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Author manuscript *Behav Sleep Med.* Author manuscript; available in PMC 2019 April 18.

Published in final edited form as:

Behav Sleep Med. 2011; 9(3): 130-143. doi:10.1080/15402002.2011.583895.

# Neuroticism Mediates the Relationship Between Childhood Adversity and Adult Sleep Quality

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# Abstract

This study investigated the relationship of childhood adversity and adult sleep quality in 327 college students (91 males), with a mean age of 18.9 years (SD = 2.1); and also examined whether neuroticism significantly mediated the observed association. Regression findings indicate that the relationship between childhood adversity and adult sleep quality is significant, and that there is a stronger association in men. Furthermore, a bootstrapping approach to testing the significance of the indirect effect (i.e., mediation) indicated that neuroticism mediated this relationship in both men and women. These data suggest that otherwise healthy young adults with a history of childhood adversity are at increased risk for sleep disturbance. Neuroticism may represent a potential target for change in future insomnia interventions, particularly in adults with a history of childhood adversity.

Adverse childhood experiences (ACEs), such as physical, sexual, and emotional abuse and neglect, are common and often associated with health and behavioral problems in children (Dubowitz, Papas, Black, & Starr, 2002; Flaherty et al., 2006; Thompson et al., 2005). Consequences of these experiences may endure—ACEs are associated with a host of physical and psychological problems later in life, including increased rates of depression and other psychiatric disorders (Chapman et al., 2004; Felitti et al., 1998), substance use (Kendler et al., 2000), obesity (Felitti et al., 1998; Thomas, Hypponen, & Power, 2008), heart disease (Felitti et al., 1998), increased health care utilization (Chartier, Walker, & Naimark, 2007), and suicide attempts (Dube et al., 2001). In fact, roughly one fourth of adult psychiatric disorders and as many as one half of all suicide attempts are attributable to ACEs (Afifi et al., 2008).

One of the most ubiquitous symptoms to emerge after exposure to a traumatic event is sleep disruption—evidence suggests that trauma exposure may confer risk for sleep difficulties even in the absence of a full-fledged posttraumatic stress disorder (PTSD) diagnosis (Mellman, Aigbogun, Graves, Lawson, & Alim, 2008; Ohayon & Shapiro, 2000; J. Rosen, Reynolds, Yeager, Houck, & Hurwitz, 1991). Most of these investigations have focused on

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traumatic events that have occurred during adulthood. Some investigators have also examined sleep in children exposed to a traumatic event (Chemtob, Nomura, & Abramovitz, 2008; Mongillo, Briggs-Gowan, Ford, & Carter, 2009), but few studies have examined sleep in adults who have experienced childhood adversity. Evidence of such a relationship would suggest long-lasting effects of adverse experiences on sleep. Two recent studies by Bader and colleagues utilized wrist actigraphy and polysomnography to examine objective sleep disturbance in adults with primary insomnia with and without ACEs (Bader, Schafer, Schenkel, Nissen, Kuhl, et al., 2007; Bader, Schafer, Schenkel, Nissen, & Schwander, 2007). In these small samples (N= 39 and N= 59, respectively), ACEs were found to correlate with worse sleep, including lower sleep efficiency and a greater number of movements during sleep. Another recent study found an association between childhood adversity and selfreported adult sleep quality in a large epidemiological sample, which remained significant even after adjusting for depressive symptoms and recent stressors (Koskenvuo, Hublin, Partinen, Paunio, & Koskenvuo, 2010). However, this investigation did not employ empirically validated measures of ACEs and sleep quality, and participants were not directly queried about exposure to childhood maltreatment including abuse and neglect. Because the effects of abuse, neglect, and other interpersonal insults have been found to be more pernicious than non-interpersonal events (Luthra et al., 2009; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993), it is desirable to explicitly assess these types of events in studies of childhood adversity. This study would permit partial replication of the previous findings, using a larger sample than that of the studies by Bader and colleagues (Bader, Schafer, Schenkel, Nissen, Kuhl, et al. (2007) and Bader, Schafer, Schenkel, Nissen, and Schwander (2007)); a sample of young adults more proximal to the events in question; and empirically validated measures of sleep quality and ACEs involving abuse and neglect.

Many studies have found that the female gender is associated with increased vulnerability to unfavorable psychosocial outcomes following traumatic events (Fisher et al., 2009; Haatainen et al., 2003; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Stein, Walker, & Forde, 2000). Of the three aforementioned studies on ACEs and adult sleep in the extant literature, two studies reported no gender differences (Bader, Schafer, Schenkel, Nissen, Kuhl, et al., 2007; Bader, Schafer, Schenkel, Nissen, & Schwander, 2007), and one study found the ACEs–adult sleep association to be stronger in women than in men when adjusting for work status and health behaviors (Koskenvuo et al., 2010). The putative female vulnerability to adverse outcomes following ACEs is again tested in this study.

A final aim of this study was to examine one potential pathway by which childhood adversity may exert its influence on adult sleep quality. We chose to examine neuroticism, which refers to one's general proneness to negative emotions. Neuroticism is one of five core personality domains that emerges during late childhood and becomes increasingly stable into adulthood (Costa & McCrae, 1988; Lamb, Chuang, Wessels, Broberg, & Hwang, 2002). Thus, neuroticism emerges, on average, at the chronological midpoint between ACEs and adult sleep, supporting its exploration as a potential mediator of the ACEs–adult sleep quality relationship. We examined neuroticism, relative to other variables of interest, as it is considered a latent factor underlying the development and maintenance of a range of psychiatric disorders, including anxiety and depression (Brown, 2007). There is evidence from animal (Carroll et al., 2007; Holmes et al., 2005) and clinical studies (Glaser, van Os,

Portegijs, & Myin-Germeys, 2006; McFarlane et al., 2005; Roy, 2002) of an association between ACEs and neuroticism in adulthood. Neuroticism has also been implicated in poor sleep (Gau, 2000; Soehner, Kenned, & Monk, 2007; Vincent, Cox, & Clara, 2009; Williams & Moroz, 2009). To our knowledge, there are no published studies examining whether neuroticism mediates the childhood adversity–adult sleep relationship. However, in previous work, the association between ACEs and sleep outcomes was found in individuals *without* current psychopathology (Bader, Schafer, Schenkel, Nissen, Kuhl, et al., 2007; Bader, Schafer, Schenkel, Nissen, & Schwander, 2007) and while adjusting for current depressive symptoms (Koskenvuo et al., 2010), suggesting that the effects of neuroticism may not fully account for this relationship. Although there may be other variables involved in the association, we would hypothesize that neuroticism partially, but significantly, mediates the ACEs–adult sleep relationship.

Our study hypotheses were as follows:

H1: There will be a significant, inverse relationship between childhood adversity and adult sleep quality.

H2: The relationship will be stronger in women than in men.

H3: Neuroticism will mediate this relationship.

We tested these hypotheses in a sample of undergraduate men and women.

# METHOD

#### **Participants**

Participants were 327 undergraduate students (236 females and 91 males) at San Diego State University (SDSU). These students completed self-report questionnaires during the first month of the semester in a mass testing session for course credit. Students were then invited to participate in additional testing that involved completion of computer tasks and additional questionnaires (including those examined for this study) if they either (a) were considered "normal" on trait anxiety (i.e., they scored within the 40th-60th percentile during the mass testing session on a measure of trait anxiety not used in this study) or (b) were considered "high trait anxious" by virtue of scoring at or above the 80th percentile on trait anxiety. This selection process was related to hypotheses for a larger study. Two hundred sixty participants (79.5%) had scores in the "normal" range, with the remaining 20.5% (67 participants) in the "high" range. The study was approved by the Human Research Protections Programs of SDSU and the University of California, San Diego; and participants gave informed, written consent to participate.

#### Measures

**ACEs.**—The 28-item Childhood Trauma Questionnaire (CTQ) contains five domains that assess levels of physical abuse, emotional abuse, sexual abuse, physical neglect, and emotional neglect experienced during childhood (Bernstein et al., 1994; Bernstein et al., 2003). These domains sum to yield a total score (CTQ Total). Higher scores indicate more

extensive endorsement of abuse or neglect. The CTQ has demonstrated adequate internal consistency and test-retest reliability (Bernstein et al., 2003).

**Sleep quality.**—The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) was used to assess sleep quality. The PSQI is a widely used and well-validated 19-item self-report measure of sleep quality. It contains seven subscales measuring domains such as subjective sleep quality, sleep latency, sleep duration, and sleep disturbance, which combine to yield a global composite score of sleep quality over the past week. Global sleep quality scores are continuous (range = 0–21), with high scores (6) reflecting poor sleep quality (Buysse et al., 1989). These guidelines were used to categorize participants as either "good sleepers" or "poor sleepers" for some analyses. The PSQI has been demonstrated to have high internal consistency (Cronbach's a = 0.83), test–retest reliability (0.85–0.87), and convergent validity (Buysse et al., 1989).

**Neuroticism.**—The Revised NEO Personality Inventory (NEO PI–R) is a 240-item measure of five broad personality domains: neuroticism, extraversion, openness to experience, agree-ableness, and conscientiousness. The NEO PI–R also measures six subordinate domains known as *facets* for each of the personality domains. It has demonstrated good internal consistency, test–retest reliability, and validity (Costa & McCrae, 1992). For this study, neuroticism was the only domain of interest. *T* scores were derived from college-age, sex-specific norms (Costa & McCrae, 1992).

#### **Statistical Analysis**

All statistical analyses were conducted using Statistical Package for the Social Sciences Version 15.0 (SPSS, Inc., Chicago, IL). Group differences (at an alphalevel of p .05) on demographic variables, CTQ Total and subscale scores, and neuroticism were assessed by sleep status (good sleepers vs. poor sleepers as defined by global PSQI score) and gender using chi-square or independent-sample t test analyses as appropriate.

A hierarchical linear regression approach was employed to test the first two study hypotheses. First, to assess the relationship between ACEs and sleep quality (H1), linear regression analyses were run with CTQ Total as the predictor and global PSQI score as the outcome variable. Next, to test a possible moderating effect of gender on the relationship between ACEs and sleep quality (H2), CTQ Total and gender were entered on Step 1 of a hierarchical linear regression model (female gender was coded "0," and male gender was coded "1"). A Gender × CTQ Total interaction term was entered on Step 2. If a significant interaction effect was detected, subsequent analyses would be stratified by gender.

Neuroticism's role as a potential mediator was assessed by directly testing the significance of its effect (i.e., the indirect effect of X on Y) on the CTQ–PSQI relationship (H3). This *indirect effect*, as depicted in Figure 1, is quantified as the product of unstandardized regression coefficients for the CTQ–neuroticism relation (Path a) and the neuroticism–PSQI relation (Path b), or *ab*. In this study, the significance of the indirect effect (*ab*) was tested using a bootstrapping approach, as recommended by Hayes and colleagues (Hayes, 2009; Preacher & Hayes, 2004). Bootstrapping is a nonparametric method considered to have several advantages over other approaches to testing mediation, such as the "causal effects"

approach popularized by Baron and Kenny (1986), which suffers from low power; and the Sobel test, which relies on an assumption that the sampling distribution of the indirect effect is normal (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). In bootstrapping, the study sample is re-sampled *with replacement* multiple times, such that an observation that appears only once in the original sample could potentially appear more than once in a given re-sample. Following recommendations by Hayes, 5,000 bootstrap re-samples were generated in this study. This procedure yields an inference about the size of the indirect effect, a standard error estimate (mean of the standard deviation across the 5,000 re-samples), and 95% confidence intervals based on the distribution of the 5,000 samples. If the confidence interval does not include zero, the indirect effect (i.e., mediation) is considered significant. Finally, for purposes of interpretability and practical significance, an estimation of the effect size of the indirect effect was calculated using methods developed by Fairchild, MacKinnon, Taborga, and Taylor (2009).

# RESULTS

#### **Demographic and Clinical Characteristics**

CTQ Total, neuroticism, and global PSQI scores for females, males, and the total sample are presented in Table 1. There were no significant gender differences on any study variables. In the total sample, 52.5% of study participants (53.2% of females and 50.6% of males  $\chi^2(1, 326) = 0.18$ , p = .669, were considered poor sleepers.

#### **Good Sleepers Versus Poor Sleepers**

Comparisons of clinical characteristics for poor sleepers relative to good sleepers are displayed in Table 2. There were significant differences on CTQ Total score, t(318) = -3.99, p < .001; and neuroticism, t(317) = -7.21, p < .001, indicating more ACEs and higher neuroticism for poor sleepers. For descriptive purposes, CTQ subscale findings are also presented. Poor sleepers were more likely to have experienced several types of ACEs compared to good sleepers.

#### **Regression Analyses Testing H1 and H2**

Table 3 displays findings for the linear regression analyses that include the total sample. The model displayed in Table 3a tests the bivariate association between CTQ Total and global PSQI (H1). CTQ Total was significantly correlated with global PSQI ( $\beta = .26$ ), t(318) = 4.81, p < .001.  $R^2$  was .07, suggesting that CTQ Total explained a small, but significant, amount of variance in sleep quality. The model displayed in Table 3b tests whether gender moderates the CTQ Total–global PSQI relationship (H2). The full model containing main and interaction effects was significant, F(3,316) = 9.65, p < .001. Step 2, which contained the interaction term, explained significantly more variance than the main effects model, F(1,316) = 5.25, p = .023 ( $R^2$  change = .02). The Gender × CTQ Total interaction term was significant ( $\beta = .518$ ), t(316) = 2.29, p = .023, indicating that the CTQ Total–global PSQI association is stronger in men.

To better understand the effects of gender on the CTQ–PSQI relationship, subsequent analyses were stratified by gender.

#### **Tests of Mediation (H3)**

Table 4 displays results for tests of the indirect effect of CTQ Total on PSQI in men and women, respectively. Note that a formal p value is not shown, as this test relies on an assumption of normality of the sampling distribution of *ab* and, as such, is not reported in favor of a bootstrapping approach to testing indirect effects, which does not make this assumption (Preacher & Hayes, 2008). As the confidence intervals for *ab* do not include zero, it can be concluded that neuroticism significantly mediates the relationship between CTQ Total and global PSQI in both men and women.

#### Interpretability and Practical Significance of the Indirect Effect

To translate the indirect effect into a value with practical significance, an estimate of effect size,  $R^2$ , was calculated for women and men using methods developed by Fairchild et al. (2009) to gauge the proportion of variance accounted for in an outcome variable by the indirect effect. In women,  $R^2$  was .029, indicating that 2.9% of the variance in PSQI is accounted for by the indirect effect of CTQ through neuroticism. As the  $R^2$  for the full model in women was .211, the proportion of variance accounted for in the model by the indirect effect of neuroticism was .029/.211 = .137; that is, 13.7% of the variance in the full model is through the mediated effect. In men,  $R^2$  of the indirect effect of neuroticism. The  $R^2$  for the full model in men was .293, and the proportion of variance accounted for in the 46.8% of the variance in the full model is due to the mediated effect. Thus, a sizeable portion of the variance in adult sleep quality is explained by the mediating effect of neuroticism.

# DISCUSSION

The goal of this study was to examine the relationship between ACEs and adult sleep, the role of gender, and the extent to which this putative relation may be mediated by neuroticism in a young adult sample. These findings suggest that there is a significant relationship between childhood adversity and adult sleep, that there are gender differences in this relationship, and that it is partially mediated by neuroticism.

As predicted in H1, a statistically significant inverse relationship was detected between ACEs and adult sleep quality in the total sample. This is notable, as few previous studies have examined the relationship between ACEs and adult sleep (Bader, Schafer, Schenkel, Nissen, Kuhl, et al., 2007; Bader, Schafer, Schenkel, Nissen, & Schwander, 2007; Koskenvuo et al., 2010). This study, which features a relatively large sample size and uses empirically validated self-report measures, corroborates the previous findings. Taken together, these data suggest that the repercussions of childhood adversity extend into early adulthood. This sample primarily consisted of college freshman, with a mean age of 18.9 years, and so one could infer that the reported ACEs occurred in the fairly recent past for participants who endorsed these events. Thus, it is unclear whether the same strength of association would be found in an older sample of adults more temporally distant from ACEs. Nonetheless, these findings are clinically significant in light of data suggesting that the presence of current sleep disturbance signifies increased risk of future onset of psychiatric

disorders, such as depression and anxiety (Chang, Ford, Mead, CooperPatrick, & Klag, 1997; Johnson, Roth, & Breslau, 2006; Ohayon & Roth, 2003). The presence of both ACEs and sleep problems could conceivably confer even greater risk for these outcomes.

H2 specified that the relationship between ACEs and adult sleep quality would be stronger in women. Contrary to our predictions, these data suggest that the ACEs-sleep relationship is more robust in men. This gender difference emerged despite the fact that there were no significant differences between men and women on any of the self-report measures included in this analysis. This is also in contrast to the three published studies on ACEs and adult sleep, of which two studies found no gender differences (Bader, Schafer, Schenkel, Nissen, Kuhl, et al., 2007; Bader, Schafer, Schenkel, Nissen, & Schwander, 2007), and one study found the relationship to be stronger in women relative to men when adjusting for work status and health behaviors (Koskenvuo et al., 2010). It may be that methodological differences between the studies, including variables assessed, led to these disparate results. This finding is also in contrast with much of the broader literature on gender differences in trauma sequelae, which suggests that adverse outcomes following trauma exposure are either more prevalent or more severe in women (Fisher et al., 2009; Haatainen et al., 2003; Kessler et al., 1995; Stein et al., 2000). However, some investigations have found no differences between men and women, particularly once the higher prevalence of reported sexual abuse in women is taken into account (L. N. Rosen & Martin, 1996; Roy & Janal, 2006; Young, Harford, Kinder, & Savell, 2007). One study actually found increased rates of depression and anxiety symptoms in men receiving treatment for sexual abuse relative to female patients (Gold, Lucenko, Elhai, Swingle, & Sellers, 1999). On the other hand, a recent metaanalysis concluded that women fared worse even when type of abuse was taken into account (Tolin & Foa, 2006). Thus, general conclusions regarding gender differences in trauma sequelae are not possible at this time, although one might question the assumption of female vulnerability to adverse outcomes following ACEs in all instances. With regard to sleep outcomes in particular, it appears that healthy young adults with histories of ACEs are at increased risk for sleep disturbance, and these findings suggest that the risk for men may equal or exceed that for women.

Finally, H3, which stated that neuroticism would mediate the relationship between childhood adversity and sleep quality, was supported by these findings. The mediating effect of neuroticism was significant and accounted for 2.9% of the variance in adult sleep quality in women and as much as 13.7% of the variance in adult sleep quality in men. These findings would perhaps suggest gender differences in the extent to which neuroticism mediates the ACEs–sleep quality relationship; however, as we had no hypotheses regarding strength of mediation in men versus women (which would be consistent with *mediated moderation* or *moderated mediation* models; e.g., Muller, Judd, & Yzerbyt, 2005), this was not tested statistically in this study. What can be said, based on these findings, is that neuroticism appears to have a significant role in the pathway from ACEs to adult sleep problems in both men and women. Extrapolating to clinical populations, neuroticism may represent a potential future target for treatment, as it is possible that interventions that change one's general tendency to experience negative affect (or, conversely, that succeed in increasing positive emotions) may lead to improvements in sleep in individuals with a history of ACEs. For example, perhaps cognitive behavioral therapy for insomnia could be augmented to

include elements that also target neuroticism. Indeed, whereas neuroticism is generally considered to be a stable personality trait in adulthood, change in levels of neuroticism during treatment has been found to be associated with improved outcomes (Quilty, Meusel, & Bagby, 2008).

Although these findings indicate that neuroticism partially mediates the ACEs-adult sleep quality relationship, what additional mechanisms might explain the association? Although a full discussion of putative mechanisms is beyond the scope of this study, there are some key possibilities that are worth mentioning. First, several neurobiological mechanisms are likely involved (although some of these may actually represent neural substrates of neuroticism). Early adversity has been linked to alterations of the hypothalamic-pituitary-adrenal (HPA) axis (Heim et al., 2000; MacMillan et al., 2009), serotonergic abnormalities (Bhansali, Dunning, Singer, David, & Schmauss, 2007; Kaufman et al., 2004), and differences in amgydala activity (Law, Pei, Feldon, Pryce, & Harrison, 2009; Sabatini et al., 2007). These systems are closely tied to the regulation of sleep, and there is evidence that each is also involved in sleep disruption (Barrett et al., 2009; Benca, Obermeyer, Shelton, Droster, & Kalin, 2000; Hatzinger, Hemmeter, Brand, Ising, & Holsboer-Trachsler, 2004; Mehlman et al., 2000). Second, the presence of physical disorders may impact the relationship between ACEs and adult sleep. ACEs are associated with increased prevalence of physical disorders in adulthood, and physical disorders are, in turn, linked with poor sleep quality (Stein, Belik, Jacobi, & Sareen, 2008). Consequences of physical disorders, such as chronic pain or breathing difficulties, have the potential to impact sleep quality. Third, an environmental explanation has been posited, which states that children who grow up with exposure to ACEs are more likely to live in chaotic family environments where few stable routines, such as regular bed times, bedtime rituals, and so forth, exist (Gregory, Caspi, Moffitt, & Poulton, 2006). Thus, these individuals, having never been taught good sleep hygiene, may continue to engage in behaviors incongruent with good sleep quality even as adults. Further research will be necessary to clarify these and other possible mechanisms involved in the ACEs-sleep quality relationship.

These findings should be interpreted in light of a number of significant limitations. First, this study was done using a relatively homogenous university student sample. Therefore, caution should be used in generalizing these findings to other age groups, to the general population, or to patient populations. Because these participants were selected based on being in the "high" or "normal" range on trait anxiety, these findings may also not be representative of a general undergraduate sample. Second, the association between ACEs and adult sleep quality found here is only one "snapshot" of the relationship in young adults. This tells us little about the strength of the association at earlier or later points in life, or whether these gender differences seen here persist over time—for example, it is quite possible that the ACEs-sleep quality relationship becomes stronger in women at a later point in life, which would be consistent with previous research that has found a stronger relationship in adult women relative to men. Longitudinal studies would be necessary to investigate the trajectory of these relationships over longer periods. Third, these findings rely on self-report measures, including retrospective reports of ACEs that may have occurred many years prior to this assessment. However, here the young ages of the participants may be advantageous; they are less temporally removed from the time of the ACEs relative to most adult samples, and so

their memory and self-reports of ACEs may be even more reliable. Furthermore, previous research suggests that retrospective reports of childhood maltreatment are valid and may actually provide an underestimation of the incidence of moderate to severe ACEs (Hardt & Rutter, 2004).

In conclusion, results suggest that childhood trauma is associated with poor sleep quality in adulthood among young men and women. This relationship may be stronger in men, and neuroticism partially mediates the association. Future studies that investigate these associations in a more diverse sample with regard to age and over longer follow-up periods would strengthen these findings. In addition, future investigators may wish to examine whether associations with adult sleep vary by *type* of ACEs. This study suggests that even otherwise healthy young adults who have experienced ACEs may be experiencing current sleep disturbance and, thus, may be at increased risk for psychiatric disorders in the future. If these findings are replicated, neuroticism may represent an additional target for change in interventions to improve sleep.

# ACKNOWLEDGMENTS

This work was funded by the following: National Institutes of Mental Health (NIMH) Grant #T32 MH018399 (Holly J. Ramsawh); National Institute of Aging Grant #R01 AG008415 and the VA Center for Stress and Mental Health (Sonia Ancoli-Israel); and NIMH Grant #K24 MH064122 (Murray B. Stein). Dr. Ancoli-Israel receives or has received research funding from the following: Sepracor, Inc. and Litebook, Inc. She currently serves or has served as a consultant on the Scientific Advisory Board for the following: Ferring Pharmaceuticals, Inc.; GlaxoSmithKline; Merck; NeuroVigil, Inc.; Orphagen Pharmaceuticals; Pfizer; Respironics; Sanofi-Aventis; Sepracor, Inc.; and Schering-Plough. Dr. Stein receives or has in the past three years received research support from the following: Eli Lilly and Company, GlaxoSmithKline, and Hoffmann-La Roche Pharmaceuticals. He is currently or in the past 3 years has been a consultant for the following: AstraZeneca; BrainCells, Inc.; Bristol-Myers Squibb; Comprehensive NeuroScience; Eli Lilly and Company; Forest Laboratories; Hoffmann-La Roche Pharmaceuticals; Jazz Pharmaceuticals; Johnson & Johnson; Mindsite; Pfizer; Sepracor, Inc.; and Transcept Pharmaceuticals, Inc.

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# FIGURE 1.

Simple mediation model depicting direct and indirect effects of adverse childhood experiences (ACEs) on adult sleep quality. The indirect effect (Path *ab*) represents the effect of the proposed mediator, neuroticism. The direct effect (Path c') shows the effects of ACEs on adult sleep quality with the indirect effect (*ab*) removed.

#### TABLE 1

Demographic and Clinical Characteristics of the Sample

Variable	Total Sample	Females	Males	
Age: M(SD)	18.9 (2.1)	19.0 (2.3)	18.7 (1.1)	
Gender (% of total sample)	327	236 (72.2)	91 (27.8)	
Ethnicity (%) <sup>a</sup>				
Caucasian	173 (52.9)	130 (55.1)	43 (47.3)	
Latino	65 (19.9)	45 (19.1)	20 (22.0)	
Asian	57 (17.4)	40 (16.9)	17 (18.7)	
Other	32 (9.8)	21 (8.9)	11 (12.1)	
CTQ Total (SD)	35.6 (12.5)	36.5 (12.8)	35.9 (12.6)	
Neuroticism (SD)	50.1 (10.5)	49.9 (9.4)	50.0 (10.2)	
PSQI global (SD)	6.2 (3.0)	6.0 (3.2)	6.1 (3.1)	
Poor sleepers (% of total)	169 (52.5)	134 (53.2)	45 (50.6)	

Note. CTQ = Childhood Trauma Questionnaire; PSQI = Pittsburgh Sleep Quality Index.

<sup>a</sup>Percentages may not equal 100 due to missing data.

#### TABLE 2

Between-Group Differences on CTQ and Neuroticism for Good Sleepers Versus Poor Sleepers

Variable	Good sleepers: PSQI 5	Poor sleepers: PSQI	5	р
CTQ Total score (SD)	32.9 (11.0)	38.4 (13.4)		<.001
CTQ subscales (%)				
Emotional abuse	21.7	40.2		< .001
Physical abuse	19.7	28.4		ns
Sexual abuse	13.2	14.8		ns
Emotional neglect	23.0	39.1		.002
Physical neglect	10.5	26.2		< .001
Neuroticism	46.0 (9.1)	53.6 (9.7)		< .001

*Note*. CTQ = Childhood Trauma Questionnaire; PSQI = Pittsburgh Sleep Quality Index.

#### TABLE 3a

Linear Regression Analyses Estimating the Relationship Between CTQ Total and Global PSQI

Dependent Variable	Predictor	<b>R</b> <sup>2</sup>	F	В	SE B	β	р
Global PSQI	CTQ	.07	23.17	.06	.01	.26	<.001

Note. CTQ = Childhood Trauma Questionnaire; PSQI = Pittsburgh Sleep Quality Index.

#### TABLE 3b

Multiple Linear Regression Analyses Estimating Main and Interactions Effects for CTQ Total and Gender on Global PSQI

Dependent Variable	Predictor	<b>R</b> <sup>2</sup>	F	В	SE B	β	р
Global PSQI	Step 1: CTQ	.07	11.70	0.06	0.01	.26	<.001
	Gender	-0.20	0.37	03	.599		
	Step 2: CTQ	.08	9.65	-0.02	0.04	09	.581
	Gender	-2.61	1.12	38	.020		
	$CTQ \times Gender$	0.07	0.03	.52	.023		

Note. CTQ = Childhood Trauma Questionnaire; PSQI = Pittsburgh Sleep Quality Index.

### TABLE 4

Test of the Significance of Neuroticism as a Mediator of the Childhood Adversity–Adult Sleep Quality Relationship in Men and Women (5,000 Bootstraps)

95% Confidence Interval						
Variable	Point Estimate of the Indirect Effect (ab)	SE	Lower	Upper		
Men	.041	.016	.015	.079		
Women	.024	.006	.012	.038		

*Note.* P values for the indirect effect are not reported here as it assumes a normal sampling distribution of the indirect effect, which tends to be skewed (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Significance of indirect effect is instead determined by the 95% confidence interval. ab = indirect effect.