <sup>36,37</sup>	<ul style="list-style-type: none"> <li>• Cognitive model by Harvey is a model centered on cognitive aspects of insomnia such as underlying critical beliefs, ruminations, worries and misperception of sleep deficits.</li> </ul>
Bottom-up model of Riemann et al. <sup>3</sup>	<ul style="list-style-type: none"> <li>• This is not in fact a theory about insomnia. However, it represents an integrated and critical view emphasizing the genetic and neurobiological vulnerabilities in causing insomnia. Notwithstanding, the behavioral and psychological variables are equally considered by the authors.</li> </ul>
The instability hypothesis of REM sleep <sup>41</sup>	<ul style="list-style-type: none"> <li>• It is suggested that one of the major causes of insomnia might be an instability in REM sleep. The micro- and macro- awakenings during REM sleep in insomnia patients appear to be an important topic to study in the next years.</li> </ul>
Metacognitive model of insomnia <sup>26</sup>	<ul style="list-style-type: none"> <li>• The metacognitive model is the most recent model on insomnia. It is the first structured perspective based on mindfulness and acceptance approaches. The aim is to give a mindfulness perspective of insomnia, but in a complementary way to the classical models.</li> </ul>