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Original Article

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Racial/ethnic disparities in the trajectories of insomnia symptoms from childhood to young adulthood

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Abstract

Study Objectives: To examine differences in the longitudinal prevalence of childhood insomnia symptoms across black/African American, Hispanic/Latinx, and non-Hispanic white groups.

Methods: Participants were 519 children from the Penn State Child Cohort (baseline [V1] from 2000–2005) who were followed up 8 years later as adolescents (V2) and 15 years later as young adults (S3). Mean age at S3 was 24.1 ± 2.7 years. Approximately, 76.5% identified as non-Hispanic white, 12.9% as black/African American, 7.1% as Hispanic/Latinx, and 3.5% as "other" race/ethnicity. Insomnia symptoms were defined as parent-reported (childhood) or self-reported (adolescence and young adulthood) moderate-to-severe difficulties initiating/maintaining sleep. Longitudinal trajectories of insomnia symptoms were identified across three-time points and the odds of each trajectory were compared between racial/ethnic groups, adjusting for sex, age, overweight, sleep apnea, periodic limb movements, psychiatric/behavioral disorders, and psychotropic medication use.

Results: Black/African Americans compared to non-Hispanic whites were at significantly higher odds of having a childhood-onset persistent trajectory through young adulthood (OR = 2.58, 95% CI [1.29, 5.14]), while Hispanics/Latinx were at nonsignificantly higher odds to have the same trajectory (OR = 1.81, 95% CI [0.77, 4.25]). No significant racial/ethnic differences were observed for remitted and waxing-and-waning trajectories since childhood or incident/new-onset trajectories in young adulthood.

Conclusions: The results indicate that disparities in insomnia symptoms among black/African American and, to a lesser extent, Hispanic/Latinx groups start early in childhood and persist into young adulthood. Identifying and intervening upon upstream determinants of racial/ethnic insomnia disparities are warranted to directly address these disparities and to prevent their adverse health sequelae.

Clinical Trial Information: N/A; Not a clinical trial.

Key words: insomnia; sleep; health disparity populations; longitudinal study; cohort study

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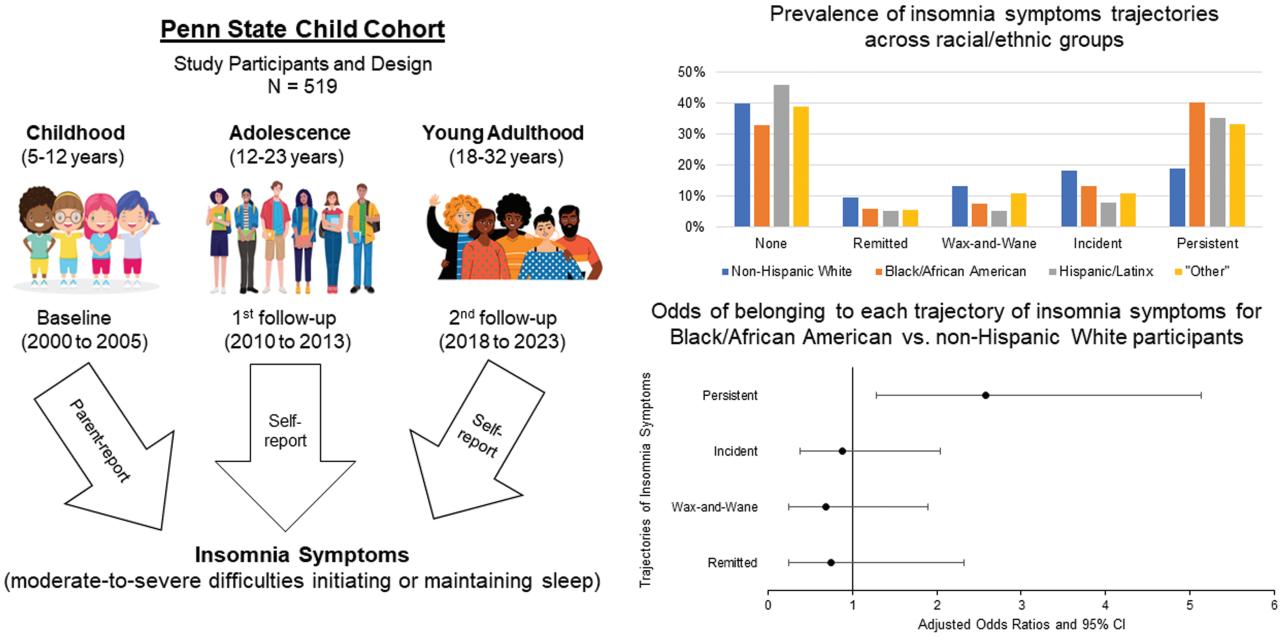
Graphical Abstract

Racial/Ethnic Disparities in the Trajectories of Insomnia Symptoms from Childhood to Young Adulthood



Statement of Significance

Insomnia prevalence is likely higher and symptoms more severe among minoritized racial/ethnic groups; yet, racial/ethnic disparities in the trajectories of insomnia symptoms from childhood through young adulthood have not been investigated. This study shows that minoritized racial/ethnic groups are disproportionately affected by persistent insomnia symptoms from childhood to young adulthood. These data underscore the importance of identifying and addressing insomnia symptoms early in life. Additionally, further research is warranted to understand the factors contributing to observed disparities in insomnia symptom trajectories, which could inform the development of multi-level interventions for minoritized racial/ethnic groups.

Introduction

Insomnia symptoms are commonly self-reported sleep problems characterized by difficulty initiating and/or maintaining sleep. The prevalence of insomnia symptoms in children ranges from 20% to 25%, increasing during adolescence to 25% to 35% [1–3]. In adulthood, 30% to 45% of adults experience insomnia symptoms, with 10% having diagnosed insomnia disorder [4–6]. Prior studies have found associations between insomnia symptoms and many adverse health as well as social outcomes, including cardiometabolic diseases, depression, impaired work/school performance, reduced quality of life, and increased health care costs [2, 7–10].

Research suggests that minoritized racial/ethnic groups may be at risk for more severe insomnia symptoms compared to non-Hispanic whites (NHWs) [11–14]. For instance, black/African American (black/AA) adults have a 67% higher risk of insomnia with objective short sleep duration compared to NHWs [14], which is the most severe phenotype [15]. Additionally, black/AA participants were twice as likely to experience chronic insomnia symptoms lasting over a year compared to NHWs [16]. The odds of severe insomnia have also been found to be greater among black/AA participants compared to white participants during the pandemic [17]. Furthermore, a retrospective analysis of electronic medical records has found that black/AA patients are less likely to receive an order for insomnia medication after insomnia diagnosis compared to white patients (6.2% vs. 8%) [18]. Moreover, in one longitudinal study examining the racial/ethnic differences in the trajectories of insomnia severity among middle-aged and older adults, the authors found that Hispanic adults experienced a greater increase in insomnia severity over time compared to NHW adults [19]. Although these findings need to be replicated, they suggest that insomnia symptoms may disproportionately impact minoritized racial/ethnic groups.

Despite the evidence of more severe and chronic insomnia symptoms among racially/ethnically minoritized groups compared to NHWs, there is a gap in the literature regarding the trajectory of insomnia symptoms from childhood through adolescence into adulthood among minoritized populations. Using 8-year longitudinal data from the Penn State Child Cohort (PSCC), Fernandez-Mendoza et al. found that 56% of participants with childhood insomnia symptoms persisted in the transition to adolescence, while only 30% experienced full remission [20]. Furthermore, the authors found racially/ethnically minoritized children, regardless of socioeconomic status (SES), and NHW children of low SES were 3–4 times more likely to have insomnia symptoms that persisted into adolescence; however, this study did not investigate whether these trajectories continued into young adulthood [20]. In another study using 15-year longitudinal data of the PSCC, Fernandez-Mendoza et al. found that

childhood insomnia symptoms persist into young adulthood in 43.3% of children, while 38.1% had experienced remission and 18.6% a waxing-and-waning course [21]. However, to the best of our knowledge, no studies have examined the trajectories of insomnia symptoms from childhood through young adulthood across various racial/ethnic groups.

Thus, we aim to address this important scientific gap by examining differences in the longitudinal trajectories of childhood insomnia symptoms among black/AA and Hispanic/Latinx individuals compared to NHW individuals using data from the PSCC. We hypothesized that a higher percentage of black/AA and Hispanic/Latinx participants with childhood insomnia symptoms would experience persistence into young adulthood compared to NHW participants. We also hypothesized that fewer black/AA and Hispanic/Latinx participants with childhood insomnia symptoms would experience remission during the transition to adolescence or young adulthood compared to their NHW counterparts.

Materials and Methods

Data source

The PSCC is a random, population-based study of 700 school-aged children. The design and protocol of the study have been described previously [20, 22–24]. Briefly, out of 5740 children (aged 5–12 years), 1000 were randomly selected to participate in an in-laboratory clinical and polysomnography (PSG) study (visit 1 [V1]), of which, 700 agreed to participate (baseline from 2000 to 2005). Between 2010 and 2013, 421 participants returned for visit 2 (V2) and underwent an in-laboratory follow-up study at ages 12–23 [22]. There were no significant differences in demographic characteristics at V1 in the 279 participants lost to follow-up at V2 [22]. The length between V1 and V2 ranged from 5.8 years to 13 years (median = 7.4 years; interquartile range [IQR], 6.8–8.7 years). Of the 700 participants who competed in V1, 519 aged 18 to 32 years also completed a self-reported survey (survey 3 [S3]) between 2018 and 2023. No differences in demographic characteristics at V1 were observed between the 700 participants and the 519 followed-up at S3 (race/ethnicity $p = 0.83$; age $p = 0.94$; overweight $p = 0.69$, psychiatric/behavioral disorder $p = 0.94$; psychotropic medication $p = 0.76$; pediatric obstructive sleep apnea (OSA) $p = 0.09$; pediatric PLMS $p = 0.40$; insomnia symptoms $p = 0.42$), except a lower proportion of males ($p < 0.001$). The length between V1 and S3 ranged from 11.5 years to 21.2 years (median = 14.9 years; IQR, 13.4–16.4 years). The current study focuses on these 519 participants with S3 data ($n = 343$ with data at all-time points) (Supplementary Figure S1).

Written informed consent from the parent or legal guardian and from participants aged ≥ 18 years, and assent from those aged < 18 years were obtained. The study was approved by Penn State College of Medicine's institutional review board.

Assessment of insomnia symptoms

Participants or their parents completed the Pediatric Behavior Scale (PBS) [25] and the Pediatric Sleep Questionnaire (PSQ) [26] at V1 and V2. Insomnia symptoms were defined as a parent report of "often/moderate" or "very often/severe" difficulty initiating and/or maintaining sleep (DIMS) on the PBS [1, 27–29] and/or sleep medication (over-the-counter or prescription) use for DIMS [4] at V1. There were 3 participants missing PBS data, for whom a positive response to DIMS on the PSQ was used [20]. During V2, insomnia symptoms were defined as a self-report of DIMS on the PSQ [26] and/or sleep medication use; there were 13 participants missing PSQ data, for whom a parent reported often/moderate or very often/severe DIMS on the PBS was used [20, 27, 30]. Only one participant was reclassified as having insomnia symptoms based on their report of sleep medication use at V2. In S3, insomnia symptoms were defined as a self-report of "moderate" or "severe" DIMS and/or sleep medication use obtained from a structured questionnaire that was developed in the Penn State Adult Cohort [5, 6]. Based on our prior study [21], insomnia symptoms trajectories (Supplementary Table S1) were clustered into five clinically meaningful groups: "None" (i.e. absence of DIMS at any time point), "Remitted" (i.e. presence of DIMS at V1 and absence at V2 and S3), "Wax-and-Wane" (i.e. intermittent absence and presence of DIMS across V1, V2 and S3), "Incident" (i.e. absence of DIMS at V1 and V2 and presence at S3), and "Persistent" (i.e. presence of DIMS at V1 through S3 or at V2 and S3).

Assessment of demographic and clinical characteristics

A standard questionnaire with demographic information (i.e. date of birth, sex, race, and ethnicity) and psychiatric history (i.e. psychiatric or learning disorders and psychotropic medication use history) was completed by participants and/or their parents at V1, V2, and S3 [22, 23]. The black/African American racial category excluded participants who identified as Hispanic/Latinx, and this latter category included those four participants who also identified as black. Given the small number of minoritized participants who did not identify as black/AA or Hispanic/Latinx, a category called "other" race/ethnicity, including individuals identified, for example, as Asian (n-13) or Native American ($n = 2$), was created and included in the analyses. Parents also reported their occupational status during V1 and V2, categorized as professional (i.e. managerial or professional occupation) or nonprofessional (i.e. secretarial or non-managerial occupation or unemployed, disabled, retired, and a student), as an indicator of SES. Data for the SES-adjusted model are presented in Supplementary Table S2. Given that the adjusted model did not change the main results and we did not have SES data at S3, results are reported for the models that did not adjust for V1 and V2 SES. During V1 and V2, height and weight were measured in the laboratory [22, 23, 29], and body mass index (BMI) percentile for sex and age was calculated using standard growth curves. The presence of overweight was defined as a BMI percentile ≥ 85%. In S3, participants self-reported their height and weight and BMI was calculated using the standard formula, with the presence of overweight defined as a BMI ≥ 25. The presence of overweight in S3 was included as the covariate because it provides a contemporaneous and temporally aligned representation of the variable, reducing measurement error and enhancing the rigor of the analysis compared to using BMI at earlier time points (i.e. V1 or V2). Participants underwent an in-lab PSG at V1 and V2, which indicated the presence of OSA and periodic limb movements (PLMS). For OSA, pediatric criteria of apnea–hypopnea index ≥ 2 per hour was used [20, 22, 24], and for PLMS, a pediatric PLM index of ≥ 5 per hour was used [20, 31, 32].

Statistical analyses

Descriptive statistics were used to estimate the characteristics (Table 1) and developmental trajectories of insomnia symptoms in the overall sample stratified by race/ethnicity (Table 2). To account for missing insomnia symptoms data ($n = 176$) during adolescence (V2), multiple imputations were performed

Table 1. Sociodemographic and Clinical Characteristics across Study Visits Among Black/African American, Hispanic/Latinx, Non-Hispanic White, and "Other" Race/Ethnicity Study Participants, *N* = 519

	V1. Childhood *N* = 519	**V2. Adolescence *N* = 343[a]**	**S3. Young Adulthood *N* = 519**
		Non-Hispanic white	
	***n* = 397**	***n* = 264**	***n* = 397**
Female sex, *n*(%)	197 (49.6%)	128 (48.5%)	197 (49.6%)
Age, years, mean ± SD	8.65 ± 1.69	16.60 ± 2.31	24.16 ± 2.77
Low SES, *n*(%)	121 (30.5%)	163 (61.7%)	—
Overweight, *n*(%)	113 (28.5%)	81 (30.7%)	184 (46.3%)
Psychiatric/behavioral disorder, *n*(%)	90 (22.7%)	89 (33.7%)	182 (45.8%)
Psychotropic medication, *n*(%)	35 (8.8%)	46 (17.4%)	91 (22.9%)
Pediatric OSA, *n*(%)	46 (11.6%)	93 (35.2%)	—
Pediatric PLMS, *n*(%)	16 (4.0%)	63 (23.9%)	—
Insomnia symptoms, *n*(%)	95 (23.9%)	93 (35.2%)	165 (41.6%)
After imputation, *n*(%)	—	120 (30.2%)	—
Length of follow-up, years			
Since V1, mean ± SD	—	7.92 ± 1.52	14.98 ± 2.12
Since V2, mean ± SD	—	—	6.89 ± 1.11
		Black/African American	
	n = 67	*n* = 45	*n* = 67
Female sex, *n*(%)	35 (52.2%)	21 (46.7%)	35 (52.2%)
Age, years, mean ± SD	8.75 ± 1.83	16.11 ± 1.85	24.40 ± 2.39
Overweight, *n*(%)	31 (46.3%)	21 (46.7%)	41 (61.2%)
Low SES, *n*(%)	27 (40.3%)	33 (73.3%)	—
Psychiatric/behavioral disorder, *n*(%)	14 (20.9%)	16 (35.6%)	35 (52.2%)
Psychotropic medication, *n*(%)	4 (6.0%)	6 (13.3%)	10 (14.9%)
Pediatric OSA, *n*(%)	16 (23.9%)	24 (53.3%)	—
Pediatric PLMS, *n*(%)	0 (0.0%)	8 (17.8%)	—
Insomnia symptoms, *n*(%)	15 (22.4%)	20 (44.4%)	36 (53.7%)
After imputation, *n*(%)	—	32 (47.8%)	—
Length of follow-up, years			
Since V1, mean ± SD	—	7.44 ± 0.69	15.13 ± 1.48
Since V2, mean ± SD	—	—	7.43 ± 1.15
		Hispanic/Latinx	
	n = 37	*n* = 25	*n* = 37
Female sex, *n*(%)	24 (64.9%)	14 (56.0%)	24 (64.9%)
Age, years, mean ± SD	8.54 ± 1.80	16.56 ± 1.98	23.89 ± 2.51
Low SES, *n*(%)	19 (51.4%)	20 (80.0%)	—
Overweight, *n*(%)	20 (54.1%)	12 (48.0%)	24 (64.9%)
Psychiatric/behavioral disorder, *n*(%)	9 (24.3%)	11 (44.0%)	15 (40.5%)
Psychotropic medication, *n*(%)	4 (10.8%)	4 (16.0%)	8 (21.6%)
Pediatric OSA, *n*(%)	6 (16.2%)	12 (48.0%)	—
Pediatric PLMS, *n*(%)	1 (2.7%)	9 (36.0%)	—
Insomnia symptoms, *n*(%)	9 (24.3%)	7 (28.0%)	17 (45.9%)
After imputation, *n*(%)	—	14 (37.8%)	—
Length of follow-up, years			
Since V1, mean ± SD	—	7.75 ± 1.06	14.91 ± 1.52
Since V2, mean ± SD	—	—	6.97 ± 1.24

Table 1. Continued

	V1. Childhood N = 519	V2. Adolescence N = 343[a]	S3. Young Adulthood N = 519
		Non-Hispanic white	
	n = 397	n = 264	n = 397
		"Other" race/ethnicity[b]	
	n = 18	n = 9	n = 18
Female sex, n(%)	11 (61.1%)	7 (77.8%)	11 (61.1%)
Age, years, mean ± SD	8.50 ± 1.38	16.67 ± 2.29	23.83 ± 2.36
Low SES, n(%)	5 (27.8%)	5 (55.6%)	—
Overweight, n(%)	5 (27.8%)	3 (33.3%)	8 (44.4%)
Psychiatric/behavioral disorder, n(%)	3 (16.7%)	2 (22.2%)	9 (50.0%)
Psychotropic medication, n(%)	1 (5.6%)	0 (0.0%)	3 (16.7%)
Pediatric OSA, n(%)	3 (16.7%)	4 (44.4%)	—
Pediatric PLMS, n(%)	1 (5.6%)	2 (22.2%)	—
Insomnia symptoms, n(%)	3 (16.7%)	5 (55.6%)	8 (44.4%)
After imputation, n(%)	—	8 (44.4%)	—
Length of follow-up, years			
Since V1, mean ± SD	—	7.29 ± 0.88	14.75 ± 1.52
Since V2, mean ± SD	—	—	7.15 ± 0.81

Data represented as n(%) or mean ± standard deviation (SD).
[a]Participants without adolescence (V2) data were imputed for their developmental trajectories; [b]Given the small distribution, participants from "other" racial or ethnic backgrounds were excluded from the analyses; OSA, obstructive sleep apnea based on pediatric criteria (i.e. ≥ 2 events per hour of sleep). PLMS, periodic limb movements based on pediatric criteria (i.e. ≥ 5 events per hour of sleep). SES, socioeconomic status (i.e. parent with a secretarial or non-managerial occupation or unemployed, disabled, retired, or student). V1, visit 1 (childhood). V2, visit 2 (adolescence). S3, survey 3 (young adulthood). The presence of overweight was defined as a BMI percentile ≥ 85%. In S3, participants self-reported on their height and weight and BMI was calculated using the standard formula, with the presence of overweight defined as a BMI ≥ 25.
"Other" race/ethnicity included participants who identified as Asian/Native Hawaiian (n = 14) and American Indian/Native American (n = 4).

for each trajectory; the detailed procedure has been described previously [21].

To examine the relative association of race/ethnicity with insomnia symptoms trajectories, while adjusting for other demographic and clinical risk factors, multivariable-adjusted odds ratios (OR) and their 95% confidence intervals (CI) were estimated using multinomial logistic regression models. Models were adjusted for sex, age, overweight, psychiatric/behavioral disorders, and psychotropic medication use at S3, as well as OSA and PLMS at V1 or V2 (backward elimination), while simultaneously adjusting for time to follow-up (forced entry). All analyses were conducted using SPSS v26.

Results

Study participant characteristics

Table 1 shows participant characteristics at each time point. Overall, about 52% of participants were female, and average ages at each time point were consistent with the developmental stages of childhood (V1), adolescence (V2), and young adulthood (S3). Majority of participants were NHW (76.5%), followed by black/AA (13%), Hispanic/Latinx (7%), and "other" race/ethnicity (3.5%). The percentage of participants with overweight, psychiatric/behavioral disorder, psychotropic medication use, and insomnia symptoms increased at each time point across different race/ethnicity. There was also a higher percentage of participants with pediatric OSA and PLMS at V2 than at V1 across race/ethnicity.

Developmental trajectories of insomnia symptoms across racial/ethnic groups

Table 2 depicts the prevalence of the developmental trajectories of insomnia symptoms across racial/ethnic groups. Overall, 23.3% of participants had persistent insomnia symptoms and 16.8% incident insomnia symptoms, while 39.3% of participants were absent of any insomnia symptoms over their life course (i.e. "None" or normal sleep trajectory). Whereas black/AA participants had the highest proportion of persistent trajectory (40.3%), normal sleep trajectory was most prevalent in NHW (39.8%), Hispanic/Latinx (45.9%), and "other" (38.9%) groups. Compared to NHW (9.6%), black/AA (6.0%), Hispanic/Latinx (5.4%), and "other" (5.6%) races/ethnicities were less likely to belong to the remitted insomnia symptoms group. In fact, the 6.0% of black/AA participants and 5.6% of "others" in the remitted trajectory all remitted their insomnia symptoms in the transition to adolescence, and the remainder of participants in these two groups who had insomnia symptoms in childhood persisted all the way to adulthood (Supplementary Table S1). Similarly, none of the Hispanic/Latinx participants remitted in the transition to adolescence. A higher proportion of NHW experienced waxing-and-waning (13.4%) and incident trajectories (18.5%) compared to the minoritized groups.

Multivariable-adjusted logistic regression results are shown in Table 3. Compared to NHWs, black/AA participants were at 2.6-fold significantly increased odds of having a persistent insomnia symptoms trajectory (OR = 2.58, 95% CI [1.29, 5.14], $p = 0.007$). In fact, the odds for black/AA participants of persisting, instead

of remitting, were 3.44-fold (95% CI [1.11, 10.70], $p = 0.033$) compared to NHW participants. Hispanic/Latinx participants were associated with 1.8-fold increased odds of having a persistent insomnia symptoms trajectory (OR = 1.81, 95% CI [0.77, 4.25]), albeit without reaching statistical significance ($p = 0.173$). There were no statistically significant differences between NHW and the three minoritized groups in the odds of having a remitted, waxing-and-waning, or incident trajectory.

Discussion

This is the first study, to the best of our knowledge, to investigate racial/ethnic disparities in the longitudinal trajectories of childhood insomnia symptoms. We found that black/AA compared to NHW participants with insomnia symptoms in childhood were 2.6 times more likely to have symptoms that persist through young adulthood, which was consistent with our hypothesis. Although non-statistically significant, there is public health significance in the finding that Hispanic/Latinx children were 1.8 times more likely to have insomnia symptoms that persisted into young adulthood compared to NHW children. black/AA (39.4%) and Hispanic/Latinx (35.1%) participants were overrepresented in the persistent insomnia symptoms trajectory compared to their NHW (18.7%) counterparts. A smaller proportion of blacks/AAs (6.1%) and Hispanic/Latinx (5.4%) compared to NHWs (9.6%) were represented in the trajectory for remitted insomnia symptoms. Furthermore, our study revealed that minoritized children whose insomnia symptoms persisted in the transition to adolescence were less likely to experience symptom remission in the transition to adulthood, which underscores the need for early-life intervention.

Our study expands upon the two prior studies that have examined childhood insomnia trajectories [20, 21]. Fernandez-Mendoza et al. found that the full remission rate of insomnia symptoms in the transition to adolescence among minoritized children was 10%, regardless of SES, compared to 25% among NH White children of low SES and 40% among NH white children of higher SES [20]. Consistently, minoritized children, regardless of SES, had higher insomnia symptoms persistence compared to NH white children with higher SES [20]. Our study further builds on another longitudinal study by Fernandez-Mendoza et al. that reported a persistence prevalence of 43% and a remission prevalence of 27% for childhood insomnia symptoms when followed up through young adulthood; however, this latter study did not examine racial/ethnic differences [21]. Our novel findings corroborate the scant longitudinal literature, which indicates that disparities in sleep begin in the early stages of development and are prevalent throughout childhood and adolescence [33, 34]. Prior studies have shown that higher cumulative risks of low SES (e.g. family poverty and maternal educational attainment) are associated with longer sleep onset latency and more frequent and longer night awakenings among infants [35]. Furthermore, children residing in lower SES neighborhoods experience delayed bedtimes, longer sleep onset latency, more night awakenings, and shorter sleep duration when compared to their counterparts in higher SES neighborhoods [36, 37].

The 3-P model of insomnia [38] includes predisposing, precipitating, and perpetuating factors that contribute to the development and persistence of insomnia, and these factors can help illuminate potential determinants of the disparities observed in the emergence and progression of childhood insomnia symptoms. Adverse childhood experiences (ACEs) and neighborhood factors, examples of precipitating and perpetuating factors centered around social and environmental determinants of health, likely influence the onset and continuation of insomnia symptoms.

Table 2. Prevalence of Natural Course Trajectories Across Racial/Ethnic Groups

	None	Remitted	Wax-and-Wane	Incident	Persistent
	204 (39.3%)	45 (8.7%)	62 (11.9%)	87 (16.8%)	121 (23.3%)
Non-Hispanic White	158 (39.8%)	38 (9.6%)	53 (13.4%)	73 (18.4%)	75 (18.9%)
Black/African American	22 (32.8%)	4 (6.0%)	5 (7.5%)	9 (13.4%)	27 (40.3%)
Hispanic/Latinx	17 (45.9%)	2 (5.4%)	2 (5.4%)	3 (8.1%)	13 (35.1%)
"Other" race/ethnicity	7 (38.9%)	1 (5.6%)	2 (11.1%)	2 (11.1%)	6 (33.3%)

All values are provided as *n*(%) unless otherwise indicated. six cells (30% have expected count lower than five (the minimum expected count is 1.56), $p = 0.028$ for likelihood ratio test. "Other" race/ethnicity included participants who identified as Asian/Native Hawaiian ($n = 14$) and American Indian/Native American ($n = 4$).

Table 3. Multivariable Unadjusted and Adjusted Odds of Each Natural Course Trajectory Associated With Racial/Ethnic Groups

	Unadjusted Model				
	None	Remitted	Wax-and-Wane	Incident	Persistent
Non-Hispanic white	1.00	1.00	1.00	1.00	1.00
Black/African American	1.00	0.76 (0.24–2.32)	0.68 (0.24–1.88)	0.89 (0.39–2.02)	2.59 (1.38–4.84)
Hispanic/Latinx	1.00	0.49 (0.11–2.21)	0.35 (0.08–1.57)	0.38 (0.11–1.34)	1.61 (0.74–3.49)
"Other" race/ethnicity	1.00	0.59 (0.07–4.97)	0.85 (0.17–4.23)	0.62 (0.13–3.01)	1.81 (0.59–5.56)
	Adjusted model				
Non-Hispanic white	1.00	1.00	1.00	1.00	1.00
Black/African American	1.00	0.75 (0.24–2.32)	0.68 (0.24–1.90)	0.88 (0.38–2.04)	2.58 (1.29–5.14)
Hispanic/Latinx	1.00	0.53 (0.12–2.40)	0.37 (0.08–1.65)	0.45 (0.12–1.61)	1.81 (0.77–4.25)
"Other" race/ethnicity	1.00	0.60 (0.07–5.10)	0.83 (0.16–4.21)	0.67 (0.13–3.36)	1.67 (0.48–5.80)

All values are provided as odds ratio (95% confidence interval). Adjusted model is adjusted for time to follow-up (forced entry) and sex, age, overweight, psychiatric/behavioral disorders, and psychotropic medication use at S3 as well as OSA and PLMS at either V1 or V2 (backward elimination). "Other" race/ethnicity included participants who identified as Asian/Native Hawaiian ($n = 14$) and American Indian/Native American ($n = 4$).

ACEs have been associated with behavioral sleep problems in childhood including difficulty falling asleep and increased nighttime awakenings [39], as well as with increased insomnia symptoms in adulthood, with the magnitude of the association being higher for individuals with multiple ACEs [40–42]. Similarly, living in disadvantaged neighborhoods has been associated with insomnia symptoms [43], with neighborhood environmental factors (e.g. noise/light pollution) having a particular impact on difficulty falling and staying asleep [43, 44]. Furthermore, racially/ethnically minoritized individuals are disproportionately impacted by ACEs, concentrated poverty and live in suboptimal environmental and social conditions (e.g. unsafe and less well-resourced neighborhoods), and are also disproportionately impacted by poor sleep health [11, 34, 40, 43, 45–54]. Specifically, studies have found that NH-black/AA and Hispanic/Latinx children are less likely to have consistent bedtime routines, as well as more irregular bedtimes and shorter sleep duration compared to NHW children [34, 50]. Among adults, NH-black/AA and Hispanic/Latinx populations consistently report shorter sleep duration and poorer sleep quality than their NHW counterparts [11, 47–49].

Our study has several limitations. First, the proportion of black/AA and Hispanic/Latinx participants were small in our sample, and the study was underpowered to detect statistical significance among Hispanic/Latinx group. However, as the first study investigating disparities in the long-term progression of insomnia symptoms, our results reveal clear racial disparities with significant public health and clinical implications, which would strengthen with a larger sample size of above 519 participants and better representation of minoritized participants. We also were unable to disaggregate the "other" category given the small sample size but have reported the composition. Further longitudinal studies with more diverse and larger sample of minoritized racial/ethnic groups are warranted. Second, although we ascertained insomnia symptoms in a developmentally appropriate manner (i.e. by parent report in childhood and self-report in adolescence and adulthood) [20, 55], we did not have structured interview or electronic medical record data to ascertain the presence of insomnia disorder as per diagnostic criteria at each time point. Additionally, using parent-reported insomnia symptoms during childhood may result in bias. However, a study evaluating concordance between child reports, parent reports, and PSG sleep data found that child and parent reports are equally valid to assess multiple sleep parameters [56], and there is no consistent evidence that parental report of sleep difficulties varies by race/ethnicity [57]. Third, we could not control for the potential effect of OSA and PLMS during young adulthood since PSG was only included in childhood and adolescence. Fourth, we could not examine the potential effect of declining or inclining trajectories of SES since this social factor was not ascertained in young adulthood. Similarly, the built and social sleep environment as well as other social factors that are likely determinants of childhood and adulthood sleep (e.g. ACEs, experienced racism) were not measured and, therefore, not directly investigated in this study. Future research that includes these contextual factors that could help inform intervention targets is warranted.

Despite the limitations, this study also has several noteworthy strengths that extend the scientific literature. First, we employed a longitudinal design that covered three distinct time points representing different maturational and developmental stages with a median age of 9, 16, and 24 years. These novel data allow for a comprehensive understanding of the trajectory of symptoms over the life course, which is crucial for identifying effective interventions. Second, we leveraged multiple data sources to determine the trajectory of symptoms, including parent reports in children, self-reports in adolescence and beyond, and PSG. This multi-method approach ensures that our findings are robust and reliable. Lastly, we collected PSG at V1 and V2, which is noteworthy as it controls for prior exposure to organic sleep disorders.

Our findings have important clinical and public health implications. This study underscores the need for early intervention strategies aimed at preventing or treating insomnia symptoms early in life. Given the tendency for insomnia symptoms to emerge and persist from childhood into young adulthood, early identification, and targeted interventions are likely critical to reduce the disproportionate long-term impacts of insomnia in racially/ethnically minoritized children. Increasing access to sleep healthcare among minoritized racial/ethnic groups is crucial given that specialized sleep healthcare access, including access to cognitive behavioral therapy for insomnia for adolescents and adults, a first-line treatment for insomnia disorder, and pediatric behavioral sleep medicine providers for children is a major barrier due to scarcity of providers [58–60]. Additionally, healthcare providers across disciplines should be aware of these disparities and adopt a culturally sensitive approach when assessing and treating individuals with insomnia symptoms [61, 62]. Recognizing and addressing the unique challenges faced by racially/ethnically minoritized individuals in managing sleep health can contribute to more effective and equitable care. Furthermore, increasing training resources and education for healthcare providers (e.g. pediatricians/primary care providers) to consistently screen and make appropriate referrals for sleep difficulties is of paramount importance for preventing the persistence of insomnia symptoms. Moreover, addressing the racial/ethnic disparities in insomnia development and persistence requires broader social and structural changes that address structural racism and improve environmental as well as social conditions to avoid ACEs and, thus, improve sleep health and prevent insomnia development. In the meantime, implementing comprehensive sleep education programs in schools and pediatric healthcare settings for youth as well as families/parents, with a particular focus on populations at higher risk, could help promote healthy sleep habits (e.g. bedtime routine) and mitigate the development of persistent insomnia symptoms.

In conclusion, racial/ethnic disparities in insomnia symptoms start early in childhood and persist into young adulthood with black/AA individuals experiencing the highest prevalence of persistent symptoms and the lowest prevalence of symptom remission. These novel findings highlight the importance of early identification and intervention of insomnia symptoms as well as the need for further investigation to elucidate the determinants leading to and mechanisms underlying the observed racial disparities in insomnia symptom trajectories. Understanding these mechanisms can inform the development of multi-level (e.g. community and personalized) interventions tailored to disadvantaged racial/ethnic populations with limited access to material and social resources, leading to the equitable improvement of sleep health outcomes.

Supplementary Material

Supplementary material is available at *SLEEP* online.

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Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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