Manuscript Title: Association Between Sleep, Childhood Trauma and Psychosis-Like Experiences

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Abstract

Psychosis-like experiences (PLEs), or attenuated positive symptoms of psychosis, present along a severity continuum and have been associated with distressing thoughts and impairments in functioning. Although knowledge of the clinical importance of PLEs is expanding, risk factors for their expression are still poorly understood. Sleep disturbances are one known factor that exacerbate PLEs expression and distress, and trauma exposure is associated with occurrence of PLEs, as well as increased risk of later sleep difficulties. This study examined the joint influences of sleep and trauma on PLEs in an undergraduate sample. Self-report questionnaires on presence and distress of PLEs, sleep problems, and occurrence of previous traumatic experiences were completed by participants (N=409). In order to determine the unique impact of sleep on PLEs, three sets of predictors: sociodemographic, psychosocial (including trauma), and sleep were entered in steps into a hierarchical multiple regression model. In the final model, specific sleep domains uniquely predicted PLEs, while previous trauma exposure, which was a significant predictor when entered in step two with other psychosocial variables, was no longer a significant predictor. Results suggest the possibility that disruptions in sleep following or occurring alongside a traumatic experience may somehow contribute to, or exacerbate the presence of PLEs.

Key Words: psychosis, psychosis-like experiences, sleep, trauma, adolescents

1.0 Introduction

Psychosis-like experiences (PLEs) occur in the general population, at times causing distress or impairment. Research indicates that experiencing a previous traumatic event may predispose individuals for expression of PLEs (Read et al., 2005). In addition, disruption in sleep, a frequent consequence of trauma, also has demonstrated associations with increases in PLEs (Reeve et al., 2015). There is little understanding; however, of possible associated risk factors, symptom overlap, or possible underlying mechanisms within and between these three factors.

1.1 Psychosis-like Experiences

Attenuated positive symptoms of psychosis, or PLEs, are present in roughly 8% of the general population (Van Os et al., 2009). Individuals experiencing PLEs share demographic, etiological, and psychopathological risk factors with those experiencing psychotic disorders (Linscott and Van Os, 2013), suggesting that a better understanding of PLEs will allow a more complete picture of the psychosis spectrum. PLEs present along a severity continuum. Some are mild and transient, not leading to distress or impairment, others are more severe and persistent but do not progress to psychosis (Yung et al., 2009), whereas still others precede the onset of clinical psychosis (Dominguez et al., 2010). Regardless of their association with later psychosis, it remains important to elucidate factors related to PLEs as they have been associated with impairments in functioning (Yung et al., 2005; Kelleher et al., 2014), help-seeking behavior (DeVylder et al., 2014), psychiatric diagnoses (Kelleher et al., 2012; DeVylder et al., 2014), and thoughts or actions of self-harm (Kelleher et al., 2013; DeVylder et al., 2015). These patterns have been observed in both community and college-ascertained samples. Individuals with PLEs are further at a greater risk to experience comorbid disturbances in mood (Calkins et al., 2014),

and higher levels of substance use (Kelleher & Cannon, 2011), and both mood and substance use has the potential to exasperate presence of PLEs (Krabbendam et al., 2005; Barkus et al., 2006). Yet, despite a growing understanding of the clinical and conceptual importance of PLEs, the joint contributions of environmental and biobehavioral risk factors for their expression are less clear.

1.2 Sleep disruption and PLEs

Sleep disturbance is one factor that appears to increase the risk for the expression of PLEs. A range of sleep problems are observed across the psychosis spectrum (Cohrs, 2008; Lunsford-Avery and Mittal, 2013; Koyanagi and Stickley, 2015; Davies et al., 2017; Poe et al., 2017), including among clinical and community samples of adults (Oh et al., 2016) and youth experiencing PLEs (Nishida et al., 2008; Oshima et al., 2010; Lee et al., 2012; Fisher et al., 2014; Jeppesen et al., 2015; Reeve et al., 2015; Taylor et al., 2015; Thompson et al., 2015). Such associations between sleep disturbance and PLEs presentation are consistent in international studies and across multiple cultural contexts (Koyanagi and Stickley, 2015; Oh et al., 2016). Several specific sleep disorders have been associated with PLEs, including insomnia, fragmented sleep, night anxiety, movement at night, and sensations at night (definitions of specific sleep disruptions are outlined in Table 1) (Kanetia et al., 2006; Oh et al., 2016; Andorko et al., 2017). These sleep disturbances are known to impact stress tolerance, immunological functioning, and cognitive functioning, as well as to exacerbate socioemotional distress (Hofstetter et al., 2005; Bromundt et al., 2011; Waters et al., 2011; Kelly and El-Sheikh, 2014; Poe et al., 2017), all of which are considered to play roles in the etiology of psychosis (Walker et al., 2008; Bergink et al., 2014; Bora and Murray, 2014). Polysomnographic and brain imaging studies further indicate that neural structures regulating sleep (thalamus, cortical gray matter) are also affected in

individuals furthest along the psychosis continuum (i.e., schizophrenia) (Lunsford-Avery and Mittal., 2013).

Additionally, acute episodes of sleep deprivation have been linked with sudden onset of PLEs in individuals with no prior history of such experiences (Orzel-Gryglewska, 2010; Petrovsky et al., 2014). In these occurrences, PLEs rarely persist following the regaining of typical sleep hours, and are likely caused by decreases in metabolism of glucose within the prefrontal cortex, a known consequence of continuous states of wakefulness (Thomas et al., 2000). The prefrontal cortex is a neural region with significant associations to PLEs and schizophrenia (Hill et al., 2004). Given such pervasive relations between sleep and PLEs, sleep disturbance is increasingly recognized as a core pathophysiological feature of the psychosis spectrum (Yates, 2016).

1.3 Trauma and PLEs

Given the proposition that the neurobiological consequences of sleep dysfunction are related to abnormalities observed in the psychosis spectrum, a useful extension of this work could be to identify shared, mechanistically plausible risk factors for the two phenomena. One such factor is childhood trauma exposure. Prevalence estimates indicate that the majority of adults (89.7%) have experienced exposure to some sort of traumatic event (Kilpatrick et al., 2013). Longitudinal, cross-sectional, and meta analytic studies indicate that physical abuse, unwanted sexual experiences, exposure to domestic violence, and bully and police victimization are associated with the occurrence and severity of PLEs (Read et al., 2005; Spauwen et al., 2006; Schreier et al., 2009; Fisher et al., 2013; Gibson et al., 2016; Grivel et al., 2017). Research suggests that trauma exposure is related to PLEs by virtue of its tendency to disrupt cognitive, emotional, and stress-regulatory systems (Morrison, Frame, and Larkin, 2003; Van Os, 2009), all

of which are also affected by sleep dysfunction. Consistent with this possibility, researchers have found that high levels of stress sensitivity (Gibson et al., 2016) and anxiety (Freeman and Fowler, 2009) – both common consequences of trauma (Cicchetti and Toth, 2005) – mediate the relation between childhood trauma exposure and PLEs. Like sleep dysfunction, childhood trauma exposure is also associated with abnormalities in immunological and cognitive functioning (Gibson et al., 2016).

1.4 Trauma, Sleep Disruption and Distress

A history of acute or chronic trauma exposure is associated with an increased risk of later sleep difficulties (Lavie, 2001; Singh and Kenney, 2013). Sleep is generally impacted both immediately following a trauma and in the long-term (Sadeh, 1996), with affected sleep domains including nightmares, fragmented sleep, initial insomnia, fatigue, sensations at night, light sleep, and night anxiety (Lavie, 2001; Germain et al., 2008; Spilsbury et al., 2014; Ho et al., 2016). These effects on normal sleep processes are considered one of the most frequent and distressing complaints following a traumatic event (Nappi et al., 2012). In addition, longitudinal studies indicate that sleep disruption, prior to and following trauma exposure leads to exacerbation of subsequent trauma-related distress (Koren et al., 2002), specifically for those with pre-existing insomnia or demonstrating less resiliency overall (Gehrman, et al., 2013; Seelig et al., 2016). Further, nightmares can serve as an indicator of the degree to which a stressful event becomes psychologically traumatic (Thompson et al., 2015). Ultimately, the direction of this signal is unclear, however, as it may be that sleep disturbances after a psychological trauma exacerbate the traumatic response, or that the severity of the trauma leads to both sleep disturbances as well as a more severe response to trauma (Koren et al., 2002).

In addition to speculating on causal relations between sleep disturbances and trauma, Thompson and colleagues (2015) report a higher prevalence of psychotic experiences at age 18 in individuals who reported nightmares at age 12. The authors suggest that psychosis and psychological trauma may share the similar mechanisms of affective network dysfunction. Further, in order to better understand the relations between all three variables, Thompson and colleagues called for future research elucidating the links between trauma, psychosis, and sleep disturbances. Despite the interrelations of sleep disturbances to both trauma exposure and PLEs, however, no study to date has examined these domains together. This remains an important gap in the literature, given the degree of overlap and potential for shared mechanisms in the development and exacerbation of PLEs.

1.5 Current study

The present study sought to examine the joint influences of childhood trauma and sleep problems on the severity of PLEs in a sample of undergraduate students, a young-adult sample at peak age to develop PLEs (Thompson et al., 2004), and who are especially vulnerable to dysregulated sleep schedules (Brown, Buboltz, & Soper, 2002). A comprehensive measure of sleep containing specific sleep scales related to insomnia, lassitude, and parasomnia was used to probe effects of specific sleep disorders. As recent research suggests trauma is associated with both sleep disruptions and increase in PLEs (Grivel et al., 2017), and sleep is independently linked with PLEs (Poe et al., 2017), and further may be a core pathophysiological feature of the psychosis spectrum that may be independent of a prior history of trauma, disordered sleep was hypothesized to account for significant variance in PLEs above and beyond prior trauma when considering both simultaneously.

2.0 Methods

2.1 Participants

A sample of undergraduate students (*N*=420) were recruited from introductory psychology courses at the University of Maryland, Baltimore County (UMBC) between Fall 2010 and Spring 2014. All participants completed a larger battery of self-report measures, which included the questionnaires reported in this manuscript among others. Eleven participants (2.6%) did not complete any of the questionnaires presented in the current study and were therefore excluded from the final sample (N = 409). There is some inconsistency in completion of the various measures, such that not all participants completed all measures. For detailed overview of missing data refer to Table 2. All participants were at least 18 years old and were offered extra credit toward their final grade for their participation. This study was approved by the UMBC institutional review board (IRB).

2.2 Procedure

This study took place within the YouthFIRST laboratory at the University of Maryland, Baltimore County (UMBC). Prior to participation, all individuals received a written and verbal overview of the study, provided a chance to ask questions, and completed written consent. As part of a larger battery of questionnaires, participants reported on demographic information and drug-use history as well as completed self-reports assessing attenuated symptoms of psychosis (the Prodromal Questionnaire-Brief; Loewy and Cannon, 2010; Loewy et al., 2011), previous traumatic experiences (General Trauma Questionnaire – Revised; Bechdolf et al., 2010), sleep disturbances (Iowa Sleep Disturbances Inventory; Koffel, 2011), and depressive symptoms (Beck Depression Inventory-II; Beck et al., 1996).

2.3 Measures

2.3.1 The Prodromal Questionnaire-Brief (PQ-B). The PQ-B is a 21-item questionnaire examining the presence/absence of attenuated symptoms of psychosis within the past four weeks. It also probes associated impacts on social functioning, academic/occupational functioning, as well as any related distress. Following the positive endorsement of an item, participants are then asked to rate the associated distress on a 5-point Likert-type scale, with higher scores reflecting higher distress. Total scores were calculated by summing positive endorsements and distress items, resulting in total scores ranging from 0-105. Participants are asked to only report on experiences occurring outside the use of drugs or alcohol. Psychometrics of the PQ-B measure are acceptable in clinical, community, and undergraduate populations (DeVylder et al., 2015; Kline et al., 2012; Kline et al., 2015; Loewy et al., 2011; Mittal et al., 2012; Mittal et al., 2013).

2.3.2 The General Trauma Questionnaire-Revised (GTQ-R). The GTQ-R is a 12-item self-report measure assessing occurrence of physical, emotional, and sexual traumas as defined by the Diagnostic and Statistical Manual, 5th Edition (DSM-5; APA, 2013). Respondents indicate whether they have experienced specific traumatic events (yes/no) for a total score ranging from 0-12. Specific traumas probed include: combat experience, life-threatening accident, fire, flood, or natural disaster, witnessing bad injury or death, rape, sexual molestation, physical attack or assault, physical abuse, serious childhood neglect, threatened with weapon, held captive or kidnapped, other (any other terrible experience that most people never experience), and suffering shock because one of these events happened to someone close to you. For the main regression analysis, overall incidence of occurrence of trauma was used. As such scores were dichotomized

into yes, presence of any previous traumatic event, versus no presence.[1] In order to better describe incidence of specific types of trauma, Pearson correlations included specific types of trauma in which each of the individual items (*n*= 12) were examined dichotomously (event occurred: yes, no). The GTQ has been used in previous studies of the psychosis spectrum (Bechdolf et al., 2010).

2.3.3 The Iowa Sleep Disturbances Inventory (ISDI). The ISDI is an 86-item self-report measure of general sleep habits. The measure probes eleven separate sleep domains, which can be considered falling into one of three sleep disturbance categories: insomnias or related scales, lassitude, and parasomnias. Specific scales include: nightmares (12 items), initial insomnia (11 items), fatigue (10 items), fragmented sleep (9 items), non-restorative sleep (8 items), anxiety at night (7 items), light sleep (6 items), movement at night (6 items), sensations at night (6 items), excessive sleep (6 items), and irregular sleep (5 items). The presence/absence of each item is probed. Total scores represent the sum of all endorsed items, resulting in a total score range of 0-86. Individual sleep subscales are calculated in the same manner. Score ranges of subscales vary based on number of items within each scale. The psychometric properties of the measure are shown to be acceptable within both clinical and community samples (Koffel and Watson, 2010; Koffel, 2011).

2.3.4 The Beck Depression Inventory – Second Edition (BDI-II). The BDI-II is a 21-item questionnaire assessing symptoms of depression within the past two weeks. Scores are obtained by summing all responses and higher scores indicate a greater severity of depressive symptoms, with a total possible range of 0-63. The BDI-II demonstrates acceptable psychometric properties in clinical and non-clinical samples as well as in an ethnically diverse sample of

[1] Trauma and sleep were also considered as total counts. Patterns of findings remained consistent with the reported findings. Data available upon request.

college students (Beck et al., 1996; Carmody, 2005). Further, the BDI-II is the superior measure of depression among youth with early psychosis (DeVylder et al., 2014).

2.3.5 Drug Use Questionnaire. This drug-use questionnaire is a self-report assessing current and past drug use. The measure details frequency, timing, and duration of use as well as any associated impairment. For this study, recent drug use was measured on a dichotomous scale: drug was used within the past 6 months (yes/no).

2.4 Statistical Analyses

Pearson correlations were examined between main study variables, including specific types of physical trauma and demographic variables (age, gender, and race). In order to determine the unique impact of sleep parameters on PLEs, two sets of predictors (psychosocial and sleep parameters) were entered into a hierarchical multiple regression model. Of note, demographic variables were not significantly correlated with any main study variables (PLEs, sleep, or trauma), thus were not included in the regression model. In the first step of the regression, we entered trauma, depressive symptoms and drug use. Trauma is the main variable of interest. Depressive symptoms and drug use have previously been linked to presence of PLEs and the experience of trauma and thus were included as other psychosocial potential confounds (Brady et al., 2004; Chapman et al., 2004; Gibb et al., 2007; Freeman and Fowler, 2009; Kelleher and Cannon, 2011). They were also found to be significantly correlated with main study variables. In the second step, we entered all 11 specific subscales of sleep. Change in R^2 was calculated to determine if the set of sleep parameters significantly added to the prediction of PLEs above beyond the effects of the demographic and psychosocial predictors. T-tests of the specific sleep parameters beta weights were used to determine if specific sleep problems were uniquely related to PLEs while controlling for the other variables in the model. Listwise deletion

was used in cases of missing data. An alpha level of .05 was used to determine statistical significance.

3.0 Results

The average sample age was 20.10 (SD = 3.22). The sample was 50.6% female, and comprised of individuals from a diverse set of ethnicities (34.5% Asian, 33.7% White, 19.8% Black, 2.4% American Indian, and 7.8% "Other"; please see Supplementary Table 1 for distributions of study measures by race). Of the sample, 48.7% endorsed at least one previous traumatic experience, and 81.5% endorsed at least one item on the PQ-B (percentages are consistent with findings from Read and colleagues, 2011; and Fonseca-Pedrero and colleagues, 2016, respectively). Main measures, with the exception of trauma, but including those for sleep and PLEs demonstrated acceptable criteria for parametric analyses (Curran et al., 1996, skewness < 2.0, kurtosis < 7.0; see Table 2 for means, standard deviations, skewness, and kurtosis of main study measures). At least one sleep problem was endorsed by every participant, most common problems include: nightmares (96.0%), night anxiety (89.4%), and non-restorative sleep (85.8%). Just over a quarter of the sample (26.4%) noted use of drugs within the past year.

Pearson product moment correlations (matrix found in Table 3) indicated that previous traumatic experiences significantly correlated with PLEs and sleep dysfunction, and sleep dysfunction significantly correlated with PLEs. When examining specific types of traumas separately, significant correlations with PLEs included: experiencing a life-threatening event, involvement in a fire, flood, or natural disaster, other (any other terrible experience), and the experience of a great shock because a traumatic event happened to someone close to you. Similarly, total sleep dysfunction was also significantly correlated with these same four traumatic experiences (life-threatening event, fire, flood, or natural disaster, other, and traumatic

event to someone close), along with witness to someone being injured or killed, and sexual molestation. Demographic variables (age, gender, and race) were not significantly correlated with any main study measure (PLEs, trauma, or sleep).

Results of the hierarchical regression analysis are reported in Table 4. Psychosocial variables predicted a significant amount of variance of PLEs, and sleep disturbances predicted an additional increment of the variance above psychosocial variables. The final model for the regression including all variables was significant. Multicollinearity between predictors within the final model was checked through examination of tolerance and variance inflation factor (VIF) and determined to be acceptable as no variable had a tolerance level lower than .300, or a VIF higher than 3.00 (Rovai, Baker, & Ponton, 2014).

In the final model, examining each predictor individually, nightmares, fragmented sleep, and depressive symptoms all uniquely predicted PLEs. Previous trauma exposure was a significant predictor within the first step when entered with other psychosocial variables; however, once sleep subscales were included, trauma was no longer a significant predictor.

4.0 Discussion

Previous studies have examined the singular effects of sleep disruption on PLEs, and prior traumatic experience on PLEs, however, to our knowledge, this is the first study to examine the unique contributions of each. Our hierarchical regression examined the effect of sleep dysfunction on presence of PLEs above and beyond that of other psychosocial (depressive symptoms, drug use, and previous traumatic experience) variables. After accounting for confounding variables, the sleep domains of nightmares and fragmented sleep were significantly associated with the presence of PLEs, while all previously entered psychosocial variables, other than depressive symptoms, no longer significantly predicted PLEs. Depression remained significant, highlighting the importance of continuing to examine the pervasive role of mood irrespective of other variables in the presence of PLEs.

Extant literature consistently indicates an association between childhood traumatic experiences and PLEs (Misiak et al., 2017), as well as between sleep disturbances and PLEs (Koyanagi and Stickley, 2015; Andorko et al., 2017). Correlations in our sample between specific types of traumatic experiences, total sleep dysfunction, and PLEs indicated the same four traumatic events (experiencing a life-threatening event; involvement in a fire, flood, or natural disaster; the experience of a great shock because a traumatic event happened to someone close to you; and other [any other terrible experience];) were correlated with both sleep and PLEs. Endorsement frequency of the other traumatic events covered in the GTQ-R were too infrequent to draw conclusions about the relations between those items and sleep and PLEs.

Research examining the mechanisms driving the associations between trauma, sleep, and PLEs is limited, as focus of the extant literature is typically on the unique dyads of these variables. Childhood traumatic experiences often lead to disordered sleeping, disruptions that are

reported as particularly distressing (De Bellis and Zisk, 2014). Assuming that the traumatic events measured in this study preceded current assessment of sleep disruption, this study provides preliminary evidence that disruptions in sleep may be one mechanism driving the emergence of PLEs in individuals following traumatic situations.

Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis may represent a plausible biological link between trauma, sleep, and PLEs. Rodent and human studies show close, bidirectional relations between HPA and sleep functioning, such that manipulation of one process acutely disrupts the other (Buckley and Schatzberg, 2005; Steiger, 2002). For example, administration of glucocorticoids suppresses REM sleep, and in contrast, sleep restriction causes elevated cortisol, the final hormonal output of the HPA axis (Steiger, 2002). These basic mechanistic studies are complimented by a large body of literature indicating that adverse early life experiences lead to later cortisol regulation deficits, including elevated resting cortisol (Gunar and Quevedo, 2007; McEwen, 2012). In turn, studies of non-human primates demonstrate that adversity-induced cortisol elevations are closely tied to lower sleep efficiency (Barnett et al., 2009). These strong, presumably causal sleep-HPA relations have implications for the diathesis-stress model of psychosis, which highlights stress-related HPA dysregulation as a key underlying mechanism contributing to psychosis-spectrum symptoms, including PLEs (Walker et al, 2008). Sleep dysfunction and elevated HPA activity may be independent consequences of trauma exposure that interact with one another to ultimately lead to PLEs and other manifestations of psychosis.

The causal mechanisms behind the relation between all three factors had not previously been studied, given the clear biological connection all three share, future studies could continue to attempt to illuminate mechanistic links. In addition, future longitudinal studies could examine the

possibility that sleep mediates the relation between trauma and PLEs, such that traumatic events lead to sleep disruptions, which ultimately exacerbate PLEs. Given the cross-sectional nature of the data from this study, however, we were unable to effectively address this possible mediation.

4.1 Clinical Implications

PLEs represent a wide range of experiences, some known to be distressing and impairing (Armando et al., 2010). Elucidating factors relating to the presence or exacerbation of PLEs is important when focusing on treating individuals with these experiences. Previous traumatic events and dysregulated sleep are both known to be clinically relevant factors affecting PLEs presence; therefore, if a client is struggling with distressing PLEs, it may be especially important for clinicians to assess sleep, specifically nightmares or fragmented sleep, and possibly a prior history of trauma that might also be related. Taken together with prior longitudinal literature, results suggest that focused treatments of nightmares and associated sleep dysregulation, particularly fragmentation, might be relevant in facilitating psychological health among those with PLEs. Given that sleep disturbances are perceived to be associated with relatively low stigma in comparison to other mental health disorders (Stinson, Tang, & Harvey, 2006), results also add support to the usefulness of probing sleep disruption during initial mental health screening.

4.2 Limitations

Our sample was composed of undergraduates, likely limiting generalizability to other populations. Nonetheless, undergraduates present at an age of elevated risk for PLEs and sleep problems (Thompson et al., 2004; Lund et al., 2010), thus, making them a relevant population for this type of work. In addition, our methodology was cross-sectional and therefore limited in terms of inferences that could be made about causality. Regardless of causal relation, or direction

of effects, the implication that PLEs occurring post-traumatic event may be partially attributed to the occurrence of sleep dysregulation is an interesting research question, and possibly clinically useful. Trauma was assessed by a self-report questionnaire probing the occurrence of past physical trauma. It is important to note that the psychological impact of the event was not measured, and thus cannot be considered in the results. Occurrences of individual traumas limited the amount of interpretation possible with respect to the possible relation between specific traumatic experiences and sleep and PLEs. Exposure to substances that might impact sleep (e.g., caffeine, SSRIs) was not measured, but given that this was a typical functioning college sample, it is not likely that these issues would systematically impact current findings. Future studies might consider expanding on this work by employing more objective measures of sleep (e.g., formal sleep study).

4.3 Conclusions

This study examines associations between previous traumatic experience, sleep disruption, and presence of PLEs. It builds on previous literature that primarily focused on only the unique dyads of these variables, and consistently indicated an association between childhood traumatic experiences and PLEs, as well as between sleep disturbances and PLEs. Results from this study extend this literature, and postulate that disruptions in sleep following or occurring alongside a traumatic experience may somehow contribute to, or exacerbate the presence of PLEs. Sleep may therefore be an accessible target in interventions following traumatic experiences in order to attenuate the possibility of future PLEs.

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Table 1.

Brief definitions of sleep subscales.

Subscale	*Definition*
Insomnia	
Initial Insomnia	Difficulty initiating sleep in the beginning of the night.
Fragmented Sleep	Difficulty staying asleep.
Night Anxiety	Excessive worrying while trying to initiate sleep.
Light Sleep	Easily awoken during the night.
Lassitude	
Non-Restorative Sleep	Feeling that sleep has been insufficiently refreshing.
Excessive Sleep	Sleeping more than recommended (>12 hours).
Fatigue	Excessive sleepiness, or low energy during the day.
Parasomnia	
Nightmares	Unpleasant, frightening, or disturbing dreams.
Movement at Night	Recurrent movement (kicking, jerking, flailing) while asleep.
Sensations at Night	Unpleasant or uncomfortable feelings in limbs at night (restless legs).
Circadian Rhythm	
Irregular Schedule	Varying sleep and wake times.

Table 2.

Means and distributions of main study measures

	N	Mean / % Yes	S.D.	Range	Skewness	Kurtosis
Psychosis-like Experience	384	13.29	15.52	0-84	1.49	1.80
Previous Traumatic Event	384	48.7%	0.50	0-1	-0.07	-2.00
Sleep Dysfunction Total	403	34.35	16.56	0-84	0.42	-0.39
Nightmares	403	2.89	2.27	0-11	1.52	2.08
Initial Insomnia	397	4.78	3.95	0-11	0.29	-1.43
Fatigue	398	4.16	3.12	0-9	0.20	-1.34
Fragmented Sleep	402	2.36	2.33	0-9	0.99	0.35
Non Restorative Sleep	404	4.31	2.89	0-8	-0.17	-1.44
Night Anxiety	400	3.88	2.33	0-7	-0.20	-1.26
Light Sleep	400	2.53	2.26	0-6	0.36	-1.36
Movement at Night	403	2.46	2.11	0-6	0.32	-1.22
Sensations at Night	403	1.21	1.70	0-6	1.42	0.94
Excessive Sleep	400	2.23	1.84	0-6	0.36	-0.95
Irregular Schedule	405	2.42	1.82	0-5	0.00	-1.34
BDI-II	389	10.36	8.41	0-43	1.21	1.52
Drug use in the past year	409	26.4%	0.44	0-1	1.07	-0.85

Table 3.

Bivariate correlation matrix of specific traumatic events and main study measures (N = 409)

Measure (positively endorsed)	(1)	(2)	(3)	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)	(3g)	(3h)	(3i)	(3j)	(3k)	(3l)	(4)	(5)	(6)	(7)
(1) PQ-B Total (n = 338)	-																		
(2) Sleep Total (n = 409)	.530**	-																	
(3) Trauma Total (n = 199)	.215**	.202**	-																
(3a) Combat (n = 5)	-.074	.037	.111*	-															
(3b) Accident (n =91)	.146**	.107*	.510**	.100*	-														
(3c) Disaster (n =52)	.102*	.163**	.364**	-.043	.186**	-													
(3d) Witness (n =70)	.090	.102*	.439**	.126*	.284**	.120*	-												
(3e) Raped (n = 7)	.003	.053	.099	-.015	-.026	.062	.039	-											
(3f) Sexually molested (n = 22)	.077	.136**	.232**	-.027	.000	.102*	.033	.302**	-										
(3g) Physical attack (n = 28)	.093	.081	.254**	-.031	.225**	.074	.158**	.038	.197**	-									
(3h) Physical abuse (n = 18)	-.004	.072	.201**	.084	.114*	.060	.122*	.155**	.054	.224**	-								
(3i) Serious neglect (n = 12)	.060	.076	.166**	-.020	.011	-.024	-.004	.088	.086	.239**	.386**	-							
(3j) Threatened weapon (n = 21)	.072	.044	.220**	.074	.195**	.042	.246**	.063	-.057	.205**	.117*	.098	-						
(3k) Other (n = 44)	.123*	.210**	.342**	.032	.177**	.012	.099*	.044	.095	.095	.049	.184**	.073	-					
(3l) Another (n = 83)	.223**	.105*	.402**	-.048	.164**	.133**	.215**	-.021	.084	.106*	.090	.036	.133**	.189**	-				
(4) Depressive symptoms	.509**	.549**	.122*	-.023	.088	.074	-.012	.152**	.092	.081	.068	.183**	.032	.161**	.115*	-			
(5) Drug Use	.117*	.129**	.117*	-.017	.132**	.105*	.080	.049	.053	.101*	.060	.027	.089	.091	.054	.071	-		
(6) Age	-.058	.001	.011	.199**	-.026	.001	.051	.041	-.022	.053	-.015	.123*	.004	-.025	-.069	-.008	-.017	-	
(7) Gender	-.086	-.087	-.002	.069	.096	.021	.138**	-.094	-.151*	.083	-.046	.061	.195**	-.077	-.045	-.180*	.101*	.072	-
(8) Race	.095	.044	.020	.058	-.014	.031	-.037	-.039	.012	.038	-.156*	-.054	.021	.020	-.028	-.067	.192**	.116*	.083

** Correlations were significant at p value <0.001 (2-tailed); *Correlations were significant at p value <0.01 (2-tailed)

Table 4.

Hierarchical regression predicting psychosis-like experiences[a]

	Block I (Psychosocial variables)					**Block II (Sleep variables)**				
	Statistic				*p*	Statistic				*p*
R^2	.290					.404				
Change in R^2	.259				.000	.114				.000
Adjusted R^2	.276					.370				
F (df)	21.04 (6, 315)				.000	11.87 (17, 315)				.000
Variable	*B*	*SE B*	Beta	t	*p*	*B*	*SE B*	Beta	t	*p*
Entered in Block I										
Previous Traumatic Experience	4.86	1.47	0.16	3.31	.001	2.46	1.43	0.08	1.73	.085
Depressive Symptoms	0.85	0.09	0.47	9.93	.000	0.52	0.10	0.29	5.02	.000
Drug Use	2.53	1.67	0.07	1.52	.130	1.96	1.61	0.06	1.22	.224
Entered in Block II										
Nightmares						1.28	0.38	0.18	3.37	.001
Initial Insomnia						0.10	0.24	0.03	0.40	.688
Fatigue						0.48	0.36	0.10	1.32	.190
Fragmented Sleep						1.35	0.38	0.20	3.60	.000
Non-Restorative Sleep						0.16	0.35	0.03	0.44	.658
Night Anxiety						0.28	0.38	0.04	0.74	.459
Light Sleep						0.29	0.35	0.04	0.82	.411
Movement at Night						0.45	0.35	0.06	1.27	.199
Sensations at Night						-0.00	0.50	0.00	-0.01	.994
Excessive Sleep						0.21	0.44	0.03	0.47	.642
Irregular Sleep						-0.46	0.47	-0.06	-0.97	.332

[a]Psychosocial variables were entered in the first block, and sleep variables were entered in the second block.