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Moderators and Mediators of the Relationship Between Stress and Insomnia: Stressor Chronicity, Cognitive Intrusion, and Coping

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Study Objectives: To assess moderators, such as stressor chronicity, and mediators, including stress response in the form of cognitive intrusion and coping behavior, of the prospective association between naturalistic stress and incident insomnia.
Design: Longitudinal.
Setting: Epidemiological.
Participants: A community-based sample of good sleepers (n = 2,892) with no lifetime history of insomnia.
Interventions: None.
Measurements and Results: Participants reported the number of stressful events they had encountered at baseline, as well as the perceived severity and chronicity of each event. Similarly, volitional stress responses such as coping, as well as more involuntary responses such as cognitive intrusion were assayed for each stressor. Follow-up assessment 1 y hence revealed an insomnia incidence rate of 9.1%. Stress exposure was a significant predictor of insomnia onset, such that the odds of developing insomnia increased by 19% for every additional stressor. Chronicity significantly moderated this relationship, such that the likelihood of developing insomnia as a result of stress exposure increased as a function of chronicity. Cognitive intrusion significantly mediated the association between stress exposure and insomnia. Finally, three specific coping behaviors also acted as mediators: behavioral disengagement, distraction, and substance use.
Conclusions: Most studies characterize the relationship between stress exposure and insomnia as a simple dose-response phenomenon. However, our data suggest that certain stressor characteristics significantly moderate this association. Stress response in the form of cognitive intrusion and specific maladaptive coping behaviors mediate the effects of stress exposure. These findings highlight the need for a multidimensional approach to stress assessment in future research and clinical practice.
Keywords: coping, insomnia, mediator, moderator, stress
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INTRODUCTION

Nearly all current conceptualizations of insomnia identify stress exposure, typically operationalized as life changes or events, as a key precipitating factor.[1-3] However, most of the empirical support for this hypothesis has emerged from studies on the association between stress exposure and acute sleep disturbance.[1] In contrast with insomnia, sleep disturbance is a nonspecific report or polysomnographic finding of difficulty sleeping (e.g., long sleep latency, frequent awakenings) that may not be associated with daytime impairment or distress. Further, sleep disturbance is a ubiquitous phenomenon affecting nearly half of the US population.[4] However, the syndrome of insomnia has a relatively lower prevalence, ranging from 4% to 22% as a function of diagnostic criteria.[5] Thus, focusing on transient sleep disturbance alone may not yield adequate insight into the causal mechanisms complicit in the association between stress exposure and the more chronic and debilitating syndrome of insomnia. Few studies have explored the role of stress exposure in the development of an insomnia disorder. Importantly, the insomnia literature has yet to discern the moderating influences of stressor characteristics, such as perceived severity and chronicity, or the mediating influences of individual responses to stress.

A number of early studies suggest that individuals with insomnia endorse a significantly higher number of stressors than do healthy controls.[6,7] However, this traditional conceptualization of stress as an objective stimulus has given way to a more nuanced view that incorporates both the stress stimulus as well as its cognitive appraisal.[8] Specifically, research in various disorders including depression and alcohol abuse has shown that the perceived severity of incident stressors is an independent predictor of pathology.[9-11] Thus, both number of stressful events, referred to as stress exposure for the remainder of this report, as well as perceived severity may play a causal role in insomnia. However, research on the relative significance of stress exposure and severity has yielded inconsistent findings. In a cross-sectional comparison of older adults with and without insomnia, Friedman et al. found that both groups reported similar levels of stress exposure and severity.[12] Conversely, in two recent studies, participants with insomnia reported significantly higher stress exposure as well as greater severity than good sleepers.[23,24] A potential explanation of these discrepancies is that the perceived severity of a stressor may either diminish or strengthen its potency in triggering insomnia. In other words, severity may moderate the effects of stress exposure on risk for insomnia. However, no study has investigated this idea.

Chronicity, conceptualized here as the duration of stress exposure, is another important stressor characteristic that clinical research in both insomnia and psychopathology in general has largely overlooked.[13] The chronicity of stress may represent

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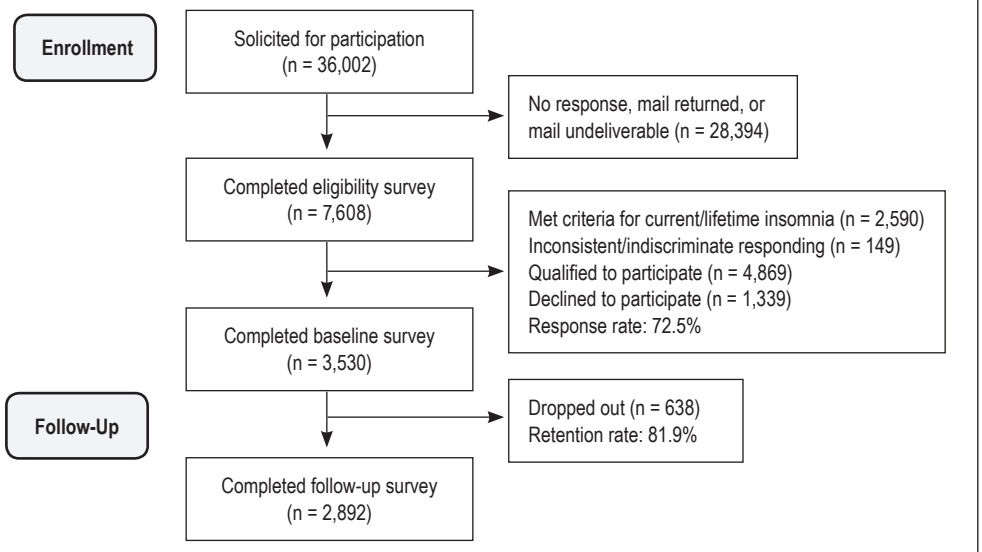

Figure 1—Flow diagram depicting flow of participants through the study.

an important source of variance in the association between stress exposure and insomnia. For instance, in their model of acute insomnia, Ellis et al. proposed that insomnia occurs when an individual's stress-response threshold is exceeded, which, in turn, is influenced by the chronicity of encountered stressors.[14] In other words, chronicity may moderate the effects of stress exposure. However, the best source of empirical support on this topic has emerged from disparate findings on the effects of acute and chronic stress on sleep disturbance.[14-17] Thus, research has yet to provide a direct comparison between the effects of chronic and transient stressors on risk for insomnia.

Though severity and chronicity together offer a richer view of the stress construct than exposure alone, the mechanisms by which stress manifests in pathology cannot be overlooked. Several influential theories highlight the significance of the individual's response to stress as a distinct and salient phenomenon. Commonly cited stress responses in the literature include autonomic nervous system (ANS) arousal, cognitive intrusion, and coping behaviors undertaken to meet the demands of the stressor.[18] Although ANS indices of stress response have been studied extensively in the context of both sleep disturbance and insomnia,[14-17] cognitive intrusion remains relatively understudied. Conceptualized as a recurrent, perseverative activation of cognitive representations of psychological stressors, intrusion has been proposed to play an important role in the development of insomnia.[19] Experimental data suggest that the cognitive load exerted by intrusion is on par with an executive/effortful attentional task,[20] a finding that may explain its wake-promoting effects. A growing body of research also suggests that intrusion may be complicit in the sleep disturbance observed in insomnia.[21,22] However, to the best of our knowledge, no study has examined intrusion as a prospective risk factor for insomnia.

Similarly, few studies have systematically examined coping behaviors in the context of insomnia risk.[10,18] In a recent prospective study, individuals with insomnia reported significantly higher trait levels of maladaptive coping than did good sleepers.[23] Further, although scores on the coping scale did not exert a main effect on sleep disturbance, they were significantly associated with stress impact and cognitive arousal in an omnibus path-analysis model predicting sleep disturbance. Although this study makes an important contribution by calling attention to the stress-coping diathesis in insomnia, the fact that coping was only measured at baseline precluded any mediational analyses between coping and stress. A second limitation is that trait-level measurements of general coping styles fail to capture the significant within-person heterogeneity in the relationship between stressors and coping.[10,11] Notably, a more recent longitudinal study of the incidence of insomnia among good sleepers found no associations between a trait-measure of coping and risk for insomnia.[24] Experience-sampling techniques suggest that stressors vary in the extent of coping they elicit from an affected individual, which, in turn, influences the risk for pathology.[25,26] Thus, a more pressing question along with individuals' general coping styles is whether and to what extent they summoned that coping style in response to a given stressor.

In summary, although stress exposure is a well-established trigger of sleep disturbance, its association with insomnia disorder is less clear. Some of the gaps in our knowledge may be filled by examining the moderating influences of stressor characteristics, such as chronicity and severity, and the mediating effects of intrusion and coping. A longitudinal study among individuals without a history of insomnia is warranted to establish a reliable temporal association between these risk factors and the onset of insomnia. The current study offers results from a year-long prospective analysis of stress and coping as predictors of new onset insomnia in a large sample of good sleepers. Participants not only reported levels of stress exposure, but also provided indices of perceived chronicity and intrusion for each stressor. Further, they indicated the nature and extent of coping in which they engaged in response to each specific stressor. This strategy allowed us to capture within-person variability in coping, in addition to establishing the temporal precedence of stress in relation to coping. We hypothesize that individuals with higher levels of stress exposure will be at a greater risk for insomnia incidence, that chronicity and severity will moderate the effects of stress exposure, and that intrusion and coping will mediate the effects of stress exposure on risk for insomnia.

METHOD

Participants

Our data are derived from the Evolution of Pathways to Insomnia Cohort (EPIC) study, a 3-y National Institute of

Mental Health-funded prospective investigation of a large sample from southeastern Michigan. Here, we report data from the first 2 y of the EPIC study. For the initial assessment (baseline), a randomly generated list of individuals (n = 36,002) from a major statewide HMO database received invitation letters to participate in the study. Of those who received these initial mailings, 7,608 completed a Web-delivered eligibility survey that assessed for history of insomnia; see Figure 1 for a detailed description of the flow of participants through the study.

At the end of this survey, participants who did not meet Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)-based diagnostic criteria[27] for current or a lifetime history of insomnia disorder (n = 4,869) were invited to participate in the present study, of whom a total 1,339 declined to participate (response rate = 72.5%). Finally, of the 3,530 participants who completed the baseline assessment, 2,892 participants completed a follow-up assessment 1 y later (retention rate: 81.9%). This final sample was predominantly white (65.3%) and female (59.3%), with a mean age of 47.9 y (Table 1). The distributions of various demographic characteristics including age, sex, and marital status in our sample were comparable to 2010 census data for the area.[28]

Table 1—Sample descriptive statistics

	Sample (n = 2,892)	Tricounty (n = 281,421,906)	United States Census (n = 308,745,538)
	%	%	%
Sex (women)	59.3	51.7	50.8
Race			
White	65.3	67.3	72.4
African American	24.4	25.2	12.6
Asian	4.8	3.5	4.8
Other	3.9	3.9	10.2
Marital status [a]			
Married	65.7	46.4	49.7
Single/divorced/separated	34.3	53.6	50.3
Employment status [a]			
Unemployed	6.2	13.2	8.7
Income [b]			
< $10k	4.9	8.3	7.1
$10k–$14,999	1.8	5.4	5.4
$15k–$24,999	3.9	10.8	10.6
$25k–$34,999	7.7	10.4	10.4
$35k–$49,999	14	13.5	13.8
$50k–$74,999	24.2	17.6	18.3
$75k–$99,999	16.0	12.3	12.4
$100k–$149,999	17.9	13.0	12.7
$150k–$199,999	5.4	4.7	4.7
> $200k	4.0	3.9	4.5

	Mean (SD)	Median	Minimum	Maximum
Age	47.9 (13.3)	51	19	70
Stress exposure (number of events)	2.2 (2.0)	2	0	14
Chronicity/duration (months)	9.4 (11.2)	5	0	37
Perceived severity	14.4 (12.5)	10	0	112
Stress-related cognitive intrusion	55.8 (60.6)	37	0	419

Data for United States and the Tricounty area are from the 2010 census. [a] United States census and Tricounty census data from these categories included individuals aged 15-17 y, which accounted for the increased percentages of nonmarried and unemployed in these categories. [b] Data from this category are presented in percentage of "households." SD, standard deviation.

Procedure

To prospectively assess the relationship between stress, coping, and insomnia incidence, data were collected in two waves 1 y apart. Data collection for baseline measures began after participants completed the eligibility survey, and was accomplished using Web-administered electronic questionnaires. Study staff sent email reminders to each participant 1 mo prior to scheduled follow-up assessments. Each assessment took approximately 30 min to complete, and preliminary analyses revealed that nearly all participants responded appropriately to item content (96%).

Measures

Stress Exposure: Number of Events

We assessed stress exposure based on the revised Social Readjustment Rating Scale (SRRS-R), an empirically validated inventory of 51 stressful life events commonly reported by US samples.[29] The SRRS-R includes stressful events across various domains, such as death and dying (e.g., death of a spouse), healthcare issues (e.g., major injury or illness), financial/economic issues (e.g., experiencing major financial problems or difficulties, foreclosure on loan/mortgage), family (e.g., divorce, infidelity), and crime/criminal justice (e.g., being a victim of a crime). The SRRS-R was recently normed in a nationally representative sample of more than 3,000 adults,[30] and is widely recognized as one of the most common stress measurement instruments.[31] In the current study, participants reported whether or not they had experienced a particular life event in the past year. The total number of endorsed events served as our operationalization of stress exposure.

Stressor Characteristics: Severity

For each endorsed stressful event on the SRRS-R, participants also reported perceived severity ("how would you rate

this overall as a stressor in your life?") on a scale from 0 (not at all stressful) to 10 (highly stressful). Consistent with techniques used by previous reports on stress and insomnia, the sum total of severity ratings comprised the stress severity score.[23]

Stressor Characteristics: Chronicity

Similarly, participants reported the duration ("for how many months has this event been an ongoing stressor in your life?") of every stressor endorsed on the SRRS-R. The arithmetic mean of the scores for each participant served as a measure of overall chronicity.

Stress-Response: Cognitive Intrusion

The impact of events scale (IES) is a 15-item questionnaire used to assess the psychological impact of stressful events along two dimensions: intrusion and avoidance.[32] The current study used the eight-item intrusion subscale of the IES, which measures the presence and pervasiveness of recurrent, intrusive ideation (e.g., "I thought about it when I didn't meant to"; "other things kept making me think about it") in response to a stressor on a four-point Likert type scale. Question stems were modified to assess levels of intrusion in the "7 days following the event." Validation studies report good internal consistency (Cronbach $\alpha = 0.86$) and excellent test-retest reliability ($r = 0.94$) for the intrusion subscale of the IES.[33]

Stress-Response: Coping

To assess levels of coping, participants completed the Brief COPE scale in response to each stressor endorsed on the SRRS-R.[34] A shortened version of the original instrument, the Brief COPE is a 26-item multifactorial questionnaire designed to assess levels of engagement in various coping techniques. This version of the scale is composed of a number of subscales, each with a distinct conceptual focus. Internal consistencies of subscales range from poor (acceptance: Cronbach $\alpha = 0.55$) to excellent (religion: Cronbach $\alpha = 0.89$; substance use: Cronbach $\alpha = 0.97$) in psychometric studies.[35] In the current study, the Brief COPE specifically referenced coping strategies in which participants engaged, 'during the 7 days following the event.'

Insomnia

DSM-IV based diagnoses of insomnia disorder were established using the following questions: "have you experienced difficulty falling asleep?"; "have you experienced difficulty staying asleep?"; "have you experienced difficulty with nonrefreshing sleep?" To earn a diagnosis, each participant had to report experiencing one or more of the aforementioned symptoms for at least 3 nights/w for a duration of 1 mo or longer. Further, they had to endorse daytime impairment or distress as measured by the following question: "to what extent do you consider your sleep problems to interfere with your daily functioning?" Responses were coded on a four-point Likert type scale ranging from 0 (not at all) to 4 (very much), such that participants who reported a score of 2 (somewhat) or higher received the diagnosis.

Data Analysis

Logistic regression analysis with maximum likelihood estimation (MLE) was used to assess risk for insomnia. To test the assumptions of logistic regression, all continuous independent variables (IV) were examined for collinearity based on bivariate correlations as well as the standard errors of parameter estimates.[36] Similarly, the large sample size (n = 2,892) and proportion of positive cases (n = 262; > 10 cases for each estimated parameter, including the intercept) afforded sufficient variance for MLE.

For all mediation analyses, we followed steps outlined by Fairchild and MacKinnon.[37] Specifically, three separate regression analyses were conducted: the dependent variable (DV) was regressed on the IV (Equation 1); the mediator (M) was regressed on the IV (Equation 2); and the DV was regressed on the M, while controlling for the IV (Equation 3),

$$\mathrm{DV} = i_1 + c(\mathrm{IV}) + e_1 \quad (1)$$

$$\mathrm{M} = i_2 + a(\mathrm{IV}) + e_2 \quad (2)$$

$$\mathrm{DV} = i_3 + c'(\mathrm{IV}) + b(\mathrm{M}) + e_3 \quad (3)$$

where c represents the relation between the IV and the DV, a denotes the association between the IV and the M, c' represents the relation between the IV and the DV adjusted for the effect of the mediator on the DV, b represents the relation between the DV and the M adjusted for the effects of the IV, e_1, e_2, and e_3 denote unexplained variability, and the intercepts are i_1, i_2, and i_3. The product of the a and b parameter estimates, ab, represents the mediated effect.

Equations 1 and 3 were estimated using logistic regression, and Equation 2 was estimated using ordinary least squares (OLS) regression. Given that the residual variance of the DV in logistic regression is constrained to $\pi^2/3$, the methods for calculating the mediated effect, ab must account for the incompatibility in the scales for the a and b parameter estimates. Hence, both parameters were standardized as follows:

$$a_z = a\,\frac{\sigma \mathrm{IV}}{\sigma \mathrm{M}}$$

$$b_z = b\,\frac{\sigma \mathrm{M}}{\sigma \mathrm{DV}}$$

where σ denotes standard deviation, and a_z and b_z represent standardized parameter estimates. Specifically, we multiplied both parameter estimates (Equations 2 and 3) with the ratio of the standard deviations of the predictor and the outcome, so that they would be on the same scale (see Mackinnon[38] for a more detailed discussion on this procedure). Next, the variance of the IV, DV, and M were calculated using equations derived by MacKinnon and Dwyer[39]:

$$\sigma^2\,\mathrm{DV} = (c^2 \times \sigma^2\,\mathrm{IV}) + \frac{\pi^2}{3}$$

$$\sigma^2\,\mathrm{M} = (a^2 \times \sigma^2\,\mathrm{IV}) + \frac{\pi^2}{3}$$

$$\sigma^2\,\mathrm{DV} = (c'^2 \times \sigma^2\,\mathrm{IV}) + (b^2 \times \sigma^2\,\mathrm{IV}) + (2 \times b \times c' \times cov(\mathrm{IV},\mathrm{M})) + \frac{\pi^2}{3}$$

where σ^2 denotes variance, and cov denotes covariance. Finally, the standard errors (SE) for the parameter estimates were calculated using the following equations:

$$SE(a_z) = SE(a) \times \frac{\sigma IV}{\sigma M}$$

$$SE(b_z) = SE(b) \times b \frac{\sigma M}{\sigma DV}$$

With regard to confidence intervals and significance testing, traditional methods are relatively underpowered and yield inaccurate confidence intervals given that mediated effects (the product of two distributions) do not follow a normal distribution.[37] Hence, the confidence interval of the mediated effect was estimated using the PRODCLIN method.[40] This method does not assume a normal distribution, yields asymmetric confidence intervals (CI), and is thus more accurate than traditional significance tests.[41,42] If the 95% CI for the mediated effect does not overlap zero, statistically significant mediation may be inferred.

RESULTS

Demographic Characteristics and Insomnia

Analyses of follow-up data revealed 262 new cases of insomnia disorder, resulting in a 1-y incidence rate of approximately 9.1%. All continuous demographic variables were normally distributed, with skewness and kurtosis within acceptable range.[43] We fit a logistic regression model predicting risk for insomnia based on the following predictors: sex, age, income, marital status, and education. A test of this model with all predictors against a constant-only model was statistically significant (χ^2 = 19.29; P < 0.01), indicating that our model reliably distinguished between participants with and without insomnia. Similarly, the Hosmer-Lemeshow test revealed that this model fit our data well (χ^2 = 6.48; P = 0.59). Sex was significantly associated with risk for insomnia (β = 0.30; odds ratio [OR] = 1.39; 95% CI = 1.01–1.83; P < 0.01), such that women exhibited a significantly higher risk for developing insomnia.

Age was another statistically significant predictor (β = -0.02; OR = 0.99; 95% CI = 0.98–1.00; P < 0.01), such that the odds for developing insomnia decreased by 2% for every 1-y increase in age. To further explore this finding, we categorized our sample into three groups based on age (y): 18 through 30, 31 through 60, and 61 and older. A chi-square test of independence between age group and insomnia suggested significant group differences (χ^2 = 8.38; P < 0.05). Analyses of standardized residuals revealed that the conditional distribution of positive insomnia cases for older adults was significantly lower (5.6%) than the other age groups. Young adults (10.6%) and middle-aged adults (9.5 %) did not differ significantly in their risk for insomnia. Finally, we also assessed age in relation to specific diagnostic questions. Sleep onset and maintenance difficulties were assessed using logistic regression because these were dichotomous variables. Daytime impairment, measured on a Likert-type scale, was tested using a one-way analysis of variance. Although age was not a significant predictor of sleep onset difficulties, it was positively associated with sleep maintenance difficulties (OR = 1.01; χ^2 = 8.38, P < 0.05). However, older adults reported significantly lower levels of daytime impairments than each of the other two groups ($F_{2, 710}$ = 6.80; P < 0.01).

None of the other demographic variables were significant predictors of insomnia.

Stressor Characteristics and Insomnia

Given that age and sex were significantly associated with insomnia, all further analyses controlled for these two demographic variables. We fit a logistic regression model with stress exposure as the IV and the presence of insomnia at follow-up as the DV. The model reliably distinguished between positive and negative cases of insomnia (χ^2 = 51.11; P < 0.01), and fit the data well per the Hosmer-Lemeshow test (χ^2 = 7.43; P = 0.49). Stress exposure was a significant predictor (β = 0.14; OR = 1.19; 95% CI = 1.13–1.26; P < 0.01) of insomnia, such that the odds of developing insomnia increased by 19% for every additional stressor.

Next, we ran a logistic regression model with insomnia as the DV, severity as the IV, and age, sex, and stress exposure as covariates. Severity (β = 0.04; OR = 1.04; 95% CI = 1.02–1.06; P < 0.01) was a significant predictor of insomnia, such that the odds of developing insomnia increased by 4% for every one-point increase on the severity scale. We then tested the interaction between stress exposure and severity using techniques outlined by Preacher et al.[44] Specifically, we repeated the aforementioned regression model with the product of severity and exposure added as a separate block. The interaction between exposure and severity was not statistically significant.

Moderation analyses for chronicity were conducted in identical fashion. Chronicity (β = 0.02; OR = 1.02; 95% CI = 1.00–1.03; P < 0.01) was a significant predictor of insomnia, such that the odds of developing insomnia increased by 2% for every 1-mo increase in average chronicity. The interaction model indicated statistically significant prediction (χ^2 = 56.67; P < 0.01); the Hosmer-Lemeshow test (χ^2 = 7.60; P = 0.47) suggested good model fit. The interaction between exposure and chronicity was statistically significant (χ^2 = 8.28; P < 0.01). To plot these effects, regression coefficients (*B*) were transformed from the logit curve scale to a probability scale using the following formula:

$$P = \frac{e^B}{1 + e^B}$$

Figure 2 depicts the association between stress exposure and insomnia as moderated by chronicity for several values of mean chronicity.

Stress-Response and Insomnia

Intrusion

Logistic regression analyses revealed that intrusion was a significant predictor (OR = 1.01; 95% CI = 1.00–1.01; P < 0.01) of insomnia, after controlling for age, sex, and stress exposure. Specifically, the odds of developing insomnia increased by 1% for each unit increase on the cognitive intrusion scale, and by 56% for a one standard deviation increase. Further, the association between stress exposure and insomnia became nonsignificant (P = 0.33) after intrusion was entered into the model. We then proceeded with the planned mediational analyses with stress exposure as the IV, intrusion as the mediator, and insomnia as the DV. Exploration of bivariate correlations (Table 2) revealed notable collinearity between stress exposure and intrusion (Pearson r = 0.75; P < 0.01). As Kenny[45] points out, multicollinearity is a requisite for

successful mediation and hence unavoidable. At worst, a mediator that is too proximal to the independent variable may diminish the power to detect statistical significance, and warrant a large sample size. However, given the large sample size in our study, threats to power because of multicollinearity are likely minimal. Finally, the standard errors associated with the parameter estimates (Table 3) did not suggest cause for concern. All mediation analyses controlled for age and sex, and followed previously outlined techniques. Intrusion mediated the association between stress exposure and insomnia, and examination of the confidence interval (95% CI = 0.08–0.12) indicated that mediation was statistically significant and accounted for 69% of the total effect of stress exposure on insomnia; see Table 3 and Figure 3 for individual analyses and adjusted parameter estimates.

Coping

Descriptive statistics for all coping variables appear in Table 4. The univariate distributions of several scales were positively skewed with significant outliers. Hence, outliers greater than three standard deviations from the mean were excluded from all further analyses. Next, we examined the bivariate correlations among these variables (Table S1, supplemental material). Two coping scales, "active coping" (e.g., Pearson r's = 0.80; 0.82; 0.88; 0.97*)* and "planning" (e.g., Pearson r's = 0.82; 0.88; 0.97*)*, exhibited a diffuse pattern of collinearity with multiple scales. Thus, these scales seemed to capture a more global index of coping. Hence, the "active coping" and "planning" scales were analyzed separately. Similarly, the emotional support and instrumental support scales were highly correlated (Pearson r = 0.96; P < 0.01). In psychometric validation studies of both the "Full Cope" and "Brief Cope" instruments, these scales loaded onto a single, distinct factor.[34,46] Hence, the arithmetic means of scores on these scales were entered as a new scale, called "support." We included this scale along with the 10 remaining coping scales in a logistic regression model to determine which coping measures independently predicted insomnia, after controlling for age and sex.

This model significantly predicted risk for insomnia (χ^2 = 75.58; P < 0.01), and the Hosmer-Lemeshow test indicated a good fit (χ^2 = 6.90; P = 0.55). The coping subscale, substance use (e.g., "I used alcohol or other drugs to help me get through it"), was a significant predictor of insomnia (OR = 1.05; 95% CI = 1.01–1.10; P < 0.05), such that a one-point increase on the substance use coping scale was associated with a 5% increase in risk for insomnia. Self-distraction (e.g., "I did something to think about it less, such as going to the movies, watch TV…") was also significantly associated with insomnia, such that the odds of developing insomnia (OR = 1.04; 95% CI = 1.00–1.08; P < 0.05) increased by 4% for every one-point increase on the scale. Finally, behavioral-disengagement (e.g., "I gave up trying to deal with it") was a significant predictor of insomnia (OR = 1.09; 95% CI = 1.03–1.15; P < 0.01); odds of developing insomnia increased by 9% for every one-point increase on this scale. Finally, in a separate model we tested the effects of "active coping" and "planning." We used the arithmetic means of scores on these two scales to construct a new scale; this scale was not significantly associated with insomnia.

Next, we assessed whether coping variables that were significantly associated with risk for insomnia mediated the relationship between stress exposure and the development of insomnia. Table 5 provides parameter estimates and omnibus significance/goodness-of-fit tests for all mediation analyses. Substance use (95% CI = 0.02–0.05*;* partial mediation: 21% of total effect), self-distraction (95% CI = 0.03–0.22*;* partial mediation: 86% of total effect), and behavioral disengagement (95% CI = 0.09–0.18; partial mediation: 91% of total effect) each acted as a significant mediator of the relationship between stress exposure and insomnia (Figure 3).

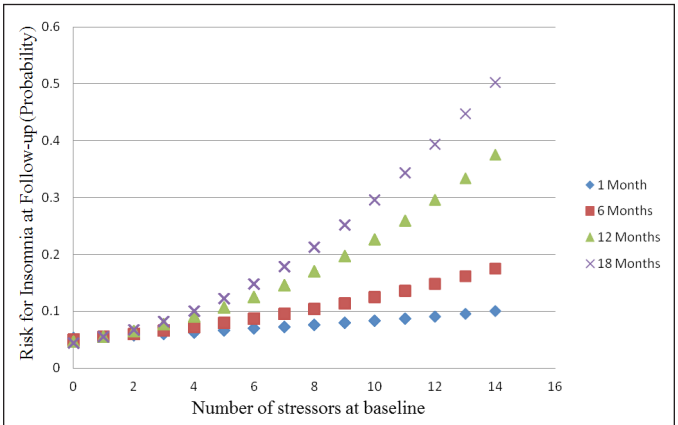

Figure 2—Moderation analyses revealed an interaction between stress exposure and chronicity for various levels of chronicity.

Table 2—Correlations among various stressor characteristics (n = 2,892)

	Exposure	Severity	Chronicity	Intrusion
Exposure	1.00	–	–	–
Severity	0.85[a]	1.00	–	–
Chronicity	0.12[a]	0.24[a]	1.00	–
Intrusion	0.75[a]	0.83[a]	0.56[a]	1.00

[a] P < 0.01.

Table 3—Intrusion as a mediator of stress exposure in predicting insomnia (n = 2,892)

Outcome	Predictor	B	SE (B)	B'	SE (B')	Model statistics
Intrusion	Exposure	24.75	0.44	0.99	0.02	F = 1093.50[a]
Insomnia	Intrusion, controlling for exposure	0.01	0.01	0.26	0.06	χ^2 = 70.83[a]

All the above models included age and sex as covariates. [a] P < 0.01. B, unstandardized regression coefficient; SE, standard error; B', adjusted for scale differences between logistic and ordinary least squares regression.

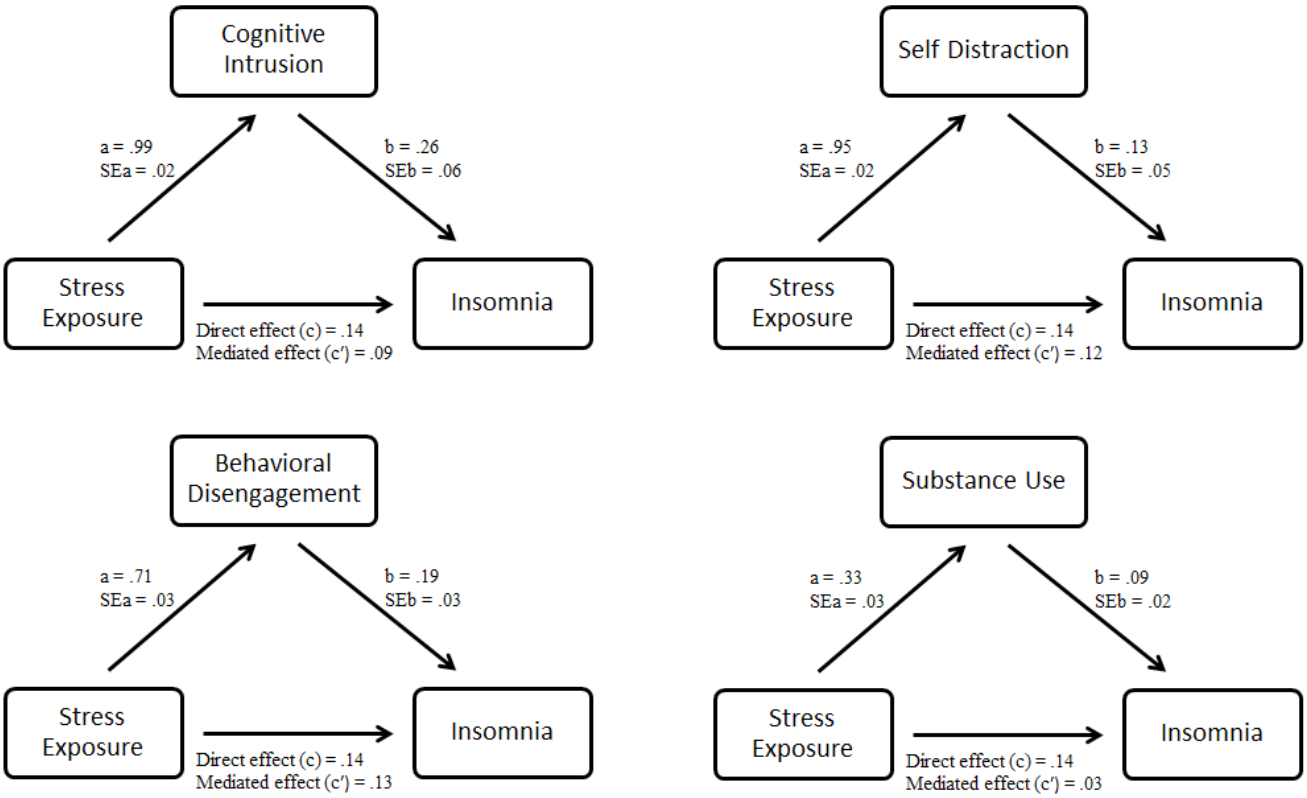

Figure 3—Intrusion and aspect of coping significantly associated with insomnia as mediators of stress exposure. All reported parameter estimates are adjusted for scale differences between logistic and ordinary least squares regression; all mediated effects were statistically significant, as evidenced by the fact that none of confidence intervals (CIs) overlapped zero.

DISCUSSION

Demographic Characteristics and Incident Insomnia

The incidence of insomnia in the current study was 9.1%; other prospective studies found similar 1-y incidence rates.[24,47] With respect to demographic characteristics, age and sex were significant predictors of insomnia. Specifically, the odds of developing insomnia for women were 1.4 times greater than those for men. A large epidemiological survey of more than 10,000 participants reported a similar finding for sex-based differences in the prevalence of insomnia (OR = 1.5).[5] With respect to age, analyses showed that risk for insomnia decreased with increasing age. This finding appeared inconsistent with prior studies, which reported either a positive association between age and insomnia[47-49] or no association.[24,50] However, a closer examination revealed that although sleep continuity problems increased with age, reports of daytime impairment were significantly lower among the elderly, as seen in other recent studies.[5,51] The question of whether this finding is related to age-related tolerance to sleep disturbance or a decline in functioning demands currently remains unanswered.

Table 4—Descriptive statistics for coping variables

	Mean (SD)	Median	Minimum	Maximum
Positive reframing	9.9 (8.6)	7	0	68
Religion	8.4 (9.7)	6	0	84
Self-distraction	7.1 (7.5)	5	0	58
Using emotional support	9.2 (8.4)	6	0	66
Using instrumental support	8.2 (8.2)	6	0	66
Planning	10.8 (9.4)	8	0	66
Substance use [a]	1.3 (4.2)	0	0	66
Venting [a]	5.2 (6.7)	3	0	58
Humor [a]	4.8 (6.5)	3	0	51
Active coping	10.5 (9.1)	8	0	66
Denial [a]	2.2 (4.7)	0	0	47
Self-blame [a]	3.1 (5.3)	1	0	49
Behavioral disengagement	1.8 (3.8)	0	0	48
Acceptance	12.4 (9.6)	10	0	81

[a] Variables that exhibited significant skewness or kurtosis. SD, standard deviation.

Stress and Insomnia

Participants with higher levels of stress exposure at baseline were more likely to develop insomnia. Importantly, this effect

Table 5—Coping as a mediator of stress exposure in predicting insomnia (n = 2,372)

Outcome	Predictor	B	SE (B)	B'	SE (B')	Model statistics [a]
SU	Exposure	0.41	0.04	0.33	0.03	$F = 53.61$
Insomnia	SU, controlling for exposure	0.06	0.01	0.09	0.02	$\chi^2 = 60.12$
SD	Exposure	2.78	0.06	0.95	0.02	$F = 810.33$
Insomnia	SD, controlling for exposure	0.03	0.01	0.13	0.05	$\chi^2 = 51.71$
BD	Exposure	0.93	0.04	0.71	0.03	$F = 225.96$
Insomnia	BD, controlling for exposure	0.09	0.02	0.19	0.03	$\chi^2 = 77.43$

Above models are presented in dyads for the three coping scales related to insomnia. Each dyad describes the effects of the independent variable (stress exposure) on the mediator (coping), and the mediator on the dependent variable (insomnia) after controlling for the independent variable. All the above models included age and sex as covariates. B, unstandardized regression coefficient; BD, behavioral disengagement; SD, self-distraction; SE, standard error; SU, substance use; B', adjusted for scale differences between logistic and ordinary least squares regression. [a] $P < 0.01$.

was significantly moderated by chronicity, such that the likelihood of developing insomnia as a result of stress exposure was higher for participants who reported greater chronicity. This finding is consistent with other data, which suggest that chronic events (≥ 1 y) are more likely to have a negative effect on health outcomes.[52-54] On the other hand, although stress severity was a predictor of insomnia, it did not interact with stress exposure. The strong correlation between exposure and severity may explain this null effect.

Intrusion and Insomnia

The effect of stress exposure on risk for insomnia was significantly mediated by cognitive intrusion. Most prior studies on the sleep-interfering effects of intrusion have measured only sleep-specific cognitions such as clock monitoring or concerns about the somatic symptoms of sleep loss, such as fatigue and achiness.[21,55,56] Presumably, this is because current research has only addressed cognitive intrusion as a maintaining factor of sleep disturbance among already affected individuals.[57] The current study is the first to examine cognitive intrusion in response to naturalistic stress as a prospective risk factor for insomnia. As such, these findings carry significant implications for future research.

Although current behavioral treatments for insomnia include a cognitive restructuring module aimed at identifying dysfunctional beliefs about sleep, it is unclear whether these interventions target cognitive intrusion.[22] A substantial body of evidence suggests that individuals with sleep difficulties typically engage in thought suppression to minimize the arousal triggered by cognitive intrusion.[58,59] However, most thought suppression techniques are not only ineffective but are also associated with poor sleep outcomes. Consistent with Wegner's Ironic Process Theory,[60] a number of intervention studies indicate that engaging in thought suppression prior to bed results in a paradoxical increase in cognitive load and thus cognitive arousal.[19] Preliminary clinical trials of mindfulness-based therapies, however, have shown considerable promise in suppressing cognitive intrusion and improving sleep.[61,62] Our data strongly stress the need for further research into the efficacy of these techniques.

Coping and Insomnia

Although several studies have implicated maladaptive coping as a mediating pathway between stress exposure and poor health outcomes,[63] the current study is the first to assess this hypothesis in relation to insomnia. Our data revealed that maladaptive coping in the form of substance use, behavioral disengagement, and distraction are mechanisms by which stress exposure precipitates insomnia. The substance use result is quite concerning, given both the high rates of self-medication among individuals with sleep disturbance[64,65] as well as the known sleep disruptive effects of substances such as alcohol. It stands to reason that among individuals without insomnia, substance use as a coping strategy leads to sleep disturbance. Sleep disturbance elicits further substance use because of the perceived efficacy of this coping strategy in promoting sleep. Increased substance use is associated with tolerance and dose escalation, and a vicious cycle leading to insomnia and substance abuse is set in motion.

Behavioral-disengagement represents another maladaptive coping style consistently cited in the stress literature; most coping theories classify specific coping behaviors based on the level of engagement or disengagement they facilitate.[66] Based on the belief that the demands of a stressor cannot be met, any attempt to mitigate its effects are forsaken.[34] A wealth of data supports the link between disengagement and poor health outcomes. For instance, behavioral-disengagement was a significant mediator of stress exposure on subsequent anxiety/depressive symptoms in a recent study of middle-school students.[8] By contrast, research on the effects of distraction as a coping technique has been less unequivocal.

Although a number of studies report the benefits of distraction,[67-69] others cite either limited[70] or no efficacy[71-73] in alleviating negative outcomes. Many of these inconsistencies are attributable to study design features. First, nearly all of the aforementioned studies were cross-sectional or short-term investigations. Conceivably, distraction may be beneficial with respect to short-term outcomes, such as negative affect[67] or acute pain.[69] However, long-term, potentially cumulative effects, as measured in the current study, are likely negative or neutral. For instance, distraction as a coping strategy has been found to be ineffective in the treatment of depression[74] and specific phobia.[71] Differences in assessment strategies across studies may be another important source of the variability in findings. In the sole study of distraction and insomnia in the literature, participants only reported their trait-level preference for this coping style.[57] Thus, this study was unable to measure

the impact of actual engagement in distraction, a key feature of our methodology.

Limitations and Future Directions

Our results should be interpreted with provisions for certain limitations. First, despite a modest response rate, a comparison of the demographic features of our sample with nationally representative data including insomnia incidence rates suggested minimal selection or recruitment bias. Further, considering the longitudinal design of this study, we had a high retention rate. Second, self-report instruments can be vulnerable to recall bias. Also, as in prior research, the current study cannot rule out confounds because of the heterogeneity inherent in insomnia symptoms. With regard to coping, past research suggests that the association between coping and health outcomes is moderated by the perceived efficacy of the coping strategy.[75] A potential explanation for some of the null findings on the relationship between positive coping behaviors and insomnia in our study may be that these strategies were deemed ineffective by participants. Hence, an important caveat in interpreting our findings on coping is that we did not probe for perceived efficacy. Similarly, coping behaviors and stressor characteristics such as chronicity were measured at the same time point in the current study. Thus, we were unable to examine the effects of coping behaviors on stress. Effective coping behaviors may attenuate the chronicity or severity of a stressful event. Daily sampling or ecological momentary assessment designs can help elucidate the dynamic interplay between stress and coping in future investigations. Finally, although none of our participants had a history of insomnia, we did not control for baseline stress levels. It is plausible that individuals at risk for insomnia are also more likely to perceive life events as stressful, such that a shared underlying factor may increase vulnerability to both stress and insomnia. Future studies should strive to examine the association between incident stressors and insomnia onset.

Despite these limitations, we believe the current study makes an important contribution to the field by identifying the effects of stress variables, such as chronicity, on risk for insomnia. This study is the first to identify process variables such as cognitive intrusion and coping through which stress exposure manifests in insomnia. As such, we believe these findings carry significant implications for future research and clinical practice. First, our results stress the need for a multidimensional approach to stress assessment that captures both stressful events as well as the personal meaning they assume for affected individuals. Further, as key mediators of the effects of stress exposure, cognitive intrusion and maladaptive coping responses represent important targets for therapeutic intervention.

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SUPPLEMENTAL MATERIAL

Table S1—Bivariate correlations among coping variables

	Humor	Denial	BD	SB	Act	PR	Rel	SD	ES	IS	Plan	Acc	Sub
Denial	0.33	–	–	–	–	–	–	–	–	–	–	–	–
BD	0.34	0.63	–	–	–	–	–	–	–	–	–	–	–
SB	0.44	0.54	0.61	–	–	–	–	–	–	–	–	–	–
Act	0.56	0.49	0.36	0.52	–	–	–	–	–	–	–	–	–
PR	0.60	0.42	0.35	0.48	0.88	–	–	–	–	–	–	–	–
Rel	0.36	0.44	0.35	0.38	0.65	0.67	–	–	–	–	–	–	–
SD	0.54	0.51	0.46	0.58	0.76	0.73	0.59	–	–	–	–	–	–
ES	0.52	0.48	0.34	0.43	0.80	0.78	0.79	0.71	–	–	–	–	–
IS	0.54	0.43	0.38	0.48	0.82	0.77	0.637	0.74	0.96	–	–	–	–
Plan	0.57	0.42	0.38	0.54	0.97	0.88	0.635	0.76	0.79	0.82	–	–	–
Acc	0.59	0.35	0.36	0.50	0.89	0.89	0.641	0.78	0.78	0.75	0.89	–	–
Sub	0.36	0.38	0.45	0.42	0.32	0.29	0.247	0.33	0.28	0.31	0.32	0.032	–
Vent	0.55	0.55	0.56	0.57	0.67	0.62	0.554	0.68	0.66	0.67	0.68	0.645	0.49

All reported correlations were significant at $P < 0.01$. Acc, Acceptance; Act, active coping; BD, behavioral disengagement; ES, emotional support; IS, instrumental support; Plan, planning; PR, positive reframing; Rel, religion; SB, self-blaming; SD, self- distraction; Sub, substance use; Vent, venting.