Nightly Sleep Duration, Momentary Perceived Stress, and Experiences of Attenuated Positive Symptoms in Daily Life in Adolescents at Clinical High-Risk for Psychosis

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Background: Sleep dysfunction and stress abnormalities are prevalent in psychotic disorders (i.e., schizophrenia), therefore it may be valuable to examine these risk markers earlier on in the disease progression to minimize poor health outcomes and the risk of developing psychosis later in life. A nuanced diathesis-stress conceptualization asserts that sleep and stress dysfunction in adolescents at clinical high-risk (CHR) for psychosis – individuals experiencing attenuated positive symptoms (e.g., perceptual abnormalities, unusual thoughts) – may contribute to symptom progression and perhaps, increase the likelihood of transitioning to a psychotic disorder in a shorter window of time. However, our understanding of how sleep and stress abnormalities influence *day-to-day experiences* of attenuated positive symptoms in CHR adolescents remains limited. Thus, the current study aims to examine the day-to-day interrelationships between sleep habits, psychological stress, and psychosis-risk symptoms in CHR and non-CHR youth, with an eye toward identifying and targeting combinations of risk markers possibly contributing to the emergence of psychotic disorders later in life.

Methods: Twelve CHR and 15 non-CHR adolescents (ages 13-20) were recruited to the University of Pittsburgh. Ecological momentary assessment (EMA) was used to collect self-reported nightly sleep duration (i.e., total time asleep), momentary perceived stress (i.e., rating of anxiousness, nervousness, or pressure), and momentary psychosis-risk symptoms (i.e., number of perceptual disturbances and/or unusual thoughts) across two-week observation periods.

Results: There were no group differences in sleep duration or momentary stress. Group status moderated the association between within-person sleep duration and momentary stress in the whole sample, such that the noted association trended toward negative in the non-CHR group but was insignificant in the CHR group. Higher momentary stress, but not shorter sleep duration, was related to increased momentary psychosis-risk symptoms in the CHR group.

Conclusion: The current study provides a novel account of the presence of sleep and stress dysfunction in general adolescents and their influence on daily experiences of attenuated positive symptoms in CHR adolescents. Findings can inform how preventative programs possibly target dysfunctional sleep and stress processes in adolescence in order to minimize experiences of psychosis-risk symptoms in daily life and the risk of developing psychosis later in life.

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Preface

My independent project was conducted as part of Dr. Leslie Horton's larger R01-funded study (MH121386) within the Youth Emotions, Thoughts, and Interactions Laboratory (YETI Lab) at the University of Pittsburgh. Additionally, I received university funding from the David C. Frederick Honors College at the University of Pittsburgh during the 2023 summer term so that I could commit full-time efforts to advancing my independent research project for the Bachelor of Philosophy degree.

I would like to thank Dr. Tina Gupta for assisting in conceptualization and data analysis, writing, and providing valuable revisions throughout this process as well as acknowledge the work and efforts of Megan Deam and Emma Headley in data collection and compilation in the YETI Lab. I would also like to give a special thanks to Dr. Horton, director of the YETI Lab, for providing me with the opportunity to obtain hands-on experience with analyzing ecological momentary assessment data in her psychiatric laboratory and for inspiring me to pursue a doctoral degree in health psychology. Lastly, I would like to thank my close friends and family for supporting me in my pursuit of the Bachelor of Philosophy and long-term career in psychological research.

1.0 Introduction

Adolescence is a critical stage of development marked by the introduction of important psychosocial, environmental, and maturational stressors – such as increased prioritization of peer relationships, academic pressure, and puberty-related hormonal/physical changes (Núñez-Regueiro & Núñez-Regueiro, 2021). The influence of these stressors on neurobiological stress mechanisms results in greater susceptibility to the emergence of mental health symptoms, including psychosis-risk symptoms (e.g. symptoms such as seeing shadows or hearing whispers and unusual thoughts that may indicate an individual is at heightened likelihood of developing a psychotic disorder in a short timeframe) (van Winkel et al., 2008; Holtzman et al., 2013). Understanding psychosis-risk symptoms during this crucial life stage is important given that psychotic disorders (e.g., schizophrenia, first-episode of psychosis) tend to have debilitating outcomes (e.g., disability), and can be exacerbated by experiences including dysfunctional sleep behaviors (i.e., disruptions in the amount, quantity, timing, or quality of sleep (Locke, 2011)) and abnormal elevations in psychophysiological stress (Lunsford-Avery et al., 2015; Lunsford-Avery et al., 2017; Zak et al., 2022; LaGoy et al., 2022; Goines et al., 2019; Nordholm et al., 2023; Poe et al., 2017; Ristanovic et al., 2020; Millman et al., 2018; Phillips et al., 2020; Pruessner et al., 2013; Sugranyes et al., 2012; Thompson et al., 2007; Trotman et al., 2014).

According to the diathesis-stress model of schizophrenia (Walker & Diforio, 1997), there is a cyclical interaction between early vulnerability factors, neurodevelopmental alterations in the body's stress response system (i.e., hypothalamic-pituitary-adrenal (HPA) axis), and the onset of psychosis-risk symptoms during adolescence and the prodrome of psychosis (i.e., a developmental window during which individuals are vulnerable to experiencing psychosis-risk symptoms (Yung & McGorry, 1996)). A recent, nuanced perspective of the diathesis-stress

model posits that sleep dysfunction plays an integral role in altering neuromaturation and reactivity to biological/psychosocial stressors, which may further exacerbate the onset of psychosis-risk symptoms in adolescents who are at-risk for psychosis as well as the risk of developing psychosis later in life (Lunsford-Avery & Mittal, 2013).

Compelling evidence supports this conceptualization of the diathesis-stress model, demonstrating that individuals at clinical high-risk (CHR) for psychosis – characterized by the presence of attenuated symptoms of psychosis but do not meet the threshold for psychotic disorders, with evidence indicating they are at elevated risk of developing a first episode of psychosis within three year of identifying CHR symptoms (Fusar-Poli et al., 2013; De Pablo et al., 2021) – are particularly vulnerable to experiencing greater sleep dysfunction and stress abnormalities compared to non-CHR peers (Lunsford-Avery et al., 2015; Lunsford-Avery et al., 2017; Zak et al., 2022; LaGoy et al., 2022; Goines et al., 2019; Nordholm et al., 2023; Poe et al., 2017; Ristanovic et al., 2020; Millman et al., 2018; Phillips et al., 2020; Pruessner et al., 2013; Sugranyes et al., 2012; Thompson et al., 2007; Trotman et al., 2014). Furthermore, these two risk factors are implicated in worsening psychosis-risk symptoms during the prodrome and exacerbating the progression to psychosis in later adolescence and early adulthood (Zak et al., 2022; van der Tuin et al., 2023; Lunsford-Avery et al., 2015; Reeve et al., 2019; Goines et al., 2019; Mayeli et al., 2021; Blanchard, 2021; Poe et al., 2017; Meyer et al., 2021; DeVylder et al., 2013; Cullen et al., 2019; Cullen et al., 2020; Chaumette et al., 2016; Carol et al., 2021; Carol & Mittal, 2015; Millman et al., 2018; Trotman et al., 2014; Cullen et al., 2022; Georgiades et al., 2023). This underscores the importance of targeting disruptions in sleep and stress processes in at-risk populations and early on in the disease progression of psychosis.

Despite the growing attention to the roles of sleep and stress dysfunction in worsening the symptomology and progression of psychosis in CHR individuals, no known studies have explored the effects of these risk factors on moment-to-moment experiences of psychosis-risk symptoms in daily life, especially during the CHR period in adolescence. Additionally, our understanding of whether the natural, day-to-day relationship between sleep dysfunction (e.g., short sleep duration, often defined as receiving less than the recommended amount of sleep (CDC, 2022)) and moment-to-moment psychological stress is maintained or impaired in CHR adolescents compared to non-CHR peers remains limited. Investigating how sleep dysfunction and psychological stress impact experiences of psychosis-risk symptoms in daily life would provide an ecologically rich perspective of how these risk markers contribute to emergence of these symptoms during the prodrome. Further, exploring the state of the natural association between nightly sleep duration and next-day perceived stress in CHR adolescents, compared to non-CHR peers, may point toward a new combination of risk factors that can be targeted at earlier stages of psychosis development. Thus, the current study aims to examine the presence and day-to-day relationships between sleep dysfunction, psychological stress, and momentary experiences of psychosis-risk symptoms in CHR and non-CHR adolescents, with an eye toward identifying combinations of psychosis-risk factors that can be targeted at earlier stages of psychosis progression through treatment and early preventative programs.

1.1 Literature Review

1.1.1 Clinical High-Risk for Psychosis

Youth at clinical high-risk (CHR) for psychosis are classified as those who endorse the presence of attenuated symptoms of psychosis but do not meet the threshold for psychotic disorders (Fusar-Poli et al., 2013; Fusar-Poli, 2017). It is estimated that 25% of individuals at CHR develop psychosis within two to three years of baseline assessment, further increasing to 35% within 10 years (De Pablo et al., 2021). Furthermore, there is a high prevalence of comorbidity with other psychiatric disorders in individuals at CHR for psychosis – such as anxiety/mood, panic, alcohol use disorders – compared to non-CHR individuals (Solmi et al., 2023; Addington et al., 2017). This highlights the importance of pinpointing risk factors of psychosis – such as sleep dysfunction and stress abnormalities – to improve early identification, prevention, and intervention strategies. Additionally, examining these factors in those who do not convert to psychosis can also provide insight into what health factors may buffer against conversion and potentially foster resilience to symptoms (DeRosse & Barber, 2021; Marulanda & Addington, 2014; Kim et al., 2013).

One hallmark characteristic of CHR symptomology is the presence of attenuated positive symptoms. Attenuated positive symptoms are symptoms including perceptual disturbances (e.g., hearing whispers or seeing shadows), unusual thought content (e.g., believing one's thoughts are being broadcasted out loud), unusual speech, suspiciousness/paranoid ideation, and grandiosity (Miller et al., 2002, Miller et al., 2003). To illustrate attenuated positive symptoms, here is a case example from Cadenhead and Mirzakhanian (2016): Aaron is a 17-year-old high school student at CHR for psychosis. Aaron reports hearing random ringing sounds, seeing dark areas out of the

corner of his eyes, experiencing tingling down the middle of his back, and feeling cold for no reason. Additionally, he reported that he consistently wondered if the TV programs he was watching were a "message just for [him]", if people knew things about him even though they could not read his mind, or that his girlfriend may have tried to poison him right before the breakup of the relationship. Despite the onset of symptoms, Aaron maintained some degree of insight that these attenuated positive symptoms may not be real. Aaron's doubt in his symptoms indicates that he had not yet reached the clinical threshold for a full-blown psychotic episode, which necessitates full conviction that one's experiences of attenuated symptoms of psychosis are real (David, 1990). Additionally, Aaron showed declines in academic, social, and overall functioning – such as drops in grades, increased isolation from peers, unemployment, and poor basic hygiene.

Although there is heterogeneity in CHR symptoms across individuals, it is well-established that the presence of CHR symptoms can have impacts on quality of life. More specifically, experiences of attenuated positive symptoms are often accompanied by high levels of subjective distress, impaired functioning, and declines in several domains of life (Rekhi et al., 2017; Rapado-Castro et al., 2015; Hazan et al., 2020; Nelson et al., 2022), all of which are predictive of conversion to psychosis (Carrión et al., 2016; Rekhi et al., 2017; Nelson et al., 2022; Rapado-Castro et al., 2015). For example, for some CHR youth, symptoms can interfere with social functioning and school performance, causing adolescents to withdraw from social relationships and academics (Addington et al., 2008; Carrión et al., 2013; Piskulic et al., 2012). CHR symptoms can also interfere with other behaviors including hobbies and even hygiene (Alderman et al., 2014; Corcoran et al., 2010; Kuhney et al., 2022). Given the impacts of CHR

symptoms, understanding factors contributing to the progression of these symptoms is a research priority in the field.

1.1.2 Neurodevelopmental Diathesis-Stress Conceptualization of Sleep Dysfunction

As briefly discussed, the neural diathesis-stress model is often used as a foundation for examining risk markers of psychosis, with an emphasis on the role of stress exposure in exacerbating the disease progression from birth to the onset of psychosis (Walker & Diforio, 1997). To further expand, this model posits that there is a pivotal interaction between early vulnerability factors (i.e., the confluence of genetic/inherited and environmental vulnerability to which individuals are predisposed during prenatal and postnatal development (Brown et al., 2000; Buka et al., 2008; Davies et al., 2020, Fusar-Poli et al., 2017; Os et al., 2010; Mittal et al., 2008)), exposure to biological/psychosocial stressors, and neuromaturational factors during adolescence. The accumulation of these interactions can lead to the development of psychopathology (Mayo et al., 2017; Fusar-Poli et al., 2013) and increases in psychosis-risk throughout this critical stage of development (Addington et al., 2019; Corcoran et al., 2003; Gibson et al., 2016; Ristanovic et al., 2020; Vargas et al., 2020, p. 20; Walker et al., 2008; Yee et al., 2019). During this period of heightened risk (i.e., CHR period), new risk factors and psychosis-risk symptoms can emerge, which may further exacerbate the onset of psychosis in vulnerable youth.

Lunsford-Avery and Mittal (2013)'s neurodevelopmental diathesis-stress conceptualization of sleep dysfunction provides a nuanced perspective of the traditional diathesis-stress model of psychosis, positing that sleep dysfunction may be an important risk factor contributing to the onset of psychosis. To elaborate, sleep dysfunction, which is affected

by early vulnerability factors (Davies et al., 2017; Wee et al., 2019), has reciprocal associations with various aspects of neurodevelopment in adolescence – such as changes in cognitive functioning, neuromaturation, and exposure to biological/psychosocial stress (Lunsford-Avery et al., 2013; Ristanovic et al., 2022; Hennig et al., 2020; Ferrarelli, 2020; Zanini et al., 2013). These cyclical interactions may further lead to greater susceptibility to the emergence of psychosis-risk symptoms in vulnerable youth, which in turn may contribute to driving the onset of psychosis in late adolescence and early adulthood (see Figure 1).

1.1.3 Evidence of Sleep Dysfunction and Stress Abnormalities Across the Psychosis Spectrum

Sleep dysfunction is broadly characterized by disruptions in the amount, quantity, timing, or quality of sleep – such as shortened sleep duration, poor sleep quality, difficulties falling asleep at night or waking up in the morning, excessive daytime exhaustion, and more (Locke, 2011). The Centers for Disease Control and Prevention (2022) recommends that teenagers receive at least 8-10 hours of sleep per night, delineating short sleep duration as any length of sleep that falls below that range – although the cutoff varies across the literature.

Compelling evidence demonstrates that sleep dysfunction is more prevalent in both CHR individuals and those diagnosed with psychotic disorders compared to general populations (Zak et al., 2022; LaGoy et al., 2022; Goines et al., 2019; Nordholm et al., 2023; Poe et al., 2017). Specifically, CHR youth display poor physiological sleep outcomes including shorter sleep duration, increased sleep latency (i.e., time it takes to fall asleep) and movements during sleep, disruptions in sleep continuity, decreased sleep efficiency, and greater overall sleep disturbances (Lunsford-Avery et al., 2013; Lunsford-Avery et al., 2015; Clarke et al., 2020; Nuzum et al.,

2022). Interestingly, Nordholm et al. (2023) found that in addition to subjectively reporting more difficulty falling asleep, problems waking up, repeated and premature awakening, and sleep as less refreshing, at-risk individuals experienced *more* sleep on average than control peers, suggesting that excessive sleep duration may be another, perhaps group-specific, health consequence of being at-risk for psychosis. These poor sleep outcomes are concerning considering that sleep dysfunction has a bidirectional relationship with psychosis-related outcomes (Zak et al., 2022; van der Tuin et al., 2023; Lunsford-Avery et al., 2015; Reeve et al., 2019; Goines et al., 2019; Mayeli et al., 2021; Blanchard, 2021; Poe et al., 2017; Meyer et al., 2021). For example, greater sleep dysfunction is associated with higher frequency and severity of attenuated positive symptoms and worse overall functioning in CHR samples (Goines et al., 2019; LaGoy et al., 2022; Lunsford-Avery et al., 2013; Lunsford-Avery et al., 2015; Poe et al., 2017; Reeve et al., 2015). Pertinent to the current study, Reeves et al., (2019a) found that shorter sleep duration was associated with more severe delusional ideas and hallucinations cross-sectionally and longitudinally in a large at-risk sample. Conversely, increased severity and frequency of perceptual abnormalities, but unusual thought content, predicted sleep problems (defined in the study as disturbed sleep and/or insomnia), and sleep problems further shortened the time to which CHR individuals transitioned to frank psychosis (Nuzum et al., 2022). In all, robust evidence supports the presence of sleep dysfunction as well as its influence on attenuated positive symptoms and the risk of transitioning to full-blown psychosis in individuals at-risk for and with psychotic disorders.

In addition to sleep dysfunction, stress abnormalities are also implicated in Lunsford-Avery and Mittal (2013)'s model due to their high prevalence in at-risk samples and psychotic disorders as well as their established contribution to the onset of psychosis. Numerous studies

have shown that CHR individuals exhibit elevated subjective stress, resting cortisol levels (a measure of physiological stress) and a blunted cortisol response to psychosocial stress (Georgiades et al., 2023; Ristanovic et al., 2020; Millman et al., 2018; Phillips et al., 2020; Pruessner et al., 2013; Sugranyes et al., 2012; Thompson et al., 2007; Trotman et al., 2014; Dauvermann & Donohoe, 2019; Söder et al., 2020; Shah et al., 2023; Carol & Mittal, 2015; Carol et al., 2021; Shah et al., 2023). Additionally, those diagnosed with and at-risk for psychotic disorders tend to display a blunted cortisol awakening response (Day et al., 2014; Cullen et al., 2014) and heightened stress sensitivity to stressors (i.e., experience more stress following stress exposure) (Ristanovic et al., 2020; van der Steen et al., 2017; Carol et al., 2021; Docherty et al., 2009), which may be indicative of dysfunction in biological stress and sleep mechanisms. Similar to sleep dysfunction, alterations in psychophysiological stress has been found to worsen the frequency and severity of psychotic symptoms in samples at-risk and diagnosed with psychotic disorders (Cullen et al., 2021; DeVylder et al., 2013; Millman et al., 2018; Muñoz-Samons et al., 2021; Palmier et al., 2012; Pruessner et al., 2011; Ristanovic et al., 2020; Zhou et al., 2024; Walker et al., 2013; Chaumette et al., 2015), as well as increase the risk of transitioning to full-blown psychosis in at-risk individuals (Georgiades et al., 2023; Walker et al., 2013). Interestingly, Cullen et al. (2022) found that the presence of daily stressors and elevations in diurnal cortisol in late childhood/early adolescence increases the risk for developing attenuated psychotic symptoms in early adulthood, underscoring how early life stressors contribute to the emergence of psychotic symptoms later in life. Colizzi et al. (2023)'s study further suggests that patients who experienced any stressful life event following the onset of first episode of psychosis had a significantly higher risk of relapse (i.e., operationalized as hospitalization) compared to those who did not experience any stressful life event. Conversely,

as previously discussed, CHR individuals often report feelings of subjective distress in response to experiences of CHR symptoms (Rekhi et al., 2017; Rapado-Castro et al., 2015; Hazan et al., 2020; Nelson et al., 2022), demonstrating the vicious cycle of biological/psychosocial stress and psychosis-risk symptoms during the prodrome of psychosis.

1.1.4 The Natural Relationship Between Sleep and Stress in the General Population

Regardless of heightened vulnerability to psychosis or underlying psychopathology, the evolution of sleep health throughout the course of adolescent development can be described as "the perfect storm" (Carskadon, 2011). Similar to Lunsford-Avery and Mittal (2013)'s diathesis-stress conceptualization, the confluence of psychosocial/environmental (e.g., technology use before bed, parental-set bedtimes, early start times in secondary educational institutions, academic and social pressure) and neurodevelopmental factors contribute to changes and disruptions in sleep patterns in general adolescents. For example, national trends suggest that there is a high prevalence of short sleep duration amongst adolescents, which gradually increased from 69.1% in 2009 to 77.9% in 2019 (Centers for Disease Control and Prevention (CDC), 2022). This high prevalence is alarming considering that short sleep duration and other indicators of sleep dysfunction have debilitating health outcomes – such as inattention, poor grades, problematic behavior, lack of behavior, and substance use (Carskadon, 2011; Crowley et al., 2019)). Pertinent to the current study, short sleep duration and general sleep dysfunction are associated with elevations in next-day psychophysiological stress (Yap et al., 2020; Kim et al., 2019; Kim & Lee, 2018; Leproult et al., 1997; Schwarz et al., 2018) and increased psychotic-like experiences (i.e., subclinical experiences of hallucinations and/or delusions that do not reach threshold or clinical relevance (Linscott & van Os, 2012)) in adult and adolescent samples not at-

risk or diagnosed with psychotic disorders (Zhou et al., 2022; Clarke et al., 2020; Koyanagi & Stickley, 2015; Lee et al., 2012). Moreover, higher perceived stress and stressful life events were also associated with increased psychotic-like experiences in general populations (Turley et al., 2019; Shakoor et al., 2018). Thus, these findings demonstrate the detrimental outcomes of disrupted sleep patterns on everyday stress processes and the emergence of psychotic-like symptoms in general populations that are not at-risk or diagnosed with psychotic disorders.

1.1.5 Ecological Momentary Assessment

One way to understand day-to-day symptoms, sleep habits, and stress levels is using methods that capture fluctuations in daily experiences. One approach is Ecological Momentary Assessment (EMA), a sampling method that involves administering multiple assessments over time and within individuals (Schiffmann et al., 2008). EMA is uniquely suited to capture day-to-day and moment-to-moment fluctuations in everyday experiences – such as thoughts, affect, behaviors, physiology, and even clinical symptoms – in real-time and in individuals' natural environment. Traditional sampling methods (e.g., cross-sectional assessments) are not as well equipped to assess these temporal processes, as they often depend on retrospective and/or aggregated reports of behaviors across an extended timeframe (e.g., "During the past month, how many hours of sleep did you usually get at night?" (Buysse et al., 1989)) and are collected outside of individuals' everyday environment (e.g., laboratory setting). Thus, EMA is often preferred to traditional methods because it maximizes ecological validity, minimizes recall bias, and allows for the examination of dynamic processes over time (Kaurin et al., 2023; Wright & Zimmermann, 2019; Trull & Ebner-Priemer, 2020). Furthermore, EMA has been shown to produce reliable data of everyday experiences in at-risk populations (Myin-Germeys et al.,

2001). Therefore, the current study uses EMA as it is an ideal method to investigate the dynamic, day-to-day fluctuations and interrelationships between sleep duration, momentary perceived stress, and momentary experiences of attenuated positive symptoms in our CHR adolescent sample.

1.1.6 Summary

It is evident that sleep dysfunction and stress abnormalities play important roles in exacerbating the symptomology and disease progression of psychosis during the prodrome. However, our understanding of how these risk factors influence momentary experiences of attenuated positive symptoms in daily life and in a psychological context remains limited, especially in adolescents at CHR for psychosis. Furthermore, having a better understanding of whether there are disruptions in the natural, day-to-day association between health sleep habits and perceived stress in CHR adolescents compared to non-CHR could improve our understanding of further disruptions in sleep and stress processes, such as dysfunctional stress reactivity following nights of poor sleep. In all, investigating the dynamic interrelationships between nightly sleep duration, perceived stress, and momentary experiences of attenuated positive symptoms in daily life in both CHR adolescents and general peers may provide insight into the presence of sleep and stress dysfunction as well as how the combination of these two risk markers influences the emergence of psychosis-risk symptoms during a crucial stage of adolescent development and the prodrome of psychosis.

1.2 Current Study: Aims and Hypotheses

The current study aimed to examine the presence of sleep and stress dysfunction in CHR adolescents and non-CHR peers, as well as their effects on experiences of attenuated positive symptoms in daily life in the CHR sample. Additionally, we sought to explore how the day-to-day relationship between nightly sleep duration and next-day psychological stress differed between CHR and non-CHR adolescents.

See Figure 2 for the conceptual model of the primary hypotheses. First, we aimed to determine whether (*H1*) CHR adolescents would report sleeping less at night and (*H2*) experiences of higher moment-to-moment stress in daily life compared to non-CHR adolescents. Second, (*H3a*) we assessed if there was an association between nightly sleep duration and next-day momentary stress in the whole sample, hypothesizing that all adolescents, regardless of risk status, would experience higher momentary stress when they received less sleep than usual the prior night. For specificity, we examined if (*H3b*) group status (i.e., CHR vs non-CHR) would moderate the association between sleep duration and momentary stress in the whole sample, such that the magnitude and/or directionality of this association would differ between the two groups. In line with the diathesis-stress model asserting the presence of stress sensitivity in at-risk samples, we expected that CHR adolescents would exhibit a stronger positive association between sleep duration and momentary stress in daily life compared to non-CHR peers. Lastly, we examined the effects of sleep duration and momentary stress on momentary experiences of attenuated positive symptoms (referred to "momentary psychosis-risk symptoms" throughout) in only the CHR sample, expecting that CHR adolescents would experience a higher frequency of momentary psychosis-risk symptoms (*H4*) on days when they slept less than usual the previous night and (*H5*) at times when they were experiencing higher momentary stress than usual.

2.0 Methods

2.1 Participants

Twelve CHR and 15 non-CHR control adolescents (*N = 27*), ages 13-20 years, were recruited from the Greater Pittsburgh area as part of a larger study in Dr. Leslie Horton's Youth Emotions, Thoughts, and Interactions Laboratory (YETI Lab) at the University of Pittsburgh. CHR participants were recruited to the study through community advertisements and referrals from clinical staff at the Hope Team Clinic, a CHR youth program directed by Dr. Horton at Western Psychiatric Hospital.

Participants were determined to be at CHR for psychosis if they met criteria for the presence of Attenuated Positive Symptom Syndrome (APSS) as operationalized in the Criteria of Psychosis Risk Syndrome: attenuated positive symptoms present at least once per week, started or worsened in the past year (unusual thought content/delusional ideas, suspiciousness/persecutory ideas, grandiosity, perceptual abnormalities/distortions, and conceptual disorganization). Additionally, participants were assessed for current attenuated symptoms of psychosis using the Scale of Prodromal Symptoms (SOPS), which is embedded within a semi-structured interview (Structured Interview for Prodromal Symptoms, SIPS (Miller et al., 2001; Miller et al., 2002)). This study used the SOPS to assess dimensional ratings of attenuated positive symptoms on a Likert scale from 0 (Absent) to 6 (Severe and psychotic).

All participants were excluded from the study if they 1) had a history of head trauma with loss of consciousness, 2) were unable to understand or speak English at the conversational level, 3) had an IQ less than 70, 4) were unable or unwilling to abstain from nicotine, alcohol, psychostimulants (e.g., Ritalin and Adderall), and caffeine on days of testing, 5) had the presence of a neurological or serious medical condition, 6) were taking medications known to influence

stress reactivity (e.g., beta-blockers, clozapine, tricyclic antidepressants, and benzodiazepines), and 7) were currently pregnant, if female. CHR participants were additionally excluded if they met current or past diagnostic criteria for a psychotic disorder. Non-CHR participants were additionally excluded if they met current or past diagnostic criteria for any psychiatric, alcohol, or substance use disorder as well as had family members (first-degree relatives) meeting current or past criteria for any psychotic disorder (i.e., excluding for those at familial high-risk for psychosis).

2.2 Procedure

The Institutional Review Board at the University of Pittsburgh approved study procedures. At baseline and follow-up timepoints, participants completed an eligibility screening visit, structured clinical interview, in-person laboratory visit, and 14-day period of at-home, ambulatory assessment. Demographic information, SIPS scores of attenuated positive symptoms, and ratings on the Perceived Stress Scale[1] (PSS-10; Cohen et al., 1983) and Global Assessment of Functioning (GAF; Aas, 2010) were obtained during the eligibility screenings and clinical interviews. All procedures were repeated at baseline, 6-month follow-up, and 1-year follow-up.

The current study used EMA (Schiffman et al., 2008) and experience sampling (ESM; Hektner et al., 2007) to collect self-reported data of nightly sleep behaviors, momentary affect (e.g., perceived stress), and momentary experiences of psychosis-risk symptoms in daily life for the 14-day, at-home portion of the study. Following an in-person study visit, all participants were

[1] Scores on the Perceived Stress Scale (PSS-10; Cohen et al., 1983) are conceptualized as a measure of trait stress, such that higher PSS scores indicate higher trait stress, and lower scores indicate lower trait stress.

provided with a Samsung Galaxy S10E smartphone with the MovisensXS (Movisens GmbH, 2016) app installed. During the 14-day observation period, participants completed mobile questionnaires through the MovisensXS app on weekdays and weekends (see Figures 3 through 5). The morning survey asked participants to report sleep behaviors from the previous night and following morning (e.g., time at which participant got into bed and tried to fall asleep, number and duration of nocturnal awakenings, wake time, etc.), and daily surveys asked participants to report momentary affect and experiences of psychosis-risk symptoms (see Figures 3, 4, and 5 for screenshots of mobile questionnaires). On weekdays, participants were prompted with a morning survey and daily survey at a fixed time before school, a daily survey at a fixed time during lunch, and four randomly prompted daily surveys during a six-hour timeframe after school. On weekends, participants were prompted with seven questionnaires at random times during designated waking hours (e.g., 9:00am – 11:00pm). While setting up the MovisensXS app before the start of the 14-day observation period, participants chose the fixed times/intervals during which they were prompted with mobile surveys so that ambulatory study procedures accommodated individuals' schedule.

In all, 4,033 EMA observations were collected across the 27 participants and three timepoints. Observations were excluded from the data if they were 1) collected after the 17th day of the observation period[2], 2) were determined to be outliers for one of the three primary study variables (sleep duration, momentary stress, or momentary psychosis-risk symptoms), or 3) were missing data. One hundred and thirty-nine observations were excluded for being collected

[2] The 17th day of the observation period was chosen as the cutoff for excluding observations because this is the day that maximized the number of EMA observations while maintaining an equal number of observation days across participants. Observations that were collected beyond the 14-day observation period were often the result of delays in equipment return or extensions in data collection requested by the experimenters (e.g., equipment malfunction).

beyond the 17th day, 1,736 excluded for missing data, and 10 excluded for being outliers (*n = 5* for sleep duration and *n = 5* for momentary psychosis-risk symptoms)[3]. The final dataset contained a total of 2,148 observations across the 27 participants.

2.3 Measures

2.3.1 Nightly Sleep Duration

The current study used sleep variables adapted from the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989). Specifically, the typical PSQI questions, which ask about usual sleep behaviors during the past month (e.g., "During the past month, what time have you usually gone to bed at night?" or "During the past month, what time have you usually gotten up in the morning?"), were modified such that the EMA surveys asked about sleep behaviors from the previous night (e.g., "Last night, I actually tried to go to sleep at: [input time]" or "This morning, I finally woke at: [input time]").

Nightly sleep duration was conceptualized as the total amount of time in bed on a given night, which was calculated by taking the difference between the time at which participants woke up in the morning and the time at which the participant reported falling asleep the prior night. Sleep duration was calculated for each morning during the 14-day period and converted from minutes to hours during data cleaning (see Figure 3 for screenshots). One hundred and twenty-

[3] Outliers were excluded if there were outside the interval of 1.46 and 13.05 hours for nightly sleep duration, -58.50 and 122.74 for momentary stress, or -1.83 and 2.16 for momentary psychosis-risk symptoms.

three observations of sleep duration were reported by the CHR group and 186 were reported by the non-CHR control group (*n = 309*).

2.3.2 Momentary Perceived Stress

Self-reported momentary stress was assessed by asking participants to rate the statement, "How anxious, nervous, or pressured do you feel right now?", on a scale from 0 (not at all) to 100 (extremely). See Figure 4 for images of the momentary stress measure on the mobile surveys. Momentary stress was evaluated on each daily survey, resulting in 763 observations from the CHR group and 1,076 observations from the non-CHR group *(n = 1,839)*.

2.3.3 Momentary Psychosis-Risk Symptoms

Psychosis-risk symptoms in daily life were measured by asking participants to indicate whether they had experienced any perceptual abnormalities (e.g., had visions or seen things that other people couldn't see) or unusual thoughts (e.g., someone or something is controlling your thoughts or actions) within the last 30 minutes (see Figure 5 for screenshots). A total score was calculated by aggregating all reported experiences of perceptual abnormalities and unusual thought content on a given daily survey. There were 37 reports of momentary psychosis-risk symptoms in the non-CHR controls and 198 reports in the CHR group (*n = 235)*. Due to the small number of momentary psychosis-risk symptoms in the non-CHR control group, analyses involving momentary psychosis-risk symptoms as the dependent variable included only CHR participants (*n = 198*).

2.4 Statistical Approach

R version 2023.09.1 (R Core Team, 2023) was used to clean EMA data (see Figure 6 for example dataset in RStudio) and run all primary analyses. Independent t-tests and chi-square tests were used to assess group differences in demographic variables, attenuated positive symptoms, and within-person means of the primary study variables (Table 1). Additionally, bivariate correlational analyses were conducted to assess relationships between demographic characteristics and the primary study variables (see Table 2).

Although there were significant group differences in participant age, sex at birth, and gender identity (see Table 1), two of which had significant bivariate correlations with at least one of the three study variables (see Table 2), including age, sex, and gender identity as covariates in the models did not change the direction, magnitude, or significance of findings. The models reported below do not include the noted covariates.

Two- and three-level mixed effect models were performed on the hierarchical EMA data to examine group differences and relationships between the primary study variables. The current study used a hierarchical data structure in which EMA observations (Level 1: assessment-level) were nested within days (Level 2: day-level) which were further nested within individuals (Level 3: person-level). See Figure 7 for a concept plot of the study's hierarchical data structure. Consistent with the recommendations of Cohen et al. (2013) and Luke (2019), Level 2 predictors (i.e., sleep duration) in the three-level models and Level 1 predictors (i.e., momentary stress, momentary psychosis-risk symptoms) in the two-level models were person-mean-centered (i.e., deviations from the individuals' mean). When Level 2 or Level 1 within-person predictors were person-mean-centered, person means (i.e., each participants' individual mean response) were grand-mean-centered (i.e., deviation from the sample mean) and added to the multilevel models

as covariates at the next level. Person-mean-centered and grand-mean-centered represent within-person fluctuations and between-person differences, respectively in the study's primary predictors.

Group differences in the nightly sleep duration and momentary stress were examined using two-level multilevel models with group status as a between-person (Level-2) predictor and sleep duration and momentary stress as within-person (Level-1) outcomes. Equation (1) and (2) provide formula of the two-level conditional models for hypothesis 1 and 2:

$$\textit{Level 1:} \quad Sleep_{ti} = \beta_{0i} + \epsilon_{ti} \qquad (1)$$

$$\textit{Level 2:} \quad \beta_{0i} = \gamma_{00} + \gamma_{01}\left(Group_i\right) + u_{0i}$$

$$\textit{Level 1:} \quad Stress_{ti} = \beta_{0i} + \epsilon_{ti} \qquad (2)$$

$$\textit{Level 2:} \quad \beta_{0i} = \gamma_{00} + \gamma_{01}\left(Group_i\right) + u_{0i}$$

The association between sleep duration and momentary stress, in the whole sample, was modeled using a three-level mixed effects model with within-person fluctuations in sleep duration as a Level 2 predictor, between-person differences in sleep duration as a Level 3 covariate, and momentary stress as a Level 1 (within-day nested within-person) outcome. An additional three-level model included group status as a between-person (level-3) moderator, with simple slopes analysis (Aiken et al., 1991) used to probe this two-way interaction. Equations (3) and (4) provide equations for these three-level models examining hypotheses 3(a) and 3(b), respectively:

$$\textit{Level 1:} \quad Stress_{mti} = \beta_{0ti} + \epsilon_{mti} \qquad (3)$$

$$\textit{Level 2:} \quad \beta_{0ti} = \delta_{00i} + \delta_{01i}\left(Sleep_{ti} - \overline{Sleep}_i\right) + u_{0ti}$$

$$\textit{Level 3:} \quad \delta_{00i} = \gamma_{000} + \gamma_{001}\left(\overline{Sleep}_i - \overline{Sleep}\right) + u_{00i}$$

$$\delta_{01i} = \gamma_{010} + u_{01i}$$

Level 1: $Stress_{mti} = \beta_{0ti} + \epsilon_{mti}$ *(4)*

Level 2: $\beta_{0ti} = \delta_{00i} + \delta_{01i}\left(Sleep_{ti} - \overline{Sleep}_i\right) + u_{0ti}$

Level 3: $\delta_{00i} = \gamma_{000} + \gamma_{001}\left(Group_i\right) + \gamma_{002}\left(\overline{Sleep}_i - \overline{Sleep}\right) + u_{00i}$

$$\delta_{01i} = \gamma_{010} + \gamma_{011}\left(Group_i\right) + u_{01i}$$

The association between sleep duration and momentary psychosis-risk symptoms in the CHR group was examined using a three-level mixed effects model with within-person fluctuations in sleep duration as a Level 2 predictor, between-person differences in sleep duration as a Level 3 covariate, and momentary psychosis-risk symptoms as a Level 1 (i.e., within-day nested within-person) outcome. See Equation (5) for the conditional three-level model for hypothesis 4:

Level 1: $Symptoms_{mti} = \beta_{0ti} + \epsilon_{mti}$ *(5)*

Level 2: $\beta_{0ti} = \delta_{00i} + \delta_{01i}\left(Sleep_{ti} - \overline{Sleep}_i\right) + u_{0ti}$

Level 3: $\delta_{00i} = \gamma_{000} + \gamma_{001}\left(\overline{Sleep}_i - \overline{Sleep}\right) + u_{00i}$

$$\delta_{01i} = \gamma_{010} + u_{01i}$$

The relationship between momentary stress and momentary psychosis-risk symptoms in the CHR group was examined using a two-level multilevel model with within-person fluctuations in momentary stress as a Level 1 predictor, between-person differences in momentary stress as a Level 2 covariate, and momentary psychosis-risk symptoms as a Level 1 (i.e., within-person) outcome. See Equation (6) for the conditional two-level model for hypothesis 5:

$$\textit{Level 1:} \quad Symptoms_{ti} = \beta_{0i} + \beta_{1i}(Stress_{ti} - \overline{Stress_i}) + \epsilon_{ti} \qquad (6)$$

$$\textit{Level 2:} \quad \beta_{0i} = \gamma_{00} + \gamma_{01}(\overline{Stress_i} - \overline{Stress}) + u_{0i}$$

$$\beta_{1i} = \gamma_{10} + u_{1i}$$

Spatial correlation structures were used to model the residual structures for level-1 variance-covariance matrices, as this type of correlation structure is optimal for modeling EMA data that is repeatedly measured at unequal time-intervals (e.g., randomly prompted surveys) (Dong et al., 2015). Multilevel modeling was accompanied by the restricted estimation maximum likelihood (REML: Goldstein, 1989) because it is the preferred estimation method when performing multilevel regressions on small samples.

3.0 Results

3.1 Descriptive Statistics of Demographic, Symptoms, and Study Variables

See Table 1 for participant demographics, symptom details, and descriptive statistics of the study variables. There were significant group differences in participant age, $t(17.34) = -2.35$, $p = .031$, and gender identity, $\chi^2(2) = 9.23$, $p < .01$. There were no significant group differences in sex assigned at birth, race/ethnicity or annual income.

As expected, there were significant group differences in most of the attenuated positive symptoms, such that he CHR group reported higher total attenuated positive symptoms, delusional thinking/unusual thought content, suspiciousness/persecutory ideation, perceptual abnormalities, and disorganized communication compared to the non-CHR group. Additionally, the CHR group reported higher trait stress, as measured by the PSS-10 (Cohen et al., 1983) compared to the non-CHR group, $t(24.34) = -3.17$, $p < .01$. The CHR and non-CHR groups did

not significantly differ on grandiosity, *t(15.73) = -1.71, p = .108*, or on global functioning, *t(15.78) = 1.93, p = .07*.

There were no significant group differences for within-person means of the primary study variables (see Table 1). Bivariate correlation analyses revealed that there were significant associations amongst the three study variables and the demographic characteristics (see Table 2).

3.2 Hypothesis 1 and 2: Group Differences in Sleep Duration and Momentary Stress

There were no significant group differences in sleep duration, γ_{01} *= .04, SE = .44, p = .928*, or momentary stress, γ_{01} *= 6.37, SE = 6.78, p = .356*. In an additional two-level multilevel model, we found that there was no group difference in momentary psychosis-risk symptoms, γ_{01} *= .09, SE = .10, p = .385*. See Table 3 for multilevel model statistics and Figure 5 for violin plots of the group differences.

3.3 Hypothesis 3: Sleep Duration Predicting Momentary Stress in the Whole Sample

Multilevel regression models (see Table 4) reveal that within-person sleep duration had no significant effect with momentary stress, γ_{010} *= -.14, SE = .95, p = .886*, including between-person sleep duration as a covariate, γ_{001} *= .91, SE = 3.23, p = .781*.

A multilevel moderation analysis (see Table 5) showed that group status significantly moderated the association between within-person sleep duration and momentary stress, γ_{011} *= 3.33, SE = 1.55, p = .031*. A simple slope analysis confirmed this finding, suggesting that the association between within-person sleep duration and momentary stress trended toward negative in the non-CHR group, β *= -1.78, SE = 1.03, p = .084*, but was not significant in the CHR group, β *= 1.55, SE = 1.55, p = .180* (See Figure 9 for the graph of the simple slopes analysis).

3.4 Hypothesis 4 and 5: Sleep Duration and Momentary Stress Predicting Momentary Psychosis-Risk Symptoms in Clinical High-Risk Group

See Table 6 for model output of the three- and two-level models in which sleep duration and momentary stress predict momentary psychosis-risk symptoms in the CHR group. A three-level multilevel model was used to assess the association between sleep duration and momentary psychosis-risk symptoms in the CHR group. The regression analysis revealed that within-person sleep duration did not have a significant relationship with momentary psychosis-risk symptoms, $\gamma_{010} < .01$, *SE = .01*, $p = .810$, including between-person sleep duration as a covariate, $\gamma_{001} = -.05$, *SE = .07*, $p = .514$.

A two-level multilevel model was used to assess the association between momentary stress and momentary psychosis-risk symptoms in the CHR group. As expected, there was a significant positive association between within-person momentary stress and momentary psychosis-risk symptoms, $\gamma_{10} < .01$, *SE <.01*, $p = .037$, such that CHR adolescents tended to report a higher frequency of psychosis-risk symptoms in daily life at times when they reported higher momentary stress than usual (see Figure 10 for simple graphs slope). Additionally, between-person momentary stress was included as a covariate and was not found to be significant in predicting momentary psychosis-risk symptoms, $\gamma_{01} < .01$, *SE <.01*, $p = .404$.

4.0 Discussion

The current study provides a novel account of the day-to-day interrelationships between sleep dysfunction, stress abnormalities, and experiences of attenuated positive symptoms in adolescents at CHR for psychosis and non-CHR peers. Findings hint at daily associations between nightly sleep duration and momentary perceived stress, which may be disrupted in CHR

adolescents compared to non-CHR peers. Furthermore, this work contributes to the growing body of work positing the role of sleep dysfunction and stress abnormalities in exacerbating experiences of attenuated positive symptoms of psychosis in daily life in CHR adolescents.

In the current study, we did not observe a significant group difference in nightly sleep duration between the CHR and non-CHR groups. While our null findings should be interpreted with caution and may be the result of not having enough power to detect effects, delving into the possible interpretations of the null findings may offer insights for future directions. The lack of a significant group difference in nightly sleep duration may reflect the fact that sleep health naturally declines over the course of normative adolescent development (Wallace et al., 2022; Maslowsky & Ozer, 2014). In conjunction with our findings that within-person averages of sleep duration do not significantly differ between our two groups (7.58 for the non-CHR group and 7.56 hours for the CHR group), it is plausible that poor sleep outcomes tend to emerge in *all* adolescents, regardless of being at-risk or underlying psychopathology. Another explanation is that our sleep duration variable was calculated using self-reports of the approximate times at which the participant fell asleep the previous night and woke up the following morning. Using approximated times to measure sleep duration may have resulted in recall bias and, in turn, a less accurate measurement of self-reported sleep duration.

Similarly, we did not find a significant group difference in momentary perceived stress between CHR and non-CHR adolescents. We would expect that there would be a group difference as previous work has shown that CHR individuals typically exhibit higher perceived stress and greater exposure to daily stressors (Ristanovic et al., 2020; Millman et al., 2018; Phillips et al., 2020; Pruessner et al., 2013; Sugranyes et al., 2012; Thompson et al., 2007; Trotman et al., 2014; Cullen et al., 2020) compared to non-CHR peers. One possible explanation

for our lack of a group difference in momentary perceived stress is that, like sleep duration, adolescence is a life stage that is naturally accompanied by an increase in exposure to various psychophysiological stressors – such as personal, family, social, academic, and other life stressors – regardless of at-risk status for psychosis or other psychopathology (Compas, 1987; Scheibe & Hannes, 2013). Seiffge-Krenke et al. (2009) showed that perceived stress tended to peak in early adolescence and begin to decrease during late adolescence. This trajectory has also been observed in adults, with maximal perceived stress occurring in early adulthood and gradually declining into later adulthood (Johnson et al., 2023; Stefaniak et al., 2021). Thus, it is plausible that the lack of a significant group difference in momentary stress may be because alterations in psychological stress naturally occur in *all* adolescents and young adults and may be further indicative of healthy changes in stress processes during these critical developmental periods. Another possible explanation, in addition to our limited sample size, is that the current study's use of self-reported momentary stress may have influenced our ability to detect a group difference, as perceived stress is a subjective concept that varies across individuals. Other studies have found that CHR adolescents tend to display elevated physiological stress (e.g., elevated cortisol levels) and stress reactivity compared to non-CHR peers (Trotman et al., 2014; Carol et al., Sugranyes et al., 2012; Lincoln et al., 2015); therefore, it may be valuable for future studies to reexamine the current study's primary analyses using physiological measures of nightly sleep duration and momentary stress (e.g., actigraphy/polysomnography, cortisol, heart rate variability).

Our moderation analysis provides a nuanced perspective in understanding the association between nightly sleep duration and next-day momentary perceived stress in daily life. While sleep duration was not significantly associated with momentary stress in the whole sample, group

status did moderate this association with the non-CHR group likely driving this pattern. Non-CHR adolescents trended (*p=.084*) toward experiencing higher momentary stress on days when they received less sleep than usual the previous night. Although the noted association was only marginally significant, we would expect our non-CHR group to exhibit this relationship between sleep duration and perceived stress in daily life based on previous literature suggesting that longer sleep duration – and other healthy sleep outcomes – typically buffers for elevated stress levels in general populations (Kim et al., 2019; Yap et al., 2020; Kim & Lee, 2018). Interestingly, there was no association between sleep duration and momentary stress in the CHR group, which contradicts our initial expectations that this association would be *stronger* in the CHR group compared to the non-CHR group. Specifically, we initially hypothesized that the noted association would be stronger in CHR adolescents compared to non-CHR peers because the diathesis-stress model (Walker & Diforio, 1997) and extended conceptualization of sleep dysfunction (Lunsford-Avery & Mittal, 2013) support the presence of increased stress sensitivity in those at-risk and diagnosed with psychotic disorders (Millman et al., 2018; Palmier-Claus et al., 2012; Philips et al., 2011; Trotman et al., 2014; Sugranyes et al., 2012; Cullen et al., 2020). However, our findings suggest that the relationship between sleep duration and momentary stress is *blunted* in the CHR group compared to in the non-CHR group. Although the noted association did not achieve statistical significance in either group, the significant moderation of group status may hint at the presence of stress habituation in the CHR group. Stress habituation is the process by which one's biological response to stress stimuli decreases with repeated exposure to stress (Grissom & Bhatnagar, 2009). Theoretically, stress habituation may serve as a coping mechanism to chronic stress so that individuals experience less stress in response to psychosocial stressors, thereby preserving more "energy" to deal with more distressing stressors in daily life

(e.g., experiences of psychosis-risk symptoms). It is possible that being at CHR for psychosis could itself be a psychological or biological stressor that is further accompanied by distressing and ongoing experiences of attenuated positive symptoms. Blunted cortisol responses to psychosocial stressors (e.g., Trier Social Stress Test) have been observed in individuals at-risk and diagnosed with psychotic disorders (Pruessner et al., 2013; Shah et al., 2023) Similarly, chronic sleep loss can be conceptualized as a psychological stressor that repeatedly activates the body's stress response mechanism (i.e., the HPA axis) during periods of sleep loss (Meerlo et al., 2020; Vargas & Lopez-Duran, 2017). As observed in psychotic disorders and at-risk states, poor sleep outcomes and acute/chronic sleep loss have been linked to blunted cortisol responses in general populations (Wright et al., 2007; Vargas & Lopez-Duran, 2017; Hansen et al., 2021; Basset et al., 2015). Thus, the combination of consistently not getting enough sleep at night during the CHR period may result in the trend towards blunted stress levels we observed on days following shorter sleep duration in the CHR group ($p = .180$). Another theoretical explanation of the positive trend observed in the CHR group ($\beta = 1.55$; i.e., trend towards experiencing less momentary stress following a night of shorter sleep duration) is that CHR adolescents may disengage in everyday stressful emotions and experiences following nights of short sleep duration. Disengagement from everyday affective experiences is a general coping strategy shown to be used by at-risk individuals (Ristanovic et al., 2022; Yee et al., 2020) as well as in response to sleep problems in samples not at-risk for psychosis (Palmer et al., 2018). Additional research using a more comprehensive battery of stress measures and larger at-risk sample is needed to further understand these possible interpretations and make definitive conclusions.

In the CHR group, sleep duration was not significantly related to experiences of psychosis-risk symptoms in daily life within or between persons. We would expect shorter sleep

duration to be associated with a higher frequency of attenuated positive symptoms in daily life in CHR adolescents based on the neurodevelopmental diathesis-stress conceptualization of sleep dysfunction (Lunsford-Avery & Mittal, 2013). Furthermore, sleep disturbances and abnormalities are consistently linked to worsening psychotic symptomology in CHR individuals, other at-risk samples, and those diagnosed with psychotic (Zak et al., 2022; van der Tuin et al., 2023; Lunsford-Avery et al., 2015; Reeve et al., 2019; Goines et al., 2019; Mayeli et al., 2021; Blanchard, 2021; Poe et al., 2017; Meyer et al., 2021). In conjunction to our small sample size, the null findings may be because the current CHR sample did not exhibit markedly worse sleep duration compared to the non-CHR group – evidenced by our insignificant group differences in EMA sleep duration and within-person averages of sleep duration. Further investigation is needed to adequately compare our findings to the current literature.

However, we did find a significant association between momentary perceived stress and experiences of psychosis-risk symptoms in daily life in the CHR group, such that CHR adolescents tended to experience a higher frequency of psychosis-risk symptoms in daily life at times when they reported higher momentary stress than usual. Findings are consistent with the diathesis-stress model (Walker & Diforio, 1997) and previous literature supporting a positive association between elevated psychophysiological stress and attenuated positive symptoms in those at-risk and diagnosed with psychotic disorders (DeVylder et al., 2013; Cullen et al., 2019; Cullen et al., 2020; Chaumette et al., 2016; Carol et al., 2021; Carol & Mittal, 2015; Millman et al., 2018; Trotman et al., 2014). Our findings advance our understanding of this mechanism as well as provide novel insight into how psychological stress worsens experiences of attenuated positive symptoms in daily life in CHR adolescents.

4.1 Clinical Implications

These findings have important clinical implications. Early treatment and preventative programs could focus on instilling healthy sleep behaviors and effective strategies for coping with stressful experiences to minimize attenuated positive symptoms in daily life. Cognitive behavioral therapy for insomnia (CBT-I) has been shown to be effective in improving sleep outcomes and daytime functioning in individuals with psychotic disorders (Waters et al., 2020; Khalid, 2022; Bradley et al., 2018). Important to our CHR sample, Waite et al. (2023) suggest that CBT-I may be a feasible treatment approach for targeting sleep problems and in turn improving clinical outcomes (e.g., anxiety, depression, paranoia) in people at-risk for psychosis. Furthermore, there is evidence that general CBT is effective in reducing rates of transition to psychosis in individuals at-risk for psychosis (Mei et al., 2021; Devoe et al., 2020), demonstrating the effectiveness of using both general and specific forms of CBT to simultaneously target poor sleep outcomes and mitigate the risk of transitioning to psychosis in at-risk individuals.

Similarly, studies have found that mindfulness-based interventions are effective in improving stress outcomes, positive symptoms, and other clinical outcomes in people with schizophrenia (Hodann-Caudevilla et al., 2020; Jansen et al., 2020; Kim et al., 2021; Liu et al., 2021). Although the effectiveness of mindfulness-based interventions has not been studied in samples at-risk for psychosis, the literature demonstrates that both treatment programs show great promise in targeting sleep dysfunction, stress abnormalities, and attenuated positive symptoms at different stages of psychosis progression.

4.2 Limitations

While there were many strengths to the study (e.g., ecological validity, repeated measures approach, longitudinal design, diverse sample), there are important limitations to discuss. Despite the advantages of using EMA data and multilevel modeling, the power of the primary analyses was likely impacted by our small sample size and data lost at follow-ups due to attrition. Additionally, we did not consider the skewness or potential for zero-inflated distribution of psychosis-risk symptoms in analyses nor a the possibility that nightly sleep duration has a bidirectional or parabolic association with psychological stress and experiences of psychosis-risk symptoms in daily life (Yap et al., 2020; Nordholm et al., 2023). Future work is needed to replicate the primary analyses using a larger at-risk sample as well as account for skewness/zero-inflated distributions in the data in order to improve the sample size, retention, and analytical accuracy. This work reports findings from preliminary analyses, and data collection for the present study is ongoing; thus, models should be re-examined in the complete adolescent sample, when available.

The second limitation is that we used total time in bed (i.e., difference between times at which participants tried to fall asleep at night and woke up the following morning) to operationalize nightly sleep duration. Time in bed is a measure of the total amount of time spent in bed at night while sleep duration reflects the total amount of time spent sleeping during that window of time. Therefore, time in bed is not an ideal measure of sleep duration as it does not capture more nuanced sleep behaviors that influence the amount of actual sleep one receives at night, such as sleep latency (i.e., time it takes to fall asleep after getting into bed) and occurrences of nocturnal awakenings (i.e., number and amount of time one is awake during the night). Moreover, sleep measures assessed with EMA were retrospective self-reports of sleep

behaviors from the previous night or following morning, which may have introduced recall bias into the sample despite using EMA. Thus, the instrumental validity of our sleep duration variable was likely compromised due to our use of self-reported time in bed in operationalizing nightly sleep duration. Relatedly, we decided to examine only sleep duration in the current study in order to reduce the number of analyses and optimize statistical power; however, sleep duration is only one component of sleep and sleep health and does not address other important dimensions of sleep (e.g., using the RU-SATED model (Buysse, 1989)) (Meltzer et al., 2021). Future studies could address this limitation by using a more accurate measurement of nightly sleep duration by accounting for sleep latency and nocturnal awakenings as well as altering the timing or format of EMA surveys in order to obtain a more accurate, self-reported account of when people fall asleep and wake up in daily life.

The final limitation is that we did not account for dependency in observations collected across the three longitudinal timepoints. Future work can use growth curve models to assess fluctuations and changes in the primary variables across the year-long study period.

4.3 Future Directions

Future studies should focus on recruiting a larger CHR sample to maximize the power of the primary analyses used in the current study. Furthermore, similar work should utilize growth curve analyses of the longitudinal data to examine fluctuations and changes in sleep, stress, and attenuated positive symptoms in not only daily life but across a prolonged observation period.

Potential areas of further investigation could use more objective measures of sleep and stress – such as actigraphy/polysomnography and cortisol, respectively – to evaluate the day-to-day association between sleep duration and momentary stress in a physiological context and in a

CHR sample. This would allow for the exploration into underlying neurobiological processes of this natural sleep-stress mechanism in individuals at-risk and diagnosed with psychotic disorders. For example, recent evidence and theoretical frameworks suggest that there may be an important role of orexin, a neuropeptide responsible for promoting wakefulness in the sleep-wake cycle (Sakurai et al., 2010), in the emergence of positive symptoms in psychotic disorders, such as schizophrenia. Orexin, in activating the HPA axis, may serve as the underlying neurobiological link between sleep dysfunction, disrupted stress processes (e.g., hyperactivity of the HPA axis), and positive symptoms in psychotic disorders (Perez & Lodge, 2021; Elam et al., 2021). Although most work investigating orexin has been done in rodent samples and there are currently limited methods for collecting and analyzing orexin activity in humans (e.g., cerebrospinal fluid, blood, serum), exploring this area of sleep-stress research may be valuable to understanding the biological mechanisms underlying the effect of sleep dysfunction on psychotic symptomology.

As previously discussed, theoretical framework and prior work have often taken a multidimensional approach when conceptualizing sleep and sleep health, with sleep duration being a single facet of the extensive and complex array of sleep behaviors (Buysse, 1989). For example, Buysse (1989)'s RU-SATED model presents six unique, yet intertwined, dimensions of sleep health: Regularity, Satisfaction, Alertness/sleepiness, Timing, Efficiency, and Duration. Similar work could use Buysse (1989)'s RU-SATED framework to model sleep health as well as develop a more comprehensive profile of sleep heath in at-risk individuals. This would advance our understanding of how various components of sleep health impact daily experiences of psychophysiological stress and psychosis-risk symptoms in CHR samples as well as inform early identification of sleep-related risk markers that characterize at-risk youth and possibly contribute to the symptomology and onset of full-blown psychotic disorders.

Moreover, it may be valuable to assess different types of relationships that may exist between sleep, stress, and psychosis-risk symptoms in daily life – such as bidirectional or higher-order associations. For instance, previous studies have found that psychosis-risk symptoms have bidirectional associations with both sleep and stress variables across the psychosis spectrum (Zak et al., 2022; Tuin et al., 2023; Meyer et al., 2022; Lee & Schepp, 2009; Renwick et al., 2009; Klippel et al., 2021) as well as in general populations, although the literature is limited (Simor et al., 2019; Wang et al., 2021). Conversely, despite a growing body of work asserting a bidirectional association between sleep and stress in general populations (Yap et al., 2020; Slavish et al., 2021; Laethem et al., 2015; LaVoy et al., 2020; Sin et al., 2017), there are no known studies investigating the noted association in people at-risk or diagnosed with psychotic disorders. There is also limited work investigating a possible U-shaped (i.e., quadratic) association between sleep behaviors, everyday stress, and psychotic symptomology across the psychosis spectrum. Overall, Geoffroy et al. (2020)'s work suggests that both restricted and excessive sleep duration is linked to a higher prevalence of psychiatric/substance-use disorders as well as higher comorbidity. There is some evidence that at-risk individuals exhibit both shorter and longer sleep duration (Georgiades et al., 2023; Morishima et al., 2020); however, there are currently no studies that provide a cohesive account of both phenomena (e.g., a U-shaped relationship). Additionally, Kim and Lee (2018) suggest that there may be a similar quadratic pattern between sleep and stress in general populations, underpinning how sleep duration may have nonlinear relationships with various facets of everyday experiences and symptomology. Future work should strive to develop a more unified and complete account of these dynamic mechanisms so that we can have a better understanding of the vicious cycle

between sleep dysfunction, psychophysiological stress, and the emergence of psychotic symptomology, especially in vulnerable youth and in a day-to-day context.

A final important, yet inconsistent, area of the literature pertaining to stress-related psychosis is the direction in which stress reactivity changes throughout the disease progression of psychosis. According to the diathesis-stress model (Walker & Diforio, 1997) and substantive empirical evidence, samples at-risk and diagnosed with psychotic disorders are expected to exhibit greater stress sensitivity (i.e., increased biological stress response to psychosocial stressors) compared to control groups (Millman et al., 2018; Palmier-Claus et al., 2012; Philips et al., 2011; Trotman et al., 2014; Sugranyes et al., 2012; Cullen et al., 2020). However, the current study and emerging literature suggests that stress habituation (i.e., decreased biological stress response to psychosocial stressors) may be another possible outcome of chronic stress exposure, sleep dysfunction, and psychosis (Wright et al., 2007; Vargas & Lopez-Duran, 2017; Hansen et al., 2021; Basset et al., 2015; Pruessner et al., 2013; Shah et al., 2023; Lam et al., 2018; Young et al., 2020; Zhang et al., 2019). Both phenomena have been observed in at-risk samples and psychotic disorders (Pruessner et al., 2013; Shah et al., 2023; Millman et al., 2018; Palmier-Claus et al., 2012; Philips et al., 2011; Trotman et al., 2014; Sugranyes et al., 2012; Cullen et al., 2020), and more studies are needed to evaluate stress sensitivity versus habituation. Given the heterogeneity of those at-risk and with psychosis, it will be important to evaluate whether stress responses differ for subgroups of individuals. The complete sample from the current study – as well as future, larger studies of young people at-risk for psychosis – will be well-poised to examine symptom and stress-related heterogeneity.

4.4 Conclusions

In all, the current study contributes to the growing literature supporting the presence of sleep dysfunction and stress abnormalities in adolescents at CHR for psychosis as well as their associations with experiences of attenuated positive symptoms in daily life. Furthermore, we provide a novel perspective of possible disruptions in the day-to-day relationship between nightly sleep duration and momentary perceived stress during the CHR period and critical developmental stage of adolescence. Although our null and marginal results should be interpreted with caution, the observed differences in the noted association between CHR and non-CHR adolescents demonstrate the need for further investigation into how psychophysiological stress sensitivity in response to sleep dysfunction changes during the prodrome of psychosis and throughout adolescence. Future research is needed to replicate the current findings in a larger adolescent and at-risk sample and to determine if these associations are maintained in a physiological context. Exploring the intersection between sleep dysfunction, stress abnormalities, and CHR status across adolescence and young adulthood can provide key insight into how sleep- and stress-related risk factors contribute to prodromal symptomology, with an eye toward improving early intervention and preventative programs that can target individual and combinations of risks markers that may further exacerbate the risk of developing psychosis later in life.

5.0 Tables

Table 1. Summary of Descriptive Statistics

Variable		Non-CHR	CHR	Statistics
		n = 15	*n = 12*	
Age, *mean (SD)*		15.33 (1.45)	17.17 (2.37)	*t(17.34) = -2.35, p = .031**
Sex at birth, *n (%)*				$\chi^2(1) = 2.43, p = .119$
	Female	7 (46.7%)	10 (83.3%)	
	Male	8 (53.3%)	2 (16.7%)	
Gender identity, *n (%)*				$\chi^2(2) = 9.23, p < .01$**
	Woman/female	7 (46.7%)	7 (58.3%)	
	Man/male	8 (53.3%)	1 (8.3%)	
	Other	0 (0.0%)	4 (33.3%)	
Race/ethnicity, *n (%)*				$\chi^2(2) = .96, p = .618$
	White/Caucasian	7 (46.7%)	7 (58.3%)	
	Non-white/minority	5 (33.3%)	2 (16.7%)	
	Biracial	3 (20.0%)	3 (25.0%)	
Income, *n (%)*				$\chi^2(4) = 2.80, p = .592$
	$20,000-$35,000	0 (0.0%)	2 (16.7%)	
	$35,000-$50,000	1 (6.7%)	1 (8.3%)	
	$50,000-$100,000	6 (40.0%)	4 (33.3%)	

$100,000-$200,000	5 (33.3%)	3 (25.0%)	
>$200,000	3 (20.0%)	2 (16.7%)	
Attenuated positive symptoms, *mean (SD)*			
Total attenuated positive symptoms	1.53 (1.85)	11.42 (2.71)	*t(18.66) = -10.78, p < .001***
Delusional thinking / unusual thought content	0.40 (.51)	3.42 (.51)	*t(23.55) = -15.23, p < .001****
Suspiciousness / persecutory ideas	0.40 (.51)	2.33 (1.50)	*t(13.03) = -4.28, p < .001****
Grandiosity	0.13 (.35)	0.50 (.67)	*t(15.73) = -1.71, p = .108*
Hallucinations / perceptual abnormalities	0.33 (.62)	3.50 (.80)	*t(20.39) = -11.31, p < .001****
Disorganized communication	0.27 (.46)	1.67 (.89)	*t(15.62) = -4.96, p < .001****
Global functioning, *mean (SD)*	83.17 (26.67)	66.67 (12.47)	*t(15.78) = 1.93, p = .07*
PSS-10 scores, *mean (SD)*	12.40 (8.09)	22.38 (7.39)	*t(24.34) = -3.17, p < .01***
Study variables, *mean (SD)*			
Nightly sleep duration	7.58 (0.9)	7.56 (1.4)	*t(18.03) = .05, p = .963*
Momentary perceived stress	28.37 (18.1)	33.51 (18.4)	*t(23.58) = -.73, p = .475*
Momentary psychosis-risk symptoms	0.09 (0.3)	0.16 (0.4)	*t(23.08) = -.07, p = .478*

Note. Baseline data of demographic information, attenuated positive symptoms (measured by the Structured Interview of Psychosis-Risk Syndrome (SIPS); Miller et al., 2002; Miller et al., 2003)), global functioning (Global Assessment of Functioning (GAF); Aas, 2010), and Perceived Stress Scale (PSS-10; Cohen et al., 1983) scores are summarized in the above table. SIPS scores measured attenuated positive symptom severity. The "other" category for gender identity denotes participants who identified as transgender or non-binary. "Non-white/minority" denotes participants who described their racial background as being anything besides

Caucasian/European decent or biracial; for example, participants who described themselves as being Asian, Black/African American, Latinx/non-European Hispanic, Native American/Pacific Islander, and so on. Income measured annual household income of either the parents – if the participant was under the age of 18 years and/or was financially dependent on parents' income – or of the participant – if the participant was above the age of 18 years and/or was financially independent of parents' income. All study variables (e.g., nightly sleep duration, momentary perceived stress, and momentary psychosis-risk symptoms) are averages of within-person means collected day-to-day during the 14-day period of at-home, ambulatory assessment. CHR: Clinical High-Risk for psychosis.

Table 2. Bivariate Correlational Analyses

	Nightly sleep duration	Momentary perceived stress	Momentary psychosis-risk symptoms
Age	-0.03	-0.01	-0.04
Sex	-0.10	-0.25***	-0.18*
Gender identity			
Male	-0.08	-0.25***	-0.18***
Other	0.02	0.05*	0.29***
Race/ethnicity			
Non-white/minority	0.15**	0.17***	-0.06*
Biracial	-0.23***	-0.29***	-0.17***
Income	-0.17**	0.10***	0.30***

Note. $p<.05$*, $p<.01$**, $p<.001$***. Sex at birth was a binary coded variable with female coded as 0 and male coded as 1. Because gender identity, race/ethnicity, and income were multi-level factored variables, they were dichotomized into separate binary variables. Gender identity was divided into two variables with female/woman coded as 0 and male/man or other coded as 1. Race/ethnicity was divided into two binary variables with white/Caucasian coded as 0 and the other levels (e.g., non-white/minority and biracial) coded as 1. The "other" category for gender identity denotes participants who identified as transgender or non-binary. "Non-white/minority" denotes participants who described their racial background as being anything besides Caucasian/European decent or biracial; for example, participants who described themselves as being Asian, Black/African American, Latinx/non-European Hispanic, Native American/Pacific Islander, and so on. Income is treated as a continuous variable as lower scores (e.g., $20,000-$30,000) correspond with lower income and higher scores correspond with higher income (e.g., <$200,000).

Table 3. Group Differences in Nightly Sleep Duration and Momentary Perceived Stress

	Nightly Sleep Duration				*Momentary Perceived Stress*			
Predictors	*Estimate*	*SE*	*p*	*95% CI*	*Estimate*	*SE*	*p*	*95% CI*
Fixed Effects								
(Intercept)	7.59	.30	<.001***	[7.01, 8.18]	27.90	4.59	<.001***	[18.90, 36.90]
Group Status	.04	.44	.928	[-.87, .95]	6.37	6.78	.356	[-7.59, 20.32]
Random Effects								
Residual (σ^2)	1.47				25.39			
Intercept (u_{00})	1.08				16.66			

Note. p<.05*, p<.01**, p<.001***. Two-level multilevel models were used to examine group differences in the primary study variables. Coefficient estimate, standard error (SE), p-value, and 95% confidence interval (CI) were reported for the fixed intercept and CHR status. The standard deviation of random intercept and residuals were also reported.

Table 4. Sleep Duration Predicting Momentary Stress in the Whole Sample

Predictors	*Estimate*	*SE*	*p*	*95% CI*
Fixed Effects				
(Intercept)	31.10	3.74	<.001***	[23.76, 38.43]
Within-person fluctuations	-.14	.95	.886	[-2.00, 1.73]
Between-person differences	.91	3.23	.781	[-5.74, 7.56]
Level 2 Random Effects				
Intercept (u_{00i})	16.78			
Slope (β_{01i})	2.53			
Level 1 Random Effects				
Intercept (u_{0ti})	5.87			
Residual (σ^2)	24.36			

Note. $p<.05$*, $p<.01$**, $p<.001$***. A three-level mixed effects model was used to examine the association between sleep duration and momentary stress within-persons (i.e., within-person fluctuations) and between-persons (i.e., between-person differences). Raw sleep duration scores (level-2 predictor) were person-mean-centered such that within-person sleep duration reflects deviations from an individual's average sleep duration and between-person sleep duration reflects how the individual's average sleep duration deviates from the sample mean. Coefficient estimate, standard error (SE), p-value, and 95% confidence interval (CI) were reported for the fixed effects. Standard deviation for the random effects at level 1 (e.g., random intercept and residual) and level 2 (e.g., random level-2 intercept and slope) were also reported.

Table 5. Group Status Moderating Association Between Sleep Duration and Momentary Stress in Whole Sample

Predictors	*Estimate*	*SE*	*p*	*95% CI*
Fixed Effects				
(Intercept)	28.79	4.94	<.001***	[19.10, 38.48]
Within-person fluctuations	-1.78	1.03	.084	[-3.80, .24]
Between-person averages	.45	3.21	.890	[-6.17, 7.07]
Group Status	5.46	7.19	.455	[-9.37, 20.30]
Within-person sleep duration × Group Status	3.33	1.55	.031*	[.30, 6.37]
Level 2 Random Effects				
Intercept (u_{00i})	16.84			
Slope (β_{01i})	1.66			
Level 1 Random Effects				
Intercept (u_{0ti})	5.94			
Residual (σ^2)	24.38			

Note. $p<.05$*, $p<.01$**, $p<.001$***. A three-level mixed effects model was used to examine group status as a moderator of the association between sleep duration and momentary stress within-persons (i.e., within-person fluctuations) and between-persons (i.e., between-person differences). Raw sleep duration scores (level-2 predictor) were person-mean-centered such that within-person sleep duration reflects deviations from an individual's average sleep duration and between-person sleep duration reflects how the individual's average sleep duration deviates from the sample mean. Group status was a level-3 moderator. Coefficient estimate, standard error (SE), p-value, and 95% confidence interval (CI) were reported for the fixed effects. Standard deviation for the random effects at level 1 (e.g., random intercept and residual) and level 2 (e.g., random level-2 intercept and slope) were also reported.

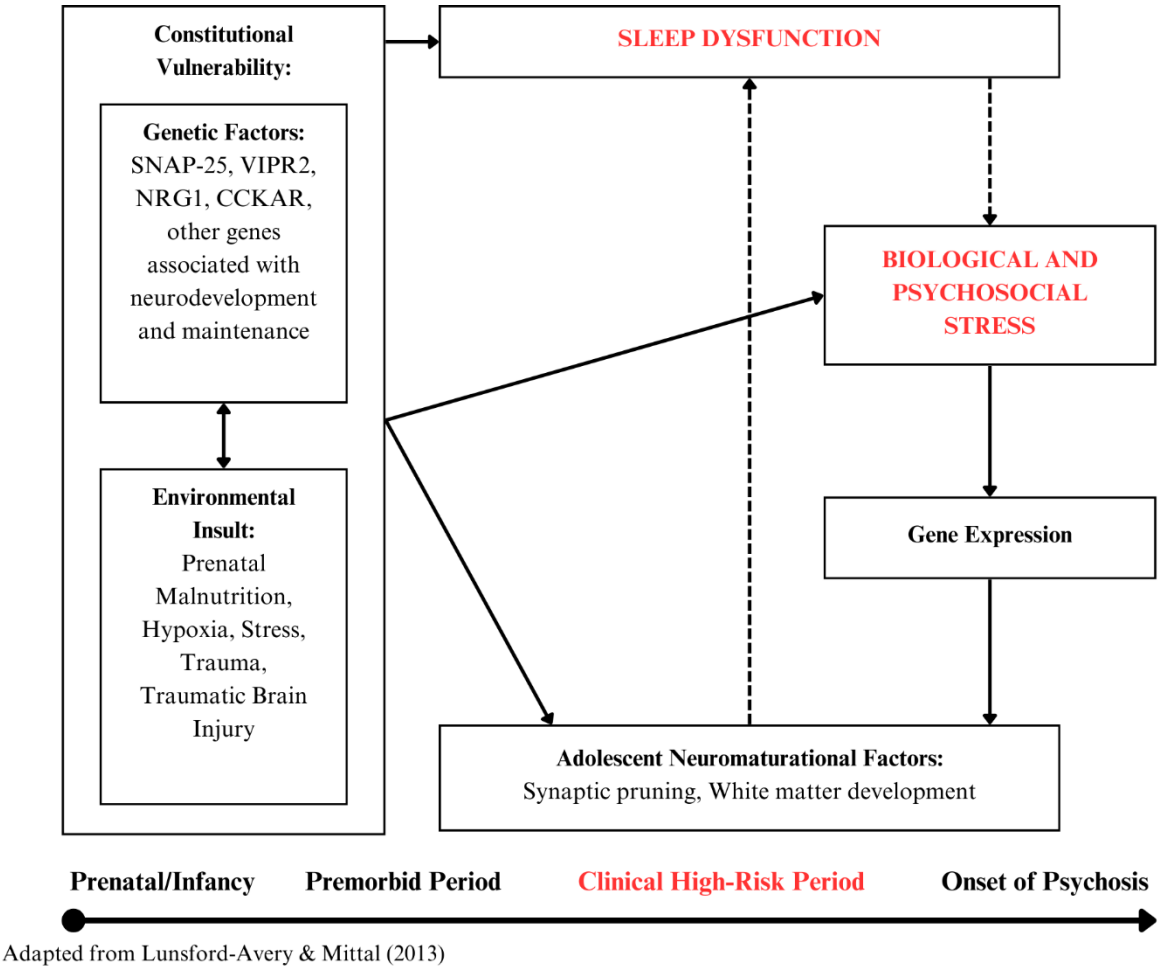
Table 6. Sleep Duration and Momentary Stress Predicting Momentary Psychosis-Risk Symptoms in the Clinical High-Risk Group

	Predictor: Sleep Duration				*Predictor: Momentary Stress*			
Predictors	*Estimate*	*SE*	*p*	*95% CI*	*Estimate*	*SE*	*p*	*95% CI*
Fixed Effects								
(Intercept)	.17	.09	.060	[-.01, .35]	.16	.08	.058	[-.005, .32]
Within-person fluctuations	<.01	.01	.810	[-.01, .02]	<.01	<.001	.037	[.00007, .002]
Between-person differences	-.05	.07	.514	[-.20, .10]	<.01	<.01	.404	[-.006, .01]
Level 2 Random Effects								
Intercept (u_{00i})	.29				---			
Slope (β_{01i})	<.001				---			
Level 1 Random Effects								
Intercept (u_{0ti})	.04				.28			
Slope (β_{1ti})	---				<.001			
Residual (σ^2)	.27				.33			

Note. p<.05*, p<.01**, p<.001***. Two- and three-level mixed effects models were used to examine the association between sleep duration and momentary psychosis-risk symptoms and momentary stress and momentary psychosis-risk symptoms, respectively, in the CHR group. Level-1 (momentary stress) and level-2 (sleep duration) predictors were person-mean-centered for their respective models, such that within-person fluctuations reflected deviations from the individual's own mean and between-person differences reflected how participants' individual means deviated from the sample mean. Coefficient estimate, standard error (SE), p-value, and 95% confidence interval (CI) were reported for the fixed effects. Standard deviation for the random effects at level 1 (e.g., random intercept and residual) and level 2 (e.g., random level-2 intercept and slope) were also reported.

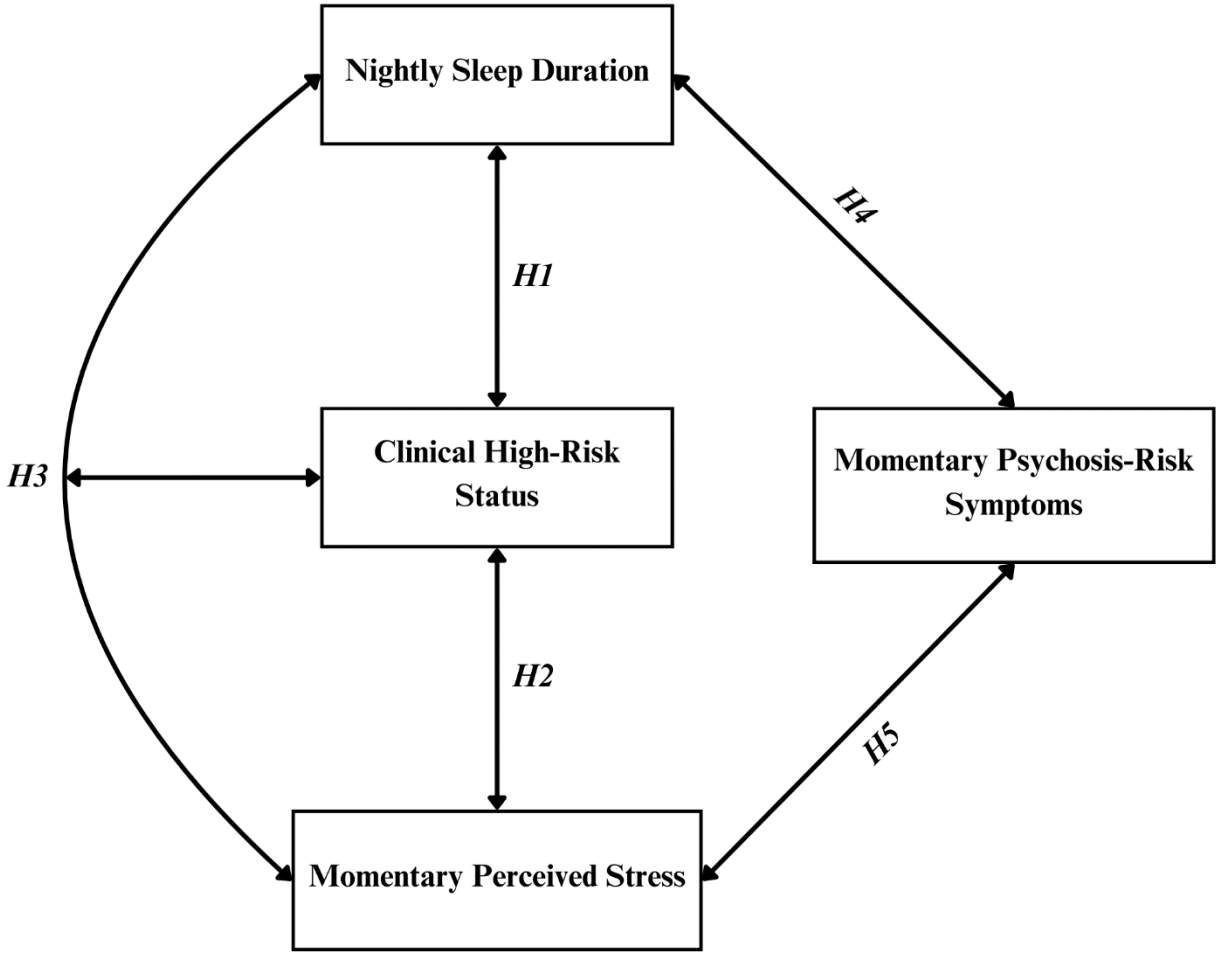
6.0 Figures

Figure 1. Neurodevelopmental Diathesis Stress Model of Sleep Dysfunction



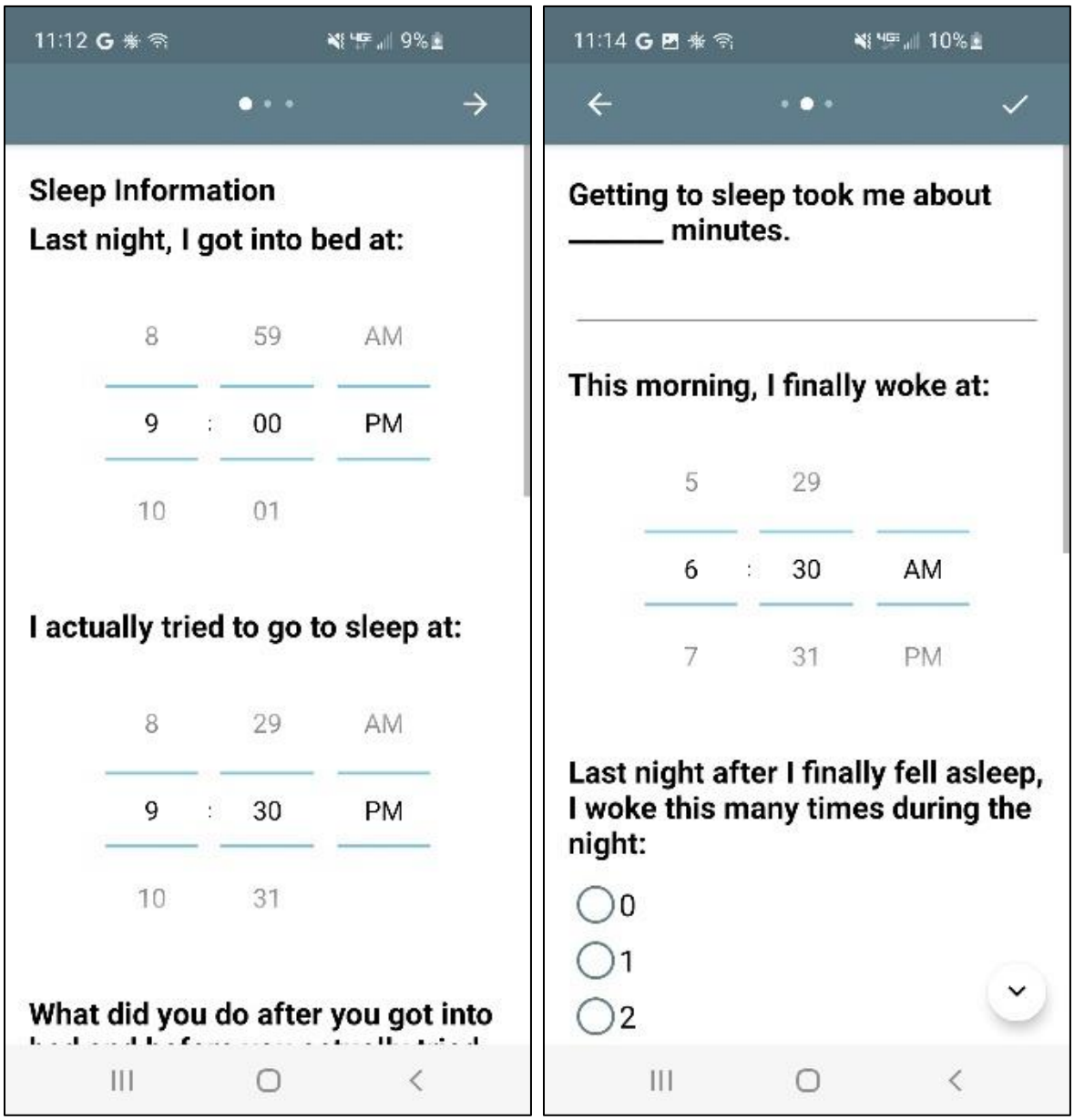
Note. This figure represents a model adapted from Lunsford-Avery & Mittal (2013) focusing on the role of sleep dysfunction in the traditional diathesis-stress model (Walker & Diforio, 1997). The bottom line displays the progression from early life to onset of psychosis. The boxes depict variables that may interact to contribute to this progression. The current study examined variables highlighted in red (clinical high-risk, sleep dysfunction, psychosocial stress, and clinical high-risk period). The dotted lines demonstrate the hypothesized interactions of sleep in the model.

Figure 2. Conceptual Model for Study Hypotheses



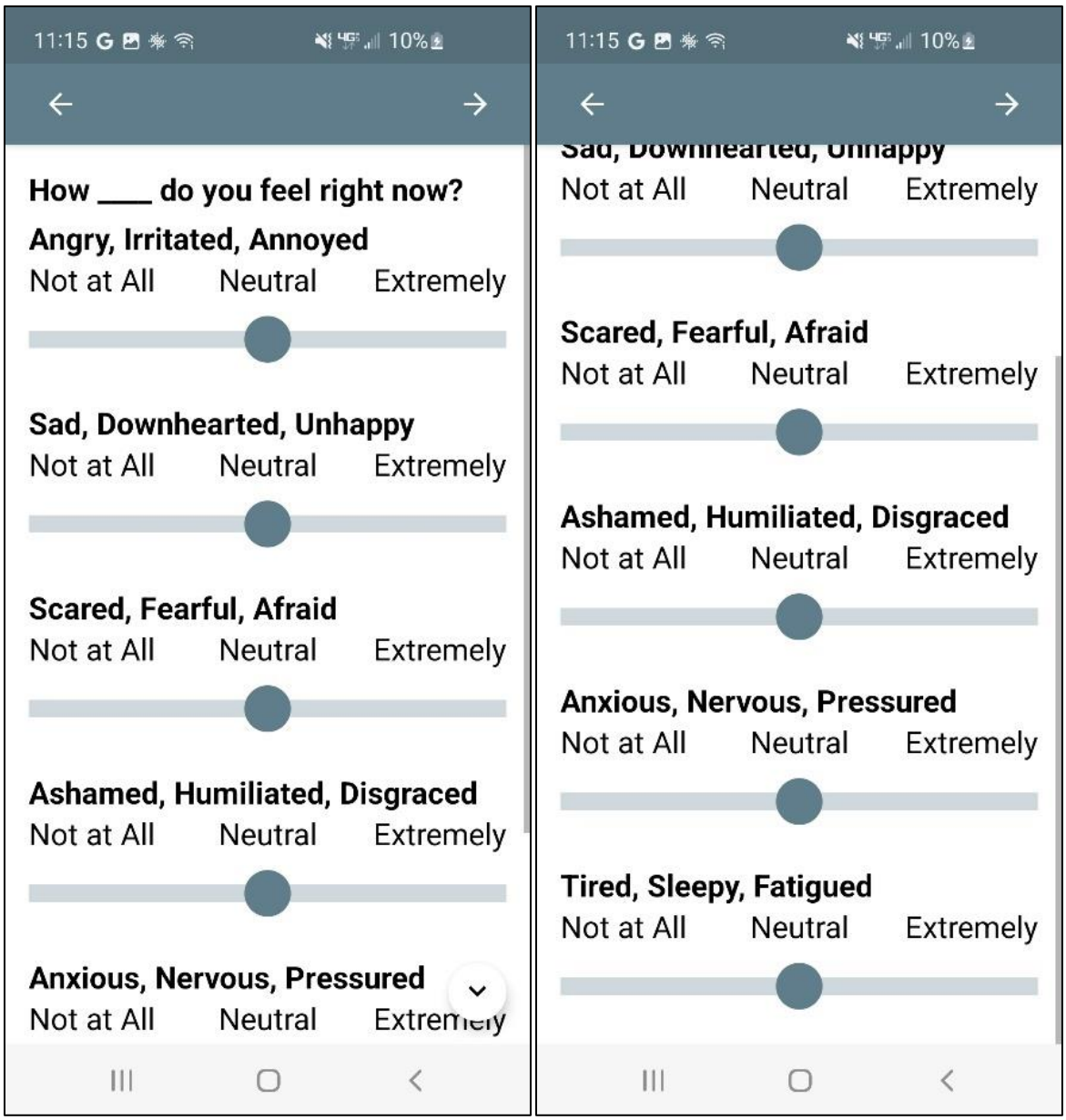
Note. A conceptual model outlining the current study's primary hypotheses and relationships between nightly sleep duration, momentary perceived stress, momentary experiences of attenuated positive symptoms, and clinical high-risk status for psychosis. We hypothesized that there would be no group differences in nightly sleep duration (H1) or momentary stress (H2); shorter sleep duration would be associated with higher momentary stress in the whole sample (H3), and group status (i.e., clinical high-risk status (CHR) vs. non-CHR) would moderate this association; shorter sleep duration (H4) and higher momentary stress (H5) would be associated with a higher frequency of momentary psychosis-risk symptoms in daily life in the CHR group. Solid lines indicate associations between nightly sleep duration, momentary perceived stress, experiences of psychosis-risk symptoms in daily life, and clinical high-risk (CHR) status.

Figure 3. Sleep-Related Questions on MovisensXS Mobile Questionnaires



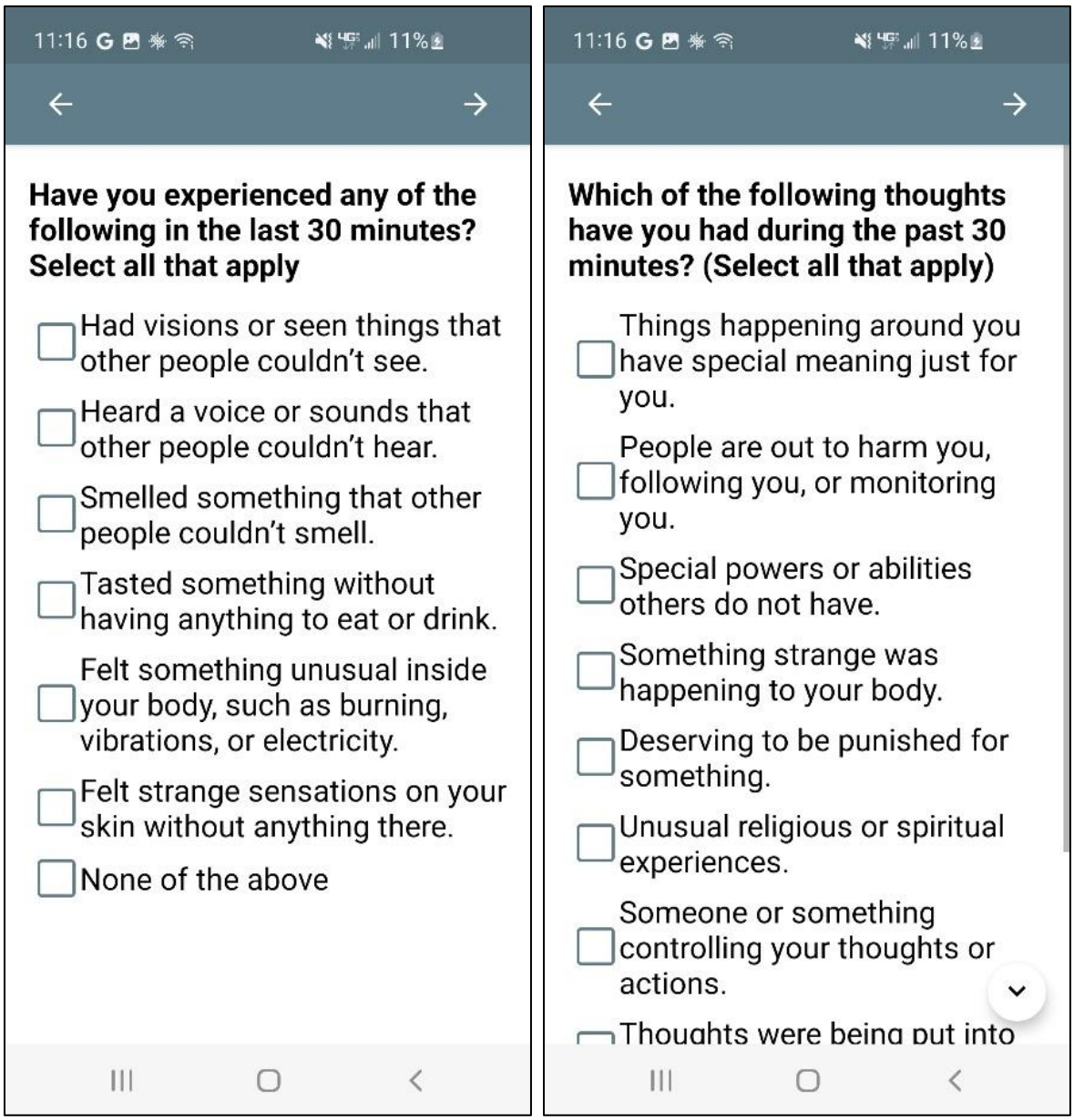
Note. The MovisensXS smartphone app (Movisens GmbH, 2016) was used to ask sleep-related questions on morning surveys. Nightly sleep duration was measured by taking the difference between the time at which participants woke up in the morning and tried to fall asleep the previous night.

Figure 4. Momentary Perceived Stress Mobile Questionnaire on MovisensXS Questionnaires



Note. Using the MovisensXS smartphone app (Movisens GmbH, 2016), momentary stress was measured by asking participants to rate the statement, "How [anxious, nervous, pressured] do you feel right now?" on a numeric scale from 0 (not at all) to 100 (extremely).

Figure 5. Momentary Psychosis-Risk Symptoms Questions on MovisensXS Questionnaire



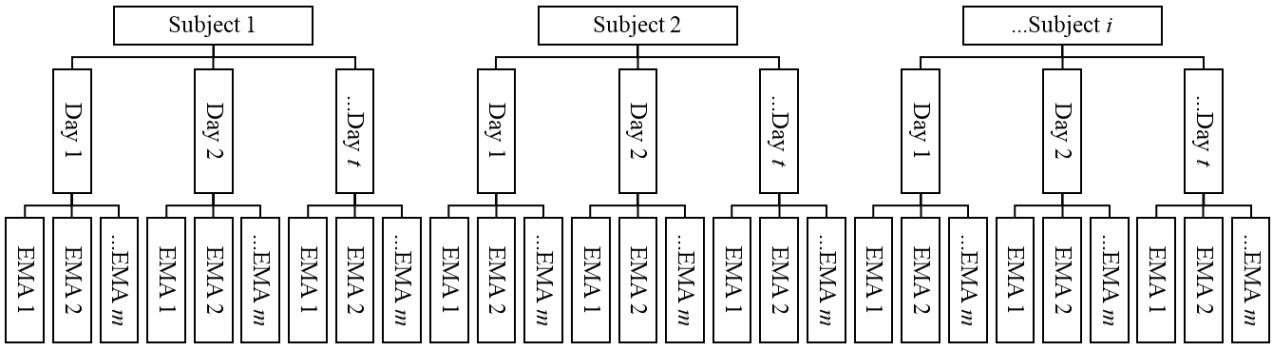
Note. Using the MovisensXS smartphone app (Movisens GmbH, 2016), momentary psychosis-risk symptoms were measured as the total number of perceptual disturbances and unusual thought content reported on a daily survey. Participants were asked to select all thoughts and/or experiences that they experienced during the past 30 minutes.

Figure 6. Example Dataset of Ecological Momentary Assessment Data

ID	Group	Timepoint	Day	Observation	Form	Sleep	Stress	APS
1001	0	0	2	1	Morning Form	6.033333	*NA*	*NA*
1001	0	0	2	2	Daily Form	*NA*	77	0
1001	0	0	2	3	Daily Form	*NA*	66	0
1001	0	0	2	4	Daily Form	*NA*	19	0
1001	0	0	2	6	Daily Form	*NA*	24	0
1001	0	0	2	7	Daily Form	*NA*	69	0
1001	0	0	2	8	Daily Form	*NA*	26	0
1001	0	0	3	1	Morning Form	5.583333	*NA*	*NA*
1001	0	0	3	2	Daily Form	*NA*	66	0
1001	0	0	3	3	Daily Form	*NA*	72	0
1001	0	0	3	5	Daily Form	*NA*	23	0
1001	0	0	3	7	Daily Form	*NA*	4	0
1001	0	0	4	2	Daily Form	*NA*	2	0
1001	0	0	4	5	Daily Form	*NA*	34	0
1001	0	0	4	6	Daily Form	*NA*	11	0
1001	0	0	5	1	Morning Form	6.516667	*NA*	*NA*
1001	0	0	5	2	Daily Form	*NA*	5	0
1001	0	0	5	3	Daily Form	*NA*	70	0
1001	0	0	5	4	Daily Form	*NA*	69	0
1001	0	0	5	5	Daily Form	*NA*	21	0
1001	0	0	5	7	Daily Form	*NA*	75	0
1001	0	0	8	1	Morning Form	6.500000	*NA*	*NA*
1001	0	0	8	2	Daily Form	*NA*	15	0
1001	0	0	8	3	Daily Form	*NA*	17	0
1001	0	0	8	4	Daily Form	*NA*	69	0
1001	0	0	8	5	Daily Form	*NA*	21	0
1001	0	0	8	6	Daily Form	*NA*	13	0
1001	0	0	8	7	Daily Form	*NA*	6	0
1001	0	0	9	1	Morning Form	7.366667	*NA*	*NA*
1001	0	0	9	2	Daily Form	*NA*	14	0

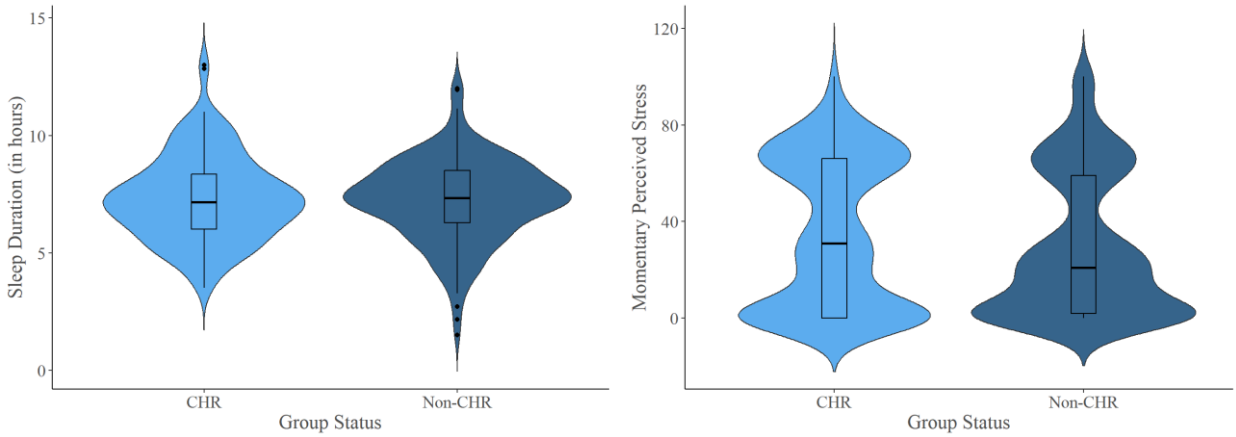
Note. R version 2023.09.1 (R Core Team, 2023) was used to clean ecological momentary assessment (EMA) data and run multilevel regression models on EMA variables.

Figure 7. Hierarchical Data Structure for Ecological Momentary Assessment Data



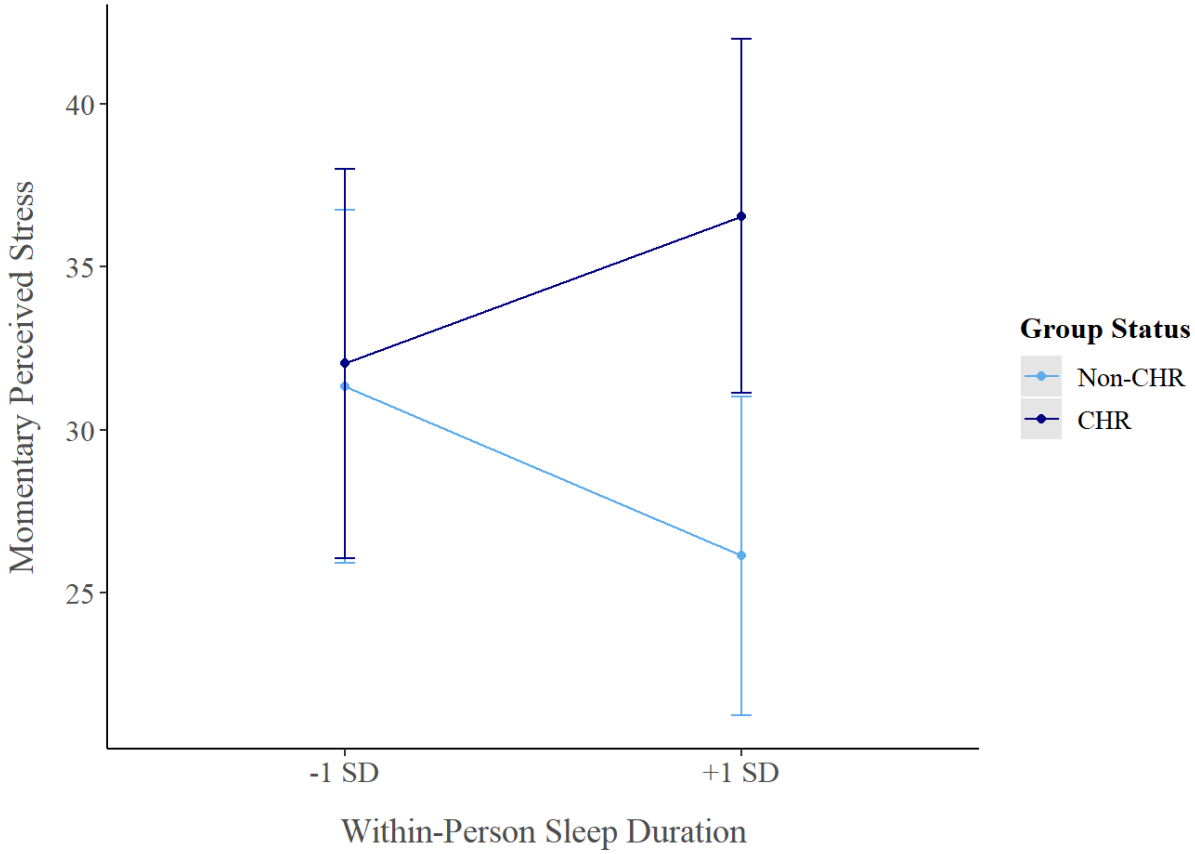
Note. Multilevel modeling was used to model hierarchical ecological momentary assessment (EMA) data across a 14-day period of at-home, ambulatory assessment. EMA observations (level-1) are nested within days (level-2) nested within subjects (level-3).

Figure 8. Violin Plots of Group Differences in Sleep Duration and Momentary Stress



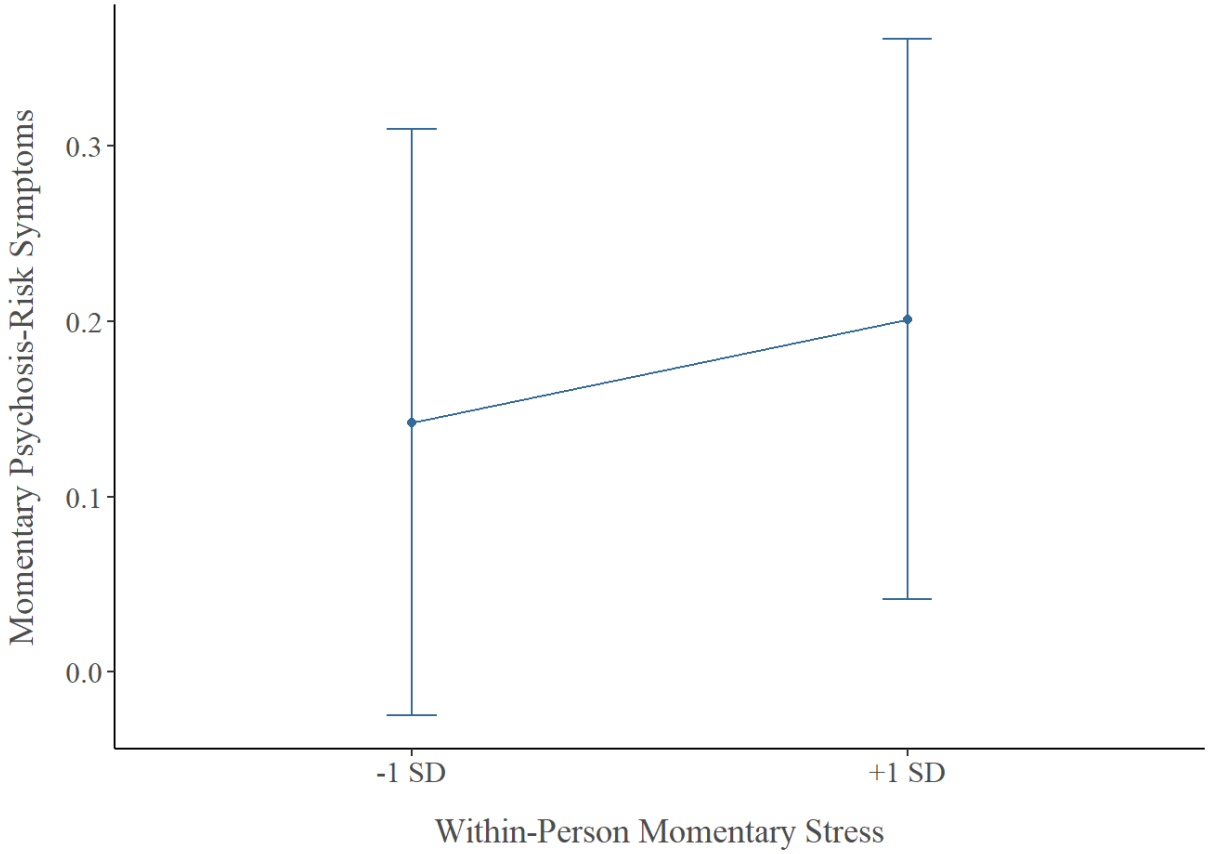
Note. The violin plots of the group differences in nightly sleep duration (left panel) and momentary stress (right panel). Multilevel regression models showed that there were no significant group differences in sleep duration and momentary stress.

Figure 9. Simple Slopes Graph of Group Status Moderating the Association Between Sleep Duration and Momentary Stress in Whole Sample



Note. Simple slopes analysis (Aiken et al., 1991) was performed to probe the two-way interaction between group status and nightly sleep duration on momentary perceived stress in the whole sample.

Figure 10. Simple Slopes Graph of Association Between Momentary Perceived Stress and Psychosis-Risk Symptoms in Clinical High-Risk Group



Note. A graph of the simple slopes demonstrates the positive association between within-person momentary stress and momentary psychosis-risk symptoms in the clinical high-risk (CHR) group.

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