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Original Article

Sleep reactivity as a potential pathway from childhood abuse to adult insomnia



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ABSTRACT

Background: Survivors of childhood abuse are prone to adult insomnia, but the mechanisms for this development are poorly understood. Abuse that occurs during sensitive developmental periods might affect risk for insomnia by impacting emerging stress regulatory processes. Sleep reactivity refers to the sensitivity of the sleep system to stress and is a robust risk factor for insomnia. Recent evidence shows stress exposure itself worsens sleep reactivity, thereby increasing insomnia vulnerability. In this preliminary study, we hypothesized the association between childhood abuse experiences and adult insomnia would be mediated through greater sleep reactivity.

Methods: Community adults were recruited from the United States during the COVID-19 pandemic between June 2020 and June 2021 (N = 241, 88% female, M_{age} = 39, SD = 13.40). Participants completed a cross-sectional survey that included the Childhood Trauma Questionnaire, Ford Insomnia Response to Stress Test, Insomnia Severity Index, and a measure of general COVID-19 stress.

Results: Reporting more frequent childhood emotional, physical, or sexual abuse was associated with more severe insomnia during the COVID-19 pandemic. Only childhood emotional and physical (but not sexual) abuse histories were associated with greater sleep reactivity, which exerted an indirect effect on the relationships between these two abuse types and insomnia symptoms. These findings were robust to the effects of gender, age, and stress about the COVID-19 pandemic.

Conclusions: This preliminary study suggests recurrent emotional and physical abuse in childhood might promote later insomnia through heightened sleep reactivity. Stress management interventions could be important to prevent insomnia for abuse survivors by bolstering resilience of the sleep system.

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1. Introduction

Childhood abuse confers risk for poorer health outcomes later in life [1,2], including insomnia symptoms independent of comorbidities [3–8]. Identifying mechanisms by which childhood abuse promotes insomnia may uncover ways to prevent this

disorder and its harmful downstream effects. Stress-diathesis models suggest insomnia develops when stress exposure activates and exploits a predispositional vulnerability to insomnia [9,10], though little is known as to how these vulnerabilities form. Major stressful events during sensitive developmental periods in early life might alter risk for insomnia through its impact on emerging stress regulatory processes. To that end, childhood abuse may set the stage for insomnia by disrupting sleep regulatory processes known to be influenced by stress [11–13] thereby heightening *sleep reactivity* [12].

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Sleep reactivity refers to the extent to which stress exposure disrupts sleep, resulting in both difficulty falling and staying asleep [14]. Individuals with highly reactive sleep systems experience drastic disruptions to sleep when stressed, whereas the sleep of those with low sleep reactivity is largely unperturbed by stress [15]. This proneness to stress-related sleep disturbances is a robust predispositional vulnerability to subsequent insomnia [15], such that reactive sleepers without insomnia are at elevated risk of developing new-onset insomnia 1 year later [16] that persists across a 3 year period [17]. Notably, sleep reactivity is expressed in response to a wide range of stressors, including environmental [14,18,19], pharmacological [18,20], and circadian challenges [18,21]. Although sleep reactivity is a trait-like characteristic [16,22,23], stress exposure has been shown to alter the sensitivity of the sleep system by increasing sleep reactivity [24]. Importantly, exposure to major life stressors can sensitize the sleep system even among those with a low genetic load for this vulnerability [24]. By extension, childhood abuse may sensitize the sleep system by increasing its reactivity to stressors throughout development and adult life, thereby increasing risk for insomnia disorder and concomitant illnesses in adulthood [24,25].

To date, research on sleep reactivity and childhood abuse is limited. One study found no differences in sleep reactivity between individuals with and without a history of any childhood maltreatment [26], prompting these researchers to suggest examining sleep reactivity as it relates to specific abuse types rather than an overall history of maltreatment. Indeed, another study of pregnant women showed a history of childhood physical and sexual abuse was associated with more than a two-fold increase in likelihood of high sleep reactivity and sleep disturbance during pregnancy [27]. Nonetheless, the mechanisms by which childhood abuse corresponds to adult insomnia, and the potential role of sleep reactivity, remains unclear.

The current cross-sectional study extends this research in two ways. First, unlike the prior study on sleep reactivity and childhood abuse in pregnant women, we assessed childhood emotional abuse in addition to physical and sexual abuse. Emotional abuse is the most common type of childhood maltreatment reported around the globe [28] and leads to significant mental health problems in adulthood [29]. Therefore, it is crucial to clarify its role in sleep reactivity and insomnia development. Second, we preliminarily tested the hypothesis that greater sleep reactivity would mediate the association between histories of these abuse types on adult insomnia symptoms.

2. Materials and methods

2.1. Participants and procedure

We analyzed data from a cross-sectional study originally designed to assess rates of physical and sexual abuse during the COVID-19 pandemic. Data were collected via an online survey between June 2020 and June 2021. Participants were required to be at least 18 years old and were recruited via email through existing volunteer databases within the Henry Ford Health System's (HFHS) Sleep Research Center in Michigan, USA, as well as the HFHS daily newsletter, word of mouth, Qualtrics panels, and the HFHS psychiatry history database. Participants were screened for a history of abuse using a single item to ensure a roughly proportionate breakdown of participants with a history of physical abuse, sexual abuse, and neither abuse type. After completing the survey, participants were debriefed and compensated. Informed consent was obtained from all participants, and all procedures were approved by HFHS's Institutional Review Board.

2.2. Measures

2.2.1. Childhood trauma questionnaire—short form (CTQ-SF)³⁰

The CTQ-SF is a 28-item, self-report questionnaire that retrospectively measures childhood maltreatment history across five scales: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Participants indicated the frequency with which they experienced instances of each maltreatment type while growing up using a five-point scale ranging from never true (1) to very often true (5). Because extant literature suggests emotional, physical, and sexual abuse are most impactful on sleep-related hyperarousal [25], we computed sum scores using the five items comprising each abuse scale, with higher scores indicating greater frequency of each abuse type in childhood. The CTQ has previously demonstrated good test-retest reliability and convergent and discriminant validity with other trauma measures [30–33]. The internal consistency for each subscale in this study was as follows: emotional abuse $\alpha = 0.93$, physical abuse $\alpha = 0.85$, and sexual abuse $\alpha = 0.94$.

2.2.2. Insomnia severity index (ISI)³⁴

The ISI is a 7-item self-report measure of insomnia symptom severity. Participants reported their insomnia severity over the past two weeks using a five-point scale ranging from no symptoms (0) to severe symptoms (4). The ISI is a validated, commonly used measure with good psychometric properties [35,36]. Scores ≥ 10 are used to detect insomnia in community samples [36]. We computed a sum ISI score to be used as our outcome variable, with higher values indicating more severe insomnia. The internal consistency for the ISI in this study was good ($\alpha = 0.87$).

2.2.3. Ford insomnia response to stress test (FIRST)¹⁴

The FIRST is a nine-item questionnaire that measures sleep reactivity as defined as vulnerability to experience sleep disturbances when faced with different stressors (eg “Before an important meeting the next day”). Participants reported the likelihood of having difficulty sleeping when experiencing such situations using a four-point scale ranging from low likelihood (1) to very likely (4). The FIRST has good psychometric properties, shows within-person stability, and has been validated across several studies [15]. Scores ≥ 18 indicate high sleep reactivity and have been shown to prospectively predict incident insomnia in community adults [37]. We computed a sum FIRST score to model the indirect effect of sleep reactivity on the relationship between childhood abuse frequency and adult insomnia, with higher values indicating greater vulnerability to stress-related sleep disturbances. The internal consistency for the FIRST in this study was good ($\alpha = 0.83$).

2.2.4. Covariates

Since sleep reactivity is exacerbated following stress [24], we controlled for COVID-19-related stress to better estimate the relationship between childhood abuse and sleep reactivity independent of concurrent stressors [25]. We used a single item to measure stress related to the COVID-19 pandemic (“Have you been worried or stressed about the coronavirus pandemic?”). Participants responded using a five-point scale ranging from not at all (1) to very much (5). We also covaried for self-reported gender identity (0 = male, 1 = female) and age (in years) given their respective associations with sleep reactivity [15] and insomnia [10]. Additionally, controlling for age accounts for the time elapsed since childhood abuse, thereby adjusting for the potentially diminishing impact of these experiences on adulthood sleep [26]. All covariates were included as antecedent variables to our mediator (sleep reactivity) and outcome (insomnia symptoms).

Table 1
Frequency of each childhood abuse type (N = 241).

Type of abuse	Frequency of abuse			
	None to low	Low to moderate	Moderate to severe	Severe to extreme
Emotional abuse	94 (39)	40 (16.6)	25 (10.4)	82 (34)
Physical abuse	126 (52.3)	34 (14.1)	27 (11.2)	54 (22.4)
Sexual abuse	141 (58.6)	22 (9.1)	16 (6.6)	62 (25.7)

Values are given as n (%).

2.3. Data analysis plan

Analyses were performed in IBM SPSS version 21 (Armonk, NY, USA). We first screened data for quality and then examined correlations and descriptive statistics. We next tested three mediation models to examine the indirect effects of each childhood abuse type on insomnia symptoms through sleep reactivity. Specifically, we examined frequency of childhood emotional, physical, and sexual abuse as separate predictors of insomnia via heightened sleep reactivity. We tested separate models for each abuse type rather than a combined abuse model to disaggregate these experiences, as research suggests important differences in how they affect sleep [25]. For each significant model, we re-ran analyses with the inclusion of covariates of gender, age, and COVID-19 stress. We also tested the assumption of no X by M interaction and probed any significant interactions by specifying X as a moderator of the effect of sleep reactivity on insomnia symptoms [38].

All mediation analyses were performed using the PROCESS macro version 4.0 [38]. PROCESS computes the product of path a ($X \rightarrow M$) and path b ($M \rightarrow Y$, controlling for X) to estimate the indirect effect of X on Y through M (ab) [38]. PROCESS generates percentile bootstrap confidence intervals (CIs) for indirect effects based on 5000 random samples of the data, with CIs that do not include zero indicating evidence of mediation [38]. We report unstandardized beta coefficients unless otherwise stated.

3. Results

3.1. Descriptive statistics and correlations

Our sample comprised 241 participants who reported residing across 35 states, with Michigan being the most frequent response ($n = 113$, 47.1%). Participants reported being mostly female (88%) with a mean age of 39 years ($SD = 13.40$, range 18–73). Most participants reported being White ($n = 182$, 75.5%), followed by Black or African American ($n = 37$, 15.4%), Asian ($n = 10$, 4.1%), more than one race ($n = 8$, 3.3%), and American Indian or Alaskan Native ($n = 2$, 0.8%); two participants preferred not to respond. Most participants reported not being of Hispanic or Latino descent (95.8%).

As the study sampled individuals with a history of childhood abuse, most participants reported experiencing at least low-to-moderate exposure to any childhood abuse ($n = 174$, 72.2%). Of the specific abuse types, most reported experiencing emotional abuse ($n = 147$, 61%), followed by physical abuse ($n = 115$, 47.7%) and sexual abuse ($n = 100$, 41.5%). The reported frequencies of each abuse type using the CTQ's recommended cut-offs are provided in Table 1 [31].

All other descriptive statistics and correlations are provided in Table 2. This sample's average FIRST score was above the 18-point cutoff for detecting high risk for insomnia, indicating high levels of sleep reactivity [37]. Unsurprisingly, then, this sample's average ISI score exceeded the 10-point cutoff for detecting insomnia,

falling within the moderately severe symptom range [34,36]. Participants reported experiencing their insomnia symptoms most nights of the week ($M_{\text{days}} = 5.15$, $SD = 1.74$), and most participants reported their insomnia symptoms lasting 3 months or longer ($n = 202$, 83.8%). We did not have more granular information on age of insomnia symptom onset, however; it is possible some individuals' experienced adult-onset insomnia whereas others developed insomnia at some point after childhood abuse that persisted into adulthood. When asked about stress over the COVID-19 pandemic, 200 participants (83.0%) reported being *somewhat* to *very much worried or stressed*, whereas just 41 participants (17.0%) reported being *not at all* or *a little worried or stressed*.

3.2. Mediation models

Our first model examined childhood emotional abuse frequency in relation to insomnia via sleep reactivity. As expected, more frequent emotional abuse was related to greater sleep reactivity ($a = 0.240$, $P < 0.001$). While controlling for emotional abuse, greater sleep reactivity was related to more severe insomnia ($b = 0.370$, $P < 0.001$). Further, emotional abuse frequency was associated with more severe insomnia after covarying for sleep reactivity (direct effect: $c' = 0.254$, $P < 0.001$). Ultimately, there was evidence of an indirect effect of emotional abuse frequency on insomnia through sleep reactivity before including covariates [completely standardized indirect effect: $ab = 0.101$, 95% CI (0.049, 0.162)]. Notably, these effects remained significant after covarying for gender, age, and COVID-19 stress (see Fig. 1 for the full standardized model after accounting for covariates).¹

A similar pattern of results emerged for childhood physical abuse. Specifically, more frequent physical abuse was related to greater sleep reactivity ($a = 0.293$, $P < 0.001$), and greater sleep reactivity was related to more severe insomnia while accounting for physical abuse ($b = 0.389$, $P < 0.001$). Physical abuse frequency was associated with more severe insomnia independent of its relationship with sleep reactivity (direct effect: $c' = 0.319$, $P < 0.001$). Finally, physical abuse frequency also had an indirect effect on insomnia through sleep reactivity before including covariates [completely standardized indirect effect: $ab = 0.092$, 95% CI (0.044, 0.148)]. These effects remained significant after covarying for gender, age, and COVID-19 stress (see Fig. 2 for the full standardized model after accounting for covariates).²

Our last model tested sleep reactivity as a mediator of childhood sexual abuse and adult insomnia. Surprisingly, more frequent

¹ There was no significant interaction between emotional abuse and sleep reactivity ($P_s > 0.05$), with or without covariates included, supporting the assumption of no X by M interaction.

² We found evidence of interaction between physical abuse and sleep reactivity (without covariates included), $F(1, 237) = 4.217$, $P = 0.041$. Subsequent probing revealed the effect of sleep reactivity remained positive and significant across all levels of physical abuse frequency (all CIs excluded zero at -1 SD below the mean, the mean, and $+1$ SD above the mean), indicating the indirect effect of sleep reactivity on insomnia does not depend on frequency of physical abuse. There was no evidence of X by M interaction after we included covariates ($P > 0.05$).

Table 2
Descriptive statistics and bivariate correlations among study variables.

Scale	1	2	3	4	5	6	7	8
1. Emotional abuse	–							
2. Physical abuse	0.70**	–						
3. Sexual abuse	0.44**	0.46**	–					
4. Sleep reactivity	0.28**	0.25**	0.08	–				
5. Insomnia severity	0.39**	0.35**	0.28**	0.44**	–			
6. Gender	0.09	0.11	0.01	0.23**	0.07	–		
7. Age	-0.09	0.08	0.08	-0.11	0.17*	-0.01	–	
8. COVID-19 stress	0.20**	0.17**	0.10	0.29**	0.37**	-0.03	0.02	–
Min	5	5	5	9	0	0	18	1
Max	25	25	25	36	28	1	73	5
Mean	12.63	9.06	8.91	25.51	16.05	0.90	39.01	3.67
Median	11.00	7.00	5.00	25.00	16.00	1.00	36.00	4.00
SD	6.84	4.87	6.11	5.82	6.00	0.30	13.40	1.15

Pearson and point-biserial (gender) correlations. Emotional, physical, and sexual abuse = frequency of each childhood abuse type using sum scores from the Childhood Trauma Questionnaire's abuse subscales; Sleep reactivity = Ford Insomnia Response to Stress Test sum score; Insomnia severity = Insomnia Severity Index sum score; Gender = self-reported gender identity (0 = Male, 1 = Female); Age = age in years; COVID-19 stress = "Have you been worried or stressed about the coronavirus pandemic?" (1 = Not at all, 2 = A little, 3 = Somewhat, 4 = Much, 5 = Very much). *P < 0.05; **P < 0.01.

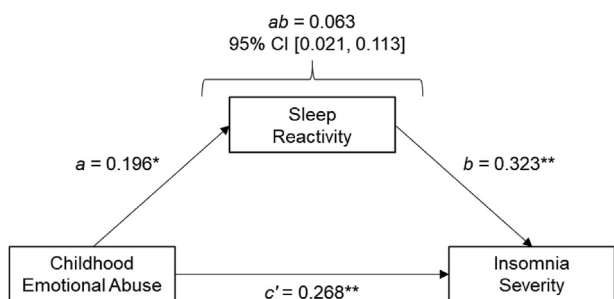


Fig. 1. The indirect effect of childhood emotional abuse frequency on insomnia symptom severity through sleep reactivity, controlling for gender, age, and COVID-19 pandemic stress (covariates not pictured). All paths represent standardized coefficients. C' = direct effect between emotional abuse and insomnia independent of sleep reactivity. CI = 95% bootstrapped confidence interval for the indirect effect (ab) based on 5000 samples. *P < 0.01, **P < 0.001.

sexual abuse was not related to sleep reactivity ($a = 0.080$, $P = 0.196$), although sleep reactivity was still related to insomnia with sexual abuse in the model ($b = 0.433$, $P < 0.001$). Consistent with the previous models, sexual abuse frequency was associated with more severe insomnia symptoms while accounting for sleep reactivity (direct effect: $c' = 0.236$, $P < 0.001$). Nonetheless, there was no evidence for sleep reactivity as a mediator of childhood

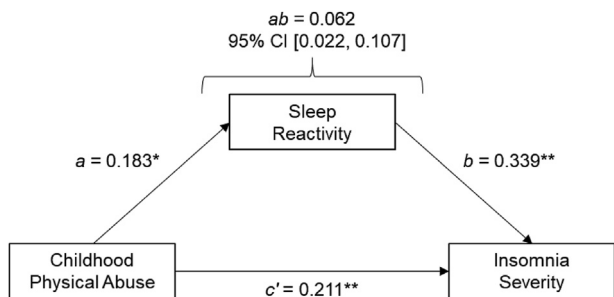


Fig. 2. The indirect effect of childhood physical abuse frequency on insomnia symptom severity through sleep reactivity, controlling for gender, age, and COVID-19 pandemic stress (covariates not pictured). All paths represent standardized coefficients. C' = direct effect between physical abuse and insomnia independent of sleep reactivity. CI = 95% bootstrapped confidence interval for the indirect effect (ab) based on 5000 samples. *P < 0.01, **P < 0.001.

sexual abuse and adult insomnia [indirect effect: $ab = 0.035$, 95% CI (-0.016, 0.085)].

4. Discussion

This preliminary study suggests sleep reactivity may be a pathway by which childhood abuse relates to adult insomnia symptoms. As predicted, more frequent childhood abuse was associated with more severe insomnia through heightened sleep reactivity, even after adjusting for gender, age, and stress about the COVID-19 pandemic. Unexpectedly, this mediation was only evident for childhood emotional and physical abuse, but not sexual abuse. Overall, this study builds on previous research by suggesting childhood emotional and physical abuse may confer risk for insomnia, at least in part, by sensitizing the sleep system [27].

Childhood abuse might exacerbate sleep reactivity due to chronic and repeated activation of the hypothalamic-pituitary-adrenal axis during sensitive developmental periods [11,12], thereby exposing the developing brain to persistently elevated stress hormones that shape it to become more responsive to threat [13]. As a result, adults with a history of childhood abuse may be more apt to activate their stress system even when doing so is unnecessary or maladaptive [13,39]. Our findings suggest this overreactive stress response sustained by childhood abuse might impact the sleep system, rendering it less capable of preserving normal sleep patterns during periods of stress, and ultimately increasing risk for developing insomnia [40]. This is consistent with evidence that childhood abuse is associated with various indices of sleep-related hyperarousal [25].

These effects might be limited to emotional and physical abuse only, as childhood sexual abuse did not exert an indirect effect on insomnia through sleep reactivity. One possibility for this null finding is that sexual abuse evokes a distinct physiological reaction that may not have the same effects on the sleep system as do stress reactions following other abuse types [41]. Nonetheless, we did find childhood sexual abuse was still related to more severe insomnia, suggesting a different mechanism may explain this association. For example, perhaps childhood sexual abuse that occurs in a sleep-related context gives rise to a fear of sleep that drives insomnia more than sleep reactivity [42–44]. More research is needed to test this assertion, however.

These findings must be interpreted within our study's limitations. Above all, these data are based on a cross-sectional survey, thus we emphasize these findings are preliminary and should be

used to guide prospective investigation using larger samples. Nonetheless, our statistical models provide initial support for this theoretical link given childhood abuse temporally preceded participants' insomnia within the past two weeks. Our findings are also strengthened by accounting for relevant covariates that may have otherwise explained our findings, particularly COVID-19 stress and age. That is, the urgency and unpredictability of the pandemic might have been expected to be a more important determinant of sleep reactivity than childhood abuse, especially for older individuals whose abuse occurred decades earlier. It is important to note, however, that our measure of COVID-related stress was limited to a single item that may not reflect the complexity of the long-lasting stressor presented by the pandemic. Further, our use of the Childhood Trauma Questionnaire may have introduced minimization biases that could have diminished the relationship between childhood sexual abuse and sleep reactivity [45]. For instance, two of the five sexual abuse items are face-valid ("Someone molested me" and "I believe that I was sexually abused"), but there is evidence to suggest individuals with a history of sexual abuse would be reluctant to acknowledge or describe their experiences in this way [46,47]. Future research could utilize clinical interviews to study sleep reactivity within abused populations more precisely.

5. Conclusions

This preliminary study expands on the nascent sleep reactivity and childhood abuse literature by using community adults during the COVID-19 pandemic and providing novel associations between emotional abuse and sleep reactivity. Ultimately, we found evidence childhood emotional and physical abuse engenders risk for adult insomnia via heightened sleep reactivity, regardless of gender, age, and COVID-19 stress. Future research is needed to identify interventions to mitigate the effects of childhood abuse on sleep system regulatory processes. As an extension of the stress system, sleep reactivity may be effectively targeted using mindfulness-based approaches given evidence they act on neuroendocrine dysfunction [48–50]. Such interventions may not only enhance resilience to insomnia, but also to other known consequences of high sleep reactivity, including posttraumatic stress disorder, depression, anxiety, psychosis, and suicidality [51–54].

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 Acquisition, Analysis, or Interpretation of data: Reffi, Arnett, Drake.
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 Critical revision of the manuscript for important intellectual content: Kalmbach, Cheng, Jovanovic, Norrholm, Sexton, Mahr, Seymour, Drake.

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Conflict of interest

None declared.

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