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Dysregulated arousal as a pathway linking childhood neglect and clinical sleep disturbances in adulthood

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ABSTRACT

Background: A history of childhood maltreatment has a well-established association with clinical sleep disturbances in adulthood, which is a transdiagnostic contributor to many chronic diseases.
Objective: Determine whether actigraphy-measured indices of dysregulated arousal during sleep explain associations between abuse or neglect in childhood and clinical sleep disturbances in adulthood.
Participants and setting: Participants were 646 individuals, ages 25–83 (59.3% female) from the MIDUS II Biomarker, Refresher studies.
Methods: Participants completed the Childhood Trauma Questionnaire, wore an actigraph for seven days, and rated sleep quality using the Pittsburgh Sleep Quality Index (PSQI).
Results: Both neglect ($b = 0.66$, $SE = 0.33$, $p = .04$) and abuse ($b = 1.09$, $SE = 0.32$, $p < .001$) were associated with clinical sleep disturbance. Actigraphy-measured sleep efficiency mediated the link between neglect and clinical sleep disturbances ($ab = 0.33$, $SE = 0.12$, *95%CI* [0.12, 0.57]). However, no such link between abuse and clinical sleep disturbances was mediated by actigraphy-measured indices. Sleep onset latency did not mediate the link between neglect or abuse and sleep disturbance. Models covaried for other maltreatment, gender, and age.
Conclusions: While the unique associations between abuse or neglect and clinical sleep disturbances were robust in this sample, only sleep efficiency emerged as a mediator linking maltreatment and clinical sleep disturbances. Critically, this mediation was specific to neglect. Abuse and neglect may lead to disease through distinct pathways. Moreover, potential dysregulation in arousal that leads to sleep inefficiency may be a specific pathway through which experiences of neglect in childhood contribute to chronic disease.

1. Introduction

Childhood maltreatment involves an act or series of acts of commission or omission by a parent, caregiver, or another person in a custodial role that results in potential or actual harm to a child (Centers for Disease Control and Prevention, 2021). The adverse consequences of childhood maltreatment can persist throughout the lifespan, including depression and substance use disorders

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(Chapman et al., 2004; Norman et al., 2012), chronic physical conditions (Walker, Gelfand, et al., 1999; Walker, Unutzer, et al., 1999), and earlier mortality (Chen et al., 2016). Sleep may be one way that maltreatment in childhood contributes to these and other health disparities in adulthood. Sleep disturbance is a transdiagnostic risk factor and contributor to chronic diseases and additional disease vulnerability (Irwin, 2015). Importantly, there is a well-established association between childhood adversity, including maltreatment, and clinical sleep disturbances in adulthood (Kajeepeta et al., 2015). This is of major public health concern because childhood maltreatment is estimated to affect up to 40% of youth in the United States (Baldwin et al., 2019; Finkelhor et al., 2009; Hussey et al., 2006). For that reason, characterizing the link between childhood maltreatment and sleep is critical to better understanding how maltreatment may lead to health disparities in adulthood.

Adversity in childhood, including maltreatment, has been associated with lower subjective sleep quality, longer sleep onset latency, trouble staying asleep throughout the night, and sleep disorders in adulthood, using both prospective longitudinal and retrospective designs (Agargun et al., 2003; Chapman et al., 2011; Greenfield et al., 2011; Gregory et al., 2006; Kajeepeta et al., 2015; Ramsawh et al., 2011). These observations may suggest that maltreatment leads to sustained difficulties regulating arousal. Arousal is a measurement domain identified by the National Institute of Mental Health's Research Domain Criteria (RDoC) as relevant to many psychiatric disorders (Insel et al., 2010). Dysregulation in arousal can be indexed as waking during the period an individual intends to be sleeping, such as the time it takes to fall asleep and the number of times a person wakes throughout the night. A high arousal state limits or prohibits sleep (Dahl, 1996), in part because sleep requires the ability to reduce vigilance and disconnect from the environment (Carskadon & Dement, 2011; Joiner, 2018). Perceptions of threat in the environment can interfere with this normative process and therefore impede sleep (Joiner, 2018). Thus, children exposed to maltreatment may be at greater risk for persistent sleep disturbances in adulthood as a result of living under potentially threatening conditions for sustained periods of development. Yet, few studies have empirically examined the mediating role of arousal dysregulation in the association between childhood maltreatment and adulthood sleep disturbances.

Childhood maltreatment is a heterogeneous construct that can vary in type, timing, or severity, which may obscure biobehavioral pathways that contribute to disease. Maltreatment is commonly categorized into acts of commission or abuse, and acts of omission or neglect (Centers for Disease Control and Prevention, 2021). This distinction is important with respect to arousal and sleep because abuse and neglect may contribute to sleep disturbances through different pathways, with neglect specifically contributing to regulation of arousal. For example, experiences of deprivation in childhood are correlated with different neurocognitive outcomes relative to experiences involving threat (McLaughlin et al., 2014; Sheridan & McLaughlin, 2014). Specifically, the absence of environmental inputs or "deprivation" contributes to a flatter trajectory of synaptogenesis and pruning across the entire central nervous system, particularly in the prefrontal cortex (PFC) (Sheridan & McLaughlin, 2014). In comparison, experiences of threat during childhood are specifically linked to changes in the morphology and effective functioning of neural structures involved in learning and memory such as the hippocampus (Sheridan & McLaughlin, 2014). Given the essential role of the PFC in modulating arousal (Phan et al., 2003; Zhang et al., 2014), experiences of deprivation (e.g., childhood emotional or physical neglect) may contribute to maturational shifts in the PFC, which could impact the development and functioning of the arousal system. This shift could decrease the capacity for an individual to modulate arousal, leading to lower sleep efficiency (i.e., percent of time asleep in bed) or increased sleep latency (i.e., time to fall asleep). Over time, these manifestations of arousal dysregulation may lead to sustained and impairing sleep disturbances.

The purpose of the present study was to determine whether dysregulated arousal was a distinct pathway through which neglect or abuse were associated with subjective clinical sleep disturbances in adulthood. Dysregulated arousal was indexed by sleep onset latency and sleep efficiency measured objectively using seven days of actigraphy. Fig. 1 provides a conceptual model of key constructs explored in the present study.

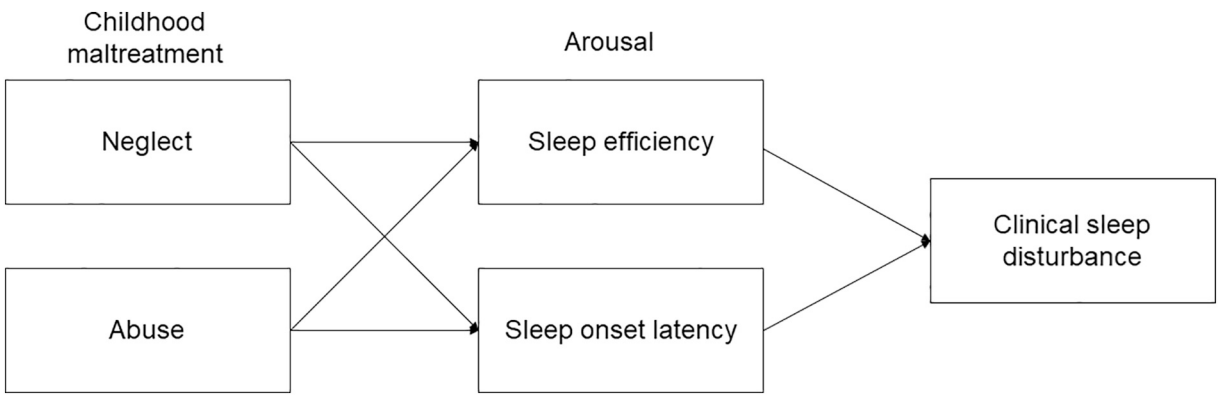

Fig. 1. Conceptual model.

2. Method

2.1. Participants

The present study included a sample of 646 individuals (59.3% female) between the ages of 25 and 83 who participated in the Biomarker projects from the 2nd wave of the Midlife in the United States (MIDUS II) study and the refresher cohort of the Midlife in the United States (MIDUS-R) study who also agreed to participate in a seven-day daily diary and actigraphy sleep study. See Table 1 for participant characteristics.

The original MIDUS study (1995–1996) is a national probability sample of 7108 noninstitutionalized, English-speaking adults ages 25–75 in the contiguous United States, recruited via random-digit dialing. A follow-up study was completed (MIDUS-II) (Ryff et al., 2007) from 2004 to 2006, with a retention rate of about 70% from the original cohort. The MIDUS-II Biomarker project was conducted 5 to 64 months after this second wave and included a total of 1255 participants who stayed overnight at one of three general clinical research centers (GCRC): University of California Los Angeles, University of Wisconsin, or Georgetown University. MIDUS-R participants were recruited from a refresher study (MIDUS-R) (Ryff et al., 2016) conducted in 2011 to 2014 to replenish the original MIDUS-I cohort with a new sample containing 3577 participants ages 24–75. The MIDUS-R Biomarker project (2012–2016) (Weinstein et al., 2017) included a total of 863 participants who stayed overnight at one of three GCRCs. A subsample of the respondents from both the MIDUS-II and MIDUS-R Biomarker projects participated in a 7-day daily diary and actigraphy sleep study. There were no differences in age, marital status, education, employment status, or clinical sleep disturbances between the present analytic sample relative to the biomarker sample overall ($p > .10$). However, the present analytic sample was less likely to be Hispanic (0.6% compared to 5.2%; $\chi^2 = 21.27$, $p < .001$) and more likely to be female (59.0% vs. 52.8%; $\chi^2 = 7.25$, $p = .007$). All included projects were approved by the Institutional Review Board at each site prior to data collection.

2.2. Procedure

Demographics and psychosocial factors, including the Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998) and Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), were completed via self-report questionnaire in their visit to one of the GCRCs. After completion of the Biomarker projects, participants were invited to participate in a seven-day sleep study. After providing informed consent to participate in the sleep study, participants were sent home with instructions to complete a daily sleep diary and continuously wear a MiniMitter Actiwatch 64 on their wrist for seven consecutive days. The Actiwatches were programmed to begin collecting data at 7:00 am on the Tuesday after the GCRC visit and stop the following Tuesday morning.

2.3. Measures

2.3.1. Neglect

Neglect was measured using retrospective self-report on the Childhood Trauma Questionnaire (CTQ) (Bernstein & Fink, 1998). This 28-item questionnaire contains five subscales: physical neglect, emotional neglect, physical abuse, sexual abuse, and emotional abuse. Each subscale includes five items, each beginning with "When I was growing up…" and proceeding to identify experiences in the childhood-rearing environment. Participants provided their responses according to a 5-point Likert scale ranging from 1 "*never true*" to 5 "*very often true*". As a result, total and subscale scores from the measure reflect a combination of maltreatment type, chronicity, and severity. Participants with scores for emotional neglect ≥15 or physical neglect ≥8 were categorized as "neglected" using established scoring thresholds (Walker, Gelfand, et al., 1999), reflecting severe or chronic exposure to emotional or physical neglect during childhood.

2.3.2. Abuse

Abuse was also measured using retrospective self-report on the CTQ. Participants with scores of emotional abuse ≥10, sexual abuse ≥8, or physical abuse ≥8 were categorized as "abused" (Walker, Unutzer, et al., 1999), reflecting severe or chronic exposure to emotional, sexual, or physical abuse during childhood.

Table 1
Participant characteristics.

	M (SD)	% (*n*)
Age	52.27 (12.32)	
Female		59.3 (383)
Childhood maltreatment		
Neglect		11.2 (72)
Abuse		17.1 (110)
Neglect and abuse		19.7 (127)
Sleep disturbances (PSQI)	6.29 (3.65)	
PSQI ≥ 5		62 (402)
Sleep efficiency	80.19 (9.18)	
Sleep onset latency	28.18 (23.33)	

2.3.3. Sleep efficiency

Sleep efficiency was measured using seven consecutive days of activity data collected via a MiniMitter Actiwatch 64, and computed by the Actiware program algorithms based on the rest intervals. Sleep efficiency refers to the percent of time an individual was asleep while in bed, and scores reflect the average across the seven days of measurement. Actigraphy is an established method for assessing objective sleep outcomes outside of the laboratory that has acceptable reliability when compared with polysomnography (Spielmanns et al., 2019).

2.3.4. Sleep onset latency

Sleep onset latency was measured using seven consecutive days of activity data collected via a MiniMitter Actiwatch 64, and computed by the Actiware program algorithms based on the rest intervals. Sleep onset latency reflects the length of time (in minutes) it took to transition from full wakefulness to sleep, and scores were averaged across the seven days of measurement.

2.3.5. Clinical sleep disturbance

Sleep disturbances were measured using the well-validated and widely used Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989). This 19-item scale assesses sleep quality over the past month and yields a global score on sleep disturbance. Global scores can range from 0 to 21, with higher scores indicating worse sleep. A global score of five or greater indicates a likely clinical sleep disturbance.

2.4. Data analysis

All predictor and outcome variables were examined for normality. In order to maximize data but also reduce the influence of plausible but extreme actigraphy-measured sleep values on our results (Ghosh & Vogt, 2012), sleep onset latency and sleep efficiency were winsorized to three standard deviations (3SD) from the mean; 8 values were winsorized for sleep onset latency, and 9 values were winsorized for sleep efficiency. To determine the unique contribution of neglect and abuse on clinical sleep disturbances, we used a single regression model predicting continuous global PSQI scores as a function of abuse, neglect, and our covariates. To test the mediating role of objectively measured sleep outcomes in this association, we used ordinary least squares regression with bootstrap resampling within the PROCESS macro for SPSS (Hayes, 2013). In these models, the predictor variable (neglect or abuse) predicted the outcome variable (global PSQI scores) through the mediator (sleep onset latency or sleep efficiency). All analyses using neglect as a predictor covaried for abuse, gender, and age, and all analyses using abuse as a predictor covaried for neglect, gender, and age.

3. Results

Sleep disturbances were common in this sample, with 62.2% ($n = 402$) of participants reporting clinical sleep disturbances (PSQI $\geq$ 5), though comparable with other community samples of adults (Buysse et al., 2008; Gasperi et al., 2017; Hinz et al., 2017; Yildirim & Boysan, 2017). Further, 11.2% ($n = 72$) of our sample were exposed to neglect alone, 17.1% ($n = 110$) were exposed to abuse alone, and 19.7% ($n = 127$) were exposed to both neglect and abuse.

Both neglect and abuse were uniquely associated with greater clinical sleep disturbances, $b = 0.66$, $SE = 0.33$, $p = .04$ and $b = 1.09$, $SE = 0.32$, $p < .001$, respectively. Tables 2 and 3 provide model fit indices and coefficient estimates for neglect and abuse as predictors of objective arousal measures and clinical sleep disturbances.

3.1. Mediation between neglect and sleep disturbances

3.1.1. Sleep efficiency mediated the association between neglect and clinical sleep disturbances

The total effect of neglect on sleep disturbances, $c = 0.66$, $SE = 0.33$, $p = .04$, was composed of a non-significant direct effect of neglect on sleep disturbances, $c' = 0.33$, $SE = 0.31$, 95% CI[−0.27, 0.94], and a significant indirect effect of neglect on sleep disturbances through sleep efficiency, $ab = 0.33$, $SE = 0.12$, 95% CI[0.12, 0.57]. See Fig. 2 for the results of the adjusted model depicting the significant indirect effect of neglect on sleep disturbance via sleep efficiency.

Table 2
Adjusted unstandardized coefficient estimates predicting objective arousal measures from abuse, neglect, and key covariates.

	Sleep efficiency				Sleep onset latency			
	b (SE)	*p*	95% CI		*b* (SE)	*p*	95% CI	
			LL	***UL***			***LL***	***UL***
Intercept	79.10 (1.71)	<.001	75.73	82.47	28.89 (4.40)	<.001	20.25	37.53
Neglect	−2.44 (0.83)	.003	−4.08	−0.80	4.01 (2.14)	.06	−0.19	8.21
Abuse	−0.66 (0.81)	.42	−2.25	0.93	2.03 (2.08)	.33	−2.07	6.12
Gender	2.12 (0.74)	.004	0.66	3.57	−3.94 (1.90)	.04	−7.68	−0.20
Age	0.02 (0.03)	.57	−0.04	0.07	−0.01 (0.07)	.91	0.15	0.14
R^2	0.03	<.001			0.01	.03		
F	5.03				2.59			

Table 3
Adjusted unstandardized coefficient estimates predicting clinical sleep disturbances from abuse, neglect, objective arousal measures, and key covariates.

	b(SE)	*p*	95% CI		*b*(SE)	*p*	95% CI	
			LL	*UL*			*LL*	*UL*
Intercept	16.57 (1.32)	<.001	13.98	19.15	4.73 (0.67)	<.001	3.42	6.05
Neglect	0.33 (0.31)	.28	−0.27	0.94	0.49 (0.32)	.12	−0.12	1.11
Abuse	0.999 (0.30)	<.001	0.41	1.59	1.0 (0.31)	.001	0.40	1.61
Sleep efficiency	−0.13 (0.01)	<.001	−0.16	−0.10				
Sleep onset latency					0.04 (0.006)	<.001	0.03	0.05
Gender	0.52 (0.27)	.059	−0.02	1.06	0.40 (0.28)	.16	−0.15	0.95
Age	−0.006 (0.01)	.59	−0.03	0.01	−0.01 (0.01)	.49	−0.03	0.01
R^2	0.15	<.001			0.11	<.001		
F	23.30				16.13			

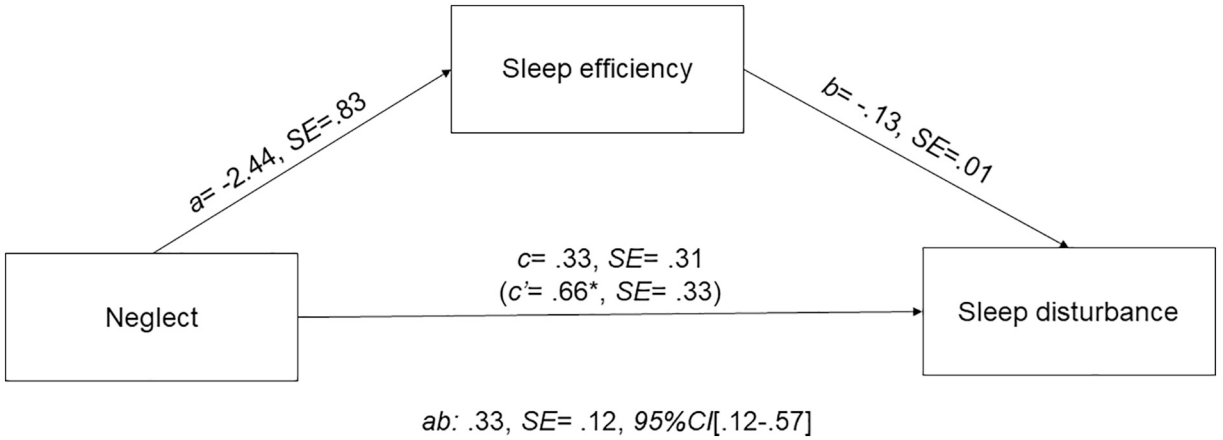

Fig. 2. Sleep efficiency mediates the association between childhood neglect and sleep disturbance in adulthood.

Sleep onset latency did not mediate the link between neglect and clinical sleep disturbances. Specifically, the total effect of neglect and sleep onset latency, $c = 0.66$, $SE = 0.33$, $p = .04$, was composed of a non-significant direct effect of neglect on sleep disturbances, $c' = 0.49$, $SE = 0.32$, 95% CI[−0.12, 1.11], and a non-significant indirect effect of neglect on sleep disturbances through sleep onset latency, $ab = 0.17$, $SE = 0.09$, 95% CI[−0.01, 0.36]. All models covaried for abuse, gender, and age.

3.1.2. Mediation between abuse and sleep disturbances

Sleep efficiency and sleep onset latency did not mediate the association between abuse and clinical sleep disturbances. The total effect of abuse and sleep efficiency, $c = 1.09$, $SE = 0.32$, $p < .001$, was composed of a significant direct effect of abuse on sleep disturbances, $c' = 1.00$, $SE = 0.30$, 95% CI [0.41, 1.59], and a non-significant indirect effect of abuse on sleep disturbances through sleep efficiency, $ab = 0.09$, $SE = 0.10$, 95% CI [−0.11, 0.28]. The total effect of abuse and sleep onset latency, $c = 1.09$, $SE = 0.32$, $p < .001$, was composed of a significant direct effect of abuse on sleep disturbances, $c' = 1.0$, $SE = 0.31$, 95% CI [0.40, 1.61], and a non-significant indirect effect of abuse on sleep disturbances through sleep onset latency, $ab = 0.08$, $SE = 0.09$, 95% CI [−0.08, 0.27]. All models covaried for neglect, gender, and age.

4. Discussion

The purpose of the present study was to explore whether objectively measured indices of dysregulated arousal at night help to explain the relatively well-established association between specific types of childhood maltreatment (neglect, abuse) and clinical sleep disturbances. While the unique associations between neglect or abuse and clinical sleep disturbances were robust in this sample, only arousal as indexed by sleep efficiency mediated the link between neglect and clinical sleep disturbances. These findings support the hypothesis that neglect and abuse may lead to sleep disturbances and disorders through distinct pathways. Moreover, the potential dysregulation in arousal that leads to waking throughout the night may be a specific pathway through which experiences of neglect in childhood contribute to chronic disease.

Sleep efficiency mediated the association between neglect and clinical sleep disturbances, whereas sleep onset latency did not. Sleep efficiency and sleep onset latency are not orthogonal, but have distinct cognitive and affective consequences (see Short & Banks, 2014 for review). What differentiates sleep efficiency from sleep onset latency is the frequency of waking throughout the night, also known as sleep fragmentation. Sleep fragmentation can have many behavioral, environmental, and neurobiological causes (Joiner,

2018; Short & Banks, 2014), but may be a critical behavioral measure of dysregulation in the sleep-wakefulness domain of arousal regulation. Our findings are consistent with at least four studies so far that have shown that maltreatment and trauma during childhood are associated specifically with lower sleep efficiency in adolescents (Turner et al., 2020), early adulthood (Greenfield et al., 2011; Teicher et al., 2017), adult military veterans (Insana et al., 2012), and adults with serious mental illness (Laskemoen et al., in press). Sleep fragmentation is a robust predictor of subjective sleep quality (Åkerstedt et al., 1994) and poorer daytime functioning (Martin et al., 1996; Stepanski, 2002), and may contribute to cognitive decline (André et al., 2019). Therefore, dysregulated arousal during sleep, as measured by sleep fragmentation, may be an important component of sleep to target in interventions aimed at preventing or mitigating chronic health problems. Among patients with insomnia, individuals with a history of childhood maltreatment evidence more beta EEG activity during sleep—an indicator of psychophysiological arousal—than those without a history of maltreatment (Bader et al., 2013). Importantly, several promising studies have shown that sleep efficiency is modifiable through transcranial magnetic stimulation (Frase et al., 2016), as well as using the benzodiazepine receptor agonist Eszopiclone (Krystal et al., 2012). Those studies together implicate an underlying dysregulation in GABA-ergic functioning. More attention to whether sleep efficiency is reliably lower relative to other sleep outcomes, and whether interventions that specifically target sleep fragmentation disproportionally benefit and lead to sustained health improvements in adults with a history of neglect is needed.

Notably, our findings were specific to chronic and severe physical or emotional neglect during childhood, not abuse. There has been long-standing interest in whether different types of childhood maltreatment have distinct developmental and health-related sequelae. From a neurophysiological perspective, experiences in childhood may differ in their biological salience to the developing individual (Kuhlman et al., 2017). For example, living with an abusive caregiver may lead a child to develop behavioral strategies for defense and physiological strategies for wound healing. However, living in an environment where there is no imminent threat, but the predictability of care is low, may lead to chronic psychophysiological states of hypervigilance. In the present sample, sleep efficiency only mediated the link between neglect and clinical sleep disturbances; no measures of arousal mediated the association between abuse and clinical sleep disturbances. Neglect may lead to sleep disturbances across the lifespan through alterations to arousal systems by promoting greater vigilance to the environment. Individuals can be vulnerable during sleep, making vigilance during this period adaptive in some environments. Indeed, threat and deprivation have been identified as distinct dimensions of childhood experience that uniquely shape neurodevelopment (Sheridan & McLaughlin, 2014). Additionally, unpredictable caregiving has separately emerged as a robust environmental predictor of neurodevelopment that has implications for later affective disorders (Glynn & Baram, 2019). The specificity of our findings to neglect when accounting for abuse in a relatively large sample supports the further interrogation of the unique psychological, neurodevelopmental, and health sequelae of different types of maltreatment.

These results should be considered in the context of the study's strengths and limitations. First, exposure to childhood maltreatment was measured via a retrospective self-report questionnaire, which may contain sources of error that differ from maltreatment measured prospectively (Baldwin et al., 2019; Monroe, 2008). Further, this measure simultaneously reflects type, severity, and chronicity of maltreatment, while other measures that distinguish between these dimensions have observed distinct behavioral consequences (Jackson et al., 2014; McGuire & Jackson, 2018; Petrenko et al., 2012). That being said, the CTQ is one of the most widely-used measures of childhood maltreatment, and therefore the results are likely generalizable to other studies linking childhood maltreatment to biobehavioral processes in adulthood. Second, the conceptual model tested in the analyses hypothesized that arousal dysregulation mediated the association between childhood maltreatment and clinical sleep disturbances, with interest in identifying potential causal pathways. However, the present data are cross-sectional, and no causal inferences can be made. Ideally, a combination of prospective longitudinal (Abajobir et al., 2017; McPhie et al., 2014; Noll et al., 2006) and experimental (Hamilton et al., 2018) research will converge to disentangle potential causal and modifiable pathways. Finally, arousal regulation was measured using actigraphy-measured sleep efficiency and sleep-onset latency. Studies confirming that sleep fragmentation mediated the link between neglect and clinical sleep disturbances using other technologies (e.g., polysomnography) and other measures of arousal—such as those identified in the National Institute of Mental Health RDoC matrix for sleep-wakefulness (Insel, 2014; Insel et al., 2010)—are needed. In particular, it will be informative to better characterize the endogenous (e.g., neurobiological) and exogenous (e.g., social and environmental) contributors to sleep efficiency as they pertain to individuals with a history of childhood maltreatment.

It is well-established that individuals exposed to maltreatment and other forms of adversity are at risk for lifelong health disparities and earlier mortality. The role of sleep in the development and maintenance of these health disparities has been relatively overlooked, despite its relevance to numerous chronic health conditions. Neglect may specifically contribute to the development of dysregulated arousal systems that lead to fragmented sleep, which may be a modifiable pathway through which the lifelong health sequelae associated with childhood maltreatment may be mitigated.

Declaration of competing interest

The authors report no conflicts of interest.

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