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NIH INSOMNIA ABSTRACT

Psychological Models of Chronic Insomnia

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A dozen hypotheses of psychological causes of primary insomnia have been advanced. This presentation will focus on the few that have gained broad acceptance: learning models, cognitive arousal, and personality traits. Primary insomnia, which assumes the absence of a disease/disorder/substance causal agent, represents about 25 percent of the insomnia population[1] (see Figure 1). Psychological factors are also contributory to both secondary insomnia and hypnotic dependent insomnia but are competing with other classes of factors, and it is difficult to parse the relative weight of causal factors in these types of insomnia.

Note a methodological caution. Most research aspiring to demonstrate casual connections in this area have been lacking in methodological purity. Salient studies generally fall into one of the following categories: (1) uniform stimuli are presented to intact groups (insomnia present or absent) and differential responses are observed; (2) characteristics of intact groups (insomnia present or absent) are assessed and related to group membership; or (3) inferences are drawn about the cause of insomnia from the success of treatments targeting particular aspects of functioning. In these cases, classical requirements for the assertion of causal inference,[2] random assignment to groups, and controls for alternative explanations are not met.

Learning Models

One of the first psychological treatments for insomnia was stimulus control,[3] and it remains one of the most effective. An operant rationale was originally presented wherein the bedroom signaled nonsleep behaviors (S^{Δ} stimulus value) due to habitual associations between that setting and sleep incompatible behaviors, such as reading, doing paperwork, watching television, and, more generally, wake time in the bedroom.

Classical conditioning has since been invoked, hypothesizing that conditioned aversion to the bedroom obstructs sleep.[4] In this model, transient insomnia introduced by intrusive events, as exemplified by bereavement or job stress, converts a neutral stimulus, the bedroom, into a conditioned stimulus evoking negative emotions. Such emotions promote deteriorating sleep that, in turn, escalates negative emotions, and this circular process sustains the conditioned stimulus properties of the bedroom.

There is indirect research to support learning models, such as the reverse first night effect in the laboratory occurring in people with insomnia. Credence is bolstered by the face validity of learning models and the success of stimulus control treatment, but there is little direct basic research validating this theory of insomnia.

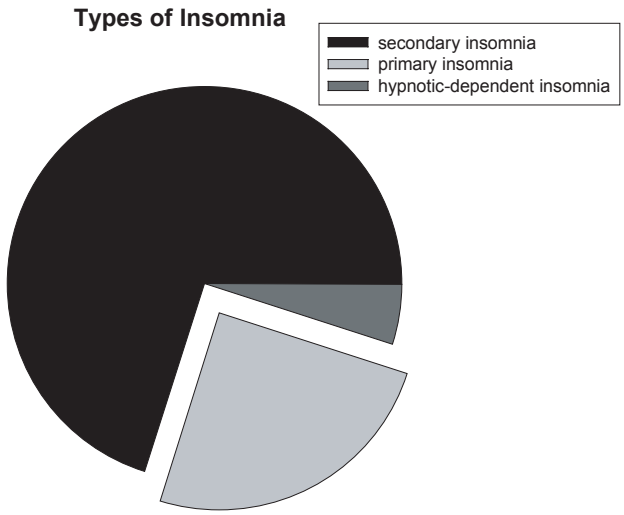

Figure 1—Prevalence of Types of Insomnia

Cognitive Arousal

Falling asleep requires quiescent somatic and cognitive processes, and active thinking at bedtime is hypothesized to be mutually exclusive of sleep. Obstructive thoughts may be laden with sleep concerns or worry about matters unrelated to sleep, may focus on temporally distant or current content, and may assume a ruminative style. Comprehensive models of cognitive causation in insomnia also recognize maladaptive sleep expectations and selective attention to negative cues.[5]

There is more evidence to support the cognitive model than any other psychological cause of insomnia. By their own account, people with insomnia strongly endorse the cognitive model above all others,[6] they are more reactive to contrived threats at bedtime than normal sleepers,[7] and their presleep images and cognitions are more negatively toned than normal sleepers.[8,9] Among the few studies that used an analogue model and randomly assigned normal sleepers to conditions, poor sleep was more likely to emerge in those individuals exposed to contrived challenges.[10]

Personality Traits

Trait personality theory could explain disturbed sleep,[11–13] and individuals with hypomanic/obsessive/anxious/depressed sub-

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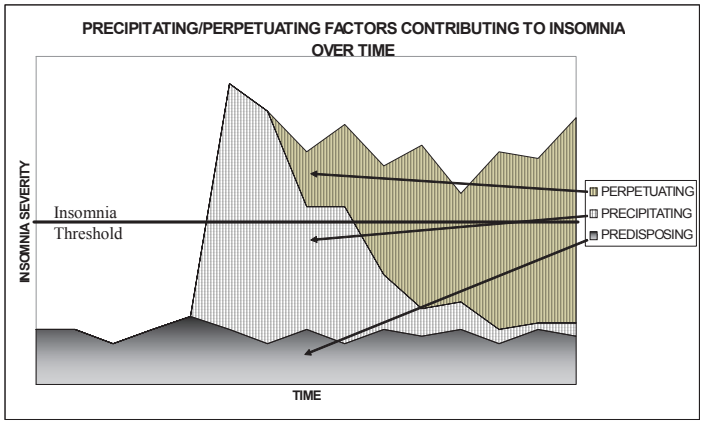

Figure 2—Spielman 3P Model

clinical tendencies could be more vulnerable to irritants provoking somatic/cognitive arousal and resulting insomnia. Depending on the strength of the disposition, such individuals may experience either chronic or intermittent insomnia when their vulnerability is stirred by environmental pressure (a diathesis-stress model).

This view has been tested mainly by correlational studies that have attempted to measure the strength of association between assessed personality traits and insomnia presence. In general, these efforts have obtained weak to moderate results.

Hybrid Model

Spielman[14] and his colleagues have advanced what has become known as the 3P model (see Figure 2), which incorporates aspects of all three models. Stable predispositions (e.g., anxiety proneness), varying precipitants (e.g., job stress), and varying perpetuating factors (e.g., conditioned aversion to the bedroom) conspire to induce and maintain insomnia. Insomnia appears when the summative influence of these factors surpasses the individual's threshold coping level. As precipitants fade, perpetuating factors may swell, and the insomnia may survive long after the demise of the precipitants. Partial support for this heuristic comes from studies showing insomnia covaries with perceived stress,[15] but overall, this model remains unvalidated.

Conclusion

Multiple psychological (and other) factors likely contribute to insomnia, the constellation of causal factors likely varies between individuals as well as within individuals over the insomnia course, and the weight of evidence supports a cognitive etiology.

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