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The Effects of Presleep Stress on Sleep-Onset Insomnia

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This study examined the effects of presleep cognitive stress on insomniac and noninsomniac subjects. Ten sleep-onset insomniacs and 11 noninsomniacs spent five nights in a sleep laboratory. They slept undisturbed on the first three nights but were exposed to brief cognitive stressors before sleep-onset on the fourth and fifth nights. Significant between-group differences were found in their responses to the cognitive stressors on self-report but not objective measures of sleep-onset latency. Noninsomniac subjects evidenced an increase in sleep-onset latency and insomniac subjects evidenced a decrease in sleep-onset latency on stress nights. Insomniac subjects demonstrated a significantly higher mean heart-rate response before and after the stressors. A variable expressing the relationship between objective and subjective measures of sleep-onset latency (pseudoidiopathic dimension) for each subject was not significantly related to the subjects' response to stress. The results of this study were inconsistent with the hypothesized role of presleep stress in sleep-onset insomnia but were consistent with other studies in indicating higher levels of physiological arousal for insomniac and for noninsomniac subjects. The implications of these findings for a cognitive theory of insomnia etiology are discussed.

A number of investigators (Feinberg, Braun, Koreski, & Gottlieb 1969; Greenberg, Pearlman & Gampel, 1972; Koulack, 1970; McGhie, 1966; Webb & Cartwright, 1978) have suggested that environmental or psychological stress might affect sleeping patterns and/or have etiological significance for insomnia. Within this model stressors are presumed to precipitate physiological or cognitive events that, in turn, inhibit sleep onset and sleep maintenance.

Indirect evidence that stress might play an etiological role in insomnia has come from several sources. A number of investigators (Baekeland & Hoy, 1971; Browman & Tepas, 1976; Cohen & Cox, 1975; De Koninck & Koulack, 1975; Greenberg et al., 1972; Hauri, 1969) have noted a positive association between the occurrence of presleep stress and sleep-onset latency in normal sleepers. In addition, Johns, Gay, Masterson, and Bruce (1971) found higher levels of urinary-free 11-hydroxy-corticosteroids, an index of adrenocortical activity, for insomniacs than for noninsomniacs. Several authors (Coursey, Buchsbaum & Frankel, 1975; Haynes, Follingstad, & McGowan, 1974; Monroe, 1967) reported higher measures of physiological arousal for insomniacs than noninsomniacs. Other studies have reported higher indexes of state or trait anxiety (Coursey et al., 1975; Haynes et al., 1974; Johns et al., 1971) and more fearful, anxious, or worrisome presleep cognitions (Coursey et al., 1975; Kamens, 1980) for insomniacs than for noninsomniacs.

Although indirect, these findings suggest that stress may be associated with various sleep inhibiting physiological and cognitive events and that it may play a significant role in the etiology of sleep-onset insomnia. However, we could not locate any study that directly examined the role of presleep stress in sleep-onset insomnia.

One factor possibly accounting for individual differences in response to stress is the discrepancy between polygraphic and self-reported measures of sleep-onset latency (Borkovec, 1979; Cohen & Cox, 1975; Frankel, Coursey, Buchbinder, & Snyder, 1976; Haynes et al., Note 1; Kamens, 1980; Lewis, 1969; Youkilis & Bootzin, 1981). Insomniacs may be classified as "idiopathic" (psychophysiologic) or "pseudo" (subjective) (Borkovec, 1979; Kamens, 1980; Youkilis & Bootzin,

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1981) as a function of the degree of agreement between these two measures. As Borkovec (1979) suggested, this dimension might have implications for the etiology and treatment of insomnia in that pseudoinsomnia and idiopathic insomnia might be a result of different causative factors.

This study was designed to investigate: (a) the relationship between presleep cognitive stress and sleep-onset latency, (b) the differential responses by insomniacs and noninsomniacs to presleep cognitive stress, and (c) the efficacy of the pseudo–idiopathic dimension in accounting for response to presleep cognitive stress.

Method

Subjects

Subjects were 10 sleep-onset insomniac (8 female, 2 male) and 11 noninsomniac (9 female, 2 male) students from Southern Illinois University recruited via campus bulletins and announcements. Diagnostic criteria for insomniacs were selected to be consistent with a "severe" classification in the behavioral treatment literature (sleeping difficulties for longer than 2 years, average sleep-onset latency greater than 60 minutes, sleep-onset difficulties more than four times per week). Noninsomniac subjects reported no history of sleeping difficulties, that it took them an average of 10 min. or less to fall asleep, and that they rarely or never experienced difficulty falling asleep. Subjects were homogenous in age (18–21 years) and all were taking no medication.

Diagnostic Reliability

Diagnostic information was acquired on three separate occasions: on a brief questionnaire, during a phone interview, and during a lengthy personal interview.[1] Any potential subject whose responses deviated from the established criteria on any of these occasions was excluded from the study.

Procedure

Following an initial interview arrangements were made for subjects to spend 5 nights (Sunday through Thursday) in the sleep research laboratory. Subjects arrived 1 hour prior to their normal bedtime each night. Data for the first night were not included in any statistical analyses.

On nights one, two, and three, subjects were allowed to fall asleep uninterrupted. On nights four and five, 5 min. after the lights were turned off, each subject was presented, on audiotape and in counterbalanced order, one of two 7.5 min. cognitive stressors. Each stressor was composed of a series of moderately difficult mental arithmetic problems (e.g., "count backwards from 347 by 18s as quickly as you can") presented every 15 sec. Subjects were informed that the purpose of these exercises was to assess the relationship between thinking abilities and sleep patterns.

Sleep Laboratory

The sleep laboratory consisted of a control room and three adjacent 2.5 × 3 m temperature-controlled, sound-attenuated subject rooms, each furnished with a bed, night table, and intercom. Electroencephalogram (EEG), electrooculogram (EOG), frontal electromyogram (EMG), and heart rate were recorded using Grass model 7D polygraphs and isolators. Standard placement, attachment, conductive media, and recording procedures were used (Rechtshaffen & Kales, 1968; Venables & Martin, 1967). Resistance readings of 10,000 Ω or less were required prior to recording. For EEG the active electrode was place on either the C3 or C4 site with an earclip electrode on the earlobe site opposite the active electrode.

Dependent Measures

Objective sleep-onset latency (OSOL). This measure was based on the beginning of Stage 2 sleep without an indication of awakening, or a return to Stage 1 within 3 min. (Rechtshaffen & Kales, 1968). Objective sleep-onset latency was the time between "lights off" and Stage 2 sleep for the three nonstress nights and the time between the termination of the stressor and Stage 2 sleep for the two stress nights. Each polygraph protocol was independently scored by at least two researchers who were blind to the subjects' group membership. Disagreements were resolved with the help of a National Institute of Mental Health consultant. Mean Pearson correlations between scorers was .97. On awakening each morning, subjects completed a brief questionnaire that included their subjective estimate of the previous night's sleep-onset latency (SSOL).

Results[2]

Objective and subjective sleep-onset latencies. Figure 1 illustrates the mean objective and sub-

[1] Copies of this interview form are available from the first author.

[2] Degrees of freedom vary slightly among the following analyses because of missing data on some measures (e.g., transient polygraph malfunctions).

jective sleep-onset latencies across the two prestress and two stress nights for insomniac and noninsomniac groups.

To examine differential effects of the cognitive stressors on the insomniac and noninsomniac groups, Group × Sex × Night repeated measures analyses of variance were conducted on objective and subjective sleep-onset latency measures. These analyses revealed: (a) significant differences between groups on objective sleep-onset latency, $F(1, 17) = 10.85, p < .01$, and on subjective sleep-onset latency, $F(1, 17) = 54.01, p < .01$, collapsing across nights; (b) a significant interaction between group and night, $F(3, 66) = 4.37, p < .05$, on subjective sleep-onset latency; (c) an interaction effect approaching significance ($p < .07$) between group and night on objective sleep-onset latency; and (d) no significant main effect for night on either dependent measure.

The analyses of primary interest were the interactions between group and night that indicated that insomniac and noninsomniac groups responded significantly differently to the stressors on subjective but not on objective measures of sleep-onset latency. Post hoc Duncan analyses on subjective measures of sleep-onset latency revealed that stress was associated with a significant reduction in self-reported sleep-onset latencies for insomniac subjects, and a significant increase in self-reported sleep-onset latencies for noninsomniac subjects.

Heart rate. Differences between insomniac and noninsomniac groups in response to cognitive stress on measures of heart rate were examined via repeated measures analyses of variance (Group × Time) on pre- and poststress heart rates (four 30-sec periods prior to and five 30-sec periods following the cognitive stressors) and heart rates during the stressor (averaged every 30 sec).

Repeated measures analyses of variance on the two sets of data revealed: (a) a significant difference between groups on pre- and poststress mean

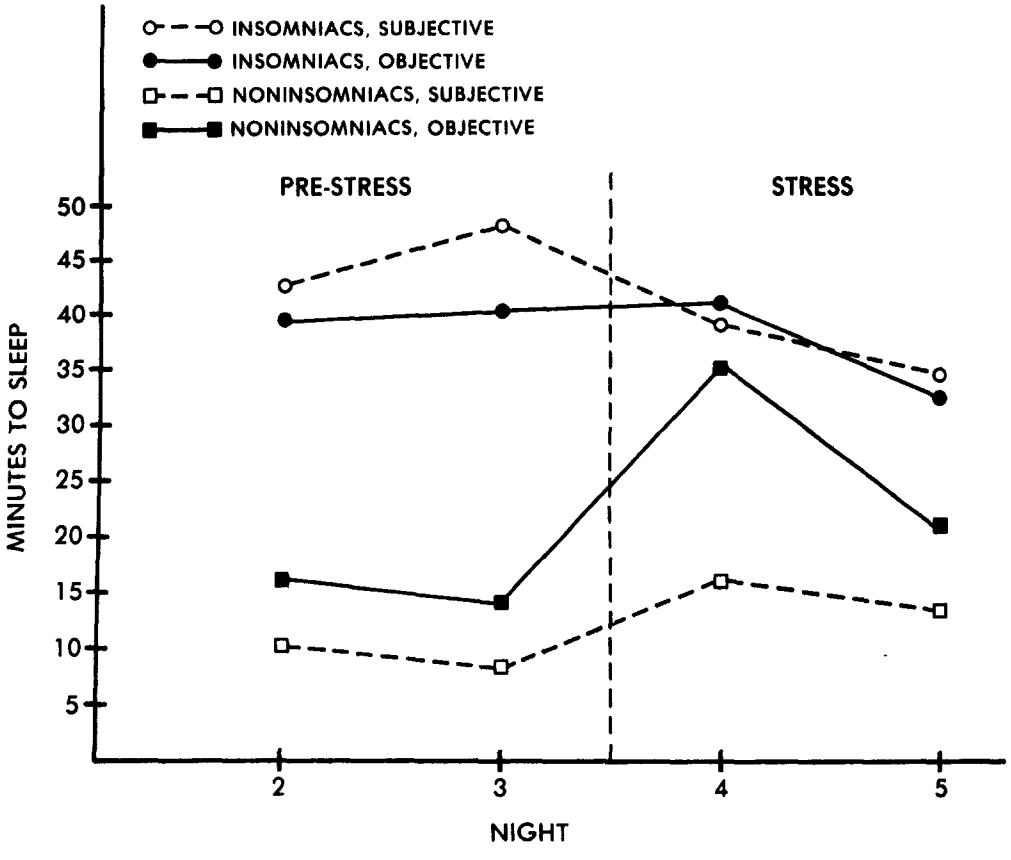

Figure 1. Mean objective and subjective sleep-onset latencies on prestress and stress nights for insomniacs and noninsomniacs.

heart rates, $F(1, 18) = 6.55$, $p < .05$ (insomniac subjects evidenced a mean heart rate 4.6 beats per minute higher than that of noninsomniacs); (b) no significant between-group differences on mean heart rates during the stressors; (c) no significant interaction between group and time on pre- and poststress mean heart rates; (d) significant, $F(8, 143) = 3.48$, $p < .05$, change across time on pre- and poststress heart rate, collapsing across groups; (e) a significant change in heart rate over time during the cognitive stressors, $F(14, 259) = 7.24$, $p < .001$, collapsing across groups; and (f) a significant, $F(1, 34) = 7.23$, $p < .05$, Group × Night interaction during the stressors, with noninsomniac subjects demonstrating a significant increase in heart rate during the second stress night.

Pseudo–idiopathic dimension. To examine the mediational effect on response to stress of the pseudo–idiopathic dimension, a ratio (OSOL/SSOL)[3] was calculated for each subject that represented the relationship between mean objective and mean subjective sleep-onset latencies. By using a ratio rather than arbitrarily dichotomizing subjects into idiopathic and pseudoinsomnia categories, it was possible to generate a more powerful estimate of the relationship among this variable and the sleep-onset variables. The relationship between this ratio and pre- to poststress change in objective and subjective sleep-onset latency was nonsignificant ($rs < .19$) for all subjects combined and for insomniac subjects separately, suggesting that subjects did not significantly differ in their responses to the cognitive stressors as a function of the relationship between objective and subjective measures of sleep-onset latency.

Comparisons with nonstressed subjects. Although the original function of the study (to compare insomniac and noninsomniac subjects' response to stress) did not necessitate the inclusion of nonstressed control groups, it was felt that comparison of the insomniac and noninsomniac groups who received the cognitive stressors to matched groups who did not receive the cognitive stressors would facilitate interpretation of the unexpected effects of stress on the insomniac group. Therefore, the objective and subjective sleep-onset latencies on nights two through five of insomniac and noninsomniac groups who received the cognitive stressors were compared to those of 8 insomniac and 10 noninsomniac subjects who had spent five nights in the same laboratory under identical conditions but without the stressors (all subjects were from the same pool and did not significantly differ on any measured characteristic). All recruitment and interview procedures, diagnostic criteria, dependent measures, and experimental procedures were identical.

Repeated measures analyses of variance on objective and subjective estimates of sleep-onset latency between the two insomniac groups and the two noninsomniac groups revealed significant Group × Night interactions on OSOL—insomniac subjects, $F(3, 25) = 3.43$, $p < .05$; noninsomniac subjects, $F(3, 33) = 3.50$, $p < .05$)—and marginally significant interactions between group and night on measures of SSOL—insomniac subjects, $F(3, 25) = 2.75$, $p < .07$; noninsomniac subjects, $F(3, 33) = 2.60$, $p < .07$. These analyses suggest that the stressors had a significant, although opposite, effect on OSOL for both groups, and an effect approaching significance but also opposite on SSOL for both groups.

Discussion

As expected, the noninsomniac subjects evidenced a significant increase in both objective and subjective measures of sleep-onset latency on the cognitive stress nights. These results were consistent with those of Baekeland and Hoy (1971), Browman and Tepas (1976), Cohen and Cox (1975), and others in suggesting that presleep stressors might serve to increase significantly sleep-onset latencies for subjects without self-defined sleeping difficulties. Insomniacs, however, demonstrated a decrease in subjective and objective sleep-onset latency on cognitive stress compared to no-stress nights. Although unexpected, the decrement in sleep-onset latencies for insomniacs is consistent with attribution and cognitive theories of sleep-onset insomnia (Kamens, 1980; Youkilis & Bootzin, 1981). If ruminative cognitive activity, sleep-related thoughts, or attributions of internal causality for sleeping difficulties serve etiological functions in sleep-onset insomnia, disruption of those cognitive events will result in reduced sleep-onset latencies. In support of this hypothesis, treatments for insomnia involving modification of cognitions and attributions have been shown to be associated with reductions in objective and subjective measures of sleep-onset latencies (Youkilis & Bootzin, 1981). Additional research on the comparative effects of cognitive and noncognitive stressful and nonstressful presleep stimuli will help identify mediating factors and mechanisms.

These results also suggest that cognitive stressors affect insomniacs' perception of sleep-onset

[3] Pilot research had suggested that although a number of ratios express the relationship between objective and subjective sleep-onset latencies, the ratio used was the most satisfactory in terms of its arithmetic properties and conceptual validity.

latency more strongly than their physiological indexes of sleep-onset latency. A number of investigators (Borkovec, Grayson, O'Brien, & Weerts, 1979; Frankel et al., 1976; Haynes, Adams, West, & Kamens, Note 1; Lewis, 1969; Monroe, 1967) have noted greater reactivity of subjective than of objective sleep-onset-latency measures to environmental and cognitive treatment interventions. Thus, the current findings of greater responsivity to stress on subjective measures is consistent with and extends the literature emphasizing the importance of subjective factors in sleep-onset insomnia.

The insomniac, compared to noninsomniac subjects, demonstrated a significantly higher mean heart rate before and after the stressors but did not respond significantly differently than the noninsomniac subjects to the stressors. The elevated mean heart rates for the insomniacs compared to noninsomniacs is consistent with the results of other investigators (Haynes et al., 1974; Johns et al., 1971; Monroe, 1967) who have noted elevated indexes of physiological activation for poor compared to good sleepers. Both groups evidenced an initial increase followed by a gradual reduction in heart rate in response to the stressors. The apparently stronger effect of stress on the heart rates of noninsomniacs did not achieve statistical significance.

The results of this study, therefore, are not consistent with the hypothesis that presleep cognitive stress contributes to the longer sleep-onset latencies of insomniacs. They do support previous findings that presleep stress may be associated with elevated indexes of physiological arousal and elevated objective and subjective sleep-onset latencies for noninsomniacs.

An evaluation of the predictive efficacy of the objective/subjective sleep-onset-latency ratio failed to reveal any significant predictive relationships. Naturally, it is impossible to exclude the possibility that the objective/subjective ratio may be significantly related to other variables or mediate the effect of other events.

It is important to emphasize the situational and population analogue-nature of the study. The samples, although representing the upper and lower 2% of the available population, did not include clinical cases. Although insomniac subjects in this study were representative of subjects in the behavioral treatment literature (Youkilis & Bootzin, 1981) and considered their sleep disturbance problematic, none had sought treatment for their sleep difficulties, and the clinical versus nonclinical nature of a subject population as well as the analogue nature of the stressor may affect the identification of causative variables.

Reference Note

1. Haynes, S. N., Adams, A. E., West, W., & Kamens, L. *Stimulus control conceptualization of sleep-onset insomnia: A reappraisal.* Unpublished manuscript, 1981. (Available from Stephen N. Haynes, Department of Psychology, Southern Illinois University at Carbondale, Carbondale, Illinois 62901.)

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